Baseline Human Health Risk Assessment

Powerhouse 2 Ash Pits
Plum Brook Ordnance Works
Sandusky, Ohio

Prepared for:

U.S. Army Corps of Engineers
P.O. Box 1070
Nashville, Tennessee 37202-1070

Prepared by:

Shaw Environmental & Infrastructure, Inc.
312 Directors Drive
Knoxville, Tennessee 37923

FUDS Project No. G0OH001822
Shaw Project No. 133422

September 2010

Final

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312 Directors Drive
Knoxville, Tennessee 37923
865.690.3211
FAX: 865.690.3626

September 29, 2010

U.S. Army Engineer District, Nashville
ATTN: CELRN-EC-E (Ms. Kathy McClanahan)
110 Ninth Avenue South, Room 682
U.S. Court House Annex
Nashville, Tennessee 37203

Submittal of the Final Baseline Human Health Risk Assessment and Screening Level Ecological Risk Assessment Power House 2 Ash Pits Former Plum Brook Ordnance Works, Sandusky, Ohio Contract No. W912QR-08-D-0013: Shaw Project Number 133422

Dear Ms. McClanahan:

In accordance with the requirements of Delivery Order No. DX05 of Contract No. W912QR-08-D-0013 Shaw is pleased to submit the draft Baseline Human Health Risk Assessment and the draft Screening Level Ecological Risk Assessment for the Power House 2 Ash Pits at the Former Plum Brook Ordnance Works (PBOW) located in Sandusky, Ohio. These reports were prepared consistent with those prepared for other PBOW sites, the agreements among the PBOW project delivery team members, and specifically the Power House 2 Ash Pits Baseline Human Health Risk Assessment and Screening Level Ecological Risk Assessment work plans.

Enclosed for your review are four copies of these reports. Copies have also been sent to those on the distribution list as indicated for their review. As requested, the document was sent to the Center of Expertise and the Restoration Advisory Board Co-Chair in electronic format only.

Sincerely,

Steven T. Downey, PE, PMP
Project Manager

Enclosures

Control Copy Distribution List

Name	Control Copy
U.S. Army Engineer District, Nashville ATTN: CELRN-EC-E (Ms. Kathy McClanahan) 110 Ninth Avenue South, Room 682 Annex Nashville, TN 37203	1-4
U.S. Army Engineer District, Huntington ATTN: CELRH-PM-PP-P (Mr. Rick Meadows) 502 Eighth Street Huntington, West Virginia 25701-2070	5-7 (CD provided)
Mr. Paul Jayko Site Coordinator Division of Emergency and Remedial Response (DERR) Ohio Environmental Protection Agency 347 North Dunbridge Road Bowling Green, Ohio 43402-0466	8, 9
Mr. Janusz Z. Byczkowski Division of Emergency and Remedial Response (DERR) Lazarus Government Center 1220 South Front Street Columbus, OH 43216-1049	10
Mr. Robert Lallier Environmental Coordinator NASA - Plum Brook Station 6100 Columbus Avenue Sandusky, Ohio 44870	11
U.S. Army Corps of Engineers (**electronic only**) HTRW Center of Expertise ATTN: CEHNC-CX-EG (Vern StJohn) 1616 Capitol Avenue Omaha, Nebraska 68144-3869	(CD provided)
Mr. John Blakeman (**electronic only**) PBOW RAB Co-chairman 2412 Scheid Road Huron, Ohio 44839	(CD provided)
Mr. Dennis Druck US Army Public Health Command (Provisional) ATTN: MCHB-TS-HER, BLDG E1675 Aberdeen Proving Grounds, MD 21010-5403	12

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DISCIPLINE SIGN-OFF REVIEW

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Project Description: Baseline Human Health Risk Assessment for Power House 2 Ash Pits Remedial Investigation
Former Plum Brook Ordnance Works, Sandusky, Ohio

Contract No. W912QR-08-D-0013 Delivery Order No. DX05

Project No. 133422 Task/Phase Number: 13000000

Document Type		Identify specific section or segment covered by this checkprint	Document Origin
☐	Technical / Cost Proposal		X Originator Developed
☐	RFP		☐ Edited Standard
☐	Contract / Subcontract		☐ Client Furnished
☐	Work Plans		
☐	Report		**Document Status**
X	Risk Assessment / Evaluation	Baseline Human Health Risk Assessment	☐ Preliminary
☐	Specifications & Plans		☐ Internal Draft
☐	Design Calculations		☐ Draft
☐	Tables		☐ Draft Final
☐	Drawings / Figures		X Final
☐	Other:		☐ Other:

	Required Person	Signature	Date
Originator	Tom Siard		9/29/10
Editor	Donald Groseclose	Donald Groseclose	9-29-10
Technical Review	Michael Gunderson	Steven T Downey for	9/29/10
Peer Review	Jon Lindberg	Jon Lindberg	9/29/10
Quality Assurance Mgr	Ken Martinez	Ken Martinez	09-29-10
Project Manager	Steven Downey	Steven T Downey	9/29/10

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List of Acronyms

ABS	dermal absorption factor
AF	soil-to-skin adherence factor
AP2	Powerhouse 2 Ash Pits
bgs	below ground surface
BHHRA	baseline human health risk assessment
BSC	background screening concentration
BTEX	benzene, toluene, ethylbenzene, and xylenes
CDI	chronic daily intake
cm^2	square centimeter
COPC	chemical of potential concern
CSEM	conceptual site exposure model
°F	degrees Fahrenheit
DA	dose absorbed per unit body surface area per day
D&M	Dames and Moore, Inc.
DM	dry matter
DNT	dinitrotoluene
DOE	U.S. Department of Energy
EPA	U.S. Environmental Protection Agency
EPC	exposure-point concentration
ET	exposure time
FI	fractional term
GAF	gastrointestinal absorption factor
gpm	gallons per minute
g	gram
g/kg-day	grams per kilogram of body weight per day
g/m^3	grams per cubic meter
GSA	General Services Administration
HHEM	human health evaluation manual
HI	hazard index
HQ	hazard quotient
ICP	inductively coupled plasma
IEUBK	Integrated Exposure Uptake Biokinetic
ILCR	incremental lifetime cancer risk
IRIS	Integrated Risk Information System
IT	IT Corporation

kg	kilogram
kg/day	kilograms per day
LMS	linearized multistage
K_p	permeability coefficient
L/cm^3	liters per cubic centimeter
L/day	liters per day
m^3	cubic meter
m^3/day	cubic meters per day
m^3/hr	cubic meters per hour
m^3/kg	cubic meters per kilogram
m/second	meters per second
MDC	maximum detected concentration
µg/m^3	micrograms per cubic meter
mg/cm^2	milligrams per square centimeter
mg/cm^2-day	milligrams per square centimeter per day
mg/m^3	milligrams per cubic meter
mg/day	milligrams per day
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram per day
µg/L	micrograms per liter
mg/L	milligrams per liter
MS	mass spectrometry
NASA	National Aeronautics and Space Administration
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
OEPA	Ohio Environmental Protection Agency
ORNL	Oak Ridge National Laboratory
PAH	polycyclic aromatic hydrocarbon
PBOW	Plum Brook Ordnance Works
PEF	particulate emission factor
PRG	preliminary remediation goal
QC	quality control
RBSC	risk-based screening concentration
RfC	reference concentration
RfD	reference dose
RI	remedial investigation

List of Acronyms *(Continued)*

RME	reasonable maximum exposure
RSL	regional screening level
SF	cancer slope factor
Shaw	Shaw Environmental, Inc.
TNT	trinitrotoluene
TNTA	TNT Area A
TNTB	TNT Area B
TNTC	TNT Area C
UCL	95th percent upper confidence limit on the arithmetic mean
UF	uncertainty factor
USACE	U.S. Army Corps of Engineers
VOC	volatile organic compound
WRS	Wilcoxon Rank Sum

Executive Summary

A baseline human health risk assessment (BHHRA) was conducted to evaluate risks associated with exposure to soil, groundwater, surface water and sediment at the Powerhouse 2 Ash Pits (AP2) at the Plum Brook Ordnance Works (PBOW), Sandusky, Ohio. The approach used in the BHHRA is consistent with methodologies described in the U.S. Environmental Protection Agency's primary risk assessment guidance documents, the site-specific work plan, and discussions and agreements between the Ohio Environmental Protection Agency, the U.S. Army Corps of Engineers Nashville and Huntington Districts, and Shaw Environmental, Inc.

Site History/Description. The PBOW facility was constructed on property comprising 9,009 acres in early 1941 as a manufacturing plant for 2,4,6-trinitrotoluene (TNT), 2,4-dinitroluene, and pentolite. Production of explosives at PBOW began in December 1941 and continued until 1945. It is estimated that more than 1 billion pounds of nitroaromatic explosives were manufactured during the 4-year operating period. After plant operations ceased, the manufacturing process lines were decontaminated by the Army in late 1945. After the property was certified as decontaminated, 3,280 acres of the property were initially transferred to the Ordnance Department, then to the War Assets Administration. In 1949, PBOW was transferred to the General Services Administration. The Department of the Army reacquired the 3,230 acres in 1954 and performed cleanup efforts from the mid-1950s until 1963.

Accountability and custody for the entire portion of the former PBOW property that had been under the accountability and custody of the Department of the Army were transferred to the National Aeronautics and Space Administration (NASA) on March 15, 1963. NASA performed further decontamination efforts during 1964. NASA has operated and maintained the former PBOW property since 1963, and the facility is currently the NASA Glenn Research Center, Plum Brook Station. NASA operates the property as a space research facility in support of their John Glenn Research Center at Lewis Field, Cleveland, Ohio. Most of the aerospace testing facilities built in the 1960s at the site are currently on standby or inactive status.

Three power stations, Powerhouse 1, Powerhouse 2, and Powerhouse 3, were constructed and utilized to support the TNT manufacturing process. The powerhouses generated steam that was used for space heating, driving compressors, and generating electrical power. Coal ash waste from each of the boilers in the powerhouses was collected in pits. Water was added to the ash, producing a slurry that flowed through a sluice trench to an ash sump located at the end of each

powerhouse. From the ash sump, the ash slurry traveled through a pipeline to a nearby surface water/ash impoundment, referred to as an "ash pit." AP2 was associated with Powerhouse 2.

AP2 is located in the western portion of PBOW, just west of Campbell Road and east of Pipe Creek. Historical drawings indicate that the AP2 surface impoundment was rectangular in shape, measuring approximately 400 feet long by 200 feet wide, and was surrounded by an earthen embankment. Based on topographical quadrangles, aerial photographs, and a visual site survey conducted in 1999, the ash pit areas are noted to have essentially remained unchanged other than vegetative growth. During a site walk conducted in October 2008, AP2 was observed to be overgrown with trees and nearly indistinguishable from surrounding forest. A nondescript, east-to-west ditch bisects AP2 and apparently drains to Pipe Creek during precipitation events.

Approach. The BHHRA evaluated exposure to chemicals in surface soil, subsurface soil, bedrock groundwater, overburden groundwater, surface water, and sediment for cancer risks and noncancer hazards. Only validated analytical data were used in the BHHRA. Validated analytical data are from samples collected during 2009, as reported in the 2010 site characterization report, and those collected in 1996 and reported in the limited site investigation report. A screening for chemicals of potential concern (COPC) was used to focus the evaluation on those chemicals most likely to present a risk to potentially exposed individuals. Each COPC in each medium was evaluated for exposure via the relevant exposure pathways and the resultant risk and hazards were estimated. One or more COPCs were found in surface soil, subsurface soil, bedrock groundwater, overburden groundwater, and sediment. No COPCs were identified for surface water, indicating that this medium does not contribute significantly to risks associated with AP2. The receptors listed below were evaluated for exposure and risk. The media evaluated for each receptor are shown in parentheses. Note that if no COPCs were found in a medium (e.g., surface water), then the relevant medium is still shown as evaluated.

- Current groundskeeper (surface soil)
- Future groundskeeper (combined surface and subsurface soil [referred to as "total soil"], bedrock groundwater, overburden groundwater)
- Indoor worker (surface soil, subsurface soil [air pathway only], bedrock groundwater, overburden groundwater)
- Construction worker (total soil, surface water, sediment)
- On-site resident (total soil, surface water, sediment, bedrock groundwater, overburden groundwater)

- Hunter (surface soil)
- Hunter's child (surface soil [venison pathway only]).

Note that there is currently no groundskeeper at AP2, but this receptor is included because current land use does not prohibit the presence of a groundskeeper or other on-site workers. The hunter and the hunter's child are both regarded as current receptors, because hunting is allowed seasonally by permit at AP2; hunting may also continue under future land use. No construction is planned for AP2, but a construction worker is assumed to be potentially exposed under current or future land use. The future groundskeeper, indoor worker, and resident are evaluated for future land use; these three receptors are also evaluated assuming groundwater use as potable water. Overburden groundwater and bedrock groundwater are evaluated for each receptor separately.

Results/Conclusions. Risks were characterized for each COPC identified in each medium for the relevant receptors. Noncancer hazards were evaluated against a target hazard index (HI) criterion of 1 and the PBOW team target incremental lifetime cancer risk (ILCR) of 1E-5 (i.e., 1 additional incidence of cancer per 100,000 individuals exposed). Cancer risk results are also compared to the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) acceptable cancer risk range of 1E-6 to 1E-4 (i.e., a rate of 1 additional incidence of cancer per 1,000,000 to 1 in 10,000 individuals exposed), which is referred to herein as the "NCP risk management range."

The overall HI and ILCR values are summarized in the following bullets; exceedances of PBOW cancer risk criteria (ILCR>1E-5) are shown as bold and exceedances of the noncancer hazard criterion (HI>1) or the NCP risk management range (1E-6 to 1E-4) are shown as bold italics:

- Current groundskeeper: ILCR = 2E-6; HI = 0.09
- Future groundskeeper – overburden groundwater use: ILCR = 2E-6; HI = 0.1
- Future groundskeeper – bedrock groundwater use: **ILCR = 7E-5**; HI = 1
- Future indoor worker – overburden groundwater use: ILCR = 2E-7; HI = 0.06
- Future indoor worker – bedrock groundwater use: **ILCR = 7E-5**; HI = 1
- Current/Future Construction worker: ILCR = 4E-7; HI = 0.4
- Resident – overburden groundwater use: ILCR = 5E-6; adult HI = 0.2; child HI = 0.9
- Resident – bedrock groundwater use: ***ILCR = 3E-4; adult HI = 3; child HI = 7***
- Current/Future Hunter: ILCR = 6E-8; HI = 0.003
- Current/Future Hunter's child: ILCR = 4E-13; HI = 0.000003.

The ILCR of the future resident that is hypothetically assumed to use bedrock groundwater as household tap water exceeds the NCP risk management range as well as the PBOW target cancer risk criterion. The HI values of the child and adult resident also exceed the target criterion of 1,

indicating that adverse human health risks under this scenario cannot be regarded as unlikely. The ILCR for each of the other future scenarios assumed to use bedrock groundwater as a potable source had an ILCR that exceeded the PBOW target cancer risk criterion, but were within the NCP risk management range.

Cancer risks and noncancer hazards to all potential receptors evaluated under current land use (i.e., current groundskeeper, construction worker, hunter, and hunter's child) are within or less than the NCP risk management range, are less than the target HI criterion, and are less than the PBOW cancer risk criterion, indicating that cancer risks are at acceptable levels and adverse noncancer effects are unlikely to occur under potential current scenarios. Similarly, all future scenarios which do not assume bedrock groundwater use meet the NCP risk management range, the target HI criterion, and the PBOW risk criterion.

Risks associated with the use of bedrock groundwater as tap water easily account for the majority of the ILCR and HI for the future groundskeeper, indoor worker, and future resident. Specifically, benzene in bedrock groundwater is by far responsible for the largest portion of cancer risk and noncancer hazards for these receptors. It is responsible for 97 to 99 percent of the total ILCR and 82 to 89 percent of the total HI for these three receptors. Benzene was identified as the only contaminant with an HI exceeding a value of 1 and an ILCR exceeding a value of 1E-5 for any receptor. Benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW, and petroleum was reported seeping at depth (47 feet and 69.5 feet) in AP2 bedrock well AP2-BEDGW-003. Without the contribution of benzene, the residual noncancer HI value for each receptor does not exceed a value of 1 and the ILCR does not exceed a value of 1E-5.

It is highly doubtful that bedrock groundwater would ever be used as potable tap water because of naturally poor water quality in the area and low, undependable groundwater yield. This poor quality is associated with the presence of hydrogen sulfide off-gassing, the associated health and aesthetic issues with high-sulfate water, and the presence of petroleum product as encountered in AP2 well AP2-BEDGW-003. Thus, if a more likely and serviceable source of tap water were to be used by a future groundskeeper or residential receptor (e.g., municipal water or perhaps even overburden groundwater), then the ILCR and HI values would meet the NCP risk management range (1E-6 to 1E-4), PBOW cancer risk criterion of 1E-5, and the target HI criterion of 1.

1.0 Introduction

This baseline human health risk assessment (BHHRA) evaluates potential human health risks associated with exposure to soil, groundwater, surface water, and sediment associated with Powerhouse 2 Ash Pits (AP2) located at the former Plum Brook Ordnance Works (PBOW), Sandusky, Erie County, Ohio. This work is being conducted for the U.S. Army Corps of Engineers (USACE) under the Defense Environmental Restoration Program-Formerly Used Defense Sites, managed by the USACE Huntington District, and technically overseen by the USACE Nashville District.

This BHHRA is consistent with U.S. Environmental Protection Agency (EPA) guidance and with the procedures established in the BHHRA for TNT Areas A and C soil (IT Corporation [IT], 2001a), the BHHRA for groundwater at PBOW (Shaw Environmental, Inc. [Shaw], 2006) and, most specifically, the AP2 BHHRA work plan (Shaw, 2010a).

1.1 Facility Location and Description

PBOW is located approximately 4 miles south of Sandusky, Ohio, and 59 miles west of Cleveland (Figure 1-1). Although located primarily in Perkins and Oxford Townships, the eastern edge of the facility extends into Huron and Milan Townships. PBOW is bounded on the north by Bogart Road, on the south by Mason Road, on the west by Patten Tract Road, and on the east by U.S. Highway 250. The areas surrounding PBOW are mostly agricultural and residential. The facility is currently surrounded by a chain-link fence, and the perimeter is regularly patrolled. Access by authorized personnel is limited to established checkpoints. Public access is restricted. Hunting is allowed by permit on portions of PBOW during the annual deer hunting season.

1.2 Facility History and Background

The PBOW facility was constructed on property comprising 9,009 acres in early 1941 as a manufacturing plant for 2,4,6-trinitrotoluene (TNT), 2,4-dinitroluene (DNT), and pentolite (USACE, 1995). Production of explosives at PBOW began in December 1941 and continued until 1945. It is estimated that more than 1 billion pounds of nitroaromatic explosives were manufactured during the 4-year operating period. The three explosive manufacturing areas were designated TNT Area A (TNTA), TNT Area B (TNTB), and TNT Area C (TNTC). Twelve process lines were used in the manufacture of TNT: four lines at TNTA, three lines at TNTB, and five lines at TNTC.

After plant operations ceased, the manufacturing process lines were decontaminated by the Army in late 1945. During decontamination, all structures, equipment, and manufacturing debris were either removed and salvaged or removed and burned. After the property was certified as decontaminated, 3,280 acres of the property were initially transferred to the Ordnance Department, then to the War Assets Administration. In 1949, PBOW was transferred to the General Services Administration (GSA). This transfer did not include the Plum Brook Depot area, which consists of 2,800 acres. The Department of the Army reacquired the 3,230 acres in 1954 and performed cleanup efforts from the mid-1950s until 1963. In 1955, the Army completed further decontamination of the manufacturing process lines. This effort included removal of contaminated surface and subsurface soil around the building and wooden and ceramic waste disposal lines containing TNT. Thousands of pounds of TNT were discovered in catch basins; this TNT was removed and burned at the burning grounds.

Two property use agreements were entered into by the Army and the National Advisory Committee of Aeronautics, the predecessor of the National Aeronautics and Space Administration (NASA), in 1956 and 1958, respectively. Accountability and custody for the entire portion of the former PBOW property (6,030 acres) that had been under the accountability and custody of the Department of the Army were transferred to NASA on March 15, 1963. NASA performed further decontamination efforts during 1964. The NASA decontamination process included removing contaminated surface soil above the drain tiles, flumes, etc.; destruction of all buildings by fire; then removal of all soil, debris, sumps, and above-grade portions of concrete foundations. Portions of the concrete foundations located below grade were left buried, and some that had been previously slightly above grade were likewise buried. All materials, including the soil in those areas, were flashed; the area was then rough-graded. The decontamination process was also to have included the burning of nitroaromatic-filled flumes that were excavated (Dames & Moore, Inc. [D&M], 1997).

NASA has operated and maintained the former PBOW property since 1963, and the facility is currently the NASA Glenn Research Center, Plum Brook Station. NASA operates the property as a space research facility in support of their John Glenn Research Center at Lewis Field, Cleveland, Ohio. Most of the aerospace testing facilities built in the 1960s at the site are currently on standby or inactive status. On April 18, 1978, NASA declared approximately 2,152 acres of PBOW as excess. This excess included former buffer areas that had not been used by the Army and were thus not subject to decontamination efforts. The Perkins Township Board of Education acquired 46 acres of the excess acreage and uses this area as a bus transportation area. The GSA retains ownership of the remaining excess acreage and currently has a use agreement

with the Ohio National Guard for 604 acres of this land. The details of land transactions are listed in the site management plan (USACE, 1995).

1.3 Powerhouse 2 Ash Pits Description and History

As noted previously, PBOW was built in early 1941 and manufactured TNT, DNT, and pentolite until 1945. Three power stations, Powerhouse 1, Powerhouse 2, and Powerhouse 3, were constructed and utilized to support the TNT manufacturing process. Each power station consisted of a main powerhouse, a coal storage area, and an aboveground fuel storage tank. The powerhouse buildings also contained two to four large coal-burning boilers, a turboelectric generator, a feed water treatment system, and several steam-driven or electric air compressors. The generated steam was used for space heating, driving compressors, and generating electrical power. Coal ash generated from each of the boilers in the powerhouse was collected in pits. Water was added to the ash, producing a slurry that flowed through a sluice trench to an ash sump located at the end of each powerhouse. From the ash sump, the ash slurry traveled through a pipeline to a nearby surface water/ash impoundment, referred to as an "ash pit" (USACE, 1995). AP2 was associated with Powerhouse 2.

AP2 is located in the western portion of PBOW, just west of Campbell Road in an area that appears to be an old surface impoundment (Figure 1-2). Pipe Creek lies just west of the western boundary of AP2. Historical drawings indicated that the surface impoundment was rectangular in shape, measuring approximately 400 feet long by 200 feet wide, and was surrounded by an earthen embankment (USACE, 1995). A nondescript, east-to-west ditch bisects AP2 and apparently drains to Pipe Creek during precipitation events. Based on topographical quadrangles (dated 1959 and 1969), aerial photographs, and a visual site survey conducted in 1999, the ash pit areas are noted to have essentially remained unchanged other than vegetative growth. As mentioned in the previous paragraph, ash from the coal-fired boilers was reportedly disposed of in the ash pit through a pipeline. During this investigation, the bermed area was observed and black to dark brown cinders were found within the bermed area at depths up to 4 feet. Environmental sampling was not conducted prior to the 1996 investigation.

During a site walk conducted in October 2008, the AP2 area was observed to be overgrown with trees and nearly indistinguishable from surrounding forest. Several moss-covered concrete slabs approximately 7 feet long and 3 feet wide were observed partially buried in the underbrush northwest of the powerhouse. A small hole constructed of mortared shale/limestone blocks and approximately 2 feet in diameter was located near the concrete slabs. Based on the construction, this appears to be a hand-dug well. The depth of the hand-dug well is 12 feet. A strong sulfur odor was noted to be emanating from it. The structure was nearly filled with water during a site

visit in December 2008. The origin of these concrete structures is not known with certainty; however, they appear to be remnants associated with a historical farmstead. A review of historical drawings and other documents indicates that a farmstead was present south of the slabs and hand-dug well. Remnants of other concrete and stone foundations are present in the former farmstead area indicated on the historic drawings. It is likely that all of the concrete and stone foundations, concrete slabs, and hand-dug well are remnants associated with agricultural activities predating the PBOW facility.

1.4 Groundwater Use and Site Use

Two groundwater aquifer systems are utilized for drinking water in the region: a carbonate aquifer to the west and a shale aquifer to the east (Shaw, 2005). PBOW is located within the transition of the two systems. Upwards of 170 private drinking water wells permitted by the Erie County Health Department are located within 4 miles of PBOW. Groundwater is not used on the PBOW facility. Permits are not required for agricultural wells. The Erie County Health Department does not permit using surface water as private drinking water. A shallow groundwater system exists within the unconsolidated material atop the bedrock under much of the site.

In this BHHRA, the term "facility" refers to the entire former PBOW property, and the term "site" refers to areas within PBOW under investigation, in this case AP2. Current use of the PBOW facility is classified as industrial for the purpose of identifying plausible human receptors and exposure pathways for evaluation in the BHHRA. D&M (1997) describes potential future uses of all or portions of the facility as follows:

- Industrial use (NASA activities and programs) may continue.
- Portions of the site may be used for recreation by hunters and fishermen.
- Portions of the site may be sold to state or local government or private individuals (no land-use restrictions were mentioned).
- Parts of the facility may be used in the future for residential or agricultural purposes.
- Parts of the facility may be used for training by the National Guard.
- Construction activities may be performed during development of any of the sites.

In summary, future site use of AP2 is considered to be industrial or residential for the purpose of developing receptor and exposure scenarios. There are no current NASA activities at AP2, but because PBOW is under NASA control, the potential for NASA human activities at AP2 may

exist. Hunting is currently allowed by permit in AP2; therefore, hunting is evaluated in this BHHRA. It is assumed that groundwater may be developed as a source of potable water in the future. Refer to Section 3.1.3 for a discussion of receptors and exposure scenarios.

1.5 Protocol for the Baseline Human Health Risk Assessment

The BHHRA was performed consistent with the AP2 BHHRA work plan (Shaw, 2010a). The AP2 BHHRA work plan was developed consistent with previous PBOW BHHRAs and is based on EPA, USACE, and Ohio Environmental Protection Agency (OEPA) guidance, including, but not limited to, the following:

- Ohio Environmental Protection Agency (OEPA), 2009a***, Use of U.S. EPA's Regional Screening Levels as Screening Values in Human Health Risk Assessments***, Technical Decision Compendium, Division of Emergency and Remedial Response, August.

- Ohio Environmental Protection Agency (OEPA), 2009b***, Human Health Cumulative Carcinogenic Risk and Non-carcinogenic Hazard Goals for the DERR Remedial Response Program***, Technical Decision Compendium, Division of Emergency and Remedial Response, August.

- U.S. Army Corps of Engineers (USACE), 1999, ***Risk Assessment Handbook, Volume I: Human Health Evaluation***, Engineer Manual EM 200-1-4.

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

- U.S. Environmental Protection Agency (EPA), 1991a, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance, Standard Default Exposure Factors***, Interim Final, Office of Solid Waste and Emergency Response, OSWER Directive: 9285.6-03.

- U.S. Environmental Protection Agency (EPA), 1992, ***Guidance on Risk Characterization for Risk Managers and Risk Assessors***, Memorandum from F. Henry Habicht II, Deputy Administrator, to Assistant Administrators, Regional Administrators, February.

- U.S. Environmental Protection Agency (EPA), 1997a, ***Exposure Factors Handbook***, Office of Research and Development, National Center for Environmental Assessment, Washington, D.C., EPA/600/P-95/002Fa, August.

- U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.

- U.S. Environmental Protection Agency (EPA), 2004a, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental***

Guidance for Dermal Risk Assessment), Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

- U.S. Environmental Protection Agency (EPA), 2009a, ***ProUCL Version 4.00.04***, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm.

- U.S. Environmental Protection Agency (EPA), 2009b, ***ProUCL Version 4.00.04 Technical Guide***, Draft, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, EPA/600/R-07/041, February.

- U.S. Environmental Protection Agency (EPA), 2009c, ***ProUCL Version 4.00.04 User Guide***, Draft, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, EPA/600/R-07/038, April.

1.6 Report Organization

The remainder of this document is organized as follows:

- ***Chapter 2.0, Data Evaluation.*** Identifies data sources, evaluates data quality, identifies chemicals of potential concern (COPC), and provides a background screening and evaluation protocol.

- ***Chapter 3.0, Exposure Assessment.*** Presents a conceptual site exposure model (CSEM), including contaminant sources, contaminant release mechanisms, receptors, and exposure pathways; describes exposure-point concentrations (EPC); and presents methods for calculating chemical intake and contact rates.

- ***Chapter 4.0, Toxicity Evaluation.*** Describes the potential for cancer and/or noncancer human health effects, provides an estimate of the quantitative relationship between the magnitude of dose or contact rate and the probability and/or severity of adverse effects, identifies the toxicity values that are used in the BHHRA, and describes the development of dermal toxicity values.

- ***Chapter 5.0, Risk Characterization.*** Combines the output of the exposure assessment and toxicity assessment to quantify the risk to each receptor at each site. Risks associated with exposure to all appropriate media for each site are evaluated.

- ***Chapter 6.0, Uncertainty Analysis.*** Identifies uncertainties in all phases of the BHHRA and discusses their individual effects on the risk assessment results, focusing on those issues that are most likely to have the greatest effect on risk estimates and/or risk management decisions.

- ***Chapter 7.0, Summary and Conclusions.*** Provides a brief summary of the BHHRA, including quantitative results, uncertainties, and pertinent site information.

Summary and discussion is focused on those results and issues that are most likely to directly affect site management decisions.

- ***Chapter 8.0, References.*** Presents the references used in the preparation of this document.

2.0 Data Evaluation

Data evaluation consists of a description of the appropriate data sources for each environmental medium sampled for each site, a discussion of data quality, a description of the methodology used for identification of the COPCs, and a summary of the COPCs for each AP2 environmental medium.

2.1 Data Sources

All soil, groundwater, sediment, and surface water samples from which the validated analytical data used in the BHHRA were derived are presented in Table 2-1. These data include surface soil, subsurface soil, surface water, sediment, and monitoring well samples. The sample summary table identifies each sample used in the BHHRA and the associated analytical suite. This includes samples collected as part of the limited site investigation for AP2 (IT, 1997a), and the remedial investigation (RI) samples (Shaw, 2010a). All sampling locations are shown on Figure 2-1.

2.2 Sorting the Analytical Data

Prior to initiation of BHHRA calculations, a database of chemicals present in site samples was compiled for each environmental medium. This database includes all chemicals detected as described in the RI report (Shaw, 2010b) and the limited site investigation report (IT, 1997a). The surface soil and subsurface soil are considered separate media. Surface and subsurface soil data are typically combined to assess exposures under the construction worker, future groundskeeper, and residential site use scenarios, which would involve excavation and mixing of surface and subsurface soil. Combined surface and subsurface soil data are termed “total soil” in the BHHRA. However, it is understood that a reference to the evaluation of exposure to total soil is actually an evaluation of exposure to both surface soil and subsurface soil. The total soil COPC list is created by combining the list of COPCs identified in surface and subsurface soil. If a chemical is either a surface soil COPC or a subsurface soil COPC (or both), then that chemical is a total soil COPC. The EPCs for total soil are typically generated from the combined data sets. As presented in Section 2.5, for AP2, a conservative determination was made to use the surface soil data set as total soil due to a lack of COPCs in subsurface soil.

Surface soil is defined as samples taken from within the interval of 0 to 1 foot below ground surface (bgs), and subsurface soil is defined as samples taken from depths greater than 1 foot bgs per the RI work plan (Shaw, 2009). The limited site investigation (IT, 1997a) historical soil samples were collected from a depth of either 0 to 0.5 or 2 to 3 feet bgs; these intervals are

consistent with surface soil and subsurface soil, respectively, as described in the RI report (Shaw, 2010b). As possible, the RI subsurface soil samples were generally collected at depths of 3 to 5 and 8 to 10 feet bgs. Note that where refusal or the water table was encountered before a depth of 10 feet bgs, samples from the deepest 2-foot interval were collected.

2.3 Evaluation of Data Quality

The quality of the analytical data was evaluated to select data for inclusion in the BHHRA. Data quality is expressed by the assignment of qualifier codes during the analytical laboratory quality control (QC) process or during third-party data evaluation. Some of the more common qualifiers and their meanings are as follows (EPA, 1989a):

U - Chemical was analyzed for but not detected; the associated value is the sample quantitation limit.

J - Value is estimated, usually below the reporting limit.

N - The analysis indicates an analyte for which there is presumptive evidence to make a tentative identification.

NJ - The analysis indicates a "tentatively identified analyte" and the reported value represents its approximate concentration.

UJ - The analyte was not detected above the reporting limit. However, the reporting limit is approximate and may or may not represent the actual limit of quantitation necessary to accurately and precisely measure the analyte in the sample.

R - QC indicates that the data are unusable (chemical may or may not be present).

B - The concentration in the sample is not sufficiently higher than concentration in the blank, using the 5-times, 10-times (5x, 10x) rule, which states that a chemical is considered a nondetect unless its concentration exceeds 5 times the blank concentration. For common laboratory contaminants (acetone, 2-butanone [methyl ethyl ketone], methylene chloride, toluene, and the phthalate esters), the sample concentration must exceed 10 times the blank concentration to be considered a detection.

"J," "N," and "NJ" qualified data are treated in the BHHRA as detected concentrations; "R" data and "B" qualified chemical data are not used. "U" qualified data (nondetects) are treated in the BHHRA as nondetections. The use of data with other, less common qualifiers is evaluated on a case-by-case basis. Generally, data for which the identity of the chemical is unclear are not used in the BHHRA. If confidence is reasonably high that the chemical is present, but the actual concentration is somewhat in question, the data generally are used in the BHHRA.

Some chemicals may be analyzed under two different analytical programs. For example, the DNT isomers are analyzed by EPA Method 8330 for nitroaromatics as well as EPA Method 8270C for semivolatile organic compounds. Risks associated with the reported values from both analyses are considered in the risk characterization (Chapter 5.0) and discussed as appropriate in the uncertainty analysis (Chapter 6.0), together with potential issues such as the relative sensitivities (i.e., differences in respective reporting limits) of the methods.

2.4 Identification of Chemicals of Potential Concern

A screening process is used to identify COPCs, which are the detected chemical analytes carried through the full risk assessment process. The objectives of COPC screening are to focus the risk assessment on those chemicals that may contribute significantly to overall risk and to remove from quantification those chemicals whose contribution is clearly inconsequential. COPC screening includes a risk-based screen which also considers status as a human nutrient (Section 2.4.1), a frequency-of-detection evaluation (Section 2.4.2), and a background screen (Section 2.4.3).

2.4.1 Risk-Based Screening

In the risk-based screen, the maximum detected concentration (MDC) of a chemical in a given medium is compared to the appropriate risk-based screening concentration (RBSC) for that chemical and medium. This is performed for each chemical in each medium. The units of the MDC and RBSC are the same for each chemical in a given medium. In groundwater, for example, both the MDC and RBSC have units of micrograms per liter (µg/L) in water.

If the MDC of a chemical is less than or equal to its RBSC, then the chemical is not considered further in the BHHRA for this medium because it is very unlikely that chemical concentrations at or below the RBSC would contribute substantially to risk. An analyte may be identified as a COPC if its MDC exceeds its RBSC. As indicated in Section 2.4, actual status as a COPC also depends on a chemical's frequency of detection (Section 2.4.2), concentration with respect to background (Section 2.4.3), and potential status as a nutrient. Groundwater RBSCs used in the BHHRA are derived from the Oak Ridge National Laboratory–EPA regional screening levels (RSL) table "tap water" values, and RBSCs for soil are derived from "residential soil" RSL values (Oak Ridge National Laboratory [ORNL]-EPA, 2009a). This is a change in the source of the RBSCs based on discussion between USACE and OEPA (2009c), and this change is consistent with recent OEPA (OEPA, 2009a) guidelines. Previously, the groundwater and soil RBSCs were derived from the corresponding EPA (2004b) Region 9 preliminary remediation goals (PRG). The soil RBSCs are applied to both surface and subsurface soil.

RSL values are based on a concentration equal to either an incremental lifetime cancer risk (ILCR) of 1E-6 or a noncancer hazard quotient (HQ) of 1, the threshold at (or below) which adverse noncancer effects are regarded as unlikely to occur. For the BHHRA, the noncancer values listed in the RSL tables are multiplied by a factor of 0.1 to provide additional protection for simultaneous exposure to multiple chemicals (OEPA, 2009a; ORNL-EPA, 2009b). This results in RBSC values associated with an HQ of 0.1. For cancer risk, the RSL values based on an ILCR of 1E-6 were used directly as RBSCs in the BHHRA. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) identifies acceptable exposure levels that are generally associated with concentration levels that represent an excess upper bound lifetime cancer risk to an individual of 1E-6 to 1E-4 (EPA, 1990). This range is hereinafter referred to as the "NCP risk management range." Cancer risks associated with RSL values represent the lower end of this range. The OEPA recognizes an overall cancer risk of 1E-5, which represents the logarithmic midpoint of the EPA risk management range, as a remedial goal (OEPA, 2009b). The RBSC for a chemical that elicits both cancer and noncancer health effects is selected based on either a cancer risk of 1E-6 or an HQ of 0.1, whichever associated concentration is lower.

Risks associated with exposure to sediment and surface water along Pipe Creek in the vicinity of AP2 are also evaluated in the BHHRA. Although RSLs have not been developed specifically for sediment and surface water, RBSCs can be derived from the RSLs based on site conditions at PBOW and the types of exposure to these media that may reasonably be anticipated. The routes by which receptors may be exposed to sediment (i.e., incidental ingestion, dermal contact) are similar to those by which receptors may be exposed to soil. However, sediment contact is expected to be appreciably less intense than soil contact, due to the lower duration and frequency of contact with sediment as compared with soil. Similarly, surface water exposure is expected to be much less intense than exposure to groundwater, as surface water from Pipe Creek is not regarded as a plausible source of drinking water, partly because the Erie County Health Department does not permit using surface water as private drinking water. Consequently, the exposure frequency is expected to be much lower for surface water, and the incidental ingestion of surface water would be much lower than the assumed intentional ingestion and use of groundwater from the tap. For these reasons, OEPA (1999) stated that unadjusted tap water PRG values (i.e., HQ = 1; ILCR = 1E-6) should be used for screening PBOW surface water. This screening protocol was adopted specifically because it was agreed that the magnitude of exposure associated with PBOW surface water exposure would be far less than that associated with household tap water. In other words, it was agreed that analytes with a maximum concentration at the PRG level would not contribute appreciably to overall risks and hazards for PBOW sites based on the exposure pathways of the surface water exposure scenarios for PBOW.

The same agreement was reached for screening sediment against unadjusted residential soil PRGs. This protocol has been updated to base RBSCs on RSLs rather than PRGs. Even though the sediment and surface water RBSCs are an order of magnitude higher for noncarcinogens than the respective soil and groundwater RBSCs, these sediment and surface water RBSCs are regarded as protective of sediment and surface water receptors for screening because of the lower exposure rate to these media. Note that this previously made PBOW Team agreement for screening surface water and sediment (OEPA, 1999), which considered site-specific conditions, takes precedence over current OEPA (2009b) guidance, which simply states that adjusted RSLs (i.e., RBSCs) for soil may be used to screen contaminants in sediment, and those for groundwater may be used to screen surface water. The surface water RBSCs also meet the outside-of-the-mixing-zone average non-drinking water concentrations for the Lake Erie Basin.

The screening of lead in soil and groundwater is a special case. The EPA (2006) Office of Water treatment technique action level of 15 µg/L for lead is listed in the RSL table, and the RSL User's Guide recommends this level for use as an RSL. Lead exposure and risk is evaluated separately from other chemicals using the EPA (2004c) Integrated Exposure Uptake Biokinetic (IEUBK) model. The selection of the action level as the drinking water RSL is based partly on IEUBK model. Section 5.2 of the RSL User's Guide states that if the average tap water concentration exceeds 15 µg/L and the average soil concentration exceeds a value of 250 milligrams per kilogram (mg/kg), then more than the IEUBK target (EPA, 2004c) of 5 percent of the population of exposed children would exceed 10 micrograms per deciliter of lead in blood. Because an RSL of 15 µg/L sufficiently screens for a potential average concentration of 15 µg/L, the RSL of 15 µg/L is used as the RBSC. However, it is possible that the residential soil RSL of 400 mg/kg, which is selected as the soil RBSC, may not screen for an average soil concentration of 250 mg/kg within a given data set. Therefore, the following conditions were placed on the screening of lead: 1) If either the soil RBSC or groundwater RBSC is exceeded, then the IEUBK blood-lead model is run using both average soil and groundwater concentrations, and 2) if the average soil concentration exceeds 250 mg/kg, then the IEUBK model is run, even if neither RBSC is exceeded, using average concentrations of lead in both soil and groundwater. Note that for AP2 soil, the MDC for lead (50.7 mg/kg) is less than both the RBSC (400 mg/kg) and the criterion for average concentration (250 mg/kg); the average lead concentration in AP2 surface soil was 20.4 mg/kg. Likewise, the lead MDC in overburden groundwater (2.4 µg/L) is less than the RBSC (15 µg/L) and lead was not detected in bedrock groundwater. Therefore, the IEUBK model was not run for the AP2 soil and groundwater.

There are no RSLs for sulfate in tap water, but the drinking water outside-of-the-mixing-zone average value of 250 milligrams per liter (mg/L) for the Lake Erie Basin is used as the RBSC.

This value is the same as the secondary drinking water regulation of 250 mg/L (EPA, 2006). Secondary drinking water regulations are non-promulgated values, based on aesthetic characteristics, which are used as guidelines for public water systems. A health-based advisory level of 500 mg/L also exists for sulfate (EPA, 2006).

The evaluation of essential nutrients is a special form of risk-based screening applied to certain ubiquitous elements that are generally considered to be required human nutrients. Essential nutrients such as calcium, chloride, iodine, magnesium, phosphorous, potassium, and sodium are generally considered innocuous at levels found in environmental media. There are no RSLs listed for these nutrients. Should any of these chemicals be identified as site related, an exposure analysis is performed whereby a daily dose of chemical from ingestion of the medium in question is calculated. The dose is compared with levels known or expected to be safe or toxic, and/or with recommended daily allowances, depending on the availability of data.

2.4.2 Frequency of Detection

When confidence that a given chemical is present is high, the data generally are used in the BHHRA. For most chemicals, their detection is presumptive evidence of their presence. As suggested by EPA (1989a), chemicals that are reported infrequently may be artifacts in the data that do not reflect the actual presence of the chemical in question. For the BHHRA, chemicals that are reported only at low concentrations in less than 5 percent of the samples from a given medium is excluded from further consideration, unless the presence of a given chemical is expected based on historical information about the site. Chemicals detected infrequently at high concentrations may identify the existence of contaminant plumes or limited “hot spots” and are retained as COPCs.

2.4.3 Comparison to Background

A number of the chemicals detected in PBOW environmental media may have MDCs that exceed RBSCs but are part of normal background concentrations. Such chemicals may include inorganics and polycyclic aromatic hydrocarbons (PAH), a class of organic compounds which form from natural or anthropogenic combustion of organic matter, including fossil fuels, and are generally ubiquitous in the environment. Airborne PAHs associated with non-U.S. Department of Defense sources may be deposited on soil and leach to groundwater. Benzene, toluene, ethylbenzene, and xylenes (BTEX) compounds, as well as PAHs, may also be associated with background concentrations due to the presence of natural petroleum-derived compounds present in the vicinity of PBOW (see Section 3.1.1).

Site concentrations of inorganic chemicals in site environmental media may be compared to those of PBOW background using a two-step approach: 1) background screening and 2) statistical data set testing. This second step (Section 2.4.3.2) is initiated only in cases where the concentration used for background screening is exceeded (refer to Section 2.4.3.1) and is performed after the risk characterization (Chapter 5.0), and the results are discussed in the uncertainty analysis (Chapter 6.0). No suitable background data set exists for overburden wells, so no background screening or statistical comparisons to background concentrations can be made for overburden groundwater samples. Similarly, no background screening or statistical evaluation can be performed for surface water or sediment analytical data, as these media lack PBOW background data sets.

Inorganics and organics are treated similarly from a quantitative perspective. However, all organics not eliminated on the basis of RBSC exceedance (Section 2.4.1) or infrequent detection (Section 2.4.2) are carried through the risk calculation process (exposure assessment, toxicity assessment, and risk characterization). As presented in Section 2.4.3.3, organic compounds are quantitatively eliminated as background related only through the uncertainty analysis if applicable.

2.4.3.1 Background Screening of Inorganics

Background screening is applied to each inorganic whose MDC in soil or limestone bedrock groundwater exceeds the RBSC and that cannot be characterized as an infrequently detected analyte. In background screening, the MDC is compared to the PBOW chemical-specific background screening concentration (BSC). The background data set and derivation of soil BSCs for all PBOW soil investigations are described in IT (1998), and the background data set and derivation of BSCs for PBOW bedrock groundwater are described in the 2004 groundwater report (Shaw, 2005). It is noted that the method agreed upon for the development of BSCs, as recorded in the September 11, 2002 PBOW Team Meeting minutes, differs from that shown in current OEPA (2004) guidance. This PBOW Team agreement, which has been used for all PBOW risk assessments to date, takes precedence over the subsequent OEPA (2004) guidance. Summary tables of the background data sets for soil and groundwater are provided as Tables 2-2 and 2-3, respectively. The background soil samples were collected from near the property boundary, away from any potential source areas, and the background groundwater wells were installed in off-site areas upgradient of PBOW sources. Briefly, BSCs were calculated for use at PBOW based on concentrations found in these background soil and bedrock monitoring well samples. Each BSC is either the MDC or the calculated 95th percent upper tolerance limit of the background data set, whichever value is lower (IT, 1998; Shaw, 2005). The background monitoring well samples were collected using low-flow samples and were unfiltered.

The background screening consists of comparing the MDC of the site data set to the BSC. The chemical may be regarded as a COPC if its MDC exceeds the BSC for that chemical or if no BSC can be determined due to a lack of detections in the background data set. COPCs are fully evaluated in the exposure assessment, toxicity assessment, and risk characterization. An inorganic analyte is not regarded as a COPC if its MDC is equal to or less than the BSC.

2.4.3.2 Statistical Data Set Testing of Inorganics

Statistical testing is performed to compare data sets of site inorganics data against the appropriate PBOW background data sets. As described in Section 2.4.3.1, the background data set for groundwater is found in the 2004 groundwater report (Shaw, 2005), and the background data set for soil is found in the site investigation for the acid areas (IT, 1998). As mentioned previously, background data sets do not exist for overburden groundwater, surface water, or sediment; therefore, a statistical background evaluation for COPCs in these media cannot be performed. The method for statistical comparison of the site data sets to the background data sets, described in Appendix M of Shaw (2005), is the Wilcoxon Rank Sum (WRS) statistical test (also known as the Mann-Whitney U test). WRS testing is performed for inorganics having MDCs that exceed the respective BSCs and are identified as COPCs based on RBSC comparison (Section 2.4.1) and frequency of detection (Section 2.4.2). All COPCs are carried through the risk characterization process; thus, statistical testing results are not used to screen out any chemicals.

Site data sets are interpreted as being significantly different from PBOW background if the associated p-level is less than 0.05. WRS statistical output and box-and-whisker plots of the various inorganic COPC data sets are appended to the BHHRA for each inorganic data set evaluated against the appropriate site background data set; the WRS results are discussed as part of the uncertainties. Analytes shown by the WRS results to exceed background (or for which the WRS testing was not run) are assumed to be site related, unless a qualitative chemical-specific explanation is presented in the uncertainties analysis as to why the analyte should not be regarded as site related. The WRS is not run if the COPC was not detected in the PBOW background data set. Data sets for which the WRS results do not suggest site relatedness (i.e., site data and background data are not statistically different) are still evaluated for risks and hazards in the risk characterization (Chapter 5.0). The WRS evaluation was not performed for AP2 because only two inorganic analytes were detected in soil or bedrock groundwater and neither of these contributed appreciably to a significant risk.

2.4.3.3 Treatment of Organics

As mentioned in Section 2.4.3, certain organic compounds (e.g., BTEX and PAHs) in site media may be attributable to background conditions. However, no organic compounds are summarily screened out. Instead, all detected organic compounds are carried through the risk assessment process (i.e., exposure assessment, toxicity assessment, risk characterization) unless screened out on the basis of comparison to RBSCs (Section 2.4.1) or characterized as infrequently detected (Section 2.4.2). Background contribution of organics are discussed in the uncertainties analysis, as applicable.

2.5 Data Evaluation Summary

Data summary tables are provided for the following media: surface soil (Table 2-4), subsurface soil (Table 2-5), bedrock groundwater (Table 2-6) and overburden groundwater (wells) (Table 2-7), sediment (Table 2-8), and surface water (Table 2-9). These tables provide the following information for each detected chemical in each environmental medium:

- Chemical name
- Frequency of detection
- Range of detected concentrations
- Range of detection limits
- Arithmetic mean of site concentrations
- 95th percent upper confidence limit on the arithmetic mean (UCL)
- Appropriate RBSC
- Appropriate BSC
- Selection/exclusion of chemical as a COPC
- EPC (for COPCs only).

The estimation of the UCL values for COPCs is discussed in Section 3.2.1. For duplicate samples, the associated values are averaged in the data summary, if both samples are detects or if both are nondetects; if only one of the duplicates is a detect, then this detected value is used in the data summary.

An analogous summary table for the overburden groundwater piezometer samples is appended (Appendix A). Note that these direct-push groundwater samples are collected for nature-and-extent purposes to determine groundwater flow direction and the placement of monitoring wells. These data are not sufficiently representative of groundwater conditions for use in risk assessment. Therefore, they are not used to identify COPCs and are not quantitatively evaluated in the BHHRA. This piezometer groundwater summary table is appended only to provide ancillary information.

The COPCs for each of the media are identified as follows:

- ***Surface Soil*** – Beryllium, thallium, Aroclor 1016, benzo(a)pyrene, benzo(b)fluoranthene
- ***Subsurface Soil*** – Thallium
- ***Bedrock Groundwater*** –Benzene, chloromethane, trans-1,3-dichloropropene, ethylbenzene, xylenes
- ***Overburden Groundwater*** – Nickel
- ***Sediment*** – Arsenic, chromium
- ***Surface Water*** – None.

As discussed in Section 2.2, the total soil COPCs include all surface soil and subsurface soil COPCs. In the case of AP2 soils, thallium is the only chemical identified as a COPC in subsurface soil. This analyte was also reported as a COPC in surface soil, in which it was reported at higher concentrations than in subsurface soil. Further observation of the surface and subsurface soil data sets revealed that all thallium RBSC exceedances were reported only in the 1996 analytical results; all 2009 soil samples were nondetect or were reported at concentrations less than the RBSC for thallium (Shaw, 2010b); also, all 2009 reporting limits were less than the RBSC. The apparent reason for the reported exceedances in the 1996 samples is that they were analyzed using inductively coupled plasma spectrometry (ICP) without mass spectrometry (MS). The 2009 samples used the combined ICP/MS method, which attained much lower reporting limits. False positives are common using ICP alone. It is likely that the reported detections among the 1996 samples were all false positives, and it is unlikely the subsurface soil truly contained any legitimate COPCs.

Because thallium is the only subsurface analyte identified as a COPC (and its presence at detectable levels is unlikely), a determination was made to conservatively assume that the surface soil data set be used in the BHHRA to represent total soil. It is recognized that this approach provides an additional high bias to the receptors evaluated for exposure to total soil.

3.0 Exposure Assessment

Exposure is the contact of a receptor with a chemical or physical agent. An exposure assessment estimates the type and magnitude of potential exposure of a receptor to COPCs found at or migrating from a site (EPA, 1989a). An exposure assessment includes the following steps:

- Characterize the physical setting.
- Identify the contaminant sources, release mechanisms, and migration pathways.
- Identify the potentially exposed receptors.
- Identify the potential exposure pathways.
- Estimate exposure concentrations.
- Estimate chemical intakes or contact rates.

The BHHRA characterizes potential exposures to COPCs in soil, groundwater, surface water, and sediment associated with AP2 media as portrayed by the CSEM in Section 3.1.

3.1 Conceptual Site Exposure Model

The CSEM provides the basis for identifying and evaluating the potential risks to human health in the BHHRA. The CSEM, graphically depicted on Figure 3-1, includes the receptors appropriate to all plausible site use scenarios and the potential exposure pathways. This presentation of all possible pathways by which a potential receptor may be exposed, including all sources, release and transport pathways, and exposure routes, facilitates consistent and comprehensive evaluation of risk to human health and helps to ensure that potential pathways are not overlooked. The elements of a CSEM include the following:

- Source
- Source media (i.e., initially contaminated environmental media)
- Contaminant release mechanisms
- Contaminant transport pathways
- Intermediate or transport media
- Exposure media
- Receptors
- Routes of exposure.

Contaminant release mechanisms and transport pathways are not relevant for direct receptor contact with a contaminated source medium (e.g., ingestion or dermal contact).

The receptors and pathways on Figure 3-1 reflect scenarios developed from information regarding site background and history, topography, climate, and demographics as presented by D&M (1997) and the sitewide groundwater investigation (IT, 1997b). On Figure 3-1, asterisks

identify exposure pathways that are complete and addressed in this BHHRA. Justification for exclusion of other pathways is provided in the Figure 3-1 footnotes, and the exclusion of other potential receptors is discussed in Section 3.1.3.8. No current or future exposure by off-site residents is evaluated. Most of the off-site residents are serviced by municipal water from surface water sources. Although there are numerous private groundwater wells in the vicinity, including eight within 1 mile of the facility boundary, none of these is used as a potable source. Based on the investigations of other PBOW sites, natural hydrocarbons and hydrogen sulfide are known to be present within the bedrock limestone, and shale formation groundwater generally provides low yields and is of low quality (e.g., Shaw [2008]); however, the groundwater underlying these sites cannot be summarily excluded for consideration as a tap water source based on natural water quality parameters or general assumptions concerning yield. Therefore, given the presence of numerous off-site wells and the assumption of unrestricted future land use on site, the development of groundwater for on-site residential (or on-site worker) use as tap water is regarded as plausible for purposes of this BHHRA.

3.1.1 Physical Setting

Climate/Meteorology. The climate in the Sandusky area is continental and strongly affected by Lake Erie. July is generally the warmest month (average high and low temperatures of 82 and 65 degrees Fahrenheit [°F], respectively), and January is generally the coldest (average high and low temperatures of 32 and 19°F, respectively) (The Weather Channel, 2004). On average, the first freezing day (low of 32°F or less) occurs in late October (average of three per month), and the last freezing day falls in early May (average of one per month) (National Oceanic and Atmospheric Administration, 1990). The average annual precipitation for Sandusky is 34.5 inches per year, with a monthly average of more than 3 inches per month falling in April through September and less than 3 inches in each of the other seven months (The Weather Channel, 2004). Precipitation is fairly evenly distributed throughout the year, with the fewest precipitation days (0.01 inch or greater) per month (10) occurring during July, August, September, and October, and the most (15) occurring in December and January (City-Data.com, 2004). The mean annual wind speed is 10.3 miles per hour (City-Data.com, 2004), with winds predominantly from the southwest (Science Applications International Corporation, 1991). Sandusky area winters are cloudy, with 33 percent sunshine during November through February, as compared with to 65 percent sunshine during the summer months (City-Data.com, 2004).

Geology. Three formations, all of Devonian Age, outcrop across PBOW, each of which was encountered in the upper 100 feet of bedrock at PBOW (Shaw, 2005). The Delaware Limestone is the lowermost formation screened by site wells. It is characterized as a hard, dense, finely

crystalline limestone and dolomite. The unit is typically buff colored and usually is described as fossiliferous. In the vicinity of PBOW, quarries mine limestone from the Delaware. Traces of natural petroleum-derived hydrocarbons and hydrogen sulfide are common in area quarries (Shaw, 2005). Hydrogen sulfide was detected in the groundwater during development of AP2 monitoring well AP2-BEDGW-003 (more than 200 parts per million). It was also detected in the breathing air in a piezometer (PZ-01) installed near the historical hand-dug farm well (Section 1.3); an obvious hydrogen sulfide odor was also noted as emanating from the hand-dug well.

Overlying the Delaware Limestone is the Olentangy Shale. Two members of the Olentangy Shale have been characterized at the site: the Plum Brook Shale and the overlying Prout Limestone. The Plum Brook Shale is interpreted to consist of approximately 35 feet of bluish-gray, soft, fossiliferous shale containing thin layers of dark, hard, fossiliferous limestone. The Prout Limestone has been described as a 15-foot-thick unit which occasionally outcrops in a 1,000- to 2,000-foot-wide, northeast-striking band across the middle portion of PBOW. It is described as a dark-gray to blue, very hard, siliceous, fossiliferous limestone or dolomitic mudstone. The uppermost formation at the site is the Ohio Shale. Only one member of the Ohio Shale, the Huron Shale, is present in the PBOW area. This unit has been described as black, thinly bedded, with abundant carbonaceous matter. Some large pyrite/carbonate concretions are also present in the Huron Shale, some as large as 6 feet in diameter (D&M, 1997). Based on drilling logs of previous boreholes in the area, the shale layer is not expected at AP2.

Soils. The bedrock overburden in Erie County is predominantly glacial till, glacial outwash, or glacial lacustrine (lake) deposits. In the vicinity of PBOW, the soil has been interpreted to be lacustrine. In many areas, the overburden also consists of highly weathered bedrock. The thickness of the overburden ranges from less than 1 foot to more than 25 feet. Overburden is thickest on the northern portion of the site in the vicinity of the Reactor Facility Area, where it has filled in a bedrock low (Shaw, 2005). In the vicinity of AP2, the overburden thickness ranges from 13 to 20 feet, based on soil boring logs.

The soil in the northwest portion of PBOW is placed within the Kibbie-Elnora-Tuscola-Colwood Association, which is described as nearly level to gently sloping. This soil is described as somewhat poorly drained, moderately well drained, and very poorly drained soils formed in outwash, lacustrine, and deltaic sediments. Along a strip from west to northeast across the site is the Castalia-Millsdale-Milton-Ritchey Association. This association is described as shallow to moderately deep, nearly level to moderately steep, well-drained and very poorly drained soils formed in glacial till, lacustrine sediments, and limestone residuum. Across much of the central portion of the site is the Hornell-Fries-Colwood Association, described as moderately deep to

deep, nearly level to gently sloping, somewhat poorly drained to very poorly drained soils formed in glacial till and lacustrine sediments over shale bedrock. At the extreme southeast portion of PBOW is the Pewamo-Bennington Association, described as nearly level to gently sloping, very poorly drained and somewhat poorly drained soils formed from glacial till and lacustrine sediments.

Hydrology. The two main water-bearing zones at PBOW are located in the overburden/shale unit and the limestone bedrock and are thus called the overburden/shale and bedrock water-bearing zones. The overburden and shale groundwater units show similar water levels in these two units, suggesting substantial vertical communication. Therefore, these two geologic units are combined for purposes of PBOW groundwater evaluation. As mentioned, shale appears to be absent at AP2. Data collected during the more recent investigations (Shaw, 2005; IT, 1997b, 1999, 2001b) indicate that groundwater in the overburden is in discontinuous pockets during dry time periods. The shallow overburden generally has low yields over most of PBOW due to the high percentage of silt and clay. In contrast, the limestone bedrock water-bearing zone is saturated year round. During periods of low precipitation, only limited migration of contaminants would occur in the overburden due to less infiltration. During a wet period, the general flow direction in the overburden water-bearing zone is to the west in the immediate vicinity of AP2. A hydrogeological study by the U.S. Geological Survey (1992) conducted in the glacial deposits of Sandusky in 1990 reported a horizontal hydraulic conductivity of 0.046 feet per day and a vertical hydraulic conductivity of 1.2 feet per day.

Regional groundwater flow in both the overburden/shale and the limestone bedrock is to the north-northeast towards Lake Erie, although local flow may vary due to local topography. Water in the limestone typically occurs in joints and along bedding planes or in solutionally enlarged openings. The conceptual model interprets that bedrock groundwater flow in the Delaware Limestone water-bearing zone is influenced by the frequency, orientation, density, and connectivity of the bedrock fractures. These fractures result in a localized groundwater flow direction that is likely to be to the west in the vicinity of AP2.

At PBOW, the bedrock groundwater has been subdivided into three zones based on location and yield. Zone 1 occurs in the north and northwestern portion of PBOW. It has been characterized as yielding from 100 to 500 gallons per minute (gpm) from karstic limestone approximately 100 feet below grade. Zone 2 is in the northern portion of PBOW and has yields of 15 gpm or less from limestone approximately 300 feet below grade. Zone 3 is located in the eastern and southern portion of the site in predominantly shale bedrock. In addition to being found in the shale, groundwater is located in thin sand and gravel horizons interbedded with silt and clay

deposits. Most Zone 3 wells are poor yielding, many of them providing less than 3 gpm (D&M, 1997). AP2 is in Zone 1.

Surface Water. No surface water bodies are located within the AP2 study area. Surface drainage in AP2 is generally westward toward Pipe Creek, which lies approximately 50 feet west of the study area. Pipe Creek is a small, perennial creek that flows south-southeast to north-northwest. AP2 contains low-lying areas in which water has been observed to pond after heavy rain events and in which water may pond seasonally.

3.1.2 Contaminant Sources, Release Mechanisms, and Migration Pathways

Contaminant sources, release mechanisms, and migration pathways are summarized on Figure 3-1. Briefly, AP2 received a coal ash slurry via a pipeline from Powerhouse 2, as described in Section 1.3. This pipeline is still in existence, as shown on Figure 1-2. This coal ash layer is present from the surface up to a depth of approximately 4 feet. Thus, the coal ash and any contaminants within it may represent surface and subsurface soil.

Leaching and downward migration may have carried contaminants from the subsurface soil to the groundwater. It is possible that that surface water and sediment at the nearby Pipe Creek may have been impacted by contaminants originating from the AP2 through surface drainage and groundwater migration.

3.1.3 Receptors and Exposure Pathways

Receptors, selected to represent the upper bound on exposure from all plausibly exposed groups of people associated with AP2 as well as the pathways by which they may be exposed to chemicals, are summarized on Figure 3-1 and in Table 3-1. The exposure variable values used in the contaminant intake models are compiled in Table 3-2. The receptors evaluated in the BHHRA include the following. Note that the current (Section 3.1.3.1) and future (Section 3.1.3.2) groundskeeper scenarios include different environmental media and are, thus, listed separately. Receptors such as the construction worker, hunter, and hunter's child may be viable under current or future land use. However, the evaluation for these receptors is exactly the same under current and future land use.

- Current groundskeeper
- Future groundskeeper
- Indoor worker (future)
- Construction worker (current/future)
- On-site resident (future)

- Hunter (current/future)
- Hunter's child (current/future).

Most BHHRAs are based on a reasonable maximum exposure (RME) assumption. The intent of the RME assumption is to estimate the highest exposure level that could reasonably be expected to occur, but not necessarily the worst possible case (EPA, 1989a; 1991a). It is interpreted as reflecting the 90 to 95th percentile on exposure. In keeping with EPA (1989a; 1991a) guidance, variables chosen for a baseline RME scenario for ingestion rate, exposure frequency, and exposure duration are generally upper bounds. Other variables, such as body weight and exposed skin surface area, are generally central or average values. In the case of contact rates consisting of multiple components, e.g., dermal contact with soil or water, which consists of a dermal absorption factor (ABS) and soil-to-skin adherence factor (AF) for soil, and permeability coefficient (K_p) and exposure time for water, only one variable, ABS or K_p, needs to be an upper bound. The conservativeness built into the individual variables ensures that the entire estimate for contact rate is sufficiently conservative.

The averaging time for noncancer evaluation is computed as the product of the exposure duration (years) multiplied by 365 days per year. The resultant noncancer averaging time is used to estimate an average daily dose over the entire exposure period (EPA, 1989a). For cancer evaluation, the averaging time is computed as the product of 70 years, the assumed human lifetime, times 365 days/year. This cancer-based averaging time is used to estimate an average daily dose prorated over a lifetime, regardless of the frequency or duration of exposure. The methodology used in deriving the averaging time for cancer risks assumes that the risk from short-term exposure to a high dose of a given carcinogen is equivalent to long-term exposure to a correspondingly lower dose, provided that the total lifetime doses are equivalent. This approach is generally consistent with the EPA (2005) policy of carcinogen evaluation, although it introduces considerable uncertainty into the BHHRA cancer risk estimates.

A fractional term (FI) is introduced into the chemical intake equations to account for scenarios in which exposure to a potentially contaminated medium associated with the site is less than total daily exposure to that medium. For example, if the site of interest is small or has unusual dimensions so that a groundskeeper would be unlikely to spend all (or nearly all) of his working time at the site, an FI value of less than 1 might be applied to the soil ingestion and dermal intake equations. An FI may also be split between two comparable media. For example, if a resident is exposed to both soil and sediment, FI values are introduced that apportion exposure between the two media such that the FI value for the two analogous media does not exceed a value of 1 (see Section 3.1.3.5).

Receptors and the associated exposure pathways are presented in the following subsections. Please note that some of the pathways considered for the receptors (e.g., surface water exposure; volatile organic compounds [VOC] in soil) could not be quantitatively evaluated because pertinent chemicals associated with these pathways were not identified as COPCs.

3.1.3.1 Current Groundskeeper

The groundskeeper scenario is designed to evaluate the upper bound for long-term site worker exposure to surface soil in the current site use scenario and total soil in the future site use scenario. It is noted that no groundskeeper is currently working at the site, but current land use does not prohibit a site worker from performing groundskeeping activities at the site.

Exposure to surface soil is evaluated for a (potential) current groundskeeper. Total soil is typically evaluated under the future groundskeeper use scenario because hypothetical future construction may include considerable excavation of subsurface soil. This soil may be spread on the surface and regraded such that some of the soil currently in the subsurface (i.e., 1 to 10 feet bgs) will be spread as surface soil (0 to 1 foot bgs). As discussed in Section 2.5, essentially no COPCs were identified for subsurface soil (aside from a likely false positive for thallium which was also reported at a higher concentration in the surface soil), so the conservative assumption was made that the surface soil data set would also be used to represent total soil. Groundwater use is also evaluated for the groundskeeper in the future site use scenario, as discussed in Section 3.1, which could theoretically be developed as a source of drinking water. It is assumed that any contact with surface water or sediment associated with Pipe Creek by this receptor would be infrequent and sporadic, because such contact would not be a part of the groundskeeper's regular duties or activities. Therefore, exposure to these media is not quantified.

Direct soil exposure pathways include incidental ingestion and dermal contact. Inhalation of dust raised by lawnmowers or other equipment is also evaluated because relatively high dust concentrations may be produced within the groundskeeper's breathing zone, with little opportunity for dilution by the large volume of ambient air.

Shaw's experience has been that surface soil that is contaminated with VOCs and that has been in place for extended periods is not a significant source of airborne VOCs, because infiltration and dissipation over time reduces residues at the surface (i.e., first few centimeters) from which volatilization would occur. However, as noted above, the data set for surface soil may include samples taken from up to 1 foot bgs, which would include the soil zone deeper than the top few centimeters, where dissipation has not reduced VOC concentrations. In other words, the surface

soil data set might indicate the presence of VOCs, although volatilization to the air is unlikely to be significant. Therefore, a surface soil-to-air volatilization model is not used in addition to the activity-based dust emissions model to estimate airborne concentrations of VOCs. Instead, the airborne concentrations estimated by the dust emissions model are assumed to sufficiently estimate levels of VOCs that may arise from volatilization, because the dust emissions model treats the VOCs as if they were located at the surface. It is assumed that VOC emissions from subsurface soil (i.e., at depths greater than 1 foot bgs) would be attenuated by the overlying soil so that concentrations in ambient air would not be toxicologically significant.

The groundskeeper is assumed to be a 70-kilogram (kg) adult who works 8 hours per day, approximately 5 days per week year-round on site for a total of 250 days/year for 25 years (EPA, 2004a). The respiratory rate for the groundskeeper is assumed to be 20 cubic meters (m^3) per 8-hour workday or 2.5 cubic meters per hour (m^3/hr) (EPA, 1991a), and the soil incidental ingestion rate is assumed to be 100 milligrams per day (mg/day) (EPA, 2002). The groundskeeper is assumed to be exposed dermally to soil. An exposed skin surface area of 3,300 square centimeters (cm^2) and a soil AF of 0.2 milligrams per square centimeter (mg/cm^2) are assumed (EPA, 2004a).

3.1.3.2 Future Groundskeeper

A future groundskeeper would be exposed to soil via the same exposure pathways as the current groundskeeper described in Section 3.1.3.1. However, the future groundskeeper scenario assumes that construction has taken place and that some of the soil currently in the subsurface has been brought to the surface during earthmoving activities. Therefore, the future groundskeeper is assumed to be exposed to a combination of surface and subsurface soil (total soil) rather than surface soil alone.

In addition, a hypothetical future groundskeeper is assumed to be exposed to groundwater, which could theoretically be developed as a source of drinking water in the future. His drinking water ingestion rate is assumed to be 1 liter per day (L/day) (EPA, 1991a). He may also experience dermal contact with groundwater used to clean equipment and to rinse dust or perspiration from his body. For this evaluation, it is assumed that the head, forearms, and hands, approximately 3,300 cm^2 of his body (EPA, 2004a), would be exposed intermittently for up to 1 hour/day. Because exposure is assumed to be intermittent rather than continuous, organic chemical uptake across the dermis would not reach steady state, which guides the selection of the EPA (2004a) model used to quantify this pathway (see Section 3.3).

3.1.3.3 Indoor Worker

This receptor scenario was created to evaluate exposure to indoor airborne VOCs entrapped in a building. VOCs released from subsurface soil may enter a building through joints or cracks in the foundation or slab. The indoor worker is also potentially exposed to surface soil via incidental ingestion. Exposure to COPCs in surface soil via dermal contact and inhalation of airborne dust and VOCs from surface soil, although plausible, are expected to be less significant than incidental ingestion, because this receptor spends his work time indoors. Therefore, dermal contact and inhalation of dust and airborne VOCs from surface soil are not quantified separately from ingestion exposure (EPA, 2002). Under a future use scenario for this receptor, construction of a building would be necessary. This would require excavation and regrading of soil. Normally, when construction is involved, such as for the future groundskeeper or resident, total soil rather than surface soil would be evaluated for ingestion exposure. However, the chief purpose for this receptor is to evaluate exposure via vapor intrusion of contaminants from subsurface soil into indoor air. Thus, the evaluation of direct contact with subsurface soil as a component of total soil would equate to "double counting" of COPCs in subsurface soil. Also, the groundskeeper reflects a worst-case exposure for a long-term worker with respect to direct contact with both surface soil and total soil. Therefore, direct contact with surface soil for the indoor worker is included to reflect a more complete exposure scenario, but direct contact of subsurface soil is most effectively addressed from an RME perspective by the groundskeeper.

The indoor worker is assumed to be a 70-kg adult who works 8 hours/day, approximately 5 days/week year-round on the site for a total of 250 days/year for 25 years (EPA, 2002). His incidental soil ingestion rate is assumed to be 50 mg/day (EPA, 2002), and his inhalation rate is assumed to be 20 m^3/8-hour workday (EPA, 1991a).

A future indoor worker is assumed to be exposed to groundwater, which could theoretically be developed as a source of drinking water (see Section 3.1). His drinking water ingestion rate is assumed to be 1 L/day (EPA, 1991a). Some indoor worker positions may require relatively frequent dermal contact with groundwater as well, e.g., a food preparer/cafeteria worker who would wash his hands, produce, equipment, etc. For this evaluation, it is assumed that the head, forearms, and hands, approximately 3,300 cm^2 of his body (EPA, 2004a), would be exposed intermittently for up to 1 hour per day. Because exposure is assumed to be intermittent rather than continuous, organic chemical uptake across the dermis would not reach steady state, which guides the selection of the EPA (2004a) model used to quantify this pathway (see Section 3.3.4).

3.1.3.4 Construction Worker

The construction worker scenario is used to evaluate short-term exposure to surface and subsurface soil (total soil) in either the current or future land-use scenario. Construction projects are expected to be infrequent. It is assumed that the construction worker participates in only one construction project on the site. Note that no construction projects are anticipated for this site, but AP2 is currently under NASA control and a construction project may be possible under current land use. Relevant exposure pathways include incidental ingestion and dermal contact, inhalation of dust raised by operating construction equipment, and inhalation of airborne VOCs released from subsurface soil during excavation and grading. Exposure to groundwater by the construction worker is also possible; however, if on-site groundwater were developed as a tap water source, other potential future groundwater receptors such as the groundskeeper would have longer and/or more frequent groundwater exposure. Therefore, groundwater exposure is not evaluated for the construction worker.

The construction worker may also be exposed to surface water and sediment at Pipe Creek during projects such as installation of underground utilities or rerouting stream flow. Dermal contact is the most significant pathway for exposure to surface water. Incidental ingestion of surface water is also possible but is not expected to be nearly as significant as dermal contact. Inhalation of VOCs from surface water is also possible, but the large volume of outdoor air and natural air currents are expected to dilute airborne concentrations, so that this pathway is expected to be less significant than dermal contact, which is quantified. For these reasons, incidental ingestion and inhalation of VOCs from surface water are not quantified separately from dermal contact. Dermal contact and incidental ingestion may be important pathways for exposure to sediment, and both are evaluated.

The construction worker is assumed to be a 70-kg adult who works 8 hours/day, approximately 5 days/week. This represents an annual exposure frequency rate of about 250 days per year, which is the same as described for the groundskeeper (Section 3.1.3.1) and indoor worker (Section 3.1.3.3). Construction projects involving soil exposure are assumed to last 6 months. The respiratory rate for the construction worker is assumed to be 20 m^3/8-hour workday (2.5 m^3/hr) (EPA, 1991a). A soil ingestion rate of 330 mg/day is assumed for the construction worker (EPA, 2002). A dermal soil AF for the construction worker of 0.3 mg/cm^2 and an exposed body surface area of 3,300 cm^2 are assumed, which represent the head, hands, and forearms (EPA, 2002; 2004a).

The construction worker may be exposed to surface water and sediment during the 6-month construction period. The construction worker dermal exposure parameters for sediment are

assumed to be exactly the same as those for soil. Dermal exposure to surface water is assumed to occur for up to 4 hours per day, or one-half the normal work day. It is assumed the exposure to surface water is intermittent during this period. An exposed body surface area of 3,300 cm^2, the same as for sediment and soil, is assumed for exposure of the construction worker to surface water. It is expected that the construction worker would wear appropriate footgear and leg protection to minimize surface water and sediment exposure to the legs.

The construction worker scenario described above provides for several different kinds of construction projects, such as upland excavation and building projects (exposure primarily to soil), as well as stream rerouting (exposure primarily to surface water and sediment). It is unlikely, however, that a single construction worker would participate in all these activities during a given project. Therefore, the evaluation described above is probably overly conservative and may represent some double counting. For example, it is unlikely that the construction worker would be simultaneously ingesting soil, sediment, and surface water. Similarly, the air in his breathing zone is not likely to contain the reasonable maximum concentrations of COPCs estimated for soil while he is exposed to surface water. The potential for double counting is not expected to contribute significantly to total risk estimates summed across chemicals, pathways, and media. Should construction worker risk estimates exceed acceptable limits, risk and hazard estimates may be performed using refined exposure assumptions based on the physical characteristics of the site. For example, an upland excavation and building project may be assumed for one or more areas of the site, and a stream rerouting project may be assumed for another. Effectively, the risks and hazards associated with surface water/sediment exposure and soil exposure could be separated. This approach would more precisely reflect plausible exposure scenarios, reduce the likelihood of double counting, and more accurately identify risk-driving media and chemicals. These refined estimates would be presented in the uncertainty analysis.

3.1.3.5 On-Site Resident

The on-site residential scenario is created to evaluate the upper bound for long-term exposure to site soil, surface water, sediment, and groundwater under the future land-use scenario. Residential land use is plausible because property surrounding the PBOW facility includes rural residential use. This type of land use is also consistent with assumed future land use of other PBOW sites such as TNTA and TNTC (IT, 2001a).

The on-site residential scenario is evaluated assuming a 30-year residential exposure scenario, considering exposure to a resident as a young child (6-year duration, ages 1 through 6 years) through the adult portion of life spent at this residence (24-year duration) (EPA, 1991a). Noncancer hazard estimates are derived separately for the child and adult life stages. Cancer risk

is estimated as the sum of the risks calculated for the adult (24 years) and the child (6 years) (EPA, 2002; ORNL-EPA, 2009b).

The resident is assumed to be exposed directly to total soil, because residential development would involve excavation and regrading, which would mix surface and subsurface soil. Relevant pathways for total soil exposure include incidental ingestion, dermal contact, and inhalation of dust and VOCs. Evaluation of VOCs from total soil is addressed during evaluation of airborne dust, as described for the groundskeeper. For evaluating inhalation of airborne dust, it is assumed that 80 percent of the soil surface is covered with pavement or vegetation. Inhalation of VOCs released from subsurface soil entrapped in indoor air is also evaluated. The resident is also assumed to be exposed to VOCs that have been released from subsurface soil through cracks in the building foundation to indoor air. It is noted that because some of the subsurface soil is expected to be brought to the surface in the future, using only subsurface soil data will conservatively result in some double counting of exposure to any VOC COPCs that may be present in the subsurface soil. This may be addressed in the uncertainty analysis should the subsurface soil-to-indoor air pathway significantly affects risk and hazard estimates. This pathway did not contribute significantly to risk at AP2, as no VOCs were identified as COPCs.

It is assumed that, under future residential land use, the overburden and limestone bedrock water units will be developed as sources of potable water (see Section 3.1). The resident is assumed to use groundwater underlying the site as the sole source of household tap water. Exposure to COPCs in groundwater would occur via ingestion, dermal contact during bathing/washing, and inhalation of VOCs released to the air during household use of tap water associated with multiple household uses.

The resident could have access to Pipe Creek in the vicinity of AP2 and could be exposed to contaminants in surface water and sediment associated with AP2. Plausible exposure pathways include dermal contact with surface water and incidental ingestion and dermal contact with sediment. Incidental ingestion of surface water in a wading scenario is considered less significant than dermal contact and is not quantified separately from dermal contact. Inhalation of VOC emissions from surface water is also possible, but the large volume of outdoor air and natural air currents are expected to dilute airborne concentrations, so that this pathway is expected to be less significant than dermal contact, which is quantified. For these reasons, the inhalation of VOC emissions from surface water is not quantified separately from dermal contact.

The adult resident is assumed to be a 70-kg person with an incidental soil ingestion rate of 100 mg/day and an inhalation rate of 20 cubic meters per day (m^3/day) or 0.83m^3/hr (EPA, 1991a). A

body surface area of 5,700 cm^2, representing the hands, forearms, head, and lower legs, is assumed to be available for dermal exposure to soil (EPA, 2004a). A soil AF of 0.07 mg/cm^2 is used as the default RME value for the adult resident (EPA, 2004a). The adult resident is assumed to be exposed for 350 days/year for 24 years (EPA, 1991a; 2002).

The child resident is assumed to be a 1- through 6-year-old child with an average body weight of 15 kg, a soil ingestion rate of 200 mg/day, and an average inhalation rate of 10 m^3/day or 0.417 m^3/hr (EPA, 2004d). An average body surface area of 2,800 cm^2 throughout the 6-year childhood exposure period, representing the head, hands, forearms, lower legs, and feet, is assumed for dermal contact with soil (EPA, 2004a). A soil AF of 0.2 mg/cm^2 is used as the default RME value for the child resident (EPA, 2004a). The child resident is assumed to be exposed for 350 days/year for 6 years (EPA, 1991a; 2002).

It is assumed that the resident would visit Pipe Creek for 8 hours/day, 2 days/week during the warmer half of the year. This resident is assumed to wade for 3 hours/day on 52 days of the year. Mechanisms of exposure to soil and sediment are likely to be similar. Therefore, the incidental soil ingestion rate of 100 mg/day, the surface area of 5,700 cm^2, and the AF of 0.07 mg/cm^2 are also applied to sediment exposure in the adult. Similarly, the resident child soil ingestion rate of 200 mg/day, skin surface area of 2,800 cm^2, and soil AF of 0.2 mg/cm^2 are applied to sediment exposure for this receptor. Pipe Creek in the vicinity of AP2 generally contains less than 1 foot of water, limiting the surface area of the body that would typically be exposed. It is assumed that an adult body surface area of 7,000 cm^2 is available for exposure to surface water. This represents the same body parts to which soil and sediment would be exposed (i.e., hands, forearms, head, and lower legs) plus the feet (EPA, 1997a; 2004a). The body surface area of 2,800 cm^2, representing the hands, forearms, head, lower legs, and feet, used for soil and sediment exposure in the young child are also used for surface water exposure for this receptor.

EPA (1989a) permits the development of an FI to reflect the proportion of total daily exposure that a receptor obtains from potentially contaminated medium (refer to Section 3.1.3). For this receptor, the FI is used to apportion the resident's time of exposure between site soil and sediment. It is assumed that the resident spends 16 hours/day awake and potentially exposed to soil or sediment. As previously noted, 350 days/year are available for contact with soil; 52 of those days are also available for contact with sediment. It is assumed that contact with soil and sediment does not occur simultaneously; i.e., on those days when the resident spends time at the streams, 8 hours would be spent in contact with soil and 8 hours would be spent in contact with sediment. Therefore, the fraction of exposure to soil is 16 hours/16 hours = 1 on the 298 days without time spent at the streams, and the fraction of exposure to soil is estimated as 8 hours/16

hours = 0.5 on the 52 days with some time spent at the streams. A weighted fraction of 0.93 (rounded to 0.9) is estimated for exposure to soil over the entire 350 days/year exposure frequency. A weighted fraction of 0.07 (rounded to 0.1) is estimated for exposure to sediment over the entire 350 days/year exposure frequency.

An adolescent resident may be the most likely individual to have regular exposure to sediment and surface water associated with Pipe Creek. It is not expected that adults would regularly visit Pipe Creek, as it does not support game fish and would seemingly not provide any attraction. It is unlikely that a young child (i.e., ages 1 through 6) would frequent Pipe Creek for substantial portions of time, because such young children (especially at the lower end of this age range) would require continued adult supervision. However, as described above, it is conservatively assumed that the resident will be regularly exposed to surface water and sediment for 30 years, 6 years assumed as a young child and 24 years assumed as an adult. For cancer effects, the 30-year exposure to surface water and sediment represented by both the young child and adult are combined. This approach is more conservative than evaluating an adolescent and is also consistent with BHHRAs performed for PBOW sites in the past.

With respect to groundwater exposure, it is assumed that an adult resident ingests 2 L/day of tap water (EPA, 1991a) and that the young child drinks 1 L/day (ORNL-EPA, 2009b). The total body surface areas of the adult and of the young child resident are assumed to be exposed to tap water while bathing/showering. The total surface area for an adult is assumed to be 20,000 cm^2 and the total surface area for the young child is assumed to be 6,600 cm^2 (EPA, 1997a). Both the child and adult resident are assumed to be dermally exposed to COPCs in groundwater while bathing/showering. The child is assumed to bathe for 20 minutes per day (0.33 hour/day) (EPA, 1997a), and the adult is assumed to shower for 12 minutes per day (0.2 hour/day) (EPA, 1997a). Inhalation rates of 0.833 m^3/hr for the adult (EPA, 1991a) and 0.416 m^3/hr for the child (EPA, 2004d) are used. Because EPA (1997a) lists a 90th percentile for time spent in a residence as over 23 hours per day, it is conservatively assumed that the resident spends 24 hours per day in the house.

3.1.3.6 Hunter

This scenario is created to evaluate the potential for contaminants in soil to affect food chain pathways. AP2 provides habitat for deer and other wildlife. Deer hunting is permitted on portions of the PBOW facility, including AP2. Therefore, a hunter who consumes his game is evaluated in the AP2 BHHRA. Hunting is also a potential land use into the future if general land use does not change at AP2.

Many kinds of game animals may be hunted and consumed (e.g., squirrel, pheasant and other upland birds, turkey, deer); however, the deer is the species most likely to contribute meaningfully to the diet. Therefore, this evaluation is limited to a deer hunter. Potential exposure pathways include incidental surface soil ingestion, dermal contact with surface soil, and ingestion of venison from deer that browse plants growing on contaminated surface soil, all of which are evaluated quantitatively. Inhalation of airborne dust from wind currents is a potentially complete exposure pathway; however, vegetation reduces dust emissions to insignificant levels (EPA, 1996), and it is assumed that the deer hunter would spend virtually all of his time on vegetated rather than bare soil. Therefore, it is assumed that inhalation exposure would contribute much less than incidental ingestion, and the inhalation exposure pathway is not quantified separately from ingestion.

Inhalation exposure to airborne VOCs from subsurface soil and surface water is not evaluated for the reasons previously explained for other receptors. Also, ingestion and dermal exposure to surface water and sediment are expected to be negligible for this receptor, as contact with these media would generally be avoided during hunting activities.

The deer hunter is assumed to be a 70-kg adult who harvests deer and consumes venison over a 30-year period. It is assumed that he spends 14 days per year hunting on PBOW. His incidental soil ingestion rate is assumed to be 100 mg/day (EPA, 1991a). Hunting at PBOW occurs in the fall and winter. Given the temperate climate of northern Ohio during hunting season, a hunter would dress appropriately, with typically only the hands and head exposed, at most. The default industrial RME exposed skin surface area of 3,300 cm^2, which represents the hands, forearms, and head (EPA, 2004a), is conservatively assumed for the hunter. The default industrial RME soil AF of 0.2 mg/cm^2 (EPA, 2004a) is also assumed.

Data regarding the rate of venison ingestion were not located; therefore, a hypothetical scenario is adapted from the assumptions applied to a similar site in West Virginia (IT, 2000) and subsequently applied to TNTA and TNTC (IT, 2001a). A highly conservative but plausible scenario consists of a hunter who kills one deer from AP2 property each year. It is assumed that the hunter eats 10 pounds (4.5 kg) of venison per year (Sharp, 1995). This consumption rate corresponds to 0.013 kilograms per day (kg/day) (0.186 grams per kilogram of body weight per day [g/kg-day]) of venison for each of the 350 days per year (EPA, 1991a) that the hunter spends at his residence.

3.1.3.7 Hunter's Child

It is likely that a successful hunter, described in Section 3.1.3.6, would share his venison with the rest of the family, which may include small children. Small children, however, would be unlikely to accompany the hunter afield. Therefore, the direct exposure pathways evaluated for the hunter (i.e., incidental ingestion and dermal contact with soil) are not evaluated for the small child.

Data regarding the rate of venison ingestion by small children were not located. However, if it is assumed that venison may replace beef in the diet, the differences in beef consumption between adults and children can be used to estimate a venison ingestion rate for children. EPA (1997a) provides per capita beef intake data for <1- to 5-year-old children ranging from 0.941 to 1.46 g/kg-day (time-weighted average of 1.296 g/kg-day). EPA (1997a) provides per capita beef intake data for 12- to 70-plus-year-old adults ranging from 0.568 to 0.83 g/kg-day (time-weighted average of 0.727 g/kg-day). From these data, it can be estimated that the rate of beef consumption for small children, expressed on a body weight basis, is approximately 1.8 times that of an adult. Therefore, a venison ingestion rate of 0.335 g/kg-day is estimated for a young child from the venison ingestion rate of 0.186 g/kg-day for the adult. Assuming that the child is 1 through 6 years old with an average body weight of 15 kg (EPA, 1991a; 2002), the child's venison ingestion rate may be expressed as 0.005 kg/day.

3.1.3.8 Other Receptors Not Considered

Another plausible receptor group is delivery personnel. These receptors, however, would be less intensively exposed to soil than the groundskeeper; therefore, their exposures are not evaluated. AP2 could become part of the area used for National Guard training activities. National Guard trainees, however, may be less exposed to any of the potentially contaminated media than the receptors identified above. Because they would likely not represent an upper bound for nonresidential exposure, these receptors are not evaluated. Pipe Creek is too small to support game fish. Therefore, fish ingestion as an indirect pathway for exposure to surface water and sediment is not evaluated. Also, as discussed in Section 3.1, off-site use of groundwater is not evaluated because nearby residents use municipal water (from surface water sources) as a potable source, and potential on-site users would be exposed to higher concentrations of contaminants in groundwater.

3.2 Quantification of Exposure-Point Concentrations

The EPC is an estimate of the concentration of a COPC in a given medium to which a receptor may be exposed over the duration of the exposure. An EPC may be based on chemical concentrations in media that have been directly measured using laboratory analysis, or it may be derived based on environmental medium-to-medium transport modeling. The EPCs of COPCs in

soil, groundwater, surface water, and sediment are derived based on measured analytical data. Note that the EPC for dermal exposure to VOCs in groundwater is based on one-half the EPC concentration derived from the measured concentrations in groundwater (Sections 3.2.1 and 3.2.2.3). This value is used because it is assumed that 50 percent of the groundwater VOC concentration is volatilized during normal household use (Section 3.2.2.2). Concentrations of COPCs in air and venison are not measured (and in some cases cannot reasonably be measured) but are based on models that use the EPCs of COPCs in the appropriate directly measured media (i.e., soil and groundwater) as input values.

Section 3.2.1 describes the approaches used to derive EPCs for direct exposure to soil, groundwater, surface water, and sediment based on analytical measurements from samples of these media. Models to derive EPCs for the air are described in Sections 3.2.2.1 and 3.2.2.2, and the model used to derive venison EPCs is described in Section 3.2.2.4.

3.2.1 Soil, Groundwater, Surface Water, and Sediment Concentrations

Exposure to an environmental medium is generally assumed to be random, and the EPC should be the arithmetic average encountered over the duration of exposure (EPA, 1989a). Therefore, the population mean concentration, if known, would be the ideal value selected as the EPC. The sample mean is an obvious estimate of the population mean. However, uncertainties exist as to how well the sample mean represents the population mean. Therefore, EPA (1989a) has recommended the inclusion of a UCL for RME evaluation as a conservative estimate of the true mean exposure concentration.

The EPA (2009a,b,c) ProUCL (Version 4.00.04) software was used to estimate UCLs for the data sets of all environmental media represented by at least five samples. If the data set consists of fewer than five data points, the MDC was selected as the EPC. Analytical data from field duplicates are averaged with originals to yield one result for use in the statistical manipulations (see Section 2.5). One-half the reporting limit is used as the ProUCL input concentration for nondetects. Nondetect sample results with aberrantly high detection limits due to matrix interferences or other sample-specific causes are included in the initial ProUCL calculations. This is a conservative approach, as EPA (1989a) recommends that nondetected results with aberrantly high detection limits be removed from the data set so that calculation of the UCL is not unduly skewed by a nondetect. Because the latest version of ProUCL (EPA, 2009a) includes mathematical manipulations that are more robust than previous versions, single elevated detection limits are less likely to skew the UCL estimates than in the past. If it is observed during the risk characterization that an elevated nondetect value skews a UCL estimate such that this value substantially affects the result of the risk estimate, the ProUCL model is rerun without the

elevated nondetect value and the risks are recalculated. If this recalculation is performed on a data set, data eliminated for the recalculation are identified in the risk characterization and discussed in the uncertainty analysis. The re-evaluation of elevated nondetects was not performed in the AP2 BHHRA.

ProUCL generates a variety of UCL estimates for each data set. The ProUCL output for each COPC is included in Appendix B. Generally, the results of one or two (sometimes more) of the UCL estimates are recommended. This recommendation is based on a variety of factors, including the distribution (i.e., normal, lognormal, gamma, or not discernable) that provides the best fit, number of nondetects, size of the data set, and skewness. In general, the UCL recommended by ProUCL is selected as the EPC. Occasionally, ProUCL recommends the 97.5 or 99 percent upper confidence limit on the arithmetic mean estimated by the Chebyshev method. In these cases, the UCL estimated by the Chebyshev method (95th percent) is selected as the EPC because this is more consistent with the intent of the RME paradigm as defined by EPA (1989a; 1991a).

The UCL generated by ProUCL or the MDC, whichever is smaller, is selected as the EPC and is understood to represent a conservative estimate of average for use in the risk assessment or in various transport models used to estimate EPCs. Unusually high detected values are included in the calculation of the UCL concentration. Inclusion of these high values increases the statistical variability and the overall conservativeness of the risk estimate.

ProUCL is a software tool that provides estimates of the UCL using a variety of mathematical approaches. As mentioned, its output includes one or more recommendations. Depending on the data set, some of the estimates generated by the various calculation methods included in ProUCL may vary by an order of magnitude. ProUCL and the decision tree on which its recommendations are based have been developed using multitudes of simulated data sets with a variety of distributions and other characteristics. There are uncertainties as to how well this decision tree derives a recommended UCL for a given data set. This uncertainty tends to increase with variability and skewness and where a large number of the samples are nondetects. For example, with respect to distribution testing, ProUCL bases the determination of distribution type only on the detected samples. The true concentrations of the nondetected values are unknown, and this lack of information can affect the distribution determination and consequently affect the ProUCL recommendation. The general uncertainties associated with the EPC values and the use of ProUCL are discussed in the uncertainty analysis (Chapter 6.0). Specific uncertainties associated with the EPC values of specific data sets may be discussed in the uncertainty analysis as appropriate.

3.2.2 Exposure-Point Concentrations in Air

The models shown in the following subsections for estimating COPC concentrations in air include only those relevant and used specifically for AP2 COPCs and media.

3.2.2.1 COPC Concentrations from Dust

Inhalation exposure to particulate (dust) emissions from soils for the groundskeeper and construction worker evaluations arises from activities that raise dust. Therefore, the most appropriate approach for estimating chemical concentrations in ambient air is the use an activity-based dust loading equation (U.S. Department of Energy [DOE], 1989):

$$C_a = (D)(C_s)(CF_1) \qquad \text{Eq. 3.1}$$

where:

C_a	=	contaminant concentration in air (milligrams per cubic meter [mg/m^3], calculated)
D	=	dust loading factor (grams [g] of soil/m^3 of air)
C_s	=	contaminant concentration in soil (mg/kg)
CF_1	=	conversion factor (1E-3 kg per g).

Plausible values for D include 2E-4 grams per cubic meter (g/m^3) for agricultural activity (DOE, 1989), 6E-4 g/m^3 for construction work (DOE, 1983), and 1E-4 g/m^3 for other activity (National Council on Radiation Protection and Measurements, 1984). The value for D of 1E-4 g/m^3 for other activity is used for the groundskeeper. It is assumed that construction activities requiring intensive contact with soil, for which D = 6E-4 g/m^3 is appropriate, may last for one-half of a construction period. The remaining one-half of the time is more realistically characterized by D = 1E-4 g/m^3. Therefore, a time-weighted average dust loading factor for construction work of 3.5E-4 g/m^3 is estimated for the construction worker.

Airborne concentrations of VOCs estimated by the dust loading model are assumed to sufficiently estimate levels of VOCs that may arise from volatilization, because the dust loading model treats the VOCs as if they were located at the ground surface.

The resident is more likely to be exposed to dust arising from wind erosion than from dust-raising activities on the site. EPA (1996) derived a model for estimating a dust particulate emission factor (PEF) based on an "unlimited reservoir" model and the assumption that the source area is square:

Eq. 3.2

$$PEF = Q/C \times \frac{3600}{0.036 \times (1 - V) \times (U_m / U_t)^3 \times F(x)}$$

where:

PEF	=	particulate emission factor (cubic meters per kilogram [m^3/kg], calculated)
Q/C	=	inverse of the mean concentration at center of square source (55.99 grams per square meter-second per kg/m^3, site-specific value from Table 3 in EPA [1996] [Zone 7, Cleveland, 5-acre site])
3600	=	seconds/hour
V	=	fraction of surface covered with vegetation (0.8, unitless, assumed)
U_m	=	mean annual wind speed (default, 4.60 meters per second [m/second] equals mean annual wind speed of 10.3 miles per hour [see Section 3.1.1])
U_t	=	equivalent threshold value of wind speed at 7 meters (default, 11.32 m/second)
F(x)	=	function dependent on U_m/U_t (default, 0.194).

The concentration of a COPC in air is calculated as follows:

Eq. 3.3

$$C_a = \frac{C_s}{PEF}$$

where:

C_a	=	contaminant concentration in air (mg/m^3, calculated)
C_s	=	contaminant concentration in soil (mg/kg)
PEF	=	particulate emission factor (m^3/kg).

Airborne concentrations of VOCs estimated by the wind erosion model are assumed to sufficiently estimate levels of VOCs that may arise from volatilization, because the wind erosion model treats the VOCs as if they were located at the ground surface.

3.2.2.2 Concentrations in Household Air from Groundwater Use

The inhalation of VOCs released from groundwater, which is assumed to be used as tap water, is evaluated for the on-site residential scenario. Chemicals that have a Henry's Law value exceeding 1E-5 atmospheres/m^3-mole and a molecular weight less than 200 g/mole are considered to be VOCs and are subject to evaluation via this pathway; Henry's Law values and molecular weights are presented in Table 3-3. Other groundwater contaminants are considered on a case-by-case basis for their potential contribution to risk via the inhalation pathway based on the degree of departure from the Henry's Law and molecular weight criteria, groundwater concentration, and toxicity.

The simple whole-house tap water-to-air model described in Part B of the Human Health Evaluation Manual (HHEM) (EPA, 1991b) was used to evaluate the tap water-to-air pathway. This model was selected based on correspondence between OEPA (2004) and USACE. Part B of the HHEM recommends a volatilization constant of 0.0005 for the total concentrations of all VOCs detected in groundwater; the conversion is characterized by the following equation:

Eq. 3.4

$$C_a = C_{gw} \times K_{wa} \times 1{,}000 \, L/m^3$$

where:

C_a = modeled concentration in air (mg/m^3)
C_{gw} = groundwater EPC (mg/L)
K_{wa} = tap water-to-air volatilization constant (0.0005 [unitless]: [EPA, 1991b])

Implicit in the HHEM Part B application of this model are the following assumptions: 1) A family of four uses the groundwater as the sole source of household tap water, 2) the volume of the house is 150 m^3, 3) the daily groundwater use is 720 L/day, 4) 50 percent of VOCs in tap water volatilize to household air, and 5) the air exchange rate of the house is 0.25 volumes per hour (EPA, 1991b).

3.2.2.3 Concentrations of VOCs in Groundwater: Resident Dermal Uptake

Volatilization of VOCs from household water reduces the remaining concentration available for dermal contact. As mentioned in Section 3.2.2.2, the HHEM Part B whole-house tap water-to-air model assumes that 50 percent of the VOC concentrations are released to household air. Thus, the concentrations of VOCs remaining in the water after volatilization occurs are calculated by difference as follows:

Eq. 3.5

$$C_d = C_{gw} \times (1 - F_v)$$

where:

C_d = concentration of VOC in household water available for dermal exposure (mg/L, calculated)
C_{gw} = concentration of VOC in groundwater (mg/L)
F_v = fraction of VOCs volatilized to air, (0.5 unitless).

Only the concentration remaining in tap water after volatilization (C_d), as applicable, is assumed to be available for contact with the skin during bathing/showering.

3.2.2.4 Exposure-Point Concentrations of COPCs in Venison

The hunter is assumed to harvest and consume game and share it with family members, including small children. The game is assumed to be venison, because the white-tailed deer is the species hunted most widely and most likely to provide a regular contribution to the diet. Data do not exist to reliably estimate contaminant concentrations in venison, but the following simplifying assumptions permit estimates sufficient for a BHHRA.

- Deer are small ruminants and, as such, are not unlike cattle; thus, it is reasonable to assume they may have similar physiological processes that could yield similar biotransfer factors. Unlike beef, however, deer meat does not undergo marbling with fat, and deer fat is quite unpalatable and is likely to be trimmed rather than consumed. Therefore, the biotransfer factors for edible venison are derived by adjusting biotransfer factors for beef to account for differences in the fat content of table-ready beef (cooked choice retail cuts trimmed to 0 inches of fat: average 14.4 percent fat) and venison (cooked boneless muscle meats: average 2.9 percent fat) (Nutrient Database, 1997).

- Deer are expected to browse a much larger area than that encompassed in AP2; therefore, the fraction of total browse consumed from AP2 is expected to be relatively small.

- Indirect food chain pathways may be significant for some metals and for those semivolatile organic compounds that persist in the environment and have the tendency to bioaccumulate. VOCs are generally mobile in the environment and labile in biological systems and do not tend to bioaccumulate.

To reflect the assumptions previously noted, venison biotransfer factors are estimated by multiplying beef biotransfer factors by 2.9/14.4 (or 0.20), and by a fraction, FI_r. This fraction reflects the areal portion of the site compared to a deer's home range area. These assumptions are captured in the following equation:

Eq. 3.6

$$B_v = 0.20(B_b)(FI_r)$$

where:

B_v	=	biotransfer factor for venison (unitless, calculated)
0.20	=	factor to reflect differences in fat content between beef and venison (0.20, unitless, see above)
FI_r	=	areal portion of site compared to a deer's home range (0.03, unitless, see below)
B_b	=	biotransfer factor for beef.

Values for B_b for metals are provided in the toxicity profiles (Appendix C). Toxicity profiles are prepared for each of the COPCs. The toxicity profiles briefly describe the uses of the chemical, its physical properties, behavior in environmental media, biotransfer capability, and toxicity values.

The AP2 area is relatively small in comparison to the home range of a white-tailed deer. The total acreage of the AP2 study area is approximately 5 acres. The home range of the white-tailed deer is between 150 and 1,280 acres (Sample and Suter, 1994). Even if the low end of this range (150 acres) is assumed for deer in northern Ohio, the area represented by AP2 is approximately 3 percent of this land area. Therefore, an FI_r value of 0.03 is used in the BHHRA.

Deer are assumed to be exposed to contaminants by ingesting browse growing on contaminated soil. It is estimated that deer consume approximately 1.74 kg of browse per day (Sample, et al., 1996), which is approximately 50 percent dry matter (DM), or 0.87 kg browse DM per day (Mautz, et al., 1976). The contaminant concentration in browse is estimated from the following equation, which was originally developed for estimating the contaminant concentration in forage to which cattle may be exposed (EPA, 1994):

Eq. 3.7

$$C_p = (CF)(C_s)(B_p)$$

where:

C_p	=	concentration of contaminant in (plant) forage DM (mg/kg, calculated)
CF	=	conversion factor to adjust for soil containing 20 percent moisture (1.25, unitless).
C_s	=	concentration of contaminant in soil (mg/kg)
B_p	=	soil-to-forage biotransfer factor (mg of chemical per kg of dry plant/mg of chemical per kg of dry soil).

Values for B_p are taken from the toxicity profiles in Appendix C. B_p values for the vegetative parts of plants, rather than the reproductive parts of plants, are selected, as possible, because deer browse year-round, and the vegetative parts are more available for the greater part of the year.

The concentration of a COPC in venison can be estimated from the following equation (adapted from EPA [1994]):

Eq. 3.8

$$C_v = (Q_p)(C_p)(B_v)$$

where:

C_v = contaminant concentration in venison (mg/kg, calculated)
Q_p = browse ingestion rate (0.87 kg DM/day)
C_p = contaminant concentration in browse DM (mg/kg)
B_v = biotransfer factor for venison (days/kg).

3.3 Quantification of Chemical Intake

This section describes the models used to quantify doses or intakes of the COPCs by the exposure pathways identified above. Models were taken or modified from EPA (1989a) unless otherwise indicated.

3.3.1 Inhalation of COPCs in Air

The inhaled dose of a COPC in air (for the groundskeeper, construction worker, on-site resident: inhalation of dust and VOCs in ambient air from surface or total soil; for the construction worker: inhalation of VOCs in ambient air from subsurface soil; for the indoor worker and on-site resident: inhalation of VOCs in indoor air from subsurface soil) is estimated as follows:

Eq. 3.9

$$I_a = \frac{(C_a)(FI_a)(IR_a)(EF)(ED)}{(BW)(AT)}$$

where:

I_a = inhaled dose of COPC (milligrams per kilograms per day [mg/kg-day], calculated)
C_a = concentration of COPC in air (mg/m^3)
FI_a = fraction of exposure attributed to site media (unitless)
IR_a = inhalation rate (m^3/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days).

3.3.2 Incidental Ingestion of COPCs in Soil or Sediment

The ingested dose of a COPC in soil (groundskeeper, construction worker, resident, indoor worker, hunter) or sediment (construction worker, resident) is estimated from the following equation:

Eq. 3.10

$$I = \frac{(C)(FI)(IR)(EF)(ED)(CF)}{(BW)(AT)}$$

where:

I	=	I_s for soil, I_{sd} for sediment, = ingested dose of COPC (mg/kg-day, calculated)
C	=	C_s for soil; C_{sd} for sediment; = concentration of COPC (mg/kg)
FI	=	FI_s for soil; FI_{sd} for sediment; = fraction of exposure attributed to site medium (unitless)
IR	=	IR_s for soil; IR_{sd} for sediment; = ingestion rate of medium (mg/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (1E-6 kg/mg)
BW	=	body weight (kg)
AT	=	averaging time (days).

3.3.3 Incidental Ingestion of COPCs in Water

The ingested dose of a COPC in groundwater (future groundskeeper, resident) is estimated from the following equation:

$$I_w = \frac{(C_w)(IR_w)(FI_w)(EF_w)(ED_w)}{(BW)(AT)} \qquad \text{Eq. 3.11}$$

where:

I_w	=	ingested dose of COPC in water (mg/kg-day, calculated)
C_w	=	concentration of COPC in water (mg/L)
IR_w	=	water ingestion rate (L/day)
FI_w	=	fraction of exposure attributed to site water (unitless)
EF_w	=	fraction of exposure attributed to site water exposure frequency (days/year)
ED_w	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days).

3.3.4 Dermal Contact with COPCs in Soil, Sediment, or Water

Unlike the methodologies for estimating inhaled or ingested doses of a COPC, which quantify the dose presented to the barrier membrane (the pulmonary or gastrointestinal mucosa, respectively), dermal dose is estimated as the dose that crosses the skin and is systemically absorbed. For this reason, dermal toxicity values are also based on absorbed dose. The absorbed dose of a COPC is estimated from the following equation (EPA, 2004a):

Eq. 3.12

$$DAD = \frac{(DA)(SA)(EF)(ED)}{(BW)(AT)}$$

where:

DAD = average dermally absorbed dose of COPC (mg/kg-day, calculated)
DA = dose absorbed per unit body surface area per day (milligrams per square centimeter per day [mg/cm^2-day])
SA = SA_s for soil, SA_{sd} for sediment, S_{gw} for groundwater, SA_{sw} for surface water, = surface area of the skin exposed (cm^2)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days).

Dose absorbed per unit body surface area per day (DA) is calculated differently for dermal uptake from soil/sediment and from water. Dermal uptake of constituents from soil (groundskeeper, construction worker, on-site resident, hunter) or sediment (construction worker, on-site resident) assumes that absorption is a function of the fraction of a dermally applied dose that is absorbed. DA is calculated from the following equation (EPA, 2004a):

Eq. 3.13

$$DA = (C_s)(FI_s)(CF)(AF)(ABS)$$

where:

DA = dose absorbed per unit body surface area per day (mg/cm^2-day, calculated)
C = C_s for soil, C_{sd} for sediment, = concentration of COPC in medium (mg/kg)
FI = FI_s for soil, FI_{sd} for sediment, = fraction of exposure attributed to site medium (unitless)
CF = conversion factor (1E-6 kg/mg)
AF = AF_s for soil, AF_{sd} for sediment, = soil- or sediment-to-skin adherence factor (mg/cm^2-day)
ABS = absorption fraction (unitless, chemical-specific).

ABS values are provided in the toxicity profiles for each COPC (Appendix C).

Quantification of dermal uptake of constituents from groundwater (future groundskeeper, resident) or surface water (construction worker, resident) depends on a K_p, which describes the rate of movement of a constituent from water across the dermal barrier to the systemic circulation (EPA, 2004a). The equation for dermal uptake of chemicals from water is the same as the equation for dermal uptake of chemicals from soil (Eq. 3.12). DA is calculated differently for inorganic and organic chemicals in water. For inorganic chemicals, DA is calculated from the following equation:

Eq. 3.14

$$DA = (C_w)(FI)(K_p)(ET_w)(CF)$$

where:

DA	=	dose absorbed per unit body surface area per day (mg/cm^2-day, calculated)
C_w	=	concentration of COPC in water (mg/L)
K_p	=	permeability coefficient (cm/hour)
ET_w	=	time of exposure (hours/day)
CF	=	conversion factor (1E-3 liters per cubic centimeter [L/cm^3]).

K_p for organic chemicals varies by several orders of magnitude and is highly dependent on lipophilicity, expressed as a function of the octanol/water partition coefficient (EPA, 2004a). Because the stratum corneum (the outer skin layer) is rich in lipid content, it may act as a sink, initially reducing the transport of chemical to the systemic circulation. With continued exposure and the attainment of steady-state conditions, the rate of dermal uptake increases. Therefore, different equations are used to estimate DA, depending on whether the exposure time is less than or greater than the estimated time to reach steady state. Non-steady-state exposures occur when either the exposure time is relatively brief (e.g., showering, for most chemicals) or when intermittent exposure occurs throughout the day (e.g., wading exposure to surface water or washing of hands). For exposure scenarios under which steady state is not reached for a given organic chemical (τ>exposure time [ET], see below), the following equation is used to calculate DA (EPA, 2004a):

Eq. 3.15

$$DA = 2(FA)(K_p)(C_w)(CF)\sqrt{\left(\frac{6\tau(ET_w)}{\pi}\right)}$$

where:

DA	=	dose absorbed per unit body surface area per day (mg/cm^2-day, calculated)
C_w	=	C_{sw} for surface water, C_{gw} for groundwater, = concentration of COPC in water (mg/L)
FA	=	fraction absorbed from the water (unitless)
K_p	=	permeability coefficient (cm/hour)
CF	=	conversion factor (1E-3 L/cm^3)
τ	=	time for concentration of contaminant in stratum corneum to reach steady state per event (hours)
ET_w	=	time of contact (hour(s)/day).

In cases where steady state is reached (τ<ET), such as where the duration of a bath exceeds the time to reach steady state for a given organic compound, the following equation is used to calculate DA (EPA, 2004a):

Eq. 3.16

$$DA = (FA)(K_p)(C_w)(CF)\left[\frac{ET_w}{1+B} + 2\tau\left(\frac{1+3B+3B^2}{(1+B)^2}\right)\right]$$

where:

DA	=	dose absorbed per unit body surface area per day (mg/cm^2-day, calculated)
C_w	=	concentration of COPC in water (mg/L)
FA	=	fraction absorbed from the water (unitless)
K_p	=	permeability coefficient (cm/hour)
CF	=	conversion factor (1E-3 L/cm^3)
τ	=	time for concentration of contaminant in stratum corneum to reach steady state per event (hours)
ET_w	=	time of contact (hour[s]/day)
B	=	Ratio of the permeability coefficient of a compound through the stratum corneum relative to its permeability coefficient across the viable epidermis (unitless).

Assuming one exposure event/day allows expressing ET as hour(s)/day, which preserves the dimensional integrity of the equation.

When available, values for K_p and τ are taken from EPA (2004a). For organics that have no K_p values listed, the values are calculated using the following equation (EPA, 2004a):

Eq. 3.17

$$\text{Log}(K_p) = -2.80 + 0.66(\log K_{ow}) - 0.0056(MW)$$

where:

K_p	=	permeability coefficient (cm/hour, calculated)
log K_{ow}	=	log of the octanol/water partition coefficient (unitless)
MW	=	molecular weight.

Where values for τ are not available, they were calculated using the following equation (EPA, 2004a).

Eq. 3.18

$$\tau = 0.105 \times 10^{(0.0056 \times MW)}$$

where:

τ = time for concentration of contaminant in stratum corneum to reach steady state (hours, calculated)
MW = molecular weight.

Values of K_p and τ used in the BHHRA are summarized in Table 3-3 and documented in Appendix C.

3.3.5 Consumption of Venison

Consumption of venison by the hunter or the hunter's child is evaluated by the following equation:

Eq. 3.19

$$I_v = \frac{(C_v)(IR_v)(EF)(ED)}{(BW)(AT)}$$

where:

I_v = ingested dose of COPC in venison (mg/kg-day, calculated)
Cv = concentration of COPC in venison (mg/kg)
IRv = venison ingestion rate (kg/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days).

4.0 Toxicity Evaluation

Toxicity is defined as the ability of a chemical to induce adverse effects in biological systems. The purpose of the toxicity assessment is two-fold:

- Identify the cancer and noncancer effects that may arise from exposure of humans to the COPC (hazard assessment).
- Provide an estimate of the quantitative relationship between the magnitude and duration of exposure and the probability or severity of adverse effects (dose-response assessment).

The latter is accomplished by the derivation of cancer and noncancer toxicity values, as described in the following sections.

4.1 Evaluation of Carcinogenicity

A few chemicals are known, and many more are suspected, to be human carcinogens. The evaluation of the potential carcinogenicity of a chemical includes both a qualitative and a quantitative aspect (EPA, 2005). The qualitative aspect is a weight-of-evidence evaluation of the likelihood that a chemical might induce cancer in humans. EPA (2005) recognizes five weight-of-evidence group classifications for carcinogenicity. Formerly, EPA (1986) used a letter-based system to describe the weight of evidence for carcinogenicity. Reference to this former system is included because many of the carcinogenicity assessments listed on the Integrated Risk Information System (IRIS) use the former letter-based system (EPA, 2010). The five EPA weight-of-evidence classifications are as follows:

- ***Carcinogenic to Humans*** (corresponds to the former Group A - Human Carcinogen).
- ***Likely to be Carcinogenic to Humans*** (includes both the former Group B1 and Group B2-Probable Human Carcinogens)
- ***Suggestive Evidence of Carcinogenic Potential*** (corresponds to the former Group C - Possible Human Carcinogen)
- ***Inadequate Information to Assess Carcinogenic Potential*** (corresponds to the former Group D - Not Classifiable as to Human Carcinogenicity)
- ***Not Likely to be Carcinogenic to Humans*** (corresponds to the former Group E - Evidence of Noncarcinogenicity to Humans).

The toxicity value for carcinogenicity, called a cancer slope factor (SF), is an estimate of potency. SFs are developed only for chemicals in the first three groups and only if the data are sufficient. The SFs are statistically derived from the dose-response curve from the best human or animal study or studies of the chemical. Although human data are often considered to be more reliable than animal data because there is no need to extrapolate the results obtained in one species to another, most human studies have one or more of the following limitations:

- The duration of exposure is usually considerably less than lifetime.
- The concentration or dose of chemical to which the humans were exposed can be approximated only crudely, usually from historical data.
- Concurrent exposure to other chemicals frequently confounds interpretation.
- Data regarding other factors (tobacco, alcohol, illicit or medicinal drug use, nutritional factors and dietary habits, heredity) are usually insufficient to eliminate or quantify confounding effects on the results.
- Most epidemiologic studies are occupational investigations of workers, which may not accurately reflect the range of sensitivities of the general population.
- Most epidemiologic studies lack the statistical power (i.e., sample size) to detect a low, but chemical-related increased incidence of tumors.

Most potency estimates are derived from animal data, which present different limitations:

- It is necessary to extrapolate from results in animals to predict results in humans, usually by estimating an equivalent human dose from the animal dose.
- The range of sensitivities arising from genotypic and phenotypic diversity in the human population is not reflected in the animal models ordinarily used in cancer studies.
- Usually, very high doses of chemical are used, which may alter normal biology, creating a physiologically artificial state and introducing substantial uncertainty regarding the extrapolation to the low-dose range expected with environmental exposure.
- Individual studies vary in quality (e.g., duration of exposure, group size, scope of evaluation, adequacy of control groups, appropriateness of dose range, absence of concurrent disease, sufficient long-term survival to detect tumors with long induction or latency periods).

The SF is usually expressed as "extra risk" per unit dose, that is, the additional risk above background in a population corrected for background incidence. It is calculated using the following equation:

Eq. 4.1

$$SF = (p_{(d)} - p_{(0)})/(1 - p_{(0)})$$

where:

SF	=	chemical-specific slope factor per unit dose $(mg/kg\text{-}day)^{-1}$
$p_{(d)}$	=	the probability of developing cancer at a dose of 1 mg/kg-day of the chemical of interest
$p_{(0)}$	=	the background probability of developing cancer at a dose of 0 mg/kg-day of the chemical of interest.

The SF is expressed as risk per mg/kg-day, shown mathematically as $(mg/kg\text{-}day)^{-1}$. To be appropriately conservative, the SF is usually the 95 percent upper bound on the slope of the dose-response curve extrapolated from high (experimental) doses to the low-dose range expected in environmental exposure scenarios. EPA (2005) assumes that there are no thresholds for carcinogenic expression; therefore, any exposure represents some quantifiable risk, however miniscule it may be.

The oral SF is usually derived directly from the experimental dose data, because oral dose is usually expressed as mg/kg-day. When the test chemical was administered in the diet or drinking water, oral dose first must be estimated from data for the concentration of the test chemical in the food or water, food or water intake data, and body weight data.

IRIS (EPA, 2010) expresses inhalation cancer potency as a unit risk based on concentration, or risk per microgram of chemical per m^3 of ambient air, shown mathematically as (micrograms per cubic meter $[\mu g/m^3])^{-1}$. Because cancer risk characterization requires an SF expressed as risk per mg/kg-day, the unit risk must be converted to the mathematical equivalent of an inhalation cancer SF, or risk per unit dose as $(mg/kg\text{-}day)^{-1}$. Because the inhalation unit risk is based on continuous lifetime exposure of an adult human (assumed to inhale 20 m^3 of air per day and to weigh 70 kg), the mathematical conversion consists of multiplying the unit risk (per $\mu g/m^3$) by 70 kg and by 1,000 µg/mg, and dividing the result by 20 m^3 per day.

4.2 Evaluation of Noncarcinogenic Effects

Many chemicals, whether or not associated with carcinogenicity, are associated with adverse noncarcinogenic effects. The evaluation of noncancer effects (EPA, 1989b) involves the following:

- Qualitative identification of the adverse effect(s) associated with the chemical; these may differ depending on the duration (acute or chronic) or route (oral or inhalation) of exposure.
- Identification of the critical effect for each duration of exposure (i.e., the first adverse effect that occurs as dose is increased).
- Estimation of the threshold dose for the critical effect for each duration of exposure.
- Development of an uncertainty factor (UF); i.e., quantification of the uncertainty associated with interspecies extrapolation, intraspecies variation in sensitivity, severity of the critical effect, slope of the dose-response curve, and deficiencies in the database, in regard to developing a reference dose (RfD) for human exposure.
- Identification of the target organ(s) for the critical effect for each route of exposure.

These information points are used to derive an exposure route- and duration-specific toxicity value called an RfD, expressed as mg/kg-day, which is considered to be the dose for humans, with uncertainty of an order of magnitude or greater, at which adverse effects are not expected to occur. Mathematically, it is estimated as the ratio of the threshold dose to the UF. For purposes of risk assessment, chronic exposure is typically defined as equal to or greater than 7 years, i.e., at least 10 percent of expected life span; subchronic exposure is typically defined as 2 weeks to 7 years. However, professional judgment may be used where exposure durations approach 10 percent of the expected life span. Also, exposure during a critical stage of development, such as a portion of early childhood, may be treated as chronic even if the anticipated exposure duration is considerably less than 10 percent of the expected life span.

IRIS (EPA, 2010) expresses the inhalation noncancer reference value as a reference concentration (RfC) in units of mg/m^3. Because noncancer hazard characterization requires a reference value expressed as mg/kg-day, the RfC must be converted to an inhalation RfD. Because the inhalation RfC is based on continuous exposure of an adult human (assumed to inhale 20 m^3 of air per day and to weigh 70 kg), the mathematical conversion consists of multiplying the RfC (mg/m^3) by 20 m^3/day and dividing the result by 70 kg.

RfD and RfC values are derived for both chronic and subchronic exposure. Under the assumption of monotonicity (incidence, intensity, or severity of effects can increase, but cannot decrease, with increasing magnitude or duration of exposure), a chronic RfD may be considered sufficiently protective for subchronic exposure, but a subchronic RfD may not be protective for chronic exposure. Currently, subchronic RfD values exist for few chemicals. Subchronic RfD values can be derived from chronic RfD values as follows:

- If the UF applied in the derivation of the chronic RfD (or RfC) does not provide for expansion from subchronic to chronic exposure (e.g., if the chronic RfD was derived from a chronic study), the chronic RfD is adopted as being sufficiently protective for subchronic exposure.

- If the UF applied in the derivation of the chronic RfD (or RfC) contains a component to expand from subchronic to chronic exposure, the subchronic RfD is derived by multiplying the chronic RfD by the factor used to expand from subchronic to chronic exposure (e.g., if a factor of 10 was used to expand from subchronic to chronic exposure, the subchronic RfD would be 10 times larger than the chronic RfD).

Only chronic RfDs and RfCs are used in the risk characterization of this BHHRA.

4.3 Dermal Toxicity Values

Dermal RfDs and SFs are derived from the corresponding oral values, provided there is no evidence to suggest that dermal exposure induces exposure route-specific effects that are not appropriately modeled by oral exposure data. In the derivation of a dermal RfD, the oral RfD is multiplied by the gastrointestinal absorption factor (GAF), expressed as a decimal fraction. The resulting dermal RfD, therefore, is based on absorbed dose. The RfD based on absorbed dose is the appropriate value with which to compare a dermal dose, because dermal doses are expressed as absorbed doses rather than exposure doses. The dermal SF is derived by dividing the oral SF by the GAF. The oral SF is divided, rather than multiplied, by the GAF because the SF is expressed as a reciprocal dose.

4.4 Target Organ Toxicity

As a matter of science policy, EPA assumes dose and effect to be additive for noncarcinogenic effects (EPA, 1989a). This assumption provides the justification for adding the HQ or hazard index (HI) values in the risk characterization for noncancer effects (Section 5.2) resulting from exposure to multiple chemicals, pathways, or media. However, EPA (1989a) acknowledges that adding all HQ or HI values may overestimate hazard, because the assumption of additivity is probably appropriate only for those chemicals that exert their toxicity by the same mechanism.

Mechanisms of toxicity data sufficient for predicting additivity with a high level of confidence are available for very few chemicals. In the absence of such data, EPA (1989a) assumes that chemicals that act on the same target organ may do so by the same mechanism of toxicity; that is, the target organ serves as a surrogate for mechanism of toxicity. When total HI for all media for a receptor exceeds 1 due to the contributions of several chemicals, it is appropriate to segregate the chemicals by route of exposure and mechanism of toxicity (i.e., target organ) and estimate separate HI values for each target organ.

As a practical matter, because human environmental exposures are likely to involve near- or sub-threshold doses, the target organ chosen for a given chemical is the one associated with the critical effect. If more than one organ is affected by a given chemical at the threshold, then all affected target organs are selected for this chemical. The target organ is also selected on the basis of duration of exposure (i.e., the target organ for chronic or subchronic exposure to low or moderate doses is selected rather than the target organ for acute exposure to high doses) and route of exposure. Because dermal RfD values are derived from oral RfD values, the oral target organ is adopted as the dermal target organ. For some chemicals, no target organ is identified. This occurs when no adverse effects are observed or when adverse effects such as reduced longevity or growth rate are not accompanied by recognized organ- or system-specific functional or morphologic alteration.

4.5 Sources of Toxicity Information Used in the Risk Assessment

Toxicity values were selected for use in the BHHRA based on EPA Office of Solid Waste and Emergency Response Directive 9285.7-53 (EPA, 2003), which prescribes the following hierarchy:

- **Tier 1** values: IRIS (EPA, 2010) database.

- **Tier 2** values: These are EPA's provisional peer-reviewed toxicity values. The provisional peer-reviewed toxicity values are developed by the Office of Research and Development, the National Center for Environmental Assessment, and the Superfund Health Risk Technical Support Center on a chemical-specific basis when requested by the Superfund program.

- **Tier 3** values: These are other toxicity values from additional EPA and non-EPA sources of toxicity information. As stated in the EPA Office of Solid Waste and Emergency Response directive, "priority should be given to those sources of information that are the most current, the basis for which is transparent and publicly available, and which have been peer reviewed." Two common examples of Tier 3 values are the EPA's Health Effects Assessment Summary Tables (EPA, 1997b) and

the California Environmental Protection Agency (2005) Office of Environmental Health Hazard Assessment Toxicity Criteria Database.

The Environmental Council of States-U.S. Department of Defense (2007) has issued a toxicity value hierarchy that basically supports the EPA (2003) hierarchy presented previously but places higher emphasis on the necessity for external peer review.

GAFs used to derive dermal RfD values and SFs from the corresponding oral toxicity values are obtained from the following sources:

- Oral absorption efficiency data compiled by the National Center for Environmental Assessment for the Superfund Health Risk Technical Support Center of EPA
- Federal agency reviews of the empirical data, such as Agency for Toxic Substances and Disease Registry toxicological profiles and various EPA criteria documents
- Other published reviews of the empirical data
- The primary literature.

GAFs obtained from reviews are compared to empirical (especially more recent) data, when possible, and are evaluated for suitability for use in deriving dermal toxicity values from oral toxicity values. The suitability of the GAF increases when the following similarities are present in the oral pharmacokinetic study from which the GAF is derived and in the key toxicity study from which the oral toxicity value is derived:

- The same strain, sex, age, and species of test animal were used.
- The same chemical form (e.g., the same salt or complex of an inorganic element or organic compound) was used.
- The same mode of administration (e.g., diet, drinking water, or gavage vehicle) was used.
- Similar dose rates were used.

Individual toxicity profiles are included in Appendix C for all of the COPCs evaluated in the BHHRA. Summary toxicity information sufficient to support the risk calculations, including toxicity values, GAFs, target organs, and sources, are provided in Table 4-1.

5.0 Risk Characterization

Risk characterization is the process of applying numerical methods and professional judgment to determine the potential for adverse human health effects to result from the presence of site-specific contaminants. This is done by combining the intake rates estimated during the exposure assessment with the appropriate toxicity information identified during the toxicity assessment. Noncancer hazards and cancer risks are characterized separately, including COPCs that induce both types of effects.

Quantitative expressions are calculated during risk characterization that describe the probability of developing cancer (i.e., ILCRs), or the nonprobabilistic comparison of estimated dose with an RfD for noncancer effects (i.e., HQs and HIs). Quantitative estimates are developed for individual chemicals, exposure pathways, and exposure media for each receptor. These quantitative risk characterization expressions, in combination with qualitative information, are used to guide risk management decisions. Risk characterization, as described in this section, is applied only to COPCs.

Generally, the risk characterization follows the methodology prescribed by EPA (1989a), as modified by more recent information and guidance. EPA methods are appropriately designed to be health protective and tend to overestimate rather than underestimate risk. The risk results, however, may be overly conservative, because risk characterization involves multiplication of the conservative assumptions built into the estimation of the EPCs, exposure (intake) estimates, and toxicity dose-response assessments.

5.1 Cancer Risk

The risk from exposure to potential chemical carcinogens is estimated as the probability of an individual developing cancer over a lifetime and is called the ILCR. In the low-dose range, which would be expected for most environmental exposures, cancer risk is estimated from the following linear equation (EPA, 1989a):

Eq. 5.1

$$ILCR = (CDI)(SF)$$

where:

ILCR = incremental lifetime cancer risk, a unitless expression of the probability of developing cancer, adjusted for background incidence, calculated

CDI = chronic daily intake, averaged over 70 years (mg/kg-day)
SF = cancer slope factor (risk per mg/kg-day).

The chronic daily intake (CDI) term in Equation 5.1 is equivalent to the "I" or "DAD" terms (intake or dose) in Equations 3.9 through 3.12 and 3.19 when these equations are evaluated for cancer intakes.

The use of Equation 5.1 assumes that chemical carcinogenesis does not exhibit a threshold and that the dose-response relationship is linear in the low-dose range. Because this equation could generate theoretical cancer risks greater than 1 for high-dose levels, it is considered to be inaccurate at cancer risks greater than 1E-2. In these cases, cancer risk is estimated by the following one-hit model (EPA, 1989a):

Eq. 5.2

$$ILCR = 1 - e^{[-(CDI)(SF)]}$$

where:

ILCR = incremental lifetime cancer risk, a unitless expression of the probability of developing cancer, adjusted for background incidence, calculated
$-e^{(CDI)(SF)}$ = the exponential of the negative of the risk calculated using Equation 5.1.

As a matter of policy, EPA (1986) considers the carcinogenic potency of simultaneous exposure to low doses of carcinogenic chemicals to be additive, regardless of the chemicals' mechanisms of toxicity or sites of action (organs of the body). Cancer risk arising from exposure to multiple chemicals in a given exposure medium and pathway is estimated from the following equation (EPA, 1989a):

Eq. 5.3

$$ILCR_p = ILCR_{(chem\,1)} + ILCR_{(chem\,2)} + \cdots ILCR_{(chem\,i)}$$

where:

$ILCR_p$ = total pathway risk of cancer incidence, calculated
$ILCR_{(chem\ i)}$ = individual chemical cancer risk for the pathway.

The sum of the ILCRs summed across pathways is the total ILCR as shown in the equation below:

Eq. 5.4

$$Total\ ILCR = ILCR_{(p\,1)} + ILCR_{(p\,2)} + \cdots ILCR_{(p\,i)}$$

where:

Total ILCR = total incremental lifetime cancer risk across all pathways
$ILCR_{pi}$ = incremental lifetime cancer risks associate with pathway "*i*."

The total ILCR represents all additional cancer risks posed to a given receptor by contact with contaminants in site environmental media.

Total ILCRs in the range of 1E-6 to 1E-4 are regarded as acceptable (EPA, 1990); as mentioned in Section 2.4.1, this range is referred to as the "NCP risk management range." Risks less than this range are regarded as negligible. A target cancer risk criterion of 1E-5 is used by OEPA and was selected by the PBOW Project Delivery Team as a basis to consider remedial action. Use of this 1E-5 criterion represents a departure from the Army's practice of generally using a cancer risk exceeding a value of 1E-4 (the upper end of the NCP risk management range) to trigger remedial action considerations.

5.2 Noncancer Effects of Chemicals

The hazards associated with noncancer effects of chemicals are evaluated by comparing an exposure level or intake with an RfD. The HQ, defined as the ratio of intake to RfD, is estimated as follows (EPA, 1989a):

Eq. 5.5

$$HQ = I \ / \ RfD$$

where:

HQ = hazard quotient (unitless, calculated)
I = intake of chemical averaged over subchronic or chronic exposure period (mg/kg-day)
RfD = reference dose (mg/kg-day).

The I term in Equation 5.4 is equivalent to the "I" or "DAD" terms (intake or dose) in Equations 3.9 through 3.12 and 3.19 when these equations are evaluated for noncancer intakes.

Chemical noncancer hazards are evaluated using chronic RfD values. This approach is different from the probabilistic approach used to evaluate cancer risks. An HQ of 0.01 does not imply a 1-in-100 chance of an adverse effect, but indicates only that the estimated intake is 100 times lower than the RfD. An HQ of unity indicates that the estimated intake equals the RfD. If the HQ is greater than unity, there may be concern for potential adverse health effects.

In the case of simultaneous exposure of a receptor to multiple chemicals, or to a given chemical by multiple pathways, an HI is calculated as the sum of the HQs by the following equation:

Eq. 5.6

$$HI = HQ_1 + HQ_2 + ... HQ_i$$

where:

HI = hazard index (unitless, calculated)
HQ_i = hazard quotient for the ith chemical, or for the ith pathway.

A total HI is calculated as the sum of all HI values, including all media and all COPCs, for a given receptor. Calculating a total HI as the sum of HQ values is based on the assumption that the potential for noncancer effects is additive. EPA (1989a), however, acknowledges that the assumption of additivity is probably appropriate only for chemicals that induce adverse effects by the same mechanism (please see Section 4.4). Therefore, if the total HI for a receptor exceeds 1, individual HI values may be calculated for each target organ.

A total target organ HI is calculated by summing the HI values (associated by target organ[s]), across exposure pathways as follows:

Eq. 5.7

$$Total\ Target\ Organ\ HI_a = HI_{p1-a} + HI_{p2-a} + ... HI_{pi-a}$$

where:

Total target organ HI_a = total hazard index for target organ "*a*" (unitless, calculated)
HI_{pi-a} = hazard index for target organ "*a*" via pathway "*i*."

HI values of 1 or less indicate that adverse noncancer health effects associated with that target organ of any individual under the exposure assumptions for that receptor are unlikely. If the total target organ HI exceeds a value of 1, then adverse noncancer health effects concerning that target organ and receptor cannot be regarded as unlikely.

5.3 Risk Characterization Results

Cancer and noncancer risk characterization results were evaluated for each receptor and each environmental medium, using the methods described in Sections 5.1 and 5.2. The following subsections describe the risk characterization results for each receptor. Risk summary tables are shown for each receptor as referenced. The detailed quantitative evaluation tables that include the exposure equations presented in Chapter 3.0 and the risk characterization equations presented in Chapter 5.0 are provided in Appendix D.

5.3.1 Current Groundkeeper

The ILCR for the current groundskeeper (Table 5-1) exposed to surface soil both through direct contact and via inhalation of suspended particulates is estimated as 2E-6. This is within the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5.

The total HI for the current groundskeeper (Table 5-1) is 0.09. This is far less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

5.3.2 Future Groundskeeper

The exposure assumptions for the future groundskeeper differ from those of the current groundskeeper (Section 5.3.1) in that the future groundskeeper is assumed to be exposed to total soil rather than surface soil, and the future groundkeeper is also assumed to be exposed to groundwater via direct contact (ingestion and dermal exposure). However, as described in Section 2.5, the surface soil data set for AP2 is also conservatively used as the total soil data set. Therefore, the only difference between the AP2 future groundkeeper and the AP2 current groundskeeper is groundwater exposure assumed for the former. The risk characterization was run separately for bedrock groundwater exposure and overburden (well) groundwater exposure.

Bedrock Groundwater Use. The total ILCR for the future groundskeeper, assuming bedrock groundwater exposure, is 7E-5 (Table 5-1). This value is within the NCP risk management range but exceeds the PBOW target cancer risk criterion of 1E-5. Over 99 percent of this ILCR is associated with benzene in groundwater. Benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and petroleum was reported seeping at depth (47 feet and 69.5 feet) in AP2 bedrock well AP2-BEDGW-003 (Shaw, 2010b).

If the contribution of benzene in groundwater were excluded, the ILCR associated with the remaining COPCs in all media would be 2E-6 (Table 5-2).

The total HI for the future groundskeeper (Table 5-1) is 1. This equals and, thus, meets the target HI value of 1, indicating that noncancer health effects are unlikely to occur. Note that more than 80 percent of this HI value is associated with benzene in groundwater. As stated above, this benzene is not associated with former PBOW operations but is associated with naturally occurring conditions. The HI for this receptor without the contributions from benzene would be 0.2.

Overburden Groundwater Use. No carcinogenic COPCs were identified for overburden groundwater. Therefore, the only cancer risks to the future groundskeeper are those associated with surface soil. The ILCR for the future groundskeeper is thus the same as for the current groundskeeper, 2E-6 (Table 5-1). This value is within the NCP cancer risk management range and meets the 1E-5 PBOW cancer risk criterion.

The total HI for the future groundskeeper is 0.1 (Table 5-1). This is less than the target HI criterion of 1, indicating that noncancer health effects are unlikely to occur.

5.3.3 Indoor Worker

The exposure assumptions for the future indoor worker include direct contact with surface soil, inhalation of volatiles from subsurface soil, and direct exposure with groundwater via direct contact (ingestion and dermal exposure). The risk characterization was run separately for bedrock groundwater exposure and overburden (well) groundwater exposure.

Bedrock Groundwater Use. The total ILCR for the future indoor worker, assuming bedrock groundwater exposure, is 7E-5 (Table 5-1). This value is within the NCP risk management range but exceeds the PBOW target cancer risk criterion of 1E-5. Over 99 percent of this ILCR is associated with benzene in groundwater. As described in Section 5.3.2, benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and petroleum was reported seeping at depth (47 feet and 69.5 feet) in AP2 bedrock well AP2-BEDGW-003 (Shaw, 2010b) (Table 5-2).

If the contributions from benzene in groundwater were excluded, the ILCR associated with the remaining COPCs in all media would be 8E-7 (Table 5-2).

The total HI for the indoor worker (Table 5-1) is 1. This equals the target HI value of 1, indicating that noncancer health effects are unlikely to occur. Note that nearly 90 percent of this HI value is associated with benzene in groundwater. As stated in Section 5.3.2, this benzene is not associated with former PBOW operations but is associated with naturally occurring conditions. The HI for this receptor without the contributions from benzene would be 0.1.

Overburden Groundwater Use. The ILCR for the future indoor worker is 2E-7 (Table 5-1). This value is less than the NCP cancer risk management range and meets the 1E-5 PBOW cancer risk criterion.

The total HI for the indoor worker is 0.06 (Table 5-1). This is far less than the target HI criterion of 1, indicating that noncancer health effects are unlikely to occur.

5.3.4 Construction Worker

The ILCR for the construction worker (Table 5-1) exposed to total soil both through direct contact and via inhalation of suspended particulates is estimated as 4E-7. This is less than the 1E-6 to 1E-4 NCP risk management range and the PBOW cancer risk criterion of 1E-5.
The total HI for the construction worker (Table 5-1) is 0.4. This is less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

5.3.5 Resident

The exposure assumptions for the resident include direct contact with total soil, inhalation of particulates from total soil, inhalation of volatiles from subsurface soil, direct exposure to sediment and surface water (ingestion and dermal exposure), and direct exposure with groundwater via direct contact (ingestion and dermal exposure). The risk characterization was run separately for bedrock groundwater exposure and overburden (well) groundwater exposure. The ILCR was calculated assuming exposure during a combined 30-year child/adult exposure duration, whereas separate noncancer HI values were calculated for the young child and adult life stages (refer to Section 3.1.3.5). The results of each are described below.

Bedrock Groundwater Use. The total ILCR for the resident, assuming bedrock groundwater exposure, is 3E-4 (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk criterion. Approximately 97 percent of this ILCR is associated with benzene in groundwater. As described in Section 5.3.2, benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and during well installation, petroleum was observed seeping at depth from bedrock well AP2-BEGW-002 (Shaw, 2010b). If this naturally occurring benzene were not present, the resulting ILCR of 1E-5 would meet the PBOW cancer risk criterion (Table 5-2).

The total HI for the child resident, assuming bedrock groundwater exposure, is 7 (Table 5-1). This exceeds the target HI value of 1, indicating that noncancer health effects cannot be regarded as unlikely to occur. Note that 86 percent of this HI value is associated with benzene (82 percent) and xylenes (4 percent) in groundwater. These two chemicals are related to naturally occurring petroleum, which has been encountered throughout PBOW, including AP2 (Section 5.3.2). Note that if the contributions of benzene are excluded, the resulting HI is 1, which equals and thus meets the noncancer target HI criteria (Table 5-2).

The total HI for the adult resident assuming bedrock groundwater exposure is 3 (Table 5-1). This exceeds the target HI value of 1, indicating that noncancer health effects cannot be regarded as unlikely to occur. Note that 92 percent of this HI value is associated with benzene (88 percent) and xylenes (4 percent) in groundwater. These two chemicals are related to naturally occurring petroleum, which has been encountered throughout PBOW, including AP2 (Section 5.3.2). Note that if the contributions of benzene are excluded, the resulting HI is 0.3, which meets the noncancer target HI criteria.

Overburden Groundwater Use. The total ILCR for the future resident, assuming overburden groundwater exposure, is 5E-6 (Table 5-1). This value is within the NCP risk management range and meets the PBOW target cancer risk criterion.

The total HI for the child resident, assuming overburden groundwater exposure, is 0.9 (Table 5-1). This meets the target HI value of 1, indicating that noncancer health effects are regarded as unlikely to occur.

The total HI for the adult resident, assuming overburden groundwater exposure, is 0.2 (Table 5-1). This meets the target HI value of 1, indicating that noncancer health effects are regarded as unlikely to occur.

5.3.6 Adult Hunter

The ILCR for the current adult hunter (Table 5-1) exposed to surface soil both through direct contact and via the ingestion of venison is estimated as 6E-8. This is less than the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5.

The total HI for the adult hunter is (Table 5-1) is 0.003. This is far less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

5.3.7 Hunter's Child

The ILCR for the current hunter's child (Table 5-1) exposed to surface soil via the ingestion of venison from deer which grazed on site is estimated as 4E-13. This is less than the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5.

The total HI for the current hunter's child is (Table 5-1) is 3E-6. This is far less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

6.0 Uncertainty Analysis

The primary objective of the BHHRA is to characterize and quantify potential human health risks. However, these risks are estimated using incomplete and imperfect information that introduces uncertainties at various stages of the risk assessment process. Uncertainties associated with earlier stages of the risk assessment become magnified when they are linked with other uncertainties in the latter stages. Reliance on a simplified numerical presentation of dose rate and risk without consideration of uncertainties, limitations, and assumptions inherent in their derivation can be misleading. For example, the calculated ILCR for a given scenario "A" may be 1E-5 (meets the OEPA risk criterion) and that of scenario "B" may be 5E-5 (exceeds the OEPA risk criterion). However, if the uncertainties associated with scenario "B," for instance, span orders of magnitude and the ILCR is regarded as biased high, it is not unlikely that scenario "A" actually presents a higher risk of developing cancer.

The chief goal of this analysis is to evaluate uncertainties and present them in context of their potential impact on the interpretation of the risk assessment results and the types of environmental management decisions that may be based on these results. The uncertainty analysis does not exhaustively describe all potential uncertainties but presents those that have the largest implications for the interpretation of the risk assessment results. This analysis also summarizes the types and, as applicable, the magnitude of the uncertainties at each stage of the risk assessment. Although the discussion in the following sections includes generic uncertainties that are common to the state of human health risk assessment practice overall (e.g., additivity of health effects in the risk characterization), the uncertainty analysis focuses on the set of uncertainties that is peculiar to AP2.

6.1 Types of Uncertainty

Uncertainties in risk assessment are categorized into two general types: 1) variability inherent in the (true) heterogeneity of the data set, measurement precision, and measurement accuracy; and 2) uncertainty that arises from data gaps. Estimates of the degree of variability tend to decrease as the sample size increases. This is because larger data sets are less impacted by individual samples/measurements and typically allow for greater accuracy. Uncertainty that arises from data gaps is addressed by applying models and assumptions. Models are applied because they represent a level of understanding to address certain exposure parameters that are impractical or impossible to measure (e.g., COPC concentrations in air that would result from groundwater use that has not yet occurred—or may never occur—at the site). Assumptions represent an educated estimate to address information that is not available (e.g., additivity of carcinogens).

6.2 Sources of Uncertainty

The discussion in the following subsections provides an overview of uncertainty, with a focus on those sources that are most likely to affect interpretation of the risk assessment results.

6.2.1 Sample Selection

Soil samples were collected from within the former AP2 area where ash waste was deposited. The well locations were selected from within and adjacent to AP2, including downgradient areas. Surface water and sediment samples were collected along areas of Pipe Creek that had potentially been impacted by AP2 materials. The sample locations appear to be representative of the site, and no information suggests that they have introduced an identifiable bias.

6.2.2 Laboratory Analysis

State-of-the-practice laboratory methods were used for analysis of the RI samples (Shaw, 2010b). Site investigation samples, also used in this AP2 BHHRA, were collected in 1996 (IT, 1997a). The ICP method used for certain metals in the 1996 data resulted in relatively high reporting limits and tended to result in false positives when compared with the ICP/MS method used in the 2009 RI samples. Among these metals is thallium, which was a COPC in AP2 soil. The RBSC for thallium in soil is 0.51 mg/kg and the BSC is 1.3 mg/kg. The MDC among the 2009 soil samples is only 0.939 mg/kg, which is less than the BSC. This means that based on the 2009 data, thallium would not be identified as a COPC. The seven reported detections of thallium among the 1996 surface soil samples ranged from 1.9 to 8.5 mg/kg. Therefore, use of the 1996 analytical data appears to have introduced a high bias to the resulting risk calculations.

6.2.3 Exposure-Point Concentration Estimates

Uncertainty is introduced in the statistical approach used to calculate the EPCs. As stated in the *Human Health Evaluation Manual (Part A)* (EPA, 1989a), the average concentration of the site should be used as the concentration term. Generally, a UCL is used to account for the uncertainty of using a sample data set to estimate the true population mean concentration for the site. ProUCL Version 4.0 software (EPA, 2009a) was used to calculate the EPCs for those media with five or more samples. However, as is readily observed by reviewing the ProUCL output (Appendix B), the calculation of the UCL can vary with methodology. It is unclear as to whether the UCL value of a specific EPC would result in an underestimate or overestimate of the true population mean. However, the general use of a UCL on all the data sets, even given the uncertainty as to whether a given method provides full coverage at 95 percent confidence, would result in general overestimation of the population mean and associated risks. Therefore, as intended by the guidance (EPA, 1989a), this practice of using the UCL as the EPC (note that the

MDC is used as the EPC if the UCL exceeds the MDC) introduces bias that tends to overestimate the population mean and the resultant risk values.

Both overburden groundwater and bedrock groundwater data sets have fewer than five samples. Thus, the MDC was used as the EPC for these data sets. Use of the MDC likely introduces as high bias to the risk values.

As discussed in Section 2.5, the surface soil data set was used to represent total soil. Thallium was the only COPC identified for subsurface soil, and it was reported at a higher concentration in surface soil than in subsurface soil (note: also see Section 6.2.2 with respect to false positives in thallium). Because the subsurface soil appears to be virtually unimpacted from site activities with respect to risk, a conservative determination was made to use the surface soil data set to represent total soil. This assumption introduces a high bias to the resulting risk values of affected receptors.

6.2.4 Land-Use Assumptions/Receptor Selection

The current groundskeeper is intended to represent an on-site worker under the current land use as NASA-controlled property. Because there is currently no identified NASA activity at AP2, the use of this receptor likely overestimates risks and hazards to a current site worker. The use of the hunter and the hunter's child are reasonable, as this area may be legally hunted by permit under current land use.

Unrestricted land use, including residential use, is a reasonable assumption for the future, given the rural residential use of property adjacent to the PBOW facility. However, the assumption that either the overburden or bedrock groundwater will be used as a potable water source in the future is unlikely for reasons described in the following paragraphs.

The use of bedrock groundwater is regarded as unlikely because it contains natural petroleum and hydrogen sulfide, which can be commonly observed in area quarries (Shaw, 2005). A strong hydrogen odor was observed in the boreholes of each of the three AP2 bedrock monitoring wells during well installation. Petroleum hydrocarbon seepage was noted on the rock cores at depth in the boring log of bedrock well AP2-BEDGW-003. The presence of the petroleum hydrocarbon at depth, but not at shallower intervals or in overburden groundwater, provides evidence that this seepage is resultant from naturally occurring petroleum that is observed elsewhere in the bedrock regionally. Additionally, bedrock well AP2-BEDGW-001 was dry during both sampling events and could not be sampled. Although the remaining two bedrock wells, AP2-BEDGW-002 and AP2-BEDGW-003, could be sampled in May 2009 using low-flow technique, samples from

these two wells had to be collected via bailer in November 2009 because of inadequate water yield for low-flow sampling.

The overburden groundwater has been found to be of low yield, as well as regionally and seasonally discontinuous throughout much of PBOW (Shaw, 2006). This also appears to be true of the AP2. Although all three of the overburden wells could be sampled using low-flow technique in May 2009, no water for sampling could be collected from two of the three wells, and the third overburden well, AP2-MW01, produced enough volume only for collection of nitroaromatics analysis, which had to be collected via bailer.

In summary, use of either the bedrock groundwater or overburden groundwater as a potable source at AP2 is regarded as implausible for the following reasons: 1) Both bedrock and overburden groundwater yield water volumes appear to be inadequate, 2) bedrock groundwater has naturally occurring petroleum and hydrodgen sulfide constituents that make the water unsuitable regardless of yield, 3) none of the private wells in the region of PBOW are used as a potable source, and 4) municipal water is available in the area.

6.2.5 Exposure Assumption Values

The exposure assumption values used in the exposure assessment (Table 3-1) are selected to represent either an upper bound (e.g., 95th percentile) or mid-range value, depending on the particular parameter. Mathematically combining these terms in exposure equations is generally thought to result in decidedly conservative exposure estimations (Cogliano, 1997; Burmaster and Harris, 1993). However, this conservativeness is consistent with the state of risk assessment practice, which attempts to focus on the upper end of exposure possibilities rather than more realistic levels of exposure, and is not resultant from assumptions made specifically for AP2.

6.2.6 Groundwater-to-Air Household Model

The groundwater-to-air household model (EPA, 1991b) has considerable uncertainties. Some parameters that can change the model include the air exchange rate of the house which can vary with the season, level of groundwater usage, and size of the house. EPA (1991b) states that the model assumes a four-member household that uses 720 liters of water per day, a home with a volume of 150 m^3, and 0.25 household air exchanges per hour. The *Exposure Factors Handbook* (EPA, 1997a) lists the following information for the average four-person home: household use of 892 L/day (4 × 223 L/day), volume of 431 m^3, and an air exchange rate of 0.63 volumes per hour. If the values from the *Exposure Factors Handbook* were used instead of the default values from EPA (1991b), the resultant estimated household air concentrations would be less than those estimated in the BHHRA by a factor of nearly 5. Therefore, the selection of the groundwater-to-

air parameter values used by EPA (1991b) may contribute a conservative bias to the resultant risk estimates.

6.2.7 Toxicity Assessment

Uncertainties associated with the toxicity assessment include those regarding development of the health effects criteria values, the classification of potential carcinogenicity, the extrapolation of exposure route-specific toxicity values to other routes of exposure, and the extrapolation of toxic effects observed in animal studies to potential adverse effects in humans. A general summary of these uncertainties is provided in the following paragraphs.

The development of health effects criteria for noncancer effects involves considerable professional judgment. An uncertainty factor of up to 10 may be applied to a toxicologically identified benchmark dose or concentration to address the unknown regarding each of the following (EPA, 1989b): lowest-observed-adverse-effects level to no-observed-adverse-effects level, subchronic-to-chronic extrapolation, route-to-route extrapolation, and species-to-species extrapolation. A "modifying factor" of 10 or less is likewise applied in the development of RfDs and RfCs, using professional judgment. This modifying factor is intended to address gaps in the database and steepness of the dose-response curve. In practice, the overall UF, derived by multiplying the individual uncertainty factors by the modifying factor, associated with RfD and RfC values may span up to four orders of magnitude.

The EPA weight-of-evidence classification system for carcinogens is used to examine and classify chemical agents with respect to their carcinogenic potential. Most EPA potential carcinogens are classified based on animal data, without sufficient human data to support a causal association (i.e., Group B2; refer to Section 4.1). Also, the linearized multistage (LMS) mathematical model was used to extrapolate values from relatively high-dose rodent studies to relatively low-dose human exposures in the development of SFs for these compounds. This application of the LMS model is the subject of much controversy. Thus, the LMS approach used to develop SFs, combined with other assumptions, tends to overestimate potential risks.

Overall, the toxicity values, assuming similar effects between humans and test species, tend to result in overestimates of noncancer hazards or cancer risks. However, it is possible that a given chemical can elicit a toxic response in humans that is not observed in the laboratory species studied or that humans may be more sensitive to a given chemical. In this instance, it is possible for the use of the toxicity values to result in underestimates of risks/hazards.

6.2.8 Risk Characterization

It is assumed that the effects of simultaneous exposures to multiple carcinogens at a site are additive. Likewise, it is assumed that noncancer effects of contaminants are additive if they have a similar mechanism of toxicity. In risk assessment practice, it is assumed that the effects of chemicals that affect the same target organ are additive unless chemical-specific information would dictate otherwise. However, chemicals in combination may act additively, synergistically, or antagonistically or may not influence one another at all. Therefore, depending on the interactive effects (if any), the risk characterization approach to multiple contaminants may lead to either underestimates or overestimates of potential risk/hazard.

7.0 Summary and Conclusions

7.1 Summary

Cancer risks and noncancer hazards were evaluated for soil, sediment, surface water, overburden groundwater, and bedrock groundwater at AP2. A risk-based screening approach was used to identify COPCs and focus the risk assessment on those chemicals detected at AP2.

The BHHRA was conducted to evaluate cancer risk and noncancer hazards associated with AP2 surface soil, subsurface soil, groundwater, surface water, and sediment. These media were screened against the respective RBSCs, and inorganics in bedrock groundwater and soil were also screened against BSCs. COPCs for the various media were identified based on this screening. Exposure and risk/hazard associated with the COPCs were evaluated using the following receptors (media evaluated in parentheses):

- Current groundskeeper (surface soil)
- Future groundskeeper (total soil, groundwater)
- Indoor worker (surface soil, subsurface soil [air pathway only], groundwater)
- Construction worker (total soil, sediment)
- Resident (total soil, groundwater, surface water, sediment)
- Adult hunter (surface soil, venison)
- Hunter's child (venison).

Each receptor scenario that includes groundwater was run separately for overburden groundwater and bedrock groundwater. The resident was evaluated for noncancer hazards separately for the young child (ages 1 through 6 years) and adult life stages.

The overall HI and ILCR values are summarized in the following bullets; exceedances of the PBOW cancer risk criteria (ILCR>1E-5) are shown as bold and exceedances of the noncancer hazard criterion (HI>1) or the NCP risk management range (1E-6 to 1E-4) are shown as bold italics:

- Current groundskeeper: ILCR = 2E-6; HI = 0.09
- Future groundskeeper – overburden groundwater use: ILCR = 2E-6; HI = 0.1
- Future groundskeeper – bedrock groundwater use: **ILCR = 7E-5**; HI = 1

- Future indoor worker – overburden groundwater use: ILCR = 2E-7; HI = 0.06
- Future indoor worker – bedrock groundwater use: **ILCR = 7E-5**; HI = 1
- Current/future construction worker: ILCR = 4E-7; HI = 0.4
- Future resident – overburden groundwater use: ILCR = 5E-6; adult HI = 0.2; child HI = 0.9
- Future resident – bedrock groundwater use: ***ILCR = 3E-4; adult HI = 3; child HI = 7***
- Current/future hunter: ILCR = 6E-8; HI = 0.003.
- Current/future hunter's child: ILCR = 4E-13; HI = 0.000003.

7.2 Conclusions

The ILCR of the future resident that is hypothetically assumed to use bedrock groundwater as household tap water exceeds the NCP risk management range as well as the PBOW target cancer risk criterion. The HI values of the child and adult resident also exceed the target criterion of 1, indicating that adverse human health risks under this scenario cannot be regarded as unlikely. The ILCR for each of the other future scenarios assumed to use bedrock groundwater as a potable source had an ILCR that exceeded the PBOW target cancer risk criterion, but were within the NCP risk management range.

Cancer risks and noncancer hazards to all potential receptors evaluated under current land use (i.e., current groundskeeper, construction worker, hunter, and hunter's child) are within or less than the NCP risk management range, are less than the target HI criterion, and are less than the PBOW cancer risk criterion, indicating that cancer risks are at acceptable levels and adverse noncancer effects are unlikely to occur under potential current scenarios. Similarly, all future scenarios which do not assume bedrock groundwater use meet the NCP risk management range, target HI criterion, and the PBOW risk criterion.

Risks associated with the use of bedrock groundwater as tap water easily account for the majority of the ILCR and HI for the future groundskeeper, indoor worker, and future resident. Specifically, benzene in bedrock groundwater is by far responsible for the largest portion of cancer risk and noncancer hazards for these receptors. It is responsible for 97 to 99 percent of the total ILCR and 82 to 89 percent of the total HI for these three receptors. Benzene was identified as the only contaminant with an HI exceeding a value of 1 and an ILCR exceeding a value of 1E-5 for any receptor. Benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW, and petroleum was reported seeping at depth (47 feet and 69.5 feet) in AP2 bedrock well AP2-BEDGW-003. Without the contribution of benzene, the residual noncancer HI value for each receptor does not exceed a value of 1 and the ILCR does not exceed a value of 1E-5.

It is highly doubtful that bedrock groundwater would ever be used as potable tap water because of naturally poor water quality in the area and low, undependable groundwater yield. This poor quality is associated with the presence of hydrogen sulfide off-gassing in the area and the associated health and aesthetic issues with high-sulfate water, and the presence of petroleum product as encountered in AP2 well AP2-BEDGW-003. Thus, if a more likely and serviceable source of tap water were to be used by a future groundskeeper or residential receptor (e.g., municipal water or perhaps even overburden groundwater), then the ILCR and HI values would meet the NCP risk management range (1E-6 to 1E-4), PBOW cancer risk criterion of 1E-5, and the target HI criterion of 1.

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TABLES

Table 2-1

Summary of Samples Evaluated in the Baseline Human Health Risk Assessment
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample Number	Sample Purpose	Sample Date	Depth (ft)	Analyses
Surface Soil					
ASH PIT 2-SB01	AP0100	REG	15-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB02	AP0103	REG	16-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB03	AP0108	REG	18-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB04	AP0111	REG	18-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB05	AP0114	REG	16-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB06	AP0117	REG	15-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB07	AP0120	REG	16-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB07	AP0121	FD	16-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 2-SB08	AP0125	REG	16-Jan-09	0 - 1	Exp, Metals, PCB, SVOC
PH2SO01	4010	REG	28-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO02	4030	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO03	4050	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO04	4070	REG	28-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO05	4090	REG	28-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO06	4110	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO07	4130	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO08	4150	REG	28-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO09	4170	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO10	4190	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO11	4210	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO12	4230	REG	29-Sep-96	0 - 0.5	Cyanide, Metals, Pest, PCB, SVOC, VOC
Subsurface Soil					
ASH PIT 2-SB01	AP0101	REG	15-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB01	AP0102	REG	15-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB02	AP0104	REG	16-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB02	AP0105	FD	16-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB02	AP0107	REG	16-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB03	AP0109	REG	18-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB03	AP0110	REG	18-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB04	AP0112	REG	18-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB04	AP0113	REG	19-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB05	AP0115	REG	16-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB05	AP0116	REG	16-Jan-09	8 - 10	Exp, Metals, SVOC

Table 2-1

Summary of Samples Evaluated in the Baseline Human Health Risk Assessment
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample Number	Sample Purpose	Sample Date	Depth (ft)	Analyses
ASH PIT 2-SB06	AP0118	REG	15-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB06	AP0119	REG	15-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB07	AP0123	REG	16-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB07	AP0124	REG	16-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB08	AP0126	REG	16-Jan-09	3 - 5	Exp, Metals, PCB, SVOC
ASH PIT 2-SB08	AP0127	REG	16-Jan-09	8 - 10	Exp, Metals, SVOC
ASH PIT 2-SB08	AP0128	FD	16-Jan-09	8 - 10	Exp, Metals, SVOC
PH2SO01	4020	REG	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO02	4040	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO03	4060	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO04	4080	REG	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO04	4081	FD	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO05	4100	REG	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO06	4120	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO07	4140	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO08	4160	REG	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO08	4161	FD	28-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO09	4180	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO10	4200	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO11	4220	REG	29-Sep-96	2 - 3	Cyanide, Metals, Pest, PCB, SVOC, VOC
PH2SO12	4240	REG	29-Sep-96	3 - 4	Cyanide, Metals, Pest, PCB, SVOC, VOC
Groundwater Samples					
Overburden Well Samples					
AP2-MW01	AP3057	REG	23-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f& uf), PCB, SVOC, VOC
AP2-MW01	AP3075	REG	13-Nov-09	NA	Exp, Field Tests
AP2-MW02	AP3058	REG	25-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f& uf), PCB, SVOC, VOC
AP2-MW02	AP3059	FD	25-May-09	NA	Exp, Gen Chem, Metals (f & uf), PCB, SVOC, VOC
AP2-MW02	AP3076	REG	13-Nov-09	NA	Exp
AP2-MW03	AP3061	REG	24-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f& uf), PCB, SVOC, VOC

Table 2-1

Summary of Samples Evaluated in the Baseline Human Health Risk Assessment
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample Number	Sample Purpose	Sample Date	Depth (ft)	Analyses
Bedrock Well Samples					
AP2-BEDGW-002	AP3063	REG	24-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), PCB, SVOC, VOC
AP2-BEDGW-002	AP3079	REG	17-Nov-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), SVOC, VOC
AP2-BEDGW-002	AP3081	FD	17-Nov-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), SVOC, VOC
AP2-BEDGW-003	AP3064	REG	23-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), PCB, SVOC, VOC
AP2-BEDGW-003	AP3065	FD	23-May-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), PCB, SVOC, VOC
AP2-BEDGW-003	AP3080	REG	16-Nov-09	NA	Exp, Field Tests, Gen Chem, Metals (f & uf), SVOC, VOC
Sediment Samples					
ASH PIT 2-SD01	AP1000	REG	24-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
ASH PIT 2-SD02	AP1001	REG	24-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
ASH PIT 2-SD03	AP1002	REG	24-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
ASH PIT 2-SD04	AP1003	REG	25-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
ASH PIT 2-SD04	AP1004	FD	25-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
ASH PIT 2-SD05	AP1006	REG	25-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
Surface Water Samples					
AP2-SW01	AP2000	REG	24-May-09	NA	Exp, Metals, PCB, SVOC
AP2-SW02	AP2001	REG	24-May-09	NA	Exp, Metals, PCB, SVOC
AP2-SW03	AP2002	REG	24-May-09	NA	Exp, Metals, PCB, SVOC
AP2-SW03	AP2003	FD	24-May-09	NA	Exp, Metals, PCB, SVOC
AP2-SW04	AP2005	REG	25-May-09	NA	Exp, Metals, PCB, SVOC
AP2-SW05	AP2006	REG	25-May-09	NA	Exp, Metals, PCB, SVOC

FD - Field duplicate.
Exp - Explosives.
Pest - Organochlorine pesticides.
PCB - Polychlorinated biphenyls.
SVOC - Semivolatile organic compounds.
VOC - Volatile organic compounds.

Table 2-2

Background Screening Concentrations of Inorganics in Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical Name (mg/kg)	Frequency of Detection	Range of Detected Concentrations	Range of Reporting Limits	Statistical Distribution	Arithmetic Mean	95% UTL [a]	Background Screening Criterion [b]
Aluminum	12 / 12	3520 - 15500	NA	L	8.43E+03	2.69E+04	15500
Antimony	9 / 25	5.9 - 9.3	5.4 - 74	NP	4.68E+00	NA	9.30
Arsenic	23 / 26	2.1 - 36.5	1.2 - 3.7	L	1.08E+01	7.10E+01	36.5
Barium	9 / 12	35.6 - 826	23.2 - 24.7	L	1.16E+02	1.30E+03	826
Beryllium	6 / 25	0.57 - 1	0.57 - 1.2	L	5.65E-01	1.17E+00	1.00
Cadmium	0 / 25	NA	0.57 1.2	L	4.49E-01	NA	NA
Calcium	12 / 12	735 - 52300	NA	L	1.13E+04	2.18E+05	52300
Chromium	25 / 26	4.4 - 29	12.3 - 12.3	NP	1.34E+01	NA	29.0
Cobalt	9 / 12	9.6 - 116	5.8 - 6.2	L	2.26E+01	2.48E+02	116
Copper	23 / 26	2.3 - 56.2	2.2 - 2.9	L	1.70E+01	1.47E+02	56.2
Iron	12 / 12	5880 - 234000	NA	L	4.01E+04	3.58E+05	234000
Lead	26 / 26	1.9 - 48.6	NA	L	1.28E+01	5.13E+01	48.6
Magnesium	12 / 12	629 - 10400	NA	L	3.26E+03	3.08E+04	10400
Manganese	26 / 26	21 - 13300	NA	L	7.29E+02	3.51E+03	3506
Mercury	2 / 26	0.085 - 0.085	0.037 - 0.3	L	9.06E-02	5.60E-01	0.085
Nickel	26 / 26	5.4 - 55.1	NA	L	2.28E+01	7.79E+01	55.1
Potassium	11 / 12	579 - 3390	617 - 617	L	1.24E+03	6.08E+03	3390
Selenium	5 / 25	0.61 - 2	0.57 - 4.9	NP	1.55E+00	NA	2.00
Silver	2 / 26	1.1 - 11.1	1.1 - 1.3	NP	1.00E+00	NA	11.1
Sodium	0 / 12	NA	566 - 663	L	3.03E+02	NA	NA
Thallium	2 / 25	1.2 - 1.3	1.1 - 6.1	NP	1.91E+00	NA	1.30
Vanadium	11 / 12	9 - 40.9	61.7 - 61.7	L	2.48E+01	8.31E+01	40.9
Zinc	26 / 26	6.6 - 655	NA	L	7.30E+01	3.22E+02	322

mg/kg - Milligrams per kilogram.
NA - Not applicable; not available.

[a] 95% UTL - 95% upper tolerance limit calculated as described in Shaw (2005).

[b] The maximum detected concentration is used as the background screening criterion for nonparametric data sets; for normal or lognormal data sets, the 95% UTL or the maximum detected concentration, whichever is less, is used.

Note: Detection limits from sample 6990 were deleted when calculating results for antimony, beryllium, cadmium, selenium, and thallium. The detection limits were elevated by dilution factors which greatly exceed any detected concentration and would bias results unrealistically high.

Source: Shaw Environmental, Inc. (shaw), 2005, 2004 Data Summary and Evaluation Report, Final, Plum Brook Ordnance Works, Sandusky, Ohio, April.

Table 2-3

Background Screening Concentrations for Inorganics and BTEX Compounds in Groundwater Powerhouse 2 Ash Pits Former Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of Values, µg/L: Detected Concentrations Minimum	Detected Concentrations Maximum	Reporting Limits Minimum	Reporting Limits Maximum	Mean µg/L	Standard Deviation	UTL [a] µg/L	BSC [b] µg/L
Metals - Unfiltered										
Aluminum	11 / 13	85	3.15E+01	3.09E+02	2.00E+02	2.00E+02	1.05E+02	6.98E+01	4.17E+02	309
Arsenic	4 / 26	15	3.30E+00	7.40E+00	1.00E+01	1.00E+01	4.99E+00	6.56E-01	7.92E+00	7.4
Barium	28 / 28	100	2.58E+01	1.18E+04	2.00E+02	2.00E+03	1.73E+03	3.77E+03	1.86E+04	11800
Calcium	28 / 28	100	1.74E+04	3.16E+05	5.00E+03	5.00E+03	1.38E+05	8.31E+04	5.09E+05	316000
Cobalt	6 / 27	22	1.00E+00	1.21E+01	5.00E+01	5.00E+01	2.05E+01	8.75E+00	5.96E+01	12.1
Copper	2 / 28	7	3.30E+00	1.98E+01	2.50E+01	2.50E+01	1.24E+01	2.26E+00	2.25E+01	19.8
Iron	24 / 27	89	3.82E+01	1.55E+03	1.00E+02	1.00E+02	4.15E+02	4.87E+02	2.59E+03	1550
Magnesium	28 / 28	100	7.28E+03	2.17E+05	5.00E+03	5.00E+03	7.17E+04	5.85E+04	3.33E+05	217000
Manganese	28 / 28	100	3.60E+00	6.88E+02	1.50E+01	1.50E+01	8.12E+01	1.24E+02	6.36E+02	636
Nickel	4 / 27	15	4.80E+00	8.60E+00	4.00E+01	4.00E+01	1.81E+01	4.67E+00	3.90E+01	8.6
Potassium	28 / 28	100	2.53E+03	1.16E+05	5.00E+03	5.00E+04	2.70E+04	3.06E+04	1.64E+05	116000
Sodium	28 / 28	100	1.33E+04	1.39E+06	5.00E+03	5.00E+04	3.55E+05	4.36E+05	2.30E+06	1390000
Zinc	14 / 19	74	8.30E-01	5.07E+02	2.00E+01	2.00E+01	5.55E+01	1.23E+02	6.06E+02	507
Voltile Organic Compounds										
Benzene	9 / 28	32	1.45E-01	2.40E+00	1.00E+00	5.00E+00	6.73E-01	5.43E-01	3.10E+00	2.4
Ethybenzene	6 / 28	21	1.30E-01	8.70E-01	1.00E+00	5.00E+00	5.82E-01	4.00E-01	2.37E+00	0.87
Toluene	8 / 28	29	1.20E-01	1.70E+00	1.00E+00	5.00E+00	4.99E-01	2.83E-01	1.76E+00	1.7
Xylenes, total	8 / 28	29	3.60E-01	5.50E+00	1.00E+00	5.00E+00	1.22E+00	1.53E+00	8.07E+00	5.5

[a] The UTL (upper tolerance limit) is calculated as described in Shaw (2005).

[b] The BSC (background screening criterion) is the calculated UTL or the maximum detected concentration, whichever is less.

µg/L - Micrograms per liter.

BTEX - Benzene, toluene, ethylbenzene, and xylenes.

Source: Shaw Environmental, Inc. (Shaw), 2005*2004 Data Summary and Evaluation Report*, Final, Plum Brook Ordnance Works, Sandusky, Ohio, April.

Table 2-4

Statistical Summary and COPC Selection of Chemicals Detected in Surface Soil
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]	95% UCL [e] mg/kg	EPC [i] mg/kg
Inorganics														
Aluminum	20 / 20	100	4.07E+03	J	1.39E+04		1.82E+00	3.57E+01	9.46E+03	1.55E+04	7.70E+03	N (b)		---
Antimony	7 / 20	35	3.14E-01	JJ	9.58E-01		4.54E-01	1.07E+01	3.02E+00	9.30E+00	3.10E+00	N (b)		---
Arsenic	20 / 20	100	6.07E+00		2.68E+01	J	9.08E-01	1.80E+00	1.46E+01	3.65E+01	3.90E-01	N (b)		---
Barium	20 / 20	100	5.06E+01		1.41E+02		1.82E-01	3.57E+01	9.88E+01	8.26E+02	1.50E+03	N (b)		---
Beryllium	20 / 20	100	7.30E-01		1.69E+01	J	1.82E-01	8.90E-01	5.36E+00	1.00E+00	1.60E+01	Y	1.14E+01	1.14E+01
Cadmium	7 / 20	35	3.81E-01	J	1.40E+00		4.54E-01	8.90E-01	4.79E-01		7.00E+00	N (a)		---
Calcium	20 / 20	100	1.65E+03		4.72E+04	J	4.54E+00	8.91E+02	7.86E+03	5.23E+04	Nutrient	N (c)		---
Chromium	20 / 20	100	1.90E+00		1.77E+01		4.54E-01	1.80E+00	1.15E+01	2.90E+01	2.90E-01	N (b)		---
Cobalt	20 / 20	100	3.90E+00	J	1.90E+01		3.63E-01	8.90E+00	1.19E+01	1.16E+02	2.30E+00	N (b)		---
Copper	20 / 20	100	1.68E+01		3.86E+01		4.54E-01	4.50E+00	2.58E+01	5.62E+01	3.10E+02	N (b)		---
Iron	20 / 20	100	1.43E+04	J	1.09E+05		3.67E+00	3.79E+01	4.78E+04	2.34E+05	5.50E+03	N (b)		---
Lead	20 / 20	100	4.64E+00		5.07E+01		3.80E-01	5.99E-01	2.04E+01	4.86E+01	4.00E+02	N (a)		---
Magnesium	20 / 20	100	3.12E+02	J	8.96E+03	J	1.82E+00	8.91E+02	2.07E+03	1.04E+04	Nutrient	N (c)		---
Manganese	20 / 20	100	9.82E+01		1.02E+03		1.82E-01	2.70E+00	3.59E+02	3.51E+03	1.80E+02	N (b)		---
Mercury	17 / 20	85	1.98E-02	JJ	1.40E-01		2.22E-02	5.90E-02	6.51E-02	8.50E-02	2.30E+00	N (a)		---
Nickel	20 / 20	100	1.05E+01		4.19E+01		2.72E-01	7.10E+00	2.47E+01	5.51E+01	1.50E+02	N (b)		---
Potassium	20 / 20	100	4.60E+02		2.26E+03		2.27E+01	8.91E+02	1.16E+03	3.39E+03	Nutrient	N (c)		---
Selenium	18 / 20	90	6.50E-01		2.60E+00		6.30E-01	1.20E+00	1.40E+00	2.00E+00	3.90E+01	N (a)		---
Silver	3 / 20	15	1.60E+00		2.00E+00		4.54E-01	1.80E+00	7.22E-01	1.11E+01	3.90E+01	N (b)		---
Sodium	8 / 20	40	2.82E+01		7.92E+01		9.08E+00	8.91E+02	2.52E+02		Nutrient	N (c)		---
Thallium	14 / 20	70	3.08E-01	J	8.50E+00		4.54E-01	1.80E+00	1.79E+00	1.30E+00	5.10E-01 f	Y	2.95E+00	2.95E+00
Vanadium	19 / 20	95	8.80E+00		2.69E+01		1.82E-01	8.90E+00	1.82E+01	4.09E+01	3.90E+01	N (b)		---
Zinc	20 / 20	100	4.14E+01		1.27E+02		2.27E+00	3.60E+00	7.40E+01	3.22E+02	2.30E+03	N (b)		---
Cyanide														
Cyanide, total	3 / 12	25	7.10E-01		9.30E-01		6.30E-01	8.90E-01	4.93E-01		1.60E+02	N (a)		---
Polychlorinated biphenyls (PCB)														
Aroclor 1016	1 / 20	5	5.25E-01	J	5.25E-01	J	4.05E-02	5.90E-02	4.91E-02		3.90E-01	Y	1.58E-01	1.58E-01
Aroclor 1260	1 / 20	5	1.62E-02	J	1.62E-02	J	4.05E-02	5.90E-02	2.38E-02		2.20E-01	N (a)		---
Organochlorine Pesticides														
DDE, 4,4'-	7 / 12	58	2.40E-03		7.40E-03		2.20E-03	3.00E-03	3.30E-03		1.40E+00	N (a)		---
DDT, 4,4'-	8 / 12	67	2.60E-03		7.10E-03		2.20E-03	3.00E-03	3.45E-03		1.70E+00	N (a)		---
Methoxychlor	2 / 12	17	6.10E-03		6.50E-03		4.20E-03	5.90E-03	3.14E-03		3.10E+01	N (a)		---
Semivolatile Organic Compounds														
Acenaphthylene	1 / 20	5	1.10E-01	J	1.10E-01	J	4.05E-01	5.90E-01	2.33E-01		1.70E+03 g	N (a)		---
Anthracene	1 / 20	5	5.80E-02	J	5.80E-02	J	4.05E-01	5.90E-01	2.31E-01		1.70E+03	N (a)		---
Benzo(a)anthracene	5 / 20	25	3.07E-02		1.30E-01	J	4.05E-01	5.90E-01	1.98E-01		1.50E-01	N (a)		---
Benzo(a)pyrene	3 / 20	15	8.44E-02	J	1.70E-01	J	4.05E-01	5.90E-01	2.21E-01		1.50E-02	Y	4.84E-01	1.70E-01
Benzo(b)fluoranthene	2 / 20	10	1.30E-01	J	1.60E-01	J	4.05E-01	5.90E-01	2.32E-01		1.50E-01	Y	4.95E-01	1.60E-01
Benzo(ghi)perylene	1 / 20	5	1.30E-01	J	1.30E-01	J	4.05E-01	5.90E-01	2.34E-01		1.70E+02 h	N (a)		---
Benzo(k)fluoranthene	1 / 20	5	2.30E-01	J	2.30E-01	J	4.05E-01	5.90E-01	2.39E-01		1.50E+00	N (a)		---
Benzoic acid	2 / 8	25	6.22E-01		1.43E+00		4.05E-01	4.99E-01	4.22E-01		2.40E+04	N (a)		---
Bis(2-ethylhexyl)phthalate	1 / 20	5	9.50E-02	J	9.50E-02	J	4.05E-01	5.90E-01	2.31E-01		3.50E+01	N (a)		---
Chrysene	4 / 20	20	6.39E-02	J	1.60E-01	J	4.05E-01	5.90E-01	2.12E-01		1.50E+01	N (a)		---

Table 2-4

Statistical Summary and COPC Selection of Chemicals Detected in Surface Soil
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]	95% UCL [e] mg/kg	EPC [f] mg/kg
Di-n-butyl phthalate	1 / 20	5	1.70E-01	J	1.70E-01	J	4.05E-01	5.90E-01	2.36E-01		6.10E+02	N (a)		---
Fluoranthene	8 / 20	40	5.99E-02	J	3.00E-01	J	4.05E-01	5.90E-01	1.93E-01		2.30E+02	N (a)		---
Indeno(1,2,3-cd)pyrene	1 / 20	5	1.20E-01	J	1.20E-01	J	4.05E-01	5.90E-01	2.34E-01		1.50E-01	N (a)		---
Naphthalene	3 / 20	15	4.44E-02	J	5.59E-02	J	4.05E-01	5.90E-01	2.14E-01		3.60E+00	N (a)		---
Phenanthrene	4 / 20	20	6.30E-02	J	1.00E-01	J	4.05E-01	5.90E-01	2.08E-01		1.70E+02 [h]	N (a)		---
Pyrene	8 / 20	40	4.40E-02	J	2.50E-01	J	4.05E-01	5.90E-01	1.82E-01		1.70E+02	N (a)		---
Volatile Organic Compounds														
Acetone	2 / 12	17	4.20E-02	J	1.20E-01	J	2.50E-02	1.80E-01	6.46E-02		6.10E+03	N (a)		---
Methylene chloride	1 / 12	8	5.20E-02	J	5.20E-02	J	6.30E-03	4.50E-02	1.85E-02		1.10E+01	N (a)		---
Toluene	1 / 12	8	1.80E-02	J	1.80E-02	J	6.30E-03	4.50E-02	1.43E-02		5.00E+02	N (a)		---

BSC - Background screening criterion.
COPC - Chemical of potential concern.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed
mg/kg - Milligrams per kilogram.
RBSC - Risk-based screening concentration.
VQ - Validation qualifier.

[a] IT Corporation (IT), 1998, *Site Investigation of Acid Areas*, *Plum Brook Ordnance Works, Sandusky, Ohio*, August.
[b] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Table (December 2009) residential s
and are based on a risk level of 1.0E-06 and a hazard index of 0.1.
[c] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = maximum detected concentration is less than the BSC.
(c) = essential nutrient.
[d] Y = Chemical is chosen as COPC.
[e] 95% UCL determined using ProUCL Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development,
Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC
[f] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower. The EPC used to evaluate lead using the Lead Model
(see text for details) is equal to the arithmetic mean.
[g] RBSC based on thallium from IRIS, 2009.
[h] RBSC based on anthracene.
[i] RBSC based on pyrene.

Table 2-5

Statistical Summary and COPC Selection of Chemicals Detected in Subsurface Soil
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC mg/kg	RBSC mg/kg	COPC? [b,c]	95% UCL [d] mg/kg	EPC [e] mg/kg
Inorganics														
Aluminum	28 / 28	100	3.52E+03		1.50E+04	JJ	1.71E+00	2.55E+01	8.33E+03	1.55E+04	7.70E+03	N (b)		---
Antimony	16 / 28	57	2.27E-01	J	6.26E-01		4.13E-01	7.60E+00	1.70E+00	9.30E+00	3.10E+00	N (b)		---
Arsenic	28 / 28	100	3.55E+00		1.26E+01		8.27E-01	1.30E+00	7.11E+00	3.65E+01	3.90E-01	N (b)		---
Barium	28 / 28	100	1.86E+01	J	9.89E+01	J	1.65E-01	2.55E+01	5.52E+01	8.26E+02	1.50E+03	N (b)		---
Beryllium	21 / 28	75	5.90E-01		8.48E+00	J	1.65E-01	6.40E-01	4.25E+00	1.00E+00	1.60E+01	N (a)		---
Cadmium	2 / 28	7	2.76E-01	J	2.93E-01	J	4.13E-01	6.40E-01	2.62E-01		7.00E+00	N (a)		---
Calcium	28 / 28	100	3.51E+02		1.53E+05		4.39E+00	6.37E+02	2.09E+04	5.23E+04	Nutrient	N (c)		---
Chromium	28 / 28	100	6.10E+00		2.22E+01		4.13E-01	1.30E+00	1.24E+01	2.90E+01	2.90E-01	N (b)		---
Cobalt	28 / 28	100	5.60E+00		2.47E+01		3.31E-01	6.40E+00	1.15E+01	1.16E+02	2.30E+00	N (b)		---
Copper	28 / 28	100	8.30E+00		3.84E+01		4.13E-01	3.20E+00	2.08E+01	5.62E+01	3.10E+02	N (b)		---
Iron	28 / 28	100	1.05E+04		5.51E+04	J	1.90E+00	1.27E+01	2.18E+04	2.34E+05	5.50E+03	N (b)		---
Lead	28 / 28	100	6.10E+00		1.89E+01	J	3.30E-01	5.41E-01	1.20E+01	4.86E+01	4.00E+02	N (b)		---
Magnesium	28 / 28	100	1.19E+03		1.20E+04	J	1.65E+00	6.37E+02	5.05E+03	1.04E+04	Nutrient	N (c)		---
Manganese	28 / 28	100	8.21E+01	J	1.56E+03	J	1.65E-01	1.90E+00	3.69E+02	3.51E+03	1.80E+02	N (b)		---
Mercury	13 / 28	46	1.12E-02	J	3.90E-02		1.94E-02	4.20E-02	1.80E-02	8.50E-02	2.30E+00	N (b)		---
Nickel	28 / 28	100	1.38E+01		3.89E+01		2.48E-01	5.10E+00	2.64E+01	5.51E+01	1.50E+02	N (b)		---
Potassium	27 / 28	96	3.23E+02		1.99E+03		2.07E+01	6.37E+02	9.46E+02	3.39E+03	Nutrient	N (c)		---
Selenium	4 / 28	14	6.40E-01		8.59E-01	J	5.40E-01	1.08E+00	4.45E-01	2.00E+00	3.90E+01	N (b)		---
Silver	2 / 28	7	1.20E+00		1.60E+00		4.13E-01	1.30E+00	4.44E-01	1.11E+01	3.90E+01	N (b)		---
Sodium	16 / 28	57	1.84E+01		1.52E+02		8.27E+00	6.37E+02	1.75E+02		Nutrient	N (c)		---
Thallium	14 / 28	50	2.93E-01	J	2.40E+00		4.13E-01	1.30E+00	6.22E-01	1.30E+00	5.10E-01 [f]	Y	7.77E-01	7.77E-01
Vanadium	28 / 28	100	7.50E+00		2.53E+01		1.65E-01	6.40E+00	1.65E+01	4.09E+01	3.90E+01	N (b)		---
Zinc	28 / 28	100	3.53E+01		8.53E+01		2.07E+00	2.71E+00	6.20E+01	3.22E+02	2.30E+03	N (b)		---
Semivolatile Organic Compounds														
Bis(2-ethylhexyl)phthalate	1 / 28	4	6.60E-02	J	6.60E-02	J	3.60E-01	4.51E-01	1.94E-01		3.50E+01	N (a)		---
Di-n-butyl phthalate	1 / 28	4	6.60E-02	J	6.60E-02	J	3.60E-01	4.51E-01	1.94E-01		6.10E+02	N (a)		---
Phenanthrene	7 / 28	25	4.03E-02	J	8.55E-02	J	3.60E-01	4.51E-01	1.65E-01		1.70E+02 [g]	N (a)		---
Volatile Organic Compounds														
Acetone	2 / 12	17	1.30E-02	J	1.20E-01		2.20E-02	1.30E-01	2.52E-02		6.10E+03	N (a)		---
Bromomethane	1 / 12	8	1.10E-03	J	1.10E-03	J	1.10E-02	6.40E-02	9.51E-03		7.30E-01	N (a)		---
Toluene	1 / 12	8	1.10E-02		1.10E-02		5.40E-03	3.20E-02	5.56E-03		5.00E+02	N (a)		---
Xylenes, total	1 / 12	8	6.50E-03		6.50E-03		5.40E-03	3.20E-02	5.18E-03		6.30E+01	N (a)		---

RBSC - Risk-based screening concentration.
COPC - Chemical of potential concern.
mg/kg - Milligrams per kilogram.
BSC - Background screening criterion.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Table (December 2009) residentia

Table 2-5

Statistical Summary and COPC Selection of Chemicals Detected in Subsurface Soil
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.

[b] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = maximum detected concentration is less than the BSC.
(c) = essential nutrient.

[c] Y = Chemical is chosen as COPC.

[d] 95% UCL determined using ProUCL Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC.

[e] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.

[f] RBSC based on thallium from IRIS, 2009.

[g] RBSC based on pyrene.

Table 2-6

Statistical Summary and COPC Selection of Chemicals Detected in Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	BSC [a] µg/L	RBSC [b] µg/L	COPC? [c,d]	EPC [e] µg/L
Metals													
Aluminum	4 / 4	100	1.74E+01	J	4.61E+01	JJ	2.00E+02	2.00E+02	3.51E+01	3.09E+02	3.70E+03	N (a)	---
Barium	4 / 4	100	2.12E+01	J/J	1.80E+02	J/J	2.00E+02	2.00E+02	9.61E+01	1.18E+04	7.30E+02	N (a)	---
Calcium	4 / 4	100	2.71E+05	J	3.89E+05	J/J	1.00E+03	1.00E+03	3.29E+05	3.16E+05	Nutrient	N (c)	---
Iron	2 / 4	50	3.30E+01	J	2.83E+02	J	3.00E+02	3.00E+02	1.54E+02	1.55E+03	2.60E+03	N (a)	---
Magnesium	4 / 4	100	1.07E+05	J	1.24E+05		5.00E+03	5.00E+03	1.16E+05	2.17E+05	Nutrient	N (c)	---
Manganese	4 / 4	100	5.65E+00	J/J	5.37E+01		1.50E+01	1.50E+01	1.93E+01	6.36E+02	8.80E+01	N (a)	---
Potassium	4 / 4	100	7.74E+03	J	1.56E+04		1.00E+04	2.00E+04	1.17E+04	1.16E+05	Nutrient	N (c)	---
Selenium	2 / 3	67	1.24E+01	J	1.95E+01	J/J	1.00E+01	1.00E+01	1.23E+01		1.80E+01	Y	1.95E+01
Sodium	4 / 4	100	2.64E+04		6.11E+04	J	1.00E+04	2.00E+04	4.43E+04	1.39E+06	Nutrient	N (c)	---
Vanadium	3 / 4	75	1.10E+00	J	1.60E+00	J	5.00E+01	5.00E+01	7.21E+00	5.01E+02	1.80E+01	N (a)	---
Semivolatile Organic Compounds													
Dimethylphenol, 2,4-	1 / 4	25	1.70E+00	J	1.70E+00	J	4.80E+00	4.80E+00	2.23E+00		7.30E+01	N (a)	---
Methylnaphthalene, 2-	4 / 4	100	1.60E+00	J/J	6.30E+00		4.80E+00	4.80E+00	3.34E+00		1.50E+01	N (a)	---
Naphthalene	4 / 4	100	1.75E+00	J/J	7.40E+00		4.80E+00	4.80E+00	3.39E+00		1.40E-01	Y	7.40E+00
Volatile Organic Compounds													
Acetone	2 / 2	100	4.25E+01	J/J	8.61E+01	J	2.50E+01	1.30E+02	6.43E+01		2.20E+03	N (a)	---
Benzene	4 / 4	100	7.80E+00	J/J	3.36E+02		1.00E+00	5.00E+00	1.11E+02		4.10E-01	Y	3.36E+02
Butanone, 2-	3 / 4	75	2.40E+00	J	7.40E+00		5.00E+00	2.50E+01	6.73E+00		7.10E+02	N (a)	---
Carbon disulfide	1 / 4	25	3.70E+00	J	3.70E+00	J	2.00E+00	1.00E+01	1.68E+00		1.00E+02	N (a)	---
Chloromethane	2 / 4	50	5.60E+00	J/J	4.49E+01	J	2.00E+00	1.00E+01	1.31E+01		1.90E+01	Y	4.49E+01
Dichloropropene, trans-1,3-	1 / 4	25	1.70E+00	J	1.70E+00	J	1.00E+00	5.00E+00	8.00E-01		4.30E-01	Y	1.70E+00
Ethylbenzene	4 / 4	100	8.15E+00	J/J	5.71E+01		1.00E+00	5.00E+00	2.31E+01		1.50E+00	Y	5.71E+01
Toluene	4 / 4	100	4.05E+00	J/J	1.47E+02		1.00E+00	5.00E+00	4.92E+01		2.30E+02	N (a)	---
Xylenes, total	4 / 4	100	4.63E+01	J/J	6.30E+02		3.00E+00	1.50E+01	2.32E+02		2.00E+01	Y	6.30E+02
General Chemistry (mg/L)													
Chloride	4 / 4	100	4.06E+01		1.18E+02		2.00E+00	2.50E+01	8.18E+01		Nutrient	N (c)	---
Cyanide, total	1 / 4	25	7.20E-03	J	7.20E-03	J	1.00E-02	1.00E-02	5.55E-03		7.30E-02	N (a)	---
Sulfate	4 / 4	100	4.96E+02		9.46E+02		1.00E+01	4.00E+01	7.16E+02		2.50E+05 [g]	N (a)	---

RBSC - Risk-based screening concentration.
COPC - Chemical of potential concern.
µg/L - Micrograms per liter.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Shaw Environmental, Inc. (Shaw), 2005b, *2004 Groundwater Data Summary and Evaluation Report* , Final, Former Plum Brook Ordnance Works, Sandusky, Ohio, April.

[b] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Table (December 2009) tap wate values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.

[c] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = maximum detected concentration is less than the BSC.
(c) = essential nutrient.

[d] Y = Chemical is chosen as COPC.

[e] Concentration used in risk assessment is equal to maximum detected concentration.

[f] Screening criteria for lead based on the action level of 15 µg/L (EPA, 1996, *Drinking Water Regulations and Health Advisories* , Office of Water, Washington, D.C., October).

[g] US Environmental Protection Agency (EPA), 2009, *2009 Edition of the Drinking Water Standards and Health Advisories* , Office of Water, October, EPA 822-R-09-011.

Table 2-7

Statistical Summary and COPC Selection of Chemicals Detected in Groundwater Overburden Wells
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	BSC [a] µg/L	RBSC [b] µg/L		COPC? [c,d]	Source-Term Concentration [e] µg/L
Inorganics														
Aluminum	3 / 3	100	4.71E+01	J	1.33E+02	J	2.00E+02	2.00E+02	8.62E+01	3.09E+02	3.70E+03		N (a)	---
Barium	3 / 3	100	2.49E+01	JJ	9.80E+01	J	2.00E+02	2.00E+02	4.94E+01	1.18E+04	7.30E+02		N (a)	---
Cadmium	1 / 3	33	1.05E+00	JJ	1.05E+00	JJ	5.00E+00	5.00E+00	2.02E+00		1.80E+00		N (a)	---
Calcium	3 / 3	100	5.44E+04		1.12E+05		1.00E+03	1.00E+03	8.95E+04	3.16E+05	Nutrient		N (c)	---
Cobalt	1 / 3	33	8.10E+00	JJ	8.10E+00	JJ	5.00E+01	5.00E+01	1.94E+01	1.21E+01	1.10E+00		N (b)	---
Iron	2 / 3	67	3.70E+01	J	8.08E+01	J	3.00E+02	3.00E+02	8.93E+01	1.55E+03	2.60E+03		N (a)	---
Lead	1 / 3	33	2.40E+00	J	2.40E+00	J	5.00E+00	5.00E+00	2.47E+00		1.50E+01	f	N (a)	---
Magnesium	3 / 3	100	1.16E+04		3.87E+04		5.00E+03	5.00E+03	2.10E+04	2.17E+05	Nutrient		N (c)	---
Manganese	3 / 3	100	8.22E+01		2.29E+02		1.50E+01	1.50E+01	1.61E+02	6.36E+02	8.80E+01		N (b)	---
Nickel	1 / 3	33	7.42E+01		7.42E+01		4.00E+01	4.00E+01	3.81E+01	8.60E+00	7.30E+01		Y	7.42E+01
Potassium	3 / 3	100	1.50E+03	J	7.55E+03	JJ	1.00E+04	2.00E+04	4.40E+03	1.16E+05	Nutrient		N (c)	---
Sodium	3 / 3	100	2.51E+03	JJ	1.02E+04	J	1.00E+04	2.00E+04	6.72E+03	1.39E+06	Nutrient		N (c)	---
Vanadium	2 / 3	67	1.40E+00	J	3.70E+00	J	5.00E+01	5.00E+01	1.00E+01		1.80E+01		N (a)	---
Zinc	1 / 3	33	3.83E+01		3.83E+01		2.00E+01	2.00E+01	1.94E+01	5.01E+02	1.10E+03		N (a)	---
General Chemistry (mg/L)														
Sulfate	3 / 3	100	81.5		162		2	10	1.13E+02		2.50E+02	g	N (a)	---

RBSC - Risk-based screening concentration.
COPC - Chemical of potential concern.
µg/L - Micrograms per liter.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Shaw Environmental, Inc. (Shaw), 2005b, *2004 Groundwater Data Summary and Evaluation Report* , Final, Former Plum Brook Ordnance Works, Sandusky, Ohio, April.

[b] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA)Regional Screening Level Table (December 2009) tap values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.

[c] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = maximum detected concentration is less than the BSC.
(c) = essential nutrient.

[d] Y = Chemical is chosen as COPC.

[e] Concentration used in risk assessment is equal to maximum detected concentration.

[f] Screening criteria for lead based on the action level of 15 µg/L (EPA, 1996, *Drinking Water Regulations and Health Advisories* , Office of Water, Washington, D.C., October).

[g] US Environmental Protection Agency (EPA), 2009, *2009 Edition of the Drinking Water Standards and Health Advisories* , Office of Water, October, EPA 822-R-09-011.

Table 2-8

Statistical Summary and COPC Selection of Chemicals Detected in Sediment
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	RBSC [a] mg/kg	COPC? [b,c]	95% UCL [d] mg/kg	EPC [e] mg/kg
Inorganics													
Aluminum	5 / 5	100	3.64E+03		7.42E+03		1.20E+01	1.50E+01	5.86E+03	7.70E+04	N (a)		---
Arsenic	5 / 5	100	3.90E+00		1.01E+01	J	4.75E-01	9.50E-01	6.35E+00	3.90E-01	Y	9.03E+00	9.03E+00
Barium	5 / 5	100	2.28E+01		6.71E+01	J	1.20E+01	1.50E+01	4.63E+01	1.50E+04	N (a)		---
Beryllium	5 / 5	100	3.60E-01		5.60E-01		3.00E-01	3.60E-01	4.70E-01	1.60E+02	N (a)		---
Cadmium	4 / 5	80	2.40E-01	J	5.65E-01		2.40E-01	4.80E-01	3.35E-01	7.00E+01	N (a)		---
Calcium	5 / 5	100	7.66E+03		2.70E+04		3.00E+02	3.60E+02	1.67E+04	Nutrient	N (b)		---
Chromium	5 / 5	100	6.40E+00		1.17E+01		5.95E-01	7.30E-01	8.97E+00	2.90E-01	Y	1.10E+01	1.10E+01
Cobalt	5 / 5	100	6.20E+00	J	1.20E+01		3.00E+00	3.60E+00	8.19E+00	2.30E+01	N (a)		---
Copper	5 / 5	100	9.60E+00		2.21E+01		1.50E+00	1.80E+00	1.51E+01	3.10E+03	N (a)		---
Iron	5 / 5	100	1.04E+04		3.91E+04	J	5.95E+00	1.20E+01	1.85E+04	5.50E+04	N (a)		---
Lead	5 / 5	100	7.90E+00		1.22E+01	JJ	5.95E+00	1.20E+01	9.65E+00	4.00E+02	N (a)		---
Magnesium	5 / 5	100	1.95E+03		7.47E+03	J	3.00E+02	3.60E+02	5.08E+03	Nutrient	N (b)		---
Manganese	5 / 5	100	1.27E+02		6.08E+02		9.90E-01	4.45E+00	2.90E+02	1.80E+03	N (a)		---
Mercury	4 / 5	80	1.60E-02	J	2.50E-02	J	9.80E-02	1.20E-01	2.62E-02	2.30E+01	N (a)		---
Nickel	5 / 5	100	1.57E+01		2.51E+01		2.40E+00	2.90E+00	1.81E+01	1.50E+03	N (a)		---
Potassium	5 / 5	100	4.98E+02	J	1.66E+03		6.00E+02	1.50E+03	1.03E+03	Nutrient	N (b)		---
Selenium	5 / 5	100	3.90E-01	J	1.00E+00	J	5.95E+00	1.20E+01	7.85E-01	3.90E+02	N (a)		---
Silver	1 / 5	20	8.30E-02	J	8.30E-02	J	5.95E-01	7.30E-01	2.79E-01	3.90E+02	N (a)		---
Sodium	5 / 5	100	5.48E+01	J	1.64E+02	JJ	6.00E+02	1.50E+03	1.17E+02	Nutrient	N (b)		---
Vanadium	5 / 5	100	9.40E+00		1.52E+01		3.00E+00	6.00E+00	1.26E+01	3.90E+02	N (a)		---
Zinc	5 / 5	100	2.70E+01	J	5.73E+01		1.20E+00	1.50E+00	4.06E+01	2.30E+04	N (a)		---
Explosives													
Trinitrotoluene, 2,4,6-	1 / 5	20	6.47E-02	J	6.47E-02	J	1.55E-01	1.90E-01	8.14E-02	1.90E+01	N (a)		---
Semivolatiles Organic Compounds													
Fluoranthene	2 / 5	40	5.25E-02	J	5.47E-02	J	2.05E-01	2.60E-01	8.49E-02	2.30E+03	N (a)		---

RBSC - Risk-based screening concentration.
COPC - Chemical of potential concern.
mg/kg - Milligrams per kilogram.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Table (December 2009) residential soil and are based on a risk level of 1.0E-06 and a hazard index of 1.

[b] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = essential nutrient.

[c] Y = Chemical is chosen as COPC.

[d] 95% UCL determined using ProUCL Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC.

[e] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.

Table 2-9

Statistical Summary and COPC Selection of Chemicals Detected in Surface Water
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Conc Minimum	VQ	Detected Conc Maximum	VQ	Reporting Limits Minimum	Reporting Limits Maximum	Arithmetic Mean µg/L	RBSC [a] µg/L		COPC? [b,c]
Metals												
Aluminum	5 / 5	100	359		1030	J	200	200	6.01E+02	3.70E+04		N (a)
Barium	5 / 5	100	43.9		50.9	J	200	200	4.70E+01	7.30E+03		N (a)
Calcium	5 / 5	100	75900		80700		1000	1000	7.83E+04	Nutrient		N (b)
Cobalt	1 / 5	20	1.2	J	1.2	J	50	50	2.02E+01	1.10E+01		N (a)
Iron	5 / 5	100	376		1060		300	300	7.22E+02	2.60E+04		N (a)
Lead	2 / 5	40	2.9	J	2.9	J	5	10	2.66E+00	1.50E+01	d	N (a)
Magnesium	5 / 5	100	19800		21400		5000	5000	2.08E+04	Nutrient		N (b)
Manganese	5 / 5	100	52.75		102		15	15	7.08E+01	8.80E+02		N (a)
Nickel	1 / 5	20	6.2	J	6.2	J	40	40	1.72E+01	7.30E+02		N (a)
Potassium	5 / 5	100	2900		4400	J	10000	20000	3.42E+03	Nutrient		N (b)
Sodium	5 / 5	100	30300		34800		10000	20000	3.26E+04	Nutrient		N (b)
Vanadium	5 / 5	100	1.65	JJ	3.1	J	50	50	2.23E+00	1.80E+02		N (a)
Zinc	5 / 5	100	8.1	J	12.4	J	20	20	1.06E+01	1.10E+04		N (a)
Semivolatile Organic Compounds												
Bis(2-ethylhexyl)phthalate	1 / 5	20	2	J	2	J	4.8	4.8	2.32E+00	4.80E+00		N (a)

RBSC - Risk-based screening concentration.
COPC - Chemical of potential concern.
µg/L - Micrograms per liter.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Tal (December 2009) tap water values are based on a risk level of 1.0E-06 and a hazard index of 1.
[b] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = essential nutrient.
[c] Y = Chemical is chosen as COPC.
[d] Screening criteria for lead based on the action level of 15 µg/L (EPA, 2009, *Drinking Water Regulations and Health Advisories*, Office of Water, Washington, D.C., October).

Table 3-1

Receptor/Exposure Scenarios
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Source Medium	Model	Exposure Medium	Exposure Route
Groundskeeper – Current			
Surface Soil	None	Soil	Incidental Ingestion
			Dermal Contact
	Dust Emissions Based on Activity	Ambient Air	Inhalation
	Volatilization from Soil	Ambient Air	Not Quantified[a]
Subsurface Soil	Not Quantified[b]		
Groundwater	Not Quantified[c]		
Surface Water	Not Quantified[b]		
Sediment	Not Quantified[b]		
Groundskeeper – Future			
Total Soil[d]	None	Soil	Incidental Ingestion
			Dermal Contact
	Dust Emissions Based on Activity	Ambient Air	Inhalation
	Volatilization from Soil	Ambient Air	Inhalation
Groundwater	None	Tap Water	Ingestion
			Dermal Contact
Surface Water	Not Quantified[b]		
Sediment	Not Quantified[b]		
Indoor Worker – Future[c]			
Surface Soil	None	Soil	Incidental Ingestion
			Dermal Contact[a]
	Dust Emissions; Volatilization	Indoor Air	Not Quantified[a]
Subsurface Soil	Volatilization from Soil	Indoor Air	Inhalation
Groundwater	None	Tap Water	Ingestion
			Dermal Contact
Surface Water	Not Quantified[b]		
Sediment	Not Quantified[b]		

Table 3-1

Receptor/Exposure Scenarios
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Source Medium	Model	Exposure Medium	Exposure Route
Construction Worker – Current/Future			
Total Soil[d]	None	Soil	Incidental Ingestion
			Dermal Contact
	Dust Emissions Based on Activity	Ambient Air	Inhalation
	Volatilization from Soil	Ambient Air	Inhalation
Groundwater	Not Quantified[b]		
Surface Water	None	Surface Water	Dermal Contact
	Volatilization from Water	Ambient Air	Not Quantified[a]
Sediment	None	Sediment	Incidental Ingestion
			Dermal Contact
On-Site Resident – Future			
Total Soil[d]	None	Soil	Incidental Ingestion
			Dermal Contact
	Dust Emissions Based on Wind Erosion	Ambient Air	Inhalation
	Volatilization from Soil	Ambient Air	Inhalation
Subsurface Soil	Volatilization from Soil	Indoor Air	Inhalation
Groundwater	None	Tap Water	Ingestion
			Dermal Contact
	Volatilization from Water	Indoor Air	Inhalation
Surface Water	None	Surface Water	Not Quantified[a]
	Volatilization from Water	Ambient Air	Dermal Contact
Sediment	None	Sediment	Incidental Ingestion
			Dermal Contact

Table 3-1

Receptor/Exposure Scenarios
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Source Medium	Model	Exposure Medium	Exposure Route
Hunter – Current/Future			
Surface Soil	None	Soil	Incidental Ingestion
			Dermal Contact
	Dust Emissions, Volatilization	Ambient Air	Not Quantified[a]
	Biouptake	Venison	Venison Consumption
Subsurface Soil	Not Quantified[c]		
Surface Water	Not Quantified[b]		
Sediment	Not Quantified[b]		
Hunter's Child – Current/Future			
Surface Soil	Not Quantified[c]		
	Not Quantified[c]		
	Biouptake	Venison	Venison Consumption
Subsurface Soil	Not Quantified[c]		
Surface Water	Not Quantified[c]		
Sediment	Not Quantified[c]		

[a] Although theoretically complete, this pathway is not quantified as explained in text.

[b] Although contact with this medium is possible, exposure would be sporadic, rather than continuous or predictable. Such exposures do not lend themselves to evaluation under the chronic toxicity paradigm used in a baseline risk assessment.

[c] There is no plausible pathway for exposure.

[d] Total soil represents a mixture of surface and subsurface soil. This is assumed for future scenarios where excavation and regrading is assumed to take place.

[e] Even though the mixing of surface and subsurface soil described in footnote "d" might otherwise be applicable, this receptor was selected primarily to evaluate exposure to indoor air resulting from subsurface soil contamination. Surface soil was used for direct contact exposure to avoid potential "double counting" of contaminants in subsurface soil (refer to Section 3.1.3.2 of text).

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
General Variables Used in All Intake Models					
Body weight (BW), kg	70[a]	70[a]	Child: 15[a] Adult: 70[a]	70[a]	Child: 15[a] Adult: 70[a]
Averaging time, noncancer (AT), days[b]	9125	183	Child: 2190 Adult: 8760	9125	Child: 2190 Adult: 10950
Averaging time, cancer (AT), days[b]	25550	25550	25550	25550	25550
Inhalation of VOCs and Resuspended Dust from Surface Soil, Total Soil or Subsurface Soil					
Fraction exposed to contaminated medium (FI_a), unitless	1[c]	1[c]	1[c]	NA	NA
Inhalation rate (IR_a), m^3/day	20[d]	20[d]	Child: 10[e] Adult: 20[d]	NA	NA
Exposure frequency (EF), days/year	250[d]	250[c]	350[a]	NA	NA
Exposure duration (ED), years	25[a]	0.5[c]	Child: 6[a] Adult: 24[a]	NA	NA

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Inhalation of VOCs in Indoor Air from Subsurface Soil					
Fraction exposed to contaminated medium (FI_a), unitless	NA	NA	1[c]	1[c]	NA
Inhalation rate (IR_a), m^3/day	NA	NA	Child: 10[e] Adult: 20[d]	20[d]	NA
Exposure frequency (EF), days/year	NA	NA	350[a]	250[a]	NA
Exposure duration (ED), years	NA	NA	Child: 6[a] Adult: 24[a]	25[a]	NA
Incidental Ingestion of Soil					
Fraction exposed to contaminated medium (FI_{so}), unitless	1[c]	1[c]	0.9[f]	1[c]	1[c]
Soil incidental ingestion rate (IR_{so}), mg/day	100[a]	330[a]	Child: 200[a] Adult: 100[a]	50[a]	Child: NA Adult: 100[a]
Exposure frequency (EF), days/year	250[d]	250[a]	350[a]	250[a]	14[d]
Exposure duration (ED), years	25[a]	0.5[c]	Child: 6[a] Adult: 24[a]	25[a]	30[a]
Dermal Contact with Soil					
Fraction exposed to contaminated medium (FI_{so}), unitless	1[c]	1[c]	1[c]	NA	1[c]
Body surface area exposed to soil (SA_{so}), cm^2	3,300[g]	3,300[g]	Child: 2,800[g] Adult: 5,700[g]	NA	Child: NA Adult: 3,300[c]
Soil-to-skin adherence factor (AF_{so}), mg/cm^2	0.2[g]	0.3[g]	Child: 0.2[g] Adult: 0.07[g]	NA	0.2[c]

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Dermal absorption factor (ABS), unitless	csv	csv	Csv	NA	csv
Exposure frequency (EF), days/year	250[d]	250[a]	350[a]	NA	14[c]
Exposure duration (ED), years	25[a]	0.5[c]	Child: 6[a] Adult: 24[a]	NA	30[a]
Inhalation of VOCs from Groundwater					
Exposure time (ET), hours/day	NA	NA	24[h]	NA	NA
Inhalation rate (IR_a), m^3/hour	NA	NA	Child: 0.416[e] Adult: 0.833[e]	NA	NA
Exposure frequency (EF), days/year	250[d]	NA	350[a]	250[a]	NA
Drinking Water Ingestion of Groundwater					
Fraction exposed to contaminated medium (Fl_{gw}), unitless	1[c]	NA	1[c]	1[c]	NA
Drinking water ingestion rate (IR_{gw}), L/day	1[d]	NA	Child: 1[e] Adult: 2[d]	1[d]	NA
Exposure frequency (EF), days/year	250[d]	NA	350[a]	250[a]	NA
Dermal Contact with Groundwater					
Fraction exposed to contaminated medium (Fl_{gw}), unitless	1[c]	NA	1[c]	1[c]	NA
Body surface area exposed to water (SA_{gw}), cm^2	3,300[c]	NA	Child: 6,600[i] Adult: 20,000[i]	3,300[i]	NA

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Permeability coefficient (PC), cm/hour	csv	NA	csv	csv	NA
Exposure time (ET_{gw}), hours/day	1[c]	NA	Child: 0.333[i] Adult: 0.2[i]	1[c]	NA
Exposure frequency (EF), days/year	250[d]	NA	350[d]	250[a]	NA
Incidental Ingestion of Sediment					
Fraction exposed to contaminated medium (FI_{sd}), unitless	NA	1[c]	0.1[f]	NA	NA
Sediment incidental ingestion rate (IR_{sd}), mg/day	NA	330[a]	Child: 200[a] Adult: 100[a]	NA	NA
Exposure frequency (EF), days/year	NA	250[c]	350[a]	NA	NA
Exposure duration (ED), years	NA	0.5[c]	Child: 6[a] Adult: 24[a]	NA	NA
Dermal Contact with Sediment					
Fraction exposed to contaminated medium (FI_{sd}), unitless	NA	1[c]	0.1[f]	NA	NA
Body surface area exposed to sediment (SA_{sd}), cm^2	NA	3,300[g]	Child: 2,800[g] Adult: 5,700[g]	NA	NA
Sediment-to-skin adherence factor (AF_{sd}), mg/cm^2	NA	0.3[g]	Child: 0.2[g] Adult: 0.07[g]	NA	NA
Dermal absorption factor (ABS), unitless	NA	csv	csv	NA	NA
Exposure frequency (EF), days/year	NA	250[c]	52[c]	NA	NA
Exposure duration (ED), years	NA	0.5[c]	Child: 6[a] Adult: 24[a]	NA	NA

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Dermal Contact with Surface Water					
Body surface area exposed to surface water (SA_{sw}), cm^2	NA	3,300[j]	Child: 2,800[j] Adult: 7,000[c]	NA	NA
Permeability coefficient (PC), cm/hour	NA	csv	csv	NA	NA
Exposure time (ET_{sw}), hour/day	NA	4[c]	3[c]	NA	NA
Exposure frequency (EF), days/year	NA	250[c]	52[c]	NA	NA
Exposure duration (ED), years	NA	0.5[c]	Child: 6[a] Adult: 24[a]	NA	NA
Venison Consumption					
Venison ingestion rate (IR_v), kg/day	NA	NA	NA	NA	Child: 0.005[c] Adult: 0.013[c]
Exposure frequency (EF), days/year	NA	NA	NA	NA	350[a]
Exposure duration (ED), years	NA	NA	NA	NA	Child: 6[a] Adult: 30[c]

[a] U.S. Environmental Protection Agency (EPA), 2002, *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.

[b] For noncancer evaluation, calculated as the product of ED (years) x 365 days/year; for cancer evaluation, calculated as the product of 70 years (assumed human lifetime) x 365 days/year. Source: U.S. Environmental Protection Agency (EPA), 1989a, *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A)*, Interim Final, Office of Emergency and Remedial Response, Washington, D.C., EPA/540/1-89/002.

[c] Assumed; see text.

[d] U.S. Environmental Protection Agency (EPA), 1991, *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance, Standard Default Exposure Factors*, Interim Final, Office of Solid Waste and Emergency Response, OSWER Directive: 9285.603.

[e] U.S. Environmental Protection Agency (EPA), 2004a, *User's Guide and Background Technical Document for Region 9 Preliminary Remediation Goals (PRG) Table*, Region 9, San Francisco, California, October, <http://www.epa.gov/region09/waste/sfund /prg/files/04usersguide.pdf>.

[f] It is assumed that on days when the resident is visiting the ditches and is exposed to sediment that half of the daily exposure via dermal contact and

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

ingestion are associated with ditch sediment (sediment FI=0.5) and half of the exposure is associated with soil (soil FI=0.5). The resident is assumed to be exposed to soil 350 days/year and to sediment 52 days/year. The FI values of 0.1 for sediment and 0.9 for soil are weighted average daily values as described in Section 3.1.3.4 of the text.

[g] U.S. Environmental Protection Agency (EPA), 2004b, *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)*, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

[h] The *Exposure Factors Handbook* (see reference i) indicates that the 90th percentile for the amount of time spent at a residence is more than 23 hours per day.

[i] U.S. Environmental Protection Agency (EPA), 1997, *Exposure Factors Handbook*, Final, National Center for Environmental Assessment, Washington, D.C., EPA/600/P-95/002Fa, August.

[j] Value for dermal soil exposure (EPA, 2004b) was selected as appropriate for exposure to this medium by this receptor; refer to text for detail.

NA – Pathway not applicable for receptor.

Table 3-3

Physical Properties of Site-Related Chemicals[a]
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical of Potential Concern	Soil-to-forage Biotransfer Factor B_p (unitless)	Biotransfer Factor for Beef B_b (days/kg)	Biotransfer Factor for Venison B_v (unitless)	Diffusivity In Air (D_i) (cm^2/second)	Soil Organic Carbon-Water Partition Coefficient K_{oc} (cm^3/g)	log K_{ow}	Absorption Fraction ABS (unitless)	Permeability Coefficient K_p (cm/hour)	*tau* (hour/event)	*t** (hour)	Fraction Absorbed from Water FA (unitless)	B (unitless)	Molecular Weight (g/mole)	Henry's Law Constant (atm-m^3/mole)
Metals														
Arsenic	NA	NA	NA	NA	2.90E+01	NA	0.03	1.00E-03	NA	NA	NA	NA	74.92	NA
Beryllium	NA	NA	NA	NA	7.90E+02	NA	NA	1.00E-03	NA	NA	NA	NA	9.01	NA
Chromium III	NA	NA	NA	NA	1.80E+06	NA	NA	1.00E-03	NA	NA	NA	NA	52	NA
Chromium VI	NA	NA	NA	NA	1.90E+01	NA	NA	2.00E-03	NA	NA	NA	NA	52	NA
Nickel	NA	NA	NA	NA	6.50E+01	NA	NA	2.00E-04	NA	NA	NA	NA	58.69	NA
Selenium	2.50E-02	1.50E-02	9.00E-05	NA	5.00E+00	NA	NA	1.00E-03	NA	NA	NA	NA	78.96	NA
Thallium	4.00E-04	4.00E-02	2.40E-04	NA	7.10E+01	NA	NA	1.00E-03	NA	NA	NA	NA	204.38	NA
Pesticides and PCBs														
Aroclor 1016	2.24E-02	1.00E-02	6.00E-05	4.69E-02	9.12E+04	5.6	0.14	4.30E-01	11.29	47.9	0.5	3.2	257.9	2.90E-04
Semivolatile Organic Compounds														
Benzo(a)pyrene	NA	NA	NA	4.30E-02	1.02E+06	6.1	0.13	7.00E-01	2.69	11.67	1.0	4.3	252.3	1.13E-06
Benzo(b)fluoranthene	NA	NA	NA	2.26E-02	1.23E+06	6.12	0.13	7.00E-01	2.77	12.03	1.0	4.3	252.3	1.11E-04
Naphthalene	NA	NA	NA	5.90E-02	2.00E+03	3.3	0.13	4.70E-02	0.56	1.34	1	0.2	128.2	4.83E-04
Volatile Organic Compounds														
Benzene	NA	NA	NA	NA	1.77E+00	2.13	NA	1.50E-02	0.29	0.70	1	0.1	78.1	5.56E-03
Chloromethane (Methyl Chloride)	NA	NA	NA	1.39E-01	1.41E+01	0.91	NA	3.30E-03	0.2	0.49	1	0	50.5	8.82E-03
Ethylbenzene	NA	NA	NA	7.50E-02	3.63E+02	3.15	NA	4.90E-02	0.42	1.01	1.0	0.2	106.2	7.88E-03
trans-1,3-Dichloropropene	NA	NA	NA	6.26E-02	8.13E+01	2	NA	4.30E-03	0.45	1.07	1	0	111	3.55E-03
Xylenes	NA	NA	NA	7.00E-02	4.07E+02	3.2	NA	5.30E-02	0.42	1.01	1.0	0.2	106.2	7.34E-03

tau - Lag time associated with rate at which chemical crosses stratum corneum per event.
t* - Time for absorption across the stratum corneum to reach steady state ; equals 2.4 times tau.
B - Ratio of the permeability coefficient of a compound through the stratum corneum relative to its permeability coefficient across the viable epidermis
kg - Kilogram.
g - Gram.
atm-m^3 - Atmospheres per cubic meter.
[a]Appendix C provides references for these values.

Table 4-1

Summary of Toxicity Assessment[a]
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical of Potential Concern	GAF	Weight of Evidence	Oral Slope Factor (mg/kg-day) $^{-1}$	Dermal Slope Factor (mg/kg-day) $^{-1}$	Weight of Evidence	Inhalation Unit Risk (µg/m^3) $^{-1}$	Inhalation Slope Factor (mg/kg-day) $^{-1}$
Metals							
Arsenic	1	A	1.5E+00	1.5E+00	A	4.3E-03	1.5E+01
Beryllium	0.007	D	NA	NA	B1	2.4E-03	8.4E+00
Chromium III	0.013	D	NA	NA	D	NA	NA
Chromium VI	0.025	D	NA	NA	A	1.2E-02	4.2E+01
Chromium, Total	0.013	D	NA	NA	A	1.2E-02	4.2E+01
Nickel	0.04	D	NA	NA	A	2.4E-04	8.4E-01
Selenium	1	D	NA	NA	D	NA	ND
Thallium	1	D	NA	NA	D	NA	ND
Pesticides and PCBs							
Aroclor 1016	1	B2	7.0E-02	7.0E-02	B2	NA	7.0E-02
Semivolatile Organic Compounds							
Benzo(a)pyrene	1	B2	7.3E+00	7.3E+00	B2	8.8E-04	3.1E+00
Benzo(b)fluoranthene	1	B2	7.3E-01	7.3E-01	B2	8.8E-05	3.1E-01
Naphthalene	1	C	ND	ND	C	3.4E-05	1.2E-01
Volatile Organic Compounds							
Benzene		A	5.5E-02	5.5E-02	A	7.8E-06	2.7E-02
Chloromethane (Methyl Chloride)	1	D	NA	NA	D	NA	NA
Ethylbenzene	1	D	NA	NA	D	NA	ND
trans-1,3-Dichloropropene	1	B2	1.0E-01	1.0E-01	B2	4.0E-06	1.4E-02
Xylenes	1	D	NA	NA	D	NA	ND

mg/kg-day - Milligrams per kilogram - day.
µg/m^3 - Milligrams per cubic meter.
GAF - Gastrointestinal absorption factor.
NA - Not available or not applicable.
ND - No data or not determined.

Weight of Evidence EPA Group:
A - Carcinogenic to humans
B - Likely to be carcinogenic to humans (includes both the former B1 and B2 probable human carcinogens as classified on IRIS)
C - Suggested evidence of cardcinogenic potential
D - Inadequate evidence to assess carcinogenic potential

Table 4-1

Summary of Toxicity Assessment[a]
Powerhouse 2 Ash Pits, Plum Brook Ordnance Works
Sandusky, Ohio

		Oral		Dermal	Reference	Inhalation	
Chemical of Potential Concern	GAF	Reference Dose mg/kg-day	Target Organs	Reference Dose mg/kg-day	Concentration mg/m^3	Reference Dose mg/kg-day	Target Organs
Metals							
Arsenic	1	3.0E-04	S	3.0E-04	NA	ND	NA
Beryllium	0.007	2.0E-03	GI	1.4E-05	2.0E-02	5.7E-06	RT
Chromium III	0.013	1.5E+00	ND	2.0E-02	NA	ND	NA
Chromium VI	0.025	3.0E-03	ND	7.5E-05	1.0E-04	2.9E-05	Lung
Chromium, Total	0.013	2.1E-02	ND	2.7E-04	7.0E-04	2.0E-04	Lung
Nickel	0.04	2.0E-02	RB/OW	8.0E-04	NA	ND	NA
Selenium	1	5.0E-03	Selenosis	5.0E-03	NA	ND	NA
Thallium	1	6.5E-05	S,L	6.5E-05	NA	ND	NA
Pesticides and PCBs							
Aroclor 1016	1	7.0E-05	F	7.0E-05	NA	ND	NA
Semivolatile Organic Compounds							
Benzo(a)pyrene	1	ND	NA	ND	NA	ND	NA
Benzo(b)fluoranthene	1	ND	NA	ND	NA	ND	NA
Naphthalene	1	2.0E-02	RB/OW	2.0E-02	3.0E-03	8.6E-04	RT
Volatile Organic Compounds							
Benzene		4.0E-03	BC	4.0E-03	3.0E-03	8.6E-03	BC
Chloromethane (Methyl Chloride)	1	ND	NA	ND	9.0E-02	2.6E-02	CNS
Ethylbenzene	1	1.0E-01	L,K	1.0E-01	1.0E+00	2.9E-01	F
trans-1,3-Dichloropropene	1	3.0E-02	Stomach Irritation	3.0E-02	2.0E-02	5.7E-03	NE
Xylenes	1	2.0E-01	RB/OW, Su	2.0E-01	1.0E-01	2.9E-02	CNS

mg/kg-day - Milligrams per kilogram - day.
mg/m^3 - Milligrams per cubic meter.
GAF - Gastrointestinal absorption factor.
NA - Not available or not applicable.
ND - No data.

Target Organs: BC - blood cells; S - skin; GI - gastrointestinal; CNS - central nervous system; L - liver; F - fetus; NE - nasal epithelium; NS - nervous system; RB/OW - reduced body/organ weight; SU - reduced survival; RT - respiratory tract.

[a]See Appendix C which provides references for these values.

Table 5-1

Summary of Risk for All Receptors
Powerhouse 2 Ash Pit
Plum Brook Ordnance Works
Sandusky, Ohio

	Groundskeeper [a]		Construction Worker		Indoor Worker		Resident			Hunter		Hunter's Child	
	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total Adult - HI	Total Child - HI	Total ILCR	Total HI	Total ILCR	Total HI
Bedrock Groundwater Use													
Exposure to:													
Soil [b]	1.56E-06	0.09	9.08E-08	0.3	2.39E-07	0.03	2.84E-06	0.07	0.6	5.97E-08	0.003	4.43E-13	0.000003
Groundwater	7.01E-05	1.0	NE	NE	7.01E-05	1.0	3.39E-04	2.9	6.5	NE	NE	NE	NE
Sediment	NE	NE	3.40E-07	0.1	NE	NE	2.15E-06	0.004	0.04	NE	NE	NE	NE
Surface Water	NE	NE	NA	NA	NE	NE	NA	NA	NA	NE	NE	NE	NE
Total ILCR or HI	**7.E-05**	**1**	**4.E-07**	**0.4**	**7.E-05**	**1**	**3.E-04**	**3**	**7**	**6.E-08**	**0.003**	**4.E-13**	**0.000003**
Overburden Groundwater Use													
Exposure to:													
Soil [b]	1.56E-06	0.09	9.08E-08	0.3	2.39E-07	0.03	2.84E-06	0.07	0.6	5.97E-08	0.003	4.43E-13	0.000003
Groundwater	NA	0.04	NE	NE	NA	0.04	NA	0.1	0.2	NE	NE	NE	NE
Sediment	NE	NE	3.40E-07	0.1	NE	NE	2.15E-06	0.004	0.04	NE	NE	NE	NE
Surface Water	NE	NE	NA	NA	NE	NE	NA	NA	NA	NE	NE	NE	NE
Total ILCR or HI	**2.E-06**	**0.1**	**4.E-07**	**0.4**	**2.E-07**	**0.06**	**5.E-06**	**0.2**	**0.9**	**6.E-08**	**0.003**	**4.E-13**	**0.000003**

HI - Hazard index.
ILCR - Incremental lifetime cancer risk.
NA - No chemicals of potential concern available for exposure evaluation.
NE - Pathway not evaluated for this receptor.

[a] Total ILCR and total HI values for the groundskeeper reflect the respective totals for the future groundskeeper assumed to use either bedrock or overburden groundwater. The total ILCR and HI values for the current groundskeeper are simply those shown for soil. The rounded current groundskeeper ILCR is 2E-6 and the HI is 0.09.
[b] Because the surface soil data set is used to represent total soil (refer to Section 2.5 of text), associated risk of total soil and surface soil are not shown separately.

Table 5-2

Summary of Risk for Receptors Exposed to Bedrock Groundwater Without the Contributions of Benzene
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works
Sandusky, Ohio

	Future Groundskeeper		Indoor Worker		Resident		
	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total Adult - HI	Total Child - HI
Bedrock Groundwater Use							
Exposure to:							
Soil	1.56E-06	0.09	2.39E-07	0.03	2.84E-06	0.07	0.6
Groundwater	6.10E-07	0.1	6.10E-07	0.1	5.44E-06	0.3	0.6
Sediment	NE	NE	NE	NE	2.15E-06	0.004	0.04
Surface Water	NE	NE	NE	NE	NA	NA	NA
Total ILCR or HI	**2.E-06**	**0.2**	**8.E-07**	**0.1**	**1.E-05**	**0.3**	**1**

HI - Hazard index.
ILCR - Incremental lifetime cancer risk.
NA - No chemicals of potential concern available for exposure evaluation.
NE - Pathway not evaluated for this receptor.

Note: Benzene is naturally occurring in bedrock groundwater underlying the Powerhouse 2 Ash Pits; refer to Section 7.2 of the text.

FIGURES

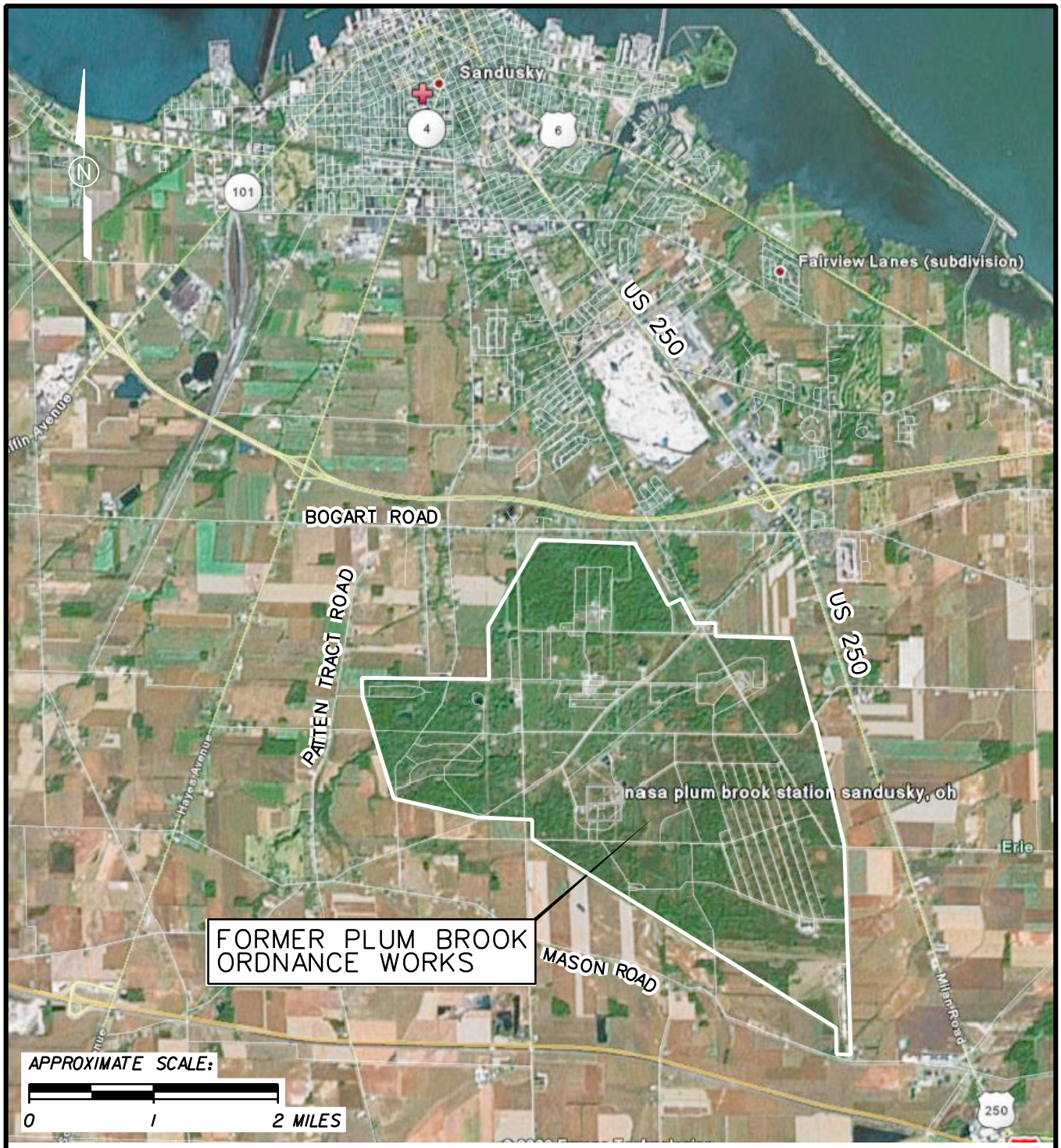

FIGURE 1-1
PBOW VICINITY MAP

POWERHOUSE 2 ASH PITS
BASELINE HUMAN HEALTH
RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

Shaw® a world of Solutions™

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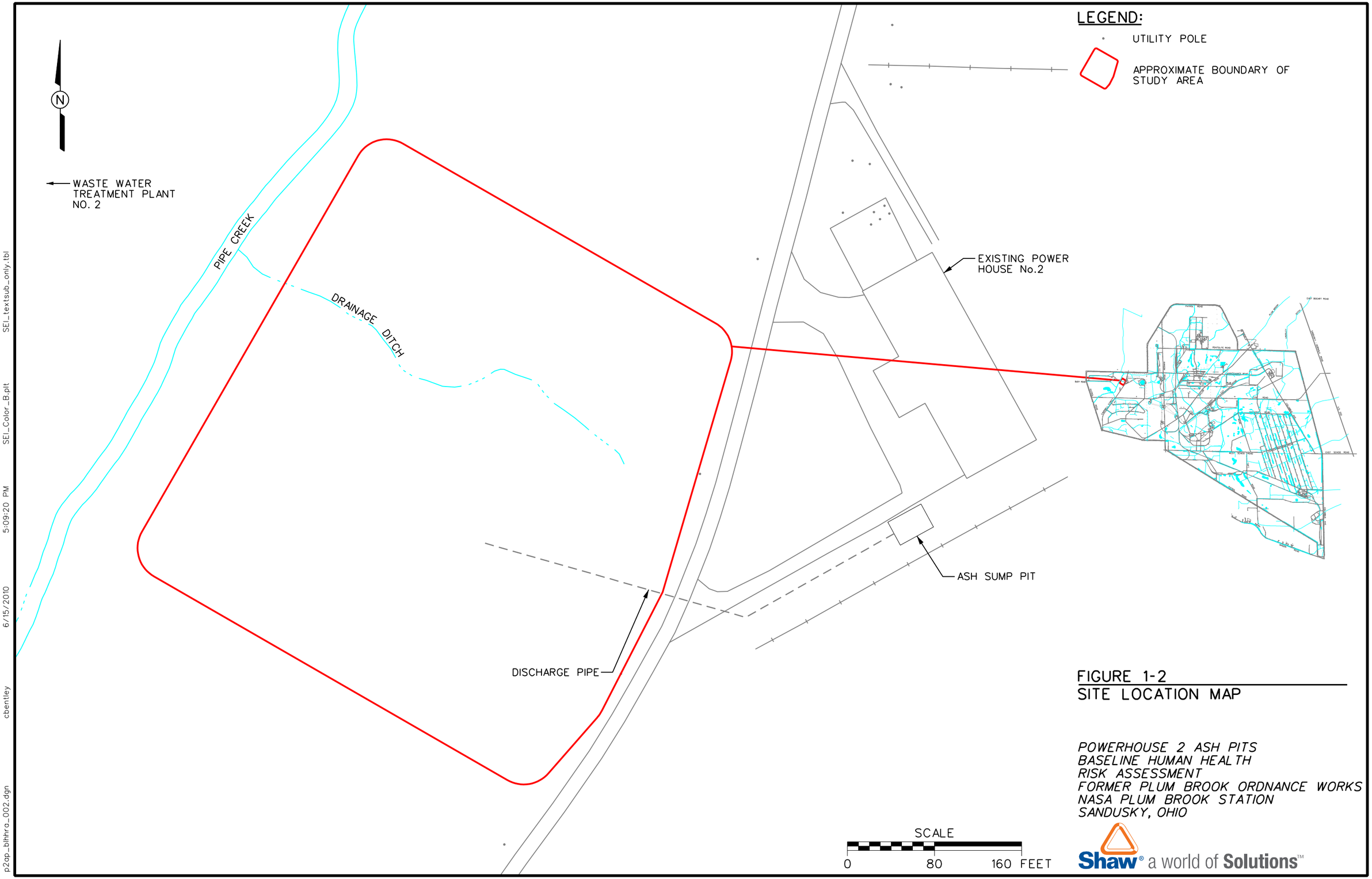

FIGURE 1-2
SITE LOCATION MAP

POWERHOUSE 2 ASH PITS
BASELINE HUMAN HEALTH
RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

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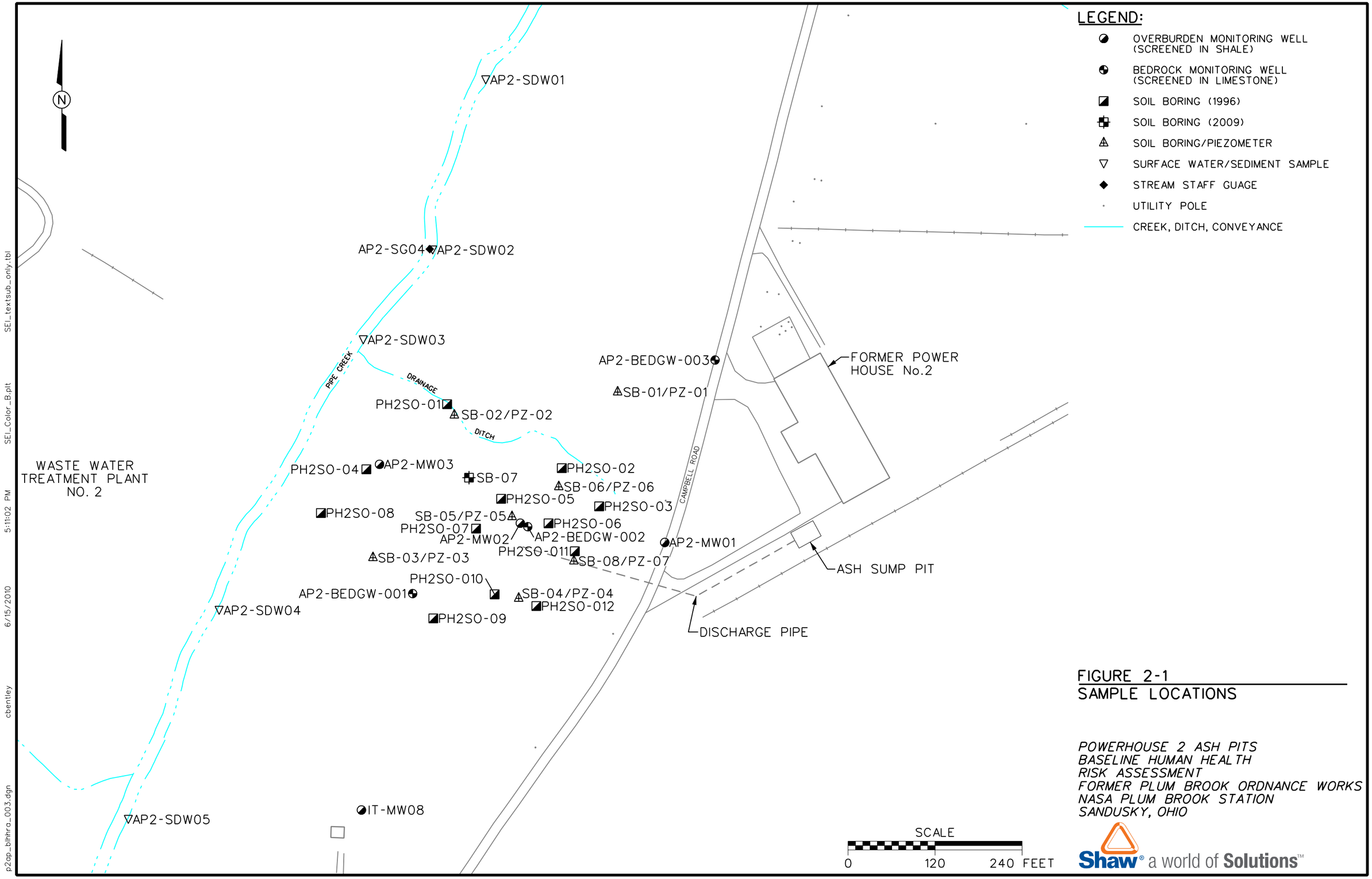
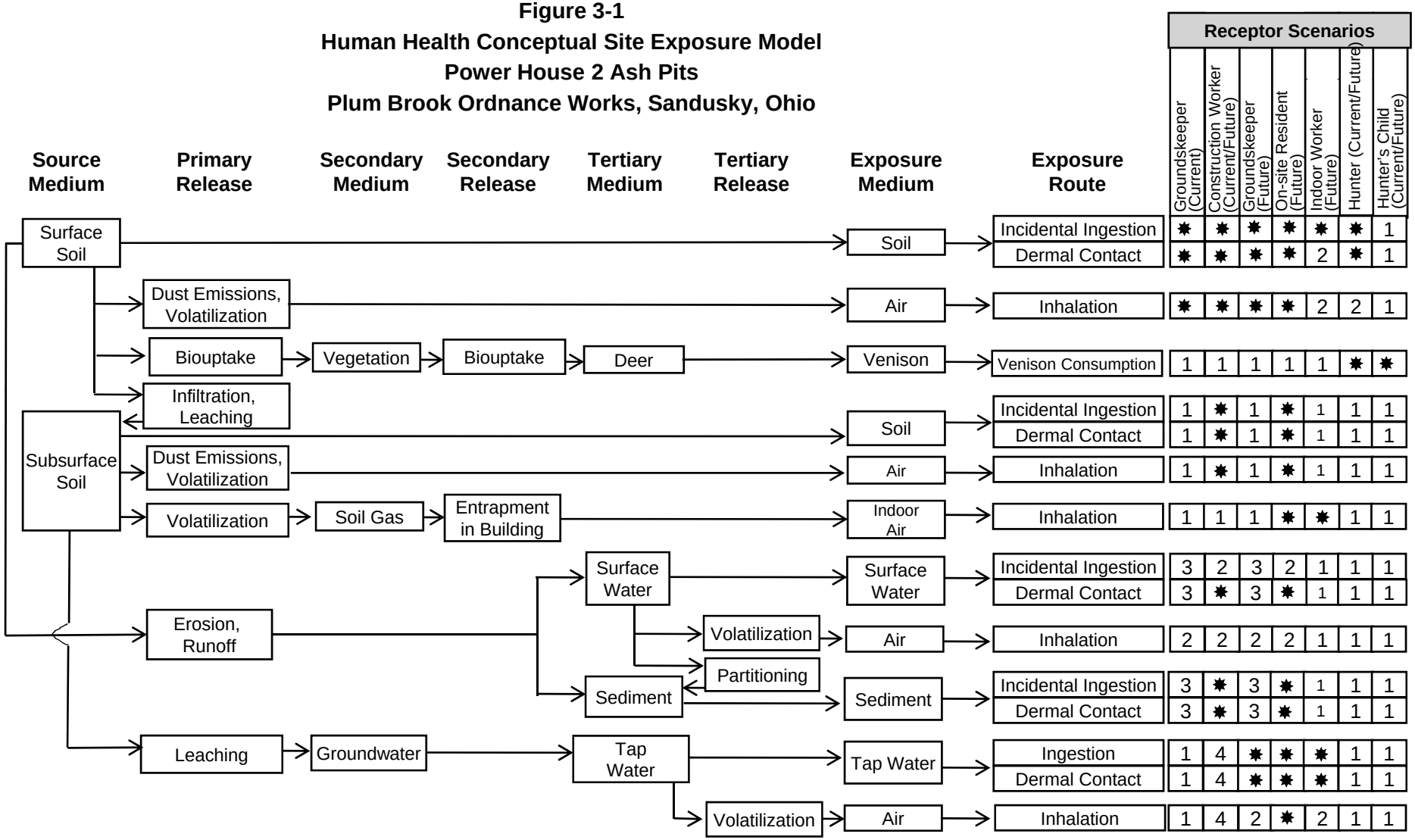


FIGURE 2-1
SAMPLE LOCATIONS

POWERHOUSE 2 ASH PITS
BASELINE HUMAN HEALTH
RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

Figure 3-1
Human Health Conceptual Site Exposure Model
Power House 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio



✸ = Complete exposure route quantified in the risk assessment.
1 = There is no plausible pathway for exposure to this medium.
2 = Although theoretically complete, this pathway is not quantified as explained in text.
3 = Contact with this medium, although plausible, is not part of this receptor's normal or expected activities; therefore contact would be sporadic and is not quantified.
4 = For current use there is no plausible exposure pathway. For future use, the pathway is potentially complete, but is not quantified as explained in the text.

KN10/PBOW/PH2/AP2/BHHRA/Draft/Fig3-1/2/17/2010 1:18 PM

APPENDIX A

DATA SUMMARY OF CHEMICALS DETECTED IN OVERBURDEN GROUNDWATER PIEZOMETERS

Table A-1

Sample Summary of Overburden Groundwater Piezometers
Powerhouse 2 Ash Pit
Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample Number	Sample Purpose	Sample Date	Analyses
ASH PIT 2-PZ03	AP3018	REG	31-Jan-09	Cyanide, Exp, Field Tests, Gen Chem, Metals, (f & uf), SVOC
ASH PIT 2-PZ04	AP3019	REG	1-Feb-09	Cyanide, Exp, Field Tests, Gen Chem, Metals, (f & uf), SVOC
ASH PIT 2-PZ07	AP3015	FD	2-Feb-09	Cyanide, Exp, Gen Chem, Metals, (f & uf), SVOC
ASH PIT 2-PZ07	AP3022	REG	2-Feb-09	Cyanide, Exp, Field Tests, Gen Chem, Metals, (f & uf), SVOC

Table A-2

Summary and Screening of Chemicals Detected in Overburden Groundwater Piezometers
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	BSC [a] µg/L	RBSC [b] µg/L		Exceeds BSC? [c,d]	Exceeds RBSC? [c,d]
Inorganics														
Aluminum	4 / 5	80	7.43E+01	J	2.40E+03		8.89E+01	2.00E+02	5.63E+02	3.09E+02	3.70E+03		Y	N
Barium	4 / 5	80	2.40E+01		5.11E+01		4.44E+00	2.00E+02	4.77E+01	1.18E+04	7.30E+02		N	N
Cadmium	4 / 5	80	3.91E+00	J	4.28E+00	J	4.44E+00	5.00E+00	3.82E+00		1.80E+00			Y
Calcium	5 / 5	100	1.01E+05		1.79E+05		1.11E+02	5.00E+03	1.27E+05	3.16E+05	Nutrient		N	N
Copper	3 / 5	60	5.63E+00	J	1.23E+01		1.11E+01	2.50E+01	8.34E+00	1.98E+01	1.50E+02		N	N
Iron	5 / 5	100	1.91E+02		5.06E+03		8.89E+01	1.00E+02	1.27E+03	1.55E+03	2.60E+03		Y	Y
Lead	1 / 5	20	3.02E+00	J	3.02E+00	J	3.00E+00	5.56E+00	2.57E+00		1.50E+01	e		N
Magnesium	5 / 5	100	2.92E+04		4.27E+04		4.44E+01	5.00E+03	3.51E+04	2.17E+05	Nutrient		N	N
Manganese	5 / 5	100	2.07E+01		5.18E+01		4.44E+00	1.50E+01	2.81E+01	6.36E+02	8.80E+01		N	N
Nickel	3 / 5	60	4.00E+00	J	6.28E+00	J	6.67E+00	4.00E+01	7.80E+00	8.60E+00	7.30E+01		N	N
Potassium	4 / 5	80	4.05E+02	J	1.26E+03		5.56E+02	5.00E+03	1.05E+03	1.16E+05	Nutrient		N	N
Selenium	1 / 5	20	7.55E+00	J	7.55E+00	J	5.00E+00	1.11E+01	5.34E+00		1.80E+01			N
Sodium	5 / 5	100	6.38E+03		1.22E+04		2.22E+02	5.00E+03	8.32E+03	1.39E+06	Nutrient		N	N
Vanadium	1 / 5	20	5.39E+00		5.39E+00		4.44E+00	5.00E+01	7.41E+00		1.80E+01			N
Inorganics - Filtered														
Barium	4 / 5	80	2.28E+01		3.49E+01		4.44E+00	2.00E+02	4.30E+01	1.08E+04	7.30E+02		N	N
Cadmium	4 / 5	80	3.90E+00	J	4.36E+00	J	4.44E+00	5.00E+00	3.77E+00		1.80E+00			Y
Calcium	5 / 5	100	1.03E+05		1.72E+05		1.11E+02	5.00E+03	1.24E+05	3.16E+05	Nutrient		N	N
Copper	1 / 5	20	6.90E+00	J	6.90E+00	J	1.11E+01	2.50E+01	7.21E+00	6.10E+00	1.50E+02		Y	N
Magnesium	5 / 5	100	2.99E+04		4.28E+04		4.44E+01	5.00E+03	3.52E+04	2.19E+05	Nutrient		N	N
Manganese	4 / 5	80	1.62E+01		3.07E+01		4.44E+00	1.50E+01	1.70E+01	6.69E+02	8.80E+01		N	N
Potassium	4 / 5	80	3.46E+02	J	5.10E+02	J	5.56E+02	5.00E+03	8.52E+02		Nutrient			N
Selenium	1 / 5	20	6.82E+00	J	6.82E+00	J	5.00E+00	1.11E+01	5.19E+00		1.80E+01			N
Sodium	5 / 5	100	6.18E+03		1.25E+04		2.22E+02	5.00E+03	8.36E+03	1.39E+06	Nutrient		N	N
Semivolatile Organic Compounds														
Dimethylphenol, 2,4-	1 / 5	20	3.38E+00	J	3.38E+00	J	1.00E+01	1.03E+01	4.70E+00		7.30E+01			N
General Chemistry (mg/L)														
Chloride	5 / 5	100	2.10E+00		1.16E+01	J	2.50E-01	1.00E+00	4.94E+00		Nutrient			N
Cyanide, total	1 / 5	20	4.40E-02		4.40E-02		1.00E-02	2.50E-02	1.88E-02		7.30E-02			N
Nitrate-Nitrite	3 / 4	75	2.80E-02	J	5.66E-01	J	4.00E-02	4.00E-01	2.11E-01		NA			Y
Sulfate	5 / 5	100	7.96E+01		3.41E+02		1.00E+00	5.00E+00	1.83E+02		2.50E+02	f		Y

RBSC - Risk based screening concentration; BSC - background screening concentration.
µg/L - micrograms per liter.
VQ - Validation qualifier.
J - The compound/analyte was positively identified; the reported result is the estimated concentration of the compound/analyte detected in the sample analyzed.

[a] Shaw Environmental, Inc. (Shaw), 2005b, 2004 Groundwater Data Summary and Evaluation Report, Final, Former Plum Brook Ordnance Works, Sandusky, Ohio, April.

[b] Risk-Based Screening Concentrations based on Oak Ridge National Laboratory-U.S. Environmental Protection Agency (ORNL-EPA) Regional Screening Level Table (December 2009) tap water values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.

[c] N = Chemical concentration does not exceed the criterion, or is an essential nutrient.

[d] Y = Chemical concentration exceeds the criterion.

[e] Screening criteria for lead based on the action level of 15 µg/L (EPA, 2009, *Drinking Water Regulations and Health Advisories* , Office of Water, Washington, DC, October).

[f] US Environmental Protection Agency (EPA), 2009, *2009 Edition of the Drinking Water Standards and Health Advisories* , Office of Water, October.

APPENDIX B

ProUCL OUTPUT FOR UPPER CONFIDENCE LIMITS ON THE MEAN

Appendix B

Surface Soil UCL Data Input
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Sample Location	Sample Number	Beryllium	D_Beryllium	Lead	D_Lead	Thallium	D_Thallium	Benzo(a)pyrene	D_Benzo(a)pyrene	Benzo(b)fluoranthene	D_Benzo(b)fluoranthene	Aroclor 1016
PH2SO01	4010	1.2	1	32.2	1	1.9	1	0.34	1	0.16	1	0.027
PH2SO02	4030	1.1	1	15.6	1	2.9	1	0.51	0	0.51	0	0.0255
PH2SO03	4050	1.2	1	25.4	1	1.7	0	0.198	1	0.57	0	0.0285
PH2SO04	4070	0.83	1	14.8	1	1.3	0	0.42	0	0.42	0	0.021
PH2SO05	4090	1.4	1	18.8	1	1.9	1	0.53	0	0.53	0	0.0265
PH2SO06	4110	1	1	15.6	1	4.4	1	0.43	0	0.43	0	0.0215
PH2SO07	4130	1.6	1	19.1	1	1.7	0	0.56	0	0.56	0	0.028
PH2SO08	4150	0.73	1	17.6	1	1.3	0	0.44	0	0.44	0	0.022
PH2SO09	4170	0.96	1	21.8	1	1.4	0	0.47	0	0.47	0	0.0235
PH2SO10	4190	1.2	1	25.5	1	5	1	0.59	0	0.59	0	0.0295
PH2SO11	4210	1.3	1	22.6	1	3.9	1	0.54	0	0.54	0	0.027
PH2SO12	4230	1.2	1	11.7	1	8.5	1	0.5	0	0.5	0	0.025
ASH PIT 2-SB01	AP0100	4.74	1	50.7	1	0.425	1	0.1688	1	0.13	1	0.0206
ASH PIT 2-SB02	AP0103	15.3	1	22.5	1	0.416	1	0.499	0	0.499	0	0.525
ASH PIT 2-SB03	AP0108	6.46	1	27.7	1	0.47	1	0.468	0	0.468	0	0.0234
ASH PIT 2-SB04	AP0111	16.3	1	10.1	1	0.468	1	0.442	0	0.442	0	0.02225
ASH PIT 2-SB05	AP0114	15.5	1	27.4	1	0.939	1	0.488	0	0.488	0	0.02405
ASH PIT 2-SB06	AP0117	12.1	1	12.5	1	0.312	1	0.417	0	0.417	0	0.0206
ASH PIT 2-SB07	AP0121AP0120	6.21	1	12.65	1	0.4585	0	0.4045	0	0.4045	0	0.020225
ASH PIT 2-SB08	AP0125	16.9	1	4.64	1	0.308	1	0.432	0	0.432	0	0.0212

Appendix B

Surface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

General UCL Statistics for Surface Soil

User Selected Options

From File	C:\Documents and Settings\debbi.freer\My Documents\PBOW\UCLss input.wst
Full Precision	OFF
Confidence Coefficient	95%
Number of Bootstrap Operations	2000

Beryllium

General Statistics

Number of Valid Observations	20	Number of Distinct Observations	17

Raw Statistics		Log-transformed Statistics	
Minimum	0.73	Minimum of Log Data	-0.315
Maximum	16.9	Maximum of Log Data	2.827
Mean	5.362	Mean of log Data	1.009
Median	1.35	SD of log Data	1.181
SD	6.14		
Coefficient of Variation	1.145		
Skewness	1.078		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.717	Shapiro Wilk Test Statistic	0.809
Shapiro Wilk Critical Value	0.905	Shapiro Wilk Critical Value	0.905
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	7.735	95% H-UCL	12.11
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	12.13
95% Adjusted-CLT UCL	7.973	97.5% Chebyshev (MVUE) UCL	15.12
95% Modified-t UCL	7.791	99% Chebyshev (MVUE) UCL	20.99

Gamma Distribution Test		Data Distribution
k star (bias corrected)	0.777	Data do not follow a Discernable Distribution (0.05)
Theta Star	6.897	
MLE of Mean	5.362	
MLE of Standard Deviation	6.081	
nu star	31.09	
Approximate Chi Square Value (.05)	19.35	**Nonparametric Statistics**
Adjusted Level of Significance	0.038	95% CLT UCL 7.62
Adjusted Chi Square Value	18.62	95% Jackknife UCL 7.735
		95% Standard Bootstrap UCL 7.556
Anderson-Darling Test Statistic	1.944	95% Bootstrap-t UCL 8.233
Anderson-Darling 5% Critical Value	0.774	95% Hall's Bootstrap UCL 7.496
Kolmogorov-Smirnov Test Statistic	0.312	95% Percentile Bootstrap UCL 7.664
Kolmogorov-Smirnov 5% Critical Value	0.2	95% BCA Bootstrap UCL 7.992
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 11.35
		97.5% Chebyshev(Mean, Sd) UCL 13.94
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 19.02
95% Approximate Gamma UCL	8.613	
95% Adjusted Gamma UCL	8.952	

Potential UCL to Use	Use 99% Chebyshev (Mean, Sd) UCL 19.02

Recommended UCL exceeds the maximum observation

Thallium

General Statistics

Appendix B

Surface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Number of Valid Observations 20

Number of Distinct Observations 17

Raw Statistics

Minimum	0.308
Maximum	8.5
Mean	1.985
Median	1.35
SD	2.076
Coefficient of Variation	1.046
Skewness	1.937

Log-transformed Statistics

Minimum of Log Data	-1.178
Maximum of Log Data	2.14
Mean of log Data	0.222
SD of log Data	1

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.772
Shapiro Wilk Critical Value	0.905

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.938
Shapiro Wilk Critical Value	0.905

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	2.787

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	2.963
95% Modified-t UCL	2.821

Assuming Lognormal Distribution

95% H-UCL	3.768
95% Chebyshev (MVUE) UCL	4.155
97.5% Chebyshev (MVUE) UCL	5.093
99% Chebyshev (MVUE) UCL	6.935

Gamma Distribution Test

k star (bias corrected)	1.068
Theta Star	1.858
MLE of Mean	1.985
MLE of Standard Deviation	1.921
nu star	42.72
Approximate Chi Square Value (.05)	28.73
Adjusted Level of Significance	0.038
Adjusted Chi Square Value	27.83
Anderson-Darling Test Statistic	0.575
Anderson-Darling 5% Critical Value	0.764
Kolmogorov-Smirnov Test Statistic	0.181
Kolmogorov-Smirnov 5% Critical Value	0.199

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	2.951
95% Adjusted Gamma UCL	3.047

Nonparametric Statistics

95% CLT UCL	2.748
95% Jackknife UCL	2.787
95% Standard Bootstrap UCL	2.718
95% Bootstrap-t UCL	3.16
95% Hall's Bootstrap UCL	3.296
95% Percentile Bootstrap UCL	2.823
95% BCA Bootstrap UCL	2.918
95% Chebyshev(Mean, Sd) UCL	4.008
97.5% Chebyshev(Mean, Sd) UCL	4.883
99% Chebyshev(Mean, Sd) UCL	6.603

Potential UCL to Use

Use 95% Approximate Gamma UCL 2.951

Appendix B

Surface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Aroclor 1016

General Statistics

Number of Valid Observations	20	Number of Distinct Observations	18

Raw Statistics		Log-transformed Statistics	
Minimum	0.0202	Minimum of Log Data	-3.901
Maximum	0.525	Maximum of Log Data	-0.644
Mean	0.0491	Mean of log Data	-3.58
Median	0.0238	SD of log Data	0.701
SD	0.112		
Coefficient of Variation	2.281		
Skewness	4.467		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.257	Shapiro Wilk Test Statistic	0.388
Shapiro Wilk Critical Value	0.905	Shapiro Wilk Critical Value	0.905
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	0.0924	95% H-UCL	0.0511
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	0.0608
95% Adjusted-CLT UCL	0.117	97.5% Chebyshev (MVUE) UCL	0.0719
95% Modified-t UCL	0.0966	99% Chebyshev (MVUE) UCL	0.0936

Gamma Distribution Test		Data Distribution
k star (bias corrected)	0.898	Data do not follow a Discernable Distribution (0.05)
Theta Star	0.0547	
MLE of Mean	0.0491	
MLE of Standard Deviation	0.0518	
nu star	35.94	
Approximate Chi Square Value (.05)	23.22	**Nonparametric Statistics**
Adjusted Level of Significance	0.038	95% CLT UCL 0.0903
Adjusted Chi Square Value	22.41	95% Jackknife UCL 0.0924
		95% Standard Bootstrap UCL 0.0889
Anderson-Darling Test Statistic	6.027	95% Bootstrap-t UCL 1.146
Anderson-Darling 5% Critical Value	0.768	95% Hall's Bootstrap UCL 0.406
Kolmogorov-Smirnov Test Statistic	0.501	95% Percentile Bootstrap UCL 0.0992
Kolmogorov-Smirnov 5% Critical Value	0.199	95% BCA Bootstrap UCL 0.125
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 0.158
		97.5% Chebyshev(Mean, Sd) UCL 0.206
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 0.298
95% Approximate Gamma UCL	0.076	
95% Adjusted Gamma UCL	0.0788	

Potential UCL to Use		Use 95% Chebyshev (Mean, Sd) UCL	0.158

Appendix B

Surface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Benzo(a)pyrene

General Statistics

Number of Valid Observations	20	Number of Distinct Observations	20

Raw Statistics		Log-transformed Statistics	
Minimum	0.169	Minimum of Log Data	-1.779
Maximum	0.59	Maximum of Log Data	-0.528
Mean	0.442	Mean of log Data	-0.855
Median	0.455	SD of log Data	0.317
SD	0.106		
Coefficient of Variation	0.241		
Skewness	-1.404		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.867	Shapiro Wilk Test Statistic	0.738
Shapiro Wilk Critical Value	0.905	Shapiro Wilk Critical Value	0.905
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	0.484	95% H-UCL	0.512
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	0.586
95% Adjusted-CLT UCL	0.474	97.5% Chebyshev (MVUE) UCL	0.646
95% Modified-t UCL	0.482	99% Chebyshev (MVUE) UCL	0.765

Gamma Distribution Test		Data Distribution
k star (bias corrected)	11	Data do not follow a Discernable Distribution (0.05)
Theta Star	0.0402	
MLE of Mean	0.442	
MLE of Standard Deviation	0.133	
nu star	440.1	
Approximate Chi Square Value (.05)	392.5	**Nonparametric Statistics**
Adjusted Level of Significance	0.038	95% CLT UCL 0.482
Adjusted Chi Square Value	388.9	95% Jackknife UCL 0.484
		95% Standard Bootstrap UCL 0.479
Anderson-Darling Test Statistic	1.594	95% Bootstrap-t UCL 0.475
Anderson-Darling 5% Critical Value	0.742	95% Hall's Bootstrap UCL 0.476
Kolmogorov-Smirnov Test Statistic	0.262	95% Percentile Bootstrap UCL 0.479
Kolmogorov-Smirnov 5% Critical Value	0.194	95% BCA Bootstrap UCL 0.473
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 0.546
		97.5% Chebyshev(Mean, Sd) UCL 0.591
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 0.679
95% Approximate Gamma UCL	0.496	
95% Adjusted Gamma UCL	0.501	

Potential UCL to Use		Use 95% Student's-t UCL	0.484
		or 95% Modified-t UCL	0.482

Appendix B

Surface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Benzo(b)fluoranthene

General Statistics

Number of Valid Observations	20	Number of Distinct Observations	20

Raw Statistics		Log-transformed Statistics	
Minimum	0.13	Minimum of Log Data	-2.04
Maximum	0.59	Maximum of Log Data	-0.528
Mean	0.45	Mean of log Data	-0.853
Median	0.469	SD of log Data	0.388
SD	0.118		
Coefficient of Variation	0.261		
Skewness	-1.79		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.796	Shapiro Wilk Test Statistic	0.638
Shapiro Wilk Critical Value	0.905	Shapiro Wilk Critical Value	0.905
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	0.495	95% H-UCL	0.545
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	0.635
95% Adjusted-CLT UCL	0.482	97.5% Chebyshev (MVUE) UCL	0.712
95% Modified-t UCL	0.494	99% Chebyshev (MVUE) UCL	0.862

Gamma Distribution Test		Data Distribution
k star (bias corrected)	8.001	Data do not follow a Discernable Distribution (0.05)
Theta Star	0.0562	
MLE of Mean	0.45	
MLE of Standard Deviation	0.159	
nu star	320	
Approximate Chi Square Value (.05)	279.6	**Nonparametric Statistics**
Adjusted Level of Significance	0.038	95% CLT UCL 0.493
Adjusted Chi Square Value	276.6	95% Jackknife UCL 0.495
		95% Standard Bootstrap UCL 0.491
Anderson-Darling Test Statistic	2.357	95% Bootstrap-t UCL 0.486
Anderson-Darling 5% Critical Value	0.742	95% Hall's Bootstrap UCL 0.485
Kolmogorov-Smirnov Test Statistic	0.317	95% Percentile Bootstrap UCL 0.488
Kolmogorov-Smirnov 5% Critical Value	0.194	95% BCA Bootstrap UCL 0.483
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 0.565
		97.5% Chebyshev(Mean, Sd) UCL 0.614
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 0.712
95% Approximate Gamma UCL	0.515	
95% Adjusted Gamma UCL	0.521	

Potential UCL to Use		Use 95% Student's-t UCL	0.495
		or 95% Modified-t UCL	0.494

Appendix B

Subsurface Soil UCL Data Input
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

		Thallium	D_Thallium
ASH PIT 2-SB01	AP0101	0.458	0
ASH PIT 2-SB01	AP0102	0.423	1
ASH PIT 2-SB02	AP0107	0.343	1
ASH PIT 2-SB03	AP0109	0.35	1
ASH PIT 2-SB03	AP0110	0.336	1
ASH PIT 2-SB04	AP0112	0.541	0
ASH PIT 2-SB04	AP0113	0.315	1
ASH PIT 2-SB05	AP0115	0.482	0
ASH PIT 2-SB05	AP0116	0.347	1
ASH PIT 2-SB06	AP0118	0.497	0
ASH PIT 2-SB06	AP0119	0.293	1
ASH PIT 2-SB07	AP0123	0.439	0
ASH PIT 2-SB07	AP0124	0.316	1
ASH PIT 2-SB08	AP0126	0.444	0
PH2SO01	4020	1.1	0
PH2SO02	4040	1.2	0
PH2SO03	4060	1.3	1
PH2SO05	4100	1.1	0
PH2SO06	4120	1.3	0
PH2SO07	4140	1.1	0
PH2SO09	4180	1.2	0
PH2SO10	4200	2	1
PH2SO11	4220	2.4	1
PH2SO12	4240	1.4	1
ASH PIT 2-SB02	AP0104	0.3	1
ASH PIT 2-SB08	AP0127AP0128	0.5015	0
PH2SO04	40804081	1.2	0
PH2SO08	4160	1.5	1

Appendix B

Subsurface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

General UCL Statistics for Subsurface Soil

User Selected Options	
From File	C:\Documents and Settings\debbi.freer\My Documents\PBOW\UCLsbs input.wst
Full Precision	OFF
Confidence Coefficient	95%
lumber of Bootstrap Operations	2000

Thallium

General Statistics

Number of Valid Data	28	Number of Detected Data	14
Number of Distinct Detected Data	14	Number of Non-Detect Data	14
		Percent Non-Detects	50.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.293	Minimum Detected	-1.228
Maximum Detected	2.4	Maximum Detected	0.875
Mean of Detected	0.83	Mean of Detected	-0.521
SD of Detected	0.736	SD of Detected	0.818
Minimum Non-Detect	0.439	Minimum Non-Detect	-0.823
Maximum Non-Detect	1.3	Maximum Non-Detect	0.262

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	23
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	5
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	82.14%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.74	Shapiro Wilk Test Statistic	0.758
5% Shapiro Wilk Critical Value	0.874	5% Shapiro Wilk Critical Value	0.874
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.622	Mean	-0.752
SD	0.567	SD	0.695
95% DL/2 (t) UCL	0.804	95% H-Stat (DL/2) UCL	0.843
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	0.538	Mean in Log Scale	-0.726
SD	0.819	SD in Log Scale	0.625
95% MLE (t) UCL	0.802	Mean in Original Scale	0.617
95% MLE (Tiku) UCL	1.236	SD in Original Scale	0.558
		95% Percentile Bootstrap UCL	0.795
		95% BCA Bootstrap UCL	0.839

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Subsurface Soil UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	1.336
Theta Star	0.622
nu star	37.4
A-D Test Statistic	1.714
5% A-D Critical Value	0.749
K-S Test Statistic	0.749
5% K-S Critical Value	0.232

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	0.293
Maximum	2.4
Mean	0.831
Median	0.833
SD	0.511
k star	2.823
Theta star	0.294
Nu star	158.1
AppChi2	130
95% Gamma Approximate UCL	1.01
95% Adjusted Gamma UCL	1.023

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	0.583
SD	0.56
SE of Mean	0.11
95% KM (t) UCL	0.77
95% KM (z) UCL	0.764
95% KM (jackknife) UCL	0.767
95% KM (bootstrap t) UCL	0.854
95% KM (BCA) UCL	0.763
95% KM (Percentile Bootstrap) UCL	0.777
95% KM (Chebyshev) UCL	1.063
97.5% KM (Chebyshev) UCL	1.27
99% KM (Chebyshev) UCL	1.678

Potential UCLs to Use

95% KM (t) UCL	0.77
95% KM (% Bootstrap) UCL	0.777

Appendix B

Sediment UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Sample Location	Sample Number	Arsenic	D_Arsenic	Chromium	D_Chromium
ASH PIT 2-SD04	AP1003AP1004	8.55	1	11.65	1
ASH PIT 2-SD01	AP1000	4.1	1	6.4	1
ASH PIT 2-SD02	AP1001	5.1	1	10.5	1
ASH PIT 2-SD03	AP1002	10.1	1	8.6	1
ASH PIT 2-SD05	AP1006	3.9	1	7.7	1

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Sediment UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

General UCL Statistics for Sediment

User Selected Options

From File	C:\Documents and Settings\debbi.freer\My Documents\PBOW\UCL input.wst
Full Precision	OFF
Confidence Coefficient	95%
Number of Bootstrap Operations	2000

Arsenic

General Statistics

Number of Valid Observations	5	Number of Distinct Observations	5

Raw Statistics		**Log-transformed Statistics**	
Minimum	3.9	Minimum of Log Data	1.361
Maximum	10.1	Maximum of Log Data	2.313
Mean	6.35	Mean of log Data	1.772
Median	5.1	SD of log Data	0.433
SD	2.808		
Coefficient of Variation	0.442		
Skewness	0.665		

Warning: A sample size of 'n' = 5 may not adequate enough to compute meaningful and reliable test statistics and estimates!

It is suggested to collect at least 8 to 10 observations using these statistical methods!
If possible compute and collect Data Quality Objectives (DQO) based sample size and analytical results.

Warning: There are only 5 Values in this data
Note: It should be noted that even though bootstrap methods may be performed on this data set,
the resulting calculations may not be reliable enough to draw conclusions

The literature suggests to use bootstrap methods on data sets having more than 10-15 observations.

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.856	Shapiro Wilk Test Statistic	0.872
Shapiro Wilk Critical Value	0.762	Shapiro Wilk Critical Value	0.762
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	9.027	95% H-UCL	11.71
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	11.65
95% Adjusted-CLT UCL	8.814	97.5% Chebyshev (MVUE) UCL	13.95
95% Modified-t UCL	9.089	99% Chebyshev (MVUE) UCL	18.47

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Sediment UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Gamma Distribution Test

k star (bias corrected)	2.812
Theta Star	2.258
MLE of Mean	6.35
MLE of Standard Deviation	3.787
nu star	28.12
Approximate Chi Square Value (.05)	17.02
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	13.39
Anderson-Darling Test Statistic	0.451
Anderson-Darling 5% Critical Value	0.68
Kolmogorov-Smirnov Test Statistic	0.261
Kolmogorov-Smirnov 5% Critical Value	0.358

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	10.49
95% Adjusted Gamma UCL	13.33

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	8.415
95% Jackknife UCL	9.027
95% Standard Bootstrap UCL	8.232
95% Bootstrap-t UCL	15.35
95% Hall's Bootstrap UCL	26.06
95% Percentile Bootstrap UCL	8.48
95% BCA Bootstrap UCL	8.28
95% Chebyshev(Mean, Sd) UCL	11.82
97.5% Chebyshev(Mean, Sd) UCL	14.19
99% Chebyshev(Mean, Sd) UCL	18.84

Potential UCL to Use

Use 95% Student's-t UCL 9.027

Chromium

General Statistics

Number of Valid Observations	5	Number of Distinct Observations	5

Raw Statistics		Log-transformed Statistics	
Minimum	6.4	Minimum of Log Data	1.856
Maximum	11.65	Maximum of Log Data	2.455
Mean	8.97	Mean of log Data	2.171
Median	8.6	SD of log Data	0.24
SD	2.114		
Coefficient of Variation	0.236		
Skewness	0.166		

Warning: A sample size of 'n' = 5 may not adequate enough to compute meaningful and reliable test statistics and estimates!

It is suggested to collect at least 8 to 10 observations using these statistical methods!
If possible compute and collect Data Quality Objectives (DQO) based sample size and analytical results.

Warning: There are only 5 Values in this data
Note: It should be noted that even though bootstrap methods may be performed on this data set,
the resulting calculations may not be reliable enough to draw conclusions

The literature suggests to use bootstrap methods on data sets having more than 10-15 observations.

Appendix B

Sediment UCL Data Output
Powerhouse 2 Ash Pits
former Plum Brook Ordnance Works
Sandusky, Ohio

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.97
Shapiro Wilk Critical Value	0.762

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.974
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	10.99

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	10.6
95% Modified-t UCL	11

Assuming Lognormal Distribution

95% H-UCL	11.85
95% Chebyshev (MVUE) UCL	13.15
97.5% Chebyshev (MVUE) UCL	14.96
99% Chebyshev (MVUE) UCL	18.52

Gamma Distribution Test

k star (bias corrected)	9.013
Theta Star	0.995
MLE of Mean	8.97
MLE of Standard Deviation	2.988
nu star	90.13
Approximate Chi Square Value (.05)	69.24
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	61.28
Anderson-Darling Test Statistic	0.209
Anderson-Darling 5% Critical Value	0.679
Kolmogorov-Smirnov Test Statistic	0.197
Kolmogorov-Smirnov 5% Critical Value	0.357

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	11.68
95% Adjusted Gamma UCL	13.19

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	10.53
95% Jackknife UCL	10.99
95% Standard Bootstrap UCL	10.36
95% Bootstrap-t UCL	11.86
95% Hall's Bootstrap UCL	12.22
95% Percentile Bootstrap UCL	10.4
95% BCA Bootstrap UCL	10.35
95% Chebyshev(Mean, Sd) UCL	13.09
97.5% Chebyshev(Mean, Sd) UCL	14.87
99% Chebyshev(Mean, Sd) UCL	18.38

Potential UCL to Use

Use 95% Student's-t UCL 10.99

APPENDIX C

TOXICOLOGICAL PROFILES

Introduction to the Toxicological Profiles

1.0 Purpose and Use of the Toxicological Profiles

Human health toxicological profiles are presented for each chemical of potential concern (COPC), each chemical for which documentation of chemical-specific values is required or about which there are chemical-specific issues that require resolution. The toxicological profiles provide documentation of the chemical-specific physical properties and toxicity values used in the risk assessment (RA). They also discuss the identity, classification and uses of the chemical, the common sources of release to the environment and the fate of the chemical in the environment, including the relevance of aquatic and terrestrial food-chain pathways to human health. A brief review of toxicokinetics discusses absorption, distribution, metabolism or biotransformation, excretion of the parent compound and metabolites, and mechanism of toxicity including identifying the ultimate toxicant – i.e., the moiety (parent chemical or metabolite) identified as most likely responsible for the adverse effects associated with the chemical. This information may be helpful to determine whether laboratory animals may serve as appropriate models for toxicity to humans, and to clarify the nature of toxicological interactions with other chemicals. A discussion of dermal exposure provides perspective on the significance of dermal uptake, and documents the development of dermal toxicity values and the extent or rate of dermal uptake from soil or water according to the latest USEPA (2004) guidance.

The noncancer effects evaluation and carcinogenicity evaluation provide an overall perspective on the nature of the adverse effects associated with the chemical, as well as documentation of the toxicity values. Chemical-specific issues or controversies that may influence the interpretation or application of the results of the RA are also discussed herein. The toxicity values used in the RA are summarized in a table.

The overall purpose of the toxicological profiles is to provide perspective on characteristics of each of the chemicals included in the RA so that the numerical risk estimates can be interpreted and applied wisely in the management of the site.

2.0 Chemical Identity

The identity of the chemical or chemical class is given in the title of each profile; additional information or clarification is provided in the *Introduction and Physical Properties* Section as required. The hyphenated number in parentheses following the chemical name is its unique Chemical Abstract Service (CAS) registration number. The CAS number may be located in the profile title, text, or in the case of multiple isomers or members of a chemical class, in the table that provides the physical properties.

3.0 Physical Properties

The toxicological profiles also provide documentation for the physical properties or constants that are important for chemical transport modeling, such as molecular weight (MW) in grams per mole (g/mole), the log of the octanol/water partition coefficient (log K_{ow}), Henry's Law constant (H) in atmosphere-cubic meters per mole (atm-m^3/mole), the soil/water partition coefficient (K_d) in liters per kilogram (L/kg) for metals, the log of the soil/organic carbon partition coefficient (log K_{oc}) (unitless) for organic chemicals, diffusivity in air (D_a) in square centimeters per second (cm^2/s), diffusivity in water (D_w) in cm^2/s, vapor pressure (VP) in atmospheres (atm), solubility in water (S) in milligrams per liter (mg/L), and, for volatile organic compounds (VOC), boiling

point of pure compound (T_b) in degrees Kelvin (°K), critical temperature (T_c) in °K, and enthalpy of vaporization at the boiling point ($\Delta H_{v,b}$) in calories per mole (cal/mol). Organic chemicals are designated as VOCs or semivolatile organic compounds (SVOC) based on their propensity to volatilize from environmental media. Chemicals designated as VOCs generally have an H greater than 1E-5 atm-m^3/mole (EPA, 2002).

The physical constants generally are taken from the most reliable source (i.e., the source that provides the highest level of documentation). Values for interrelated properties are usually taken from the same source (e.g., H is often estimated from VP and S; therefore, the same source is generally used for all three property values). When one source provides several values for a given property, professional judgment is used to select the most appropriate. Obvious outliers may be dropped from consideration. The average or the midpoint of a range of values may be selected. K_d values for metals and K_{oc} values for ionizing organic compound are based on a default pH of 6.8 (EPA, 2002) if the data are available. VP, S and H values are limited to those provided for normal ambient temperatures (0 to 30 °C) and the reference temperature is provided.

When values for H are not located, they are calculated as follows, provided the requisite information is available (EPA, 1998):

$$H = \frac{VP \bullet MW}{S}$$

where:

H	=	Henry's law constant (atm-m^3/mole, calculated)
VP	=	vapor pressure (atm)
MW	=	molecular weight (g/mole)
S	=	solubility in water (mg/L).

When values for K_{oc} are not located, they are calculated as follows for phthalates and polyaromatic aromatic hydrocarbons (PAH) provided log K_{ow} is available (EPA, 1998):

$$\log K_{oc} = 0.97 \log K_{ow} - 0.094$$

where:

K_{oc}	=	soil/organic carbon partition coefficient (unitless, calculated)
K_{ow}	=	octanol/water partition coefficient (unitless).

K_{oc} for other organic chemicals is calculated as follows provided log K_{ow} is available (EPA, 1998):

$$\log K_{oc} = 0.78 \log K_{ow} + 0.151$$

where:

K_{oc} = soil/organic carbon partition coefficient (unitless, calculated)
K_{ow} = octanol/water partition coefficient (unitless).

When values for D_a are not located, they are calculated as follows (EPA, 1998):

$$D_a = \frac{1.9}{MW^{2/3}}$$

where:

D_a = diffusivity in air (cm^2/s, calculated)
MW = molecular weight (g/mole).

When values for D_w are not located, they are calculated as follows (EPA, 1998):

$$D_w = \frac{22E{-}5}{MW^{2/3}}$$

where:

D_w = diffusivity in water (cm^2/s, calculated)
MW = molecular weight (g/mole).

Values for T_b, T_c and $\Delta H_{v,b}$ for VOCs are preferentially taken from EPA (2004), unless there is evidence that values provided by other sources (HSDB, 2010; NIST, 2010) are more reliable. If values are not located for these variables, they may be estimated by the most appropriate technique for the specific chemical or class to which it belongs as recommended by Lyman et al. (1990). The estimation method used is identified by footnote in the table of physical properties.

4.0 Environmental Release, Fate and Transport

The toxicological profiles also present the predominant sources of release to the environment as well as a qualitative or semi-quantitative description of the fate and transport of the chemical in air, water, sediment, and soil. No attempt is made to present detailed quantitative data because environmental fate is usually highly dependent on climatic conditions and the characteristics of the medium of interest, both of which may differ from location to location and change from time to time. The source and fate information may provide perspective regarding the likelihood that the chemical's presence is related to site activities, that the chemical will migrate across media, or that the chemical will persist at toxicologically significant levels.

Biotransfer factors are provided for chemicals for which food-chain pathways may be significant, which includes a few inorganic chemicals and those organic chemicals that are highly lipophilic, persist in the environment, and are resistant to metabolism by lower trophic organisms in the food chain. High lipophilicity is indicated by a log K_{ow} greater than 3 (Lyman et al., 1990). Lipophilicity enhances partitioning to biomedia and passage across biological

membranes. Persistence in the environment is evidence that the chemical might resist biotransformation and, therefore, remain in edible tissues at toxicologically significant concentrations. Toxicokinetic data, when available, clarify the potential for biotransfer. Biotransformation products may be more toxic than the parent compound, but they tend to be short-lived or more quickly removed from the body, reducing the likelihood of significant bioaccumulation. Similarly, VOCs tend to be mobile and labile (i.e., subject to rapid and extensive biotransformation and excretion), and generally do not participate significantly in food-chain pathways. Therefore, biotransfer factors are not estimated for VOCs, with few exceptions. Some SVOCs, however, are highly lipophilic and may persist. Biotransfer factors generally are not estimated unless empirical data suggest that participation in food-chain pathways is likely to be significant.

The biotransfer factors of interest are water-to-fish bioconcentration factors (BCF) or bioaccumulation factors (BAF), or biota-sediment accumulation factors (BSAF), soil-to-plant biotransfer factors (Bp), cattle ingestion-to-beef factors (Bb) and cattle ingestion-to-milk factors (Bm). Separate soil-to-plant biotransfer factors are available for the reproductive parts of plants (e.g., fruits, seeds) and the vegetative parts of plants (e.g., stems, leaves) for inorganic and some organic chemicals. Soil-to-plant factors for the reproductive parts of plants are designated Bpr; soil-to-plant factors for the vegetative parts of plants are designated Bpv.

Most chemicals in surface water bodies remain predominantly in the dissolved phase in the water column or partition to sediment, which generally settles to the bottom. The difference in concentration between surface water and sediment is usually upwards of an order of magnitude. Theoretically it is appropriate to evaluate biotransfer from both media, using BCF to quantify uptake from water and BSAF to quantify uptake from sediment. Quantification of biotransfer, however, is accompanied by considerable uncertainty and variability because field conditions cannot be readily duplicated in the laboratory where BCF values are generally measured. Therefore, the biotransfer models for either medium are intentionally designed to be very conservative to ensure protection of human health. Consequently, evaluating biotransfer from both water and sediment captures the conservatism of both models and is likely to grossly overestimate total biotransfer. Therefore, most chemicals with the potential to participate significantly in aquatic food chain pathways are evaluated for bioconcentration from surface water *or* bioaccumulation from sediment, depending on which pathway is expected to predominate, but not both.

BCF values are adopted from empirical data when the data are clearly the best choice (i.e., similar results in multiple species of fin fish). When the empirical data are few or inconsistent, BCFs for inorganic chemicals are taken from various sources. BCF values for organic chemicals are generally estimated from the Bintein and Devillers model (Devillers et al., 1996):

$$\log BCF = 0.910 \log K_{ow} - 1.975 \log(6.8E{-}7 K_{ow} + 1) - 0.786$$

where:

BCF = bioconcentration factor (L/kg, calculated)
K_{ow} = octanol/water partition coefficient (unitless).

The Bintein and Devillers model is selected over simpler models adopted by the EPA largely because the Bintein and Devillers approach has undergone scrutiny since first published (Bintein and Devillers, 1993), and considerable effort was expended to validate the model; i.e., to compare modeled and empirical results. The Bintein and Devillers model is probably more realistic than simpler models, particularly for chemicals with higher log K_{ow} values. BSAF values are chosen instead of BCFs for those very highly lipophilic chemicals such as the polychlorinated biphenyls (PCB), polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) and selected organochlorine pesticides expected to partition almost entirely to benthic sediment.

Soil-to-plant biotransfer factors (Bp) for SVOCs are estimated by a simple model by Travis and Arms (1988), which is based only on log K_{ow}:

$$\log Bp = 1.588 - 0.578 \log K_{ow}$$

where:

Bp = soil-to-plant biotransfer factor (unitless, calculated)
K_{ow} = octanol/water partition coefficient (unitless).

Separate biotransfer factors are not estimated for the reproductive parts of plants (Bpr) and the vegetative parts of plants (Bpv) by the Travis and Arms model.

Cattle ingestion-to-beef factors (Bb) for SVOCs are estimated by a simple model by Travis and Arms (1988), which is based only on log K_{ow}:

$$\log Bb = -7.6 + \log K_{ow}$$

where:

Bb = cattle ingestion-to-beef biotransfer factor (days/kg, calculated)
K_{ow} = octanol/water partition coefficient (unitless).

Cattle ingestion-to-milk factors (Bm) for SVOCs are estimated by a simple model by Travis and Arms (1988), which is based only on log K_{ow}:

$$\log Bm = -8.10 + \log K_{ow}$$

where:

Bm = cattle ingestion-to-milk biotransfer factor (days/kg, calculated)
K_{ow} = octanol/water partition coefficient (unitless).

It should be understood that the biotransfer models described above depend only on one or two physical property values that are related largely to the propensity for transfer across biological membranes. Many compounds have relatively large log K_{ow} values, indicating ready passage across membranes; however, they are sufficiently volatile, or are otherwise mobile or labile so

they are unable to persist in biological tissues. For example, many constituents with high log K_{ow} values are efficiently metabolized by lower trophic organisms in the food chain so that significant human exposure does not occur. Such is the case for practically all VOCs and most SVOCs; relatively few compounds participate significantly in food-chain pathways. Therefore, biotransfer is evaluated as a potential pathway only for those chemicals with empirical data that indicate food-chain exposure could be significant.

5.0 Toxicokinetics

A toxicokinetics section was added to the toxicological profiles in early 2004. Toxicokinetics describes the uptake or absorption of the chemical from contact media, the distribution of the chemical (or its metabolites) within the body following absorption, the metabolism or biotransformation of the chemical, and the mechanisms of excretion of the parent compound and its biotransformation products from the body. The toxicokinetics section documents the gastrointestinal (GI) absorption factor (GAF) required to develop toxicity factors for dermal exposure, because dermal risk characterization depends on an absorbed dose rather than an exposure dose. The toxicokinetics section also identifies the compounds that are rapidly metabolized or eliminated, which justifies excluding their evaluation in food-chain pathways even though they may have a large K_{ow} that indicates ready passage across cell membranes. This section also tries to identify the ultimate toxicant and elucidate the most important mechanism(s) of toxicity.

6.0 Dermal Exposure

The toxicological profiles provide the documentation for the GAF, which is used to develop the dermal toxicity values. The toxicological profiles also provide documentation for the dermal absorption factor (ABS), which describes the extent of dermal uptake from soil, and the time for dermal uptake to reach steady state (t*), the permeability coefficient (K_p), the lag time for chemical to cross the stratum corneum (τ), the fraction absorbed (FA), and the ratio of the permeability coefficient for passage across the stratum corneum to the permeability coefficient for passage across the viable epidermis (B), all which are used to estimate the rate of dermal uptake from water. These values are taken from EPA (2010) if available. Otherwise, they are estimated by the EPA (2004) methods.

7.0 Toxicity Evaluation

The toxicological profile contains a brief description of the nature of the adverse effects associated with the chemical. It is important to note that a discussion of adverse effects without a discussion of dose is incomplete and potentially misleading, because virtually any chemical may be toxic at some dose, and many chemicals (e.g., nutritionally required minerals, vitamins, amino acids, etc.) enhance human health at some low dose. An ever growing and compelling body of evidence suggests that many environmental contaminants also enhance health at low doses (Hart and Frame, 1996).

7.1 Noncancer Evaluation

The toxicity values for noncancer effects include a reference dose (RfD) expressed in milligrams per kilogram per day (mg/kg-day) for chronic or subchronic oral exposure, and a reference concentration (RfC), in milligrams per cubic meter (mg/m^3), for chronic or subchronic inhalation exposure. The inhalation RfC in units of mg/m^3 may be converted to an equivalent inhalation RfD by assuming continuous chronic exposure of humans with a body weight of 70 kg and an

inhalation rate of 20 m^3/day (EPA, 1989, 1991). In other words, the RfC expressed as mg/m^3 is multiplied by the inhalation rate of 20 m^3/day, and the result is divided by the assumed adult body weight of 70 kg to yield an inhalation RfD expressed as mg/kg-day.

RfD and RfC values are usually derived from empirical benchmark doses (BMD) or concentrations called no-observed-effect levels (NOEL) or no-observed-adverse-effect levels (NOAEL) from animal toxicity or human epidemiology studies. If the data do not permit identifying a NOEL or NOAEL, a lowest-observed-adverse-effect level (LOAEL) or lowest-effect level (LEL) may be used. A frank-effect level (FEL), e.g., mortality, shortened life span or serious physiologic, neurologic or behavioral disturbances, is generally considered an inappropriate benchmark from which to develop an RfD or RfC. Some RfD and RfC derivations employ a BMD that is a statistically estimated dose for humans at which some low proportion of the population may experience some minimally adverse effect. A BMD at which 10 percent of the population may be expected to experience such an effect is expressed as BMD_{10}. The RfD or RfC is derived by dividing the benchmark level (e.g., NOAEL or BMD_{10}) by a series of uncertainty and modifying factors, which collectively are designated the uncertainty factor (UF).

RfD and RfC values are not currently available for acute toxicity and acute exposure is not evaluated in the RA. Nonetheless, the levels associated with acute lethality and data regarding the effects of acute exposure to levels higher than ordinarily observed in chronic environmental exposure provide additional perspective regarding the toxicity of the chemical. Therefore, information regarding acute toxicity, when available, is included in the profiles. Lethality data for laboratory animals are generally expressed as the oral dose associated with lethality of 50 percent of an exposed population (LD_{50}) or the concentration in air associated with lethality of 50 percent of an exposed population (LC_{50}). Occasionally the dose associated with lethality in a low percentage of an exposed population (LD_{LO}) is presented.

RfD and RfC values are derived for both chronic and subchronic exposure. For purposes of the RA, chronic exposure is defined as equal to or greater than 7 years, i.e., at least 10 percent of expected life span; subchronic exposure is defined as 2 weeks to 7 years.

Under the assumption of monotonicity (i.e., incidence, intensity, or severity of effects can increase, but cannot decrease, with increasing magnitude or duration of exposure), a chronic RfD may be considered sufficiently protective for subchronic exposure, but a subchronic RfD may not be protective for chronic exposure. Currently, verified subchronic RfD values are unavailable. Provisional subchronic RfDs exist for few chemicals and are compiled in the Health Effects Assessment Summary Tables (HEAST) (EPA, 1997) and some National Center for Environmental Assessment (NCEA) or Provisional Peer Reviewed Toxicity Value (PPRTV) derivations. Although once updated semi-annually, the HEAST is no longer updated, because the EPA (2005) Integrated Risk Information System (IRIS) is generally considered the source of the highest quality toxicity values. For some chemicals the PPRTV derivations are more recent and it is known that summaries on IRIS will be replaced with the PPRTV derivations. In these cases the PPRTV values will be selected preferentially to those on IRIS. Subchronic RfD and RfC values are generally obtained from EPA (1997) or more recent NCEA or PPRTV evaluations, or they may be derived *de novo* from the toxicological data set or from chronic RfD values as follows:

- If the UF applied in the derivation of the chronic RfD does not provide for expansion from subchronic to chronic exposure (e.g., if the chronic RfD was derived from a chronic study), the chronic RfD is adopted as being sufficiently protective for subchronic exposure.

- If the UF applied in the derivation of the chronic RfD contains a component to expand from subchronic to chronic exposure, the subchronic RfD is derived by multiplying the chronic RfD by the component of the UF used to expand from subchronic to chronic exposure (e.g., if a factor of 10 was used to expand from subchronic to chronic exposure, the subchronic RfD will be 10 times larger than the chronic RfD).

7.2 Carcinogenicity Evaluation

The evaluation of the potential carcinogenicity of a chemical includes both a qualitative and a quantitative aspect (EPA, 1986, 2005). EPA (2005) recognizes five weight-of-evidence group classifications for carcinogenicity. Formerly, EPA (1986) used a letter-based system to describe the weight of evidence for carcinogenicity. Reference to this former system is included because many of the carcinogenicity assessments listed on the Integrated Risk Information System (IRIS) use the former letter-based system (EPA, 2010). The five EPA weight-of-evidence classifications are as follows:

- ***Carcinogenic to Humans*** (corresponds to the former Group A - Human Carcinogen).

- ***Likely to be Carcinogenic to Humans*** (includes both the former Group B1 and Group B2-Probable Human Carcinogens)

- ***Suggestive Evidence of Carcinogenic Potential*** (corresponds to the former Group C - Possible Human Carcinogen)

- ***Inadequate Information to Assess Carcinogenic Potential*** (corresponds to the former Group D - Not Classifiable as to Human Carcinogenicity)

- ***Not Likely to be Carcinogenic to Humans*** (corresponds to the former Group E - Evidence of Noncarcinogenicity to Humans).

Toxicity values for cancer risk include a slope factor (SF) for oral exposure, expressed as the risk per mg/kg-day ingested dose, and a unit risk factor (URF) for inhalation exposure, expressed as the risk per microgram per cubic meter ($\mu g/m^3$) in ambient air. These quantitative estimates are generally provided for chemicals in EPA weight-of-evidence Groups A, B and C if the data are adequate. The SF or URF is usually estimated as an upper bound on the slope of the dose- or concentration-response curve from animal toxicity or human epidemiology studies. The inhalation URF in units of risk per $\mu g/m^3$ may be converted to an equivalent inhalation SF in units of risk per mg/kg-day by assuming continuous lifetime exposure of humans with a body weight to 70 kg and an inhalation rate of 20 m^3/day. In other words, the URF expressed as risk per $\mu g/m^3$ is divided by the inhalation rate of 20 m^3/day, and multiplied by the assumed body weight of 70 kg and a conversion factor of 1000 μg/mg.

EPA (1986, 2005) generally assumes that there are no thresholds for carcinogenic expression; therefore, any exposure represents some quantifiable risk. A few potential carcinogens are understood to require a threshold for carcinogenic expression. Such chemicals are more appropriately evaluated with an RfD developed as described above.

7.3 Hierarchy for Selecting Toxicity Data

Toxicity values generally are chosen using the following hierarchy:

- The EPA's (2010) on-line IRIS database containing toxicity values that have undergone the most rigorous Agency review.
- PPRTV derivations for the Superfund Technical Support Center (STSC); there may be individual chemicals for which the PPRTV derivations supercede those on IRIS.
- Older NCEA derivations for the STSC, HEAST (EPA, 1997), or other EPA documents or memoranda.

When EPA-derived toxicity values are not located in any of the above sources, Minimal Risk Levels (MRL) from the Agency for Toxic Substances Disease Registry (ATSDR) toxicological profiles may be adopted or adapted for use in the RA. MRLs are derived by a methodology similar to the EPA methodology for RfD derivation. ATSDR toxicological profiles generally identify levels significant to human health with particular emphasis on target organ and mechanism of toxicity. Also, the U.S. Army Center for Health Promotion and Preventive Medicine (CHPPM) has derived toxicity values for certain chemicals (and their degradation products) associated with military use. Finally, the primary literature may be surveyed to determine whether sufficient data exist to derive a toxicity value using the EPA methodology. The use of surrogate chemicals is also considered if the chemical structure, adverse effects, and toxic potency of the surrogate and chemical of interest are judged to be sufficiently similar.

GAFs, used to derive dermal RfD values and SFs from the corresponding oral toxicity values, are obtained from the following hierarchy:

- EPA (2004)
- Empirical data
- PPRTV summaries or NCEA position papers
- Federal agency reviews of the empirical data, such as ATSDR toxicological profiles and various EPA criteria documents
- Other published reviews of the empirical data.

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METALS

ARSENIC (7440-38-2)

1.0 Introduction and Physical Properties

Arsenic is a natural **metalloid**, the 20th most abundant element in the earth's crust that occurs in both inorganic and organic forms (ATSDR, 2000; HSDB, 2010). Elemental arsenic is a steel-grey material that may occur naturally. However, arsenic is usually found in the environment combined with other elements such as oxygen, chlorine, and sulfur. Most inorganic and organic arsenic compounds are non-volatile, odorless and tasteless white or colorless powders. Inorganic arsenic occurs naturally in soil and in many kinds of rock, especially in minerals and ores that contain copper or lead. Inorganic arsenic is more toxic than organic forms. Major uses of arsenic in the U.S. include its incorporation into wood preservatives and other agricultural chemicals. It is also used as a metal in various electrical devices and is alloyed with lead and copper in the manufacture of lead bullets or shot (Lewis, 1997). Arsenic is also a component of the chemical warfare agent Lewisite (Opresko et al., 1998). Lewisite is manufactured by the condensation of arsenic trichloride with acetylene in the presence of aluminum, copper or mercuric chloride (Lewis, 1997). Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
74.92[a]	NA	NA	2.9E+1[b]	NA	NA	Note 1[c]	Note 2[c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.
Note 1: Variable: inorganic arsenic compounds are not likely to volatilize; some organic arsenic compounds are low-boiling liquids or gases at normal temperatures.
Note 2: Variable: inorganic arsenic compounds range from practically insoluble to freely miscible in water; most organic arsenic compounds are not readily soluble, most arsenic acid compounds are soluble to freely miscible.
[a] Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 21 April.
[b]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***,, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.
[c]Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Arsenic***, U.S. Public Health Service, Atlanta, Georgia, September, on line.

2.0 Environmental Fate and Transport

The production of arsenic and its use in nonferrous alloys and in the manufacture of semiconductors may result in its release to the environment through various waste streams HSDB, 2010). Other important anthropogenic releases are metal smelting, coal burning, and other industrial activities. Arsenic may be present at military sites as a result of training with or demilitarization or disposal of Lewisite. Most (approximately 80 percent) of anthropogenic releases are initially to soil (ATSDR, 2000).

Arsenic occurs in the air as a combination of trivalent and pentavalent forms almost entirely adsorbed to small particles that permit dispersion over long distances (ATSDR, 2000). Residence time in the atmosphere averages approximately nine days. Removal is largely by wet and dry deposition. Arsenic in surface water can undergo a variety of reactions and exist as several different soluble compounds. Sorption to sediment is often an important removal process, but biotransformation in sediment may return soluble forms to the water. Arsenic in soil generally exists as insoluble forms sorbed to clay or organic matter or complexed with calcium

or iron. Mobility is low and leaching is not generally significant, except that increasing soil pH can dramatically increase solubility and mobility.

Arsenic has been shown to bioconcentrate in aquatic organisms, mainly at the water-algae interface (ATSDR, 2000). There is no evidence for biomagnification through the various trophic levels, nor do there appear to be significant differences between bottom-feeders and predatory fish. Arsenic is among the metals listed as being of no concern for bioaccumulation in fish (EPA, 1995). Empirical data suggest that bioconcentration through terrestrial food chain pathways is unlikely to be significant (ATSDR, 2000). Therefore, biotransfer factors are not developed for arsenic.

3.0 Toxicokinetics

All forms of inorganic arsenic are readily absorbed by the lungs and GI tract; the extent of absorption is greater for more soluble compounds than for more insoluble compounds (ATSDR, 2000). Dermal absorption has not been well characterized but is significantly less than inhalation or oral absorption. GI and dermal absorption of arsenic from soil is much lower than from aqueous solution. Distribution is generally widespread throughout the body following absorption.

Metabolism of inorganic arsenic involves reduction of As^{+5} to As^{+3}, and oxidation of As^{+3} to As^{+5} so that arsenic is present systemically as a mixture of arsenate and arsenite (ATSDR, 2000). Arsenite is readily oxidized and methylated primarily in the liver to form the organic compounds monomethyl arsonic acid and dimethyl arsinic acid, which are rapidly excreted in the urine.

4.0 Dermal Exposure

EPA (2004) concluded that a GAF should not be estimated for the purpose of adjusting oral toxicity values for dermal exposure. Therefore, the oral toxicity values described below are used directly without adjustment for evaluating dermal exposure.

EPA (2004) cites empirical data indicating that dermal uptake of arsenic from soil approximates 3 percent. The ABS of 0.03 recommended by EPA (2004) is used herein. Empirical data regarding the uptake of soluble forms of arsenic from water were not located. The EPA (2004) default K_p for inorganic chemicals of 1E-3 cm/hour is selected for arsenic.

5.0 Noncancer Effects Evaluation

Inorganic arsenic may be an essential nutrient, at least for food-producing domestic animals, exerting beneficial effects on growth, health and feed conversion efficiency (Underwood, 1977). A lethal dose of arsenic trioxide in humans is 70-180 mg, approximately 50 to 140 mg arsenic (Ishinishi et al., 1986). Acute oral exposure of humans to high doses of arsenic produces liver swelling, skin lesions, disturbed heart function and neurological effects. The only noncancer effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyper pigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (EPA, 2010). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico. EPA (2010) verified an RfD of 3E-4 mg/kg-day for chronic oral exposure, based on a NOAEL of 8E-4 mg/kg-day for hyper pigmentation and kertatosis of the skin from the Chinese data. An uncertainty factor of 3 was applied. An increased incidence of Blackfoot disease was also

observed, which may not be related to arsenic alone. Goyer (1991) describes black-foot disease as a peripheral vascular disorder manifested as acrocyanosis and Raynaud's disease, which may progress to gangrene. EPA (2010) notes that the skin is the more sensitive target organ. Nonetheless, in keeping with EPA (1989) guidance regarding selection of target organ, both the skin and peripheral vascular system are selected as target organs for prolonged oral exposure to arsenic. Confidence in the RfD is medium.

The available data do not suggest a significant difference between chronic and subchronic exposure regarding the threshold for noncancer effects. Therefore, EPA (1997) adopted the chronic oral RfD as the provisional subchronic oral RfD for arsenic.

Occupational (predominantly inhalation) exposure is also associated with neurological deficits, anemia, and vascular effects (Ishinishi et al., 1986). However, concomitant exposure to other chemicals cannot be ruled out in the occupational studies. Therefore, the data are not sufficient for estimation of an inhalation RfC.

6.0 Carcinogenicity Evaluation

Inorganic arsenic is clearly a carcinogen in humans. Inhalation exposure is associated with increased risk of lung cancer in persons employed as smelter workers, in arsenical pesticide applicators, and in a population residing near a pesticide manufacturing plant (EPA, 2010). Oral exposure to high levels in well water is associated with increased risk of skin cancer and several forms of internal cancer, although the role of other chemicals in the internal cancers is unclear. Extensive animal testing with various forms of arsenic given by many routes of exposure to several species, however, has not demonstrated the carcinogenicity of arsenic, indicating that the common laboratory animals are not good models for carcinogenicity to humans. EPA (2010) classified inorganic arsenic in cancer weight-of-evidence Group A (human carcinogen), and recommended an oral SF of 1.5E+0 per mg/kg-day, based on the incidence of skin cancer in the Chinese study. EPA (200) noted that arsenic probably functions via several different mechanisms of toxicity not including direct interaction with DNA. These appear to obey thresholds or to generate a non-linear slope that approaches 0 in the low-dose range. The SF probably exaggerates cancer risk in the low dose range associated with most environmental exposures, although the extent is unclear.

An inhalation URF of 4.3E-3 per $\mu g/m^3$, equivalent to an inhalation SF of 1.5E+1 per mg/kg-day, was derived for inorganic arsenic from the incidence of lung cancer in occupationally exposed men (EPA, 2010).

7.0 Toxicity Summary

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
3E-4	3E-4	S, PVS	ND	ND	NA	A	1.5E+0	A	4.3E-3	1.5E+1

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable. Target organ or critical effect abbreviations: S = skin; PVS = peripheral vascular system. [a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.										

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BERYLLIUM

Beryllium is a naturally occurring **metal**, which is used in its pure state or as alloys in a wide variety of electrical components, tools, optical components, precision instruments, structural components of aircraft, missiles and satellites, and other metal-fabricating applications (ATSDR, 2000; HSDB, 2010. Beryllium oxide is used in high-technology ceramics, electronic heat sinks, and several refractory applications. Relevant physical properties are compiled below:

MW (g/mole)	log K_{ow} (unitless)	H (atm-m^3/mole)	K_d (L/kg)	D_a (cm^2/s)	D_w (cm^2/s)	VP (atm)	S (mg/L)
9.01[a] (element)	NA	NA	7.9E+2[b]	NA	NA	ND	Note 1[c]

NA = not applicable, ND = no data.
Note 1: Beryllium chloride (typical soluble salt): very soluble.
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
[b]U.S. Environmental Protection Agency (EPA), 2002 ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.
[c]Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Beryllium***, U.S. Public Health Service, Atlanta, Georgia, September, on line.

Although beryllium is a naturally occurring metal, the predominant release to the atmosphere is emissions from fossil fuel combustion, particularly coal (ATSDR, 2000; HSDB, 2010). Beryllium production and use in alloys, microelectronics, aerospace technology, as a solid-propellant in rocket fuels, in aircraft brakes, X-ray windows, and neutron reflectors contribute small amounts to the environment. Beryllium exists in air as particulates that are subject to wet and dry deposition. Smaller particles may travel large distances before removal from the air.

Deposition from the atmosphere and waste disposal contributes beryllium to soil (ATSDR, 2000; HSDB, 2010). Beryllium is expected to be essentially immobile in soil regardless of pH or the presence or absence of specific soil constituents; therefore, leaching to groundwater is not expected to be significant. Beryllium in surface water tends to form insoluble beryllium hydroxide or sorb to suspended mineral solids and sediments in water; very little beryllium exists in a soluble form.

Beryllium is not expected to bioconcentrate or reach significance in aquatic or terrestrial chain pathways (ATSDR, 2000). Therefore, biotransfer factors for beryllium are not compiled. A GAF of 0.01 is chosen for this evaluation (EPA, 2004).

Data were not located regarding the extent of dermal uptake of beryllium from soil, and EPA (2004) provides no estimate of the extent of dermal absorption (ABS). EPA (2004) recommends that dermal uptake from soil should not be quantified for chemicals without ABS recommendations, but that these chemicals should be discussed qualitatively as a source of uncertainty. Empirical data regarding the uptake of soluble forms of beryllium from water were not located. The EPA (2004) default PC for inorganic chemicals of 1E-3 cm/hour is selected for beryllium.

A (172-week) dietary study in dogs identified GI lesions as the critical effect of chronic ingestion exposure to beryllium (EPA, 2010). A verified chronic oral RfD of 2E-3 mg/kg-day was based

on a BMD_{10} (statistical estimate of a dose level associated with a 10 percent dose rate) of 4.6E-1 mg/kg-day in the dog study. An uncertainty factor of 300 was applied. Confidence in the RfD is low to medium. The GI tract is the target organ for chronic oral exposure.

Occupational exposure is associated with dermatitis, acute pneumonitis and chronic pulmonary granulomatosis (Reeves, 1986), also known as berylliosis or chronic beryllium disease. Berylliosis has also been observed in humans living in the vicinity of a beryllium plant. Similar pulmonary effects were observed in laboratory animals subjected to inhalation exposure. A verified chronic inhalation RfC of 2E-2 mg/m^3 was based on a LOAEL of 2.0E-1 µg/m^3 in an occupational study, and a NOAEL of 1.0E-2 to 1.0E-1 µg/m^3 in a community exposure study (EPA, 2010). The critical endpoint is beryllium sensitization and progression to berylliosis. An uncertainty factor of 10 was used. The target organ for inhalation exposure to airborne beryllium is the lung. Confidence in the inhalation RfC is medium. The RfC is equivalent to a chronic inhalation RfD of 5.7E-6 mg/kg-day.

EPA (2010) classified beryllium in cancer weight-of-evidence Group B1 (probable human carcinogen) based on limited human (occupational) cancer data and sufficient animal data. A significant increase in lung tumors occurred in rats and in rhesus monkeys subjected to inhalation exposure or intratracheal instillation of a variety of beryllium compounds. Osteogenic sarcomas were induced in rabbits and mice, but not in rats or guinea pigs, injected intravenously with various beryllium compounds. A verified inhalation URF of 2.4E-3 per µg/m^3 equivalent to an SF of 8.4E+0 per mg/kg-day was derived from an occupational study. The B1 classification applies only to inhalation exposure. Oral studies in animals yielded inconclusive results. EPA (2010) concluded that the potential carcinogenicity of oral exposure of humans to beryllium cannot be determined.

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CHROMIUM (7440-47-3)

1.0 Introduction and Physical Properties

Chromium is a naturally occurring **metal** (ATSDR, 2000). It occurs in several valence states; chromium (III) (16065-83-1) and chromium (VI) (18540-29-9) are the forms most commonly encountered in environmental media. Chromium is used largely in the metallurgical, refractory and chemical industries. The largest amount is used in the metallurgical industry in various steels and nonferrous alloys. The second largest use is by the chemical industry in pigments, metal finishing, leather tanning and wood treatment. Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
52.00[a] (element)	NA	NA	1.8E+6 (CrIII) 1.9E+1 (CrVI)[b]	NA	NA	ND	Note 1[a]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable; ND = no data.

Note 1: Chromium compounds vary from insoluble to highly soluble.

[a]Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Toxicological Profile for Chromium***, U.S. Public Health Service, Atlanta, Georgia, September, on line.

U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.

2.0 Environmental Fate and Transport

Chromium is released into the atmosphere from natural gas, oil and coal combustion, and from use by the industries mentioned above (ATSDR, 2000). Other sources include wind transport from road dust, cement producing plants, the wearing down of asbestos brake linings from automobiles, incineration of municipal refuse and sewage sludge, exhaust emission from automotive catalytic converters, and emissions from cooling towers that use chromium compounds as rust inhibitors. Significant quantities of chromium are released to surface water from industrial use. Land disposal of chromium-containing commercial products, solid waste and slag from chromate manufacture, and coal ash, primarily from electric utilities and other operations that burn coal, are the major releases to soil.

Chromium releases from combustion processes and ore processing are generally in the form of chromium (III) oxide; however, chromium (VI) has been identified in fly ash from coal combustion at chromate manufacturing and user sites (ATSDR, 2000). Airborne chromium exists in particulate form that may travel great distances from the point of emissions; wet and dry deposition account for the majority of removal from air.

Most chromium released to surface water eventually adsorbs to particles and becomes deposited in bottom sediment, but small quantities may remain in the water in both soluble and insoluble forms (ATSDR, 2000). The soluble forms are usually chromium (VI) salts and soluble chromium (III) compounds. Chromium (VI) may be found in water, but eventually organic matter and other reducing agents will reduce chromium (VI) to chromium (III). Chromium in

soil is present mainly as insoluble chromium (III) carbonates and oxides that are unlikely to be mobile to any significant extent. However, soluble forms may also be present, depending on the form of the chemical released, that may be quite mobile in soil. Also, lower pH facilitates complexation with organic matter and increases mobility.

Empirical evidence suggests that chromium is unlikely to participate significantly in food chain pathways (ATSDR, 2000; EPA, 1995); therefore, biotransfer factors are not compiled.

3.0 Toxicokinetics

Identification of chromium in urine, serum and tissues of occupationally exposed humans confirms that soluble chromium (III) and chromium (VI) compounds are absorbed from the lung (ATSDR, 2000). The rate of absorption, however, depends largely on the physical and chemical characteristics of the chromium compound involved, as shown in numerous animal inhalation studies. Chromium (VI) compounds are more readily absorbed because the chromate anion participates in facilitated diffusion mechanisms not available to chromium (III).

Absorption of dietary sources of chromium from the GI tract in humans ranges from approximately 0.5 to 2 percent, as inferred from urinary excretion data (ATSDR, 2000). The extent of absorption appears to be inversely related to dietary chromium content. A review of several studies suggests that 0.4 to 14.5 percent of orally administered chromium not related to dietary sources is absorbed by the GI tract of humans, but valence and chemical form and nutritional status (fed vs. fasting) appear to influence efficiency of absorption (ATSDR, 2000). Chromium (VI) sources are more efficiently absorbed than chromium (III) sources as described above for inhalation exposure. Experiments with laboratory animals showed that the stomach has the ability to reduce chromium (VI) to chromium (III), reducing the extent of absorption when chromium (VI) compounds were administered *per os* compared with injection directly into the jejunum.

Both chromium (III) and chromium (VI) can penetrate intact human skin, and dermal uptake is increased if the skin is damaged (ATSDR, 2000). In some cases, dermal uptake was sufficient to result in signs of toxicity. The form and the vehicle greatly influence the rate of dermal uptake.

Inhaled particles of chromium compounds can remain in the lungs for years (ATSDR, 2000). Chromium absorbed into the blood is distributed throughout the body with highest levels frequently located in the kidneys. Chromium has been shown to cross the placenta and to be excreted via lactation. Autopsy studies in the US show that highest concentrations in newborns occur in the kidney, liver, aorta, heart, pancreas and spleen, and that tissue levels decline with age.

The main feature of the metabolism of chromium involves reduction of chromium (VI) to chromium (III) via the formation of chromium (V) and chromium (IV) intermediates (ATSDR, 2000). Reduction of chromium (VI) to chromium (III) occurs in the acid milieu of the stomach, primarily by reaction with ascorbate. Ascorbate, glutathione and other substrates effectively reduce absorbed chromium (VI) to chromium (III). Chromium (III) is considered a nutritionally essential element required for the proper metabolism of glucose, proteins and lipids. It acts as part of a complex known as GTF, which facilitates the action of insulin. Although chromium (III) complexes are generally thought to be inert, there is recent evidence that chromium (III)

may be reduced to chromium (II), which could catalyze the Haber-Weiss reaction resulting in the production of genotoxic hydroxyl radicals.

Absorbed chromium is excreted primarily in the urine (ATSDR, 2000).

4.0 Dermal Exposure

EPA (2004a) recommends GAFs of 0.013 for chromium (III) and 0.025 for chromium (VI), generally consistent with the toxicokinetic data reviewed above, which are used in this evaluation. The GAF of 0.013 for chromium (III) should be used for total chromium, which is assumed to consist of 6 parts of chromium (III) to 1 part of chromium (VI) (EPA, 2004b, 2010).

Data were not located regarding the extent of dermal uptake of chromium from soil, and EPA (2004a) provides no estimate of the extent of dermal absorption (ABS). EPA (2004a) recommends that dermal uptake from soil should not be quantified for chemicals without ABS recommendations, but that these chemicals should be discussed qualitatively as a source of uncertainty. EPA (2004a) compiled *in vivo* and *in vitro* data from human experiments of dermal uptake of chromium from water. Average K_p values of 1E-4 cm/hour and 1E-3 cm/hour are estimated for chromium (III) and chromium (VI), respectively. The K_p of 1E-4 cm/hour for chromium (III) should be used for total chromium as explained above.

5.0 Noncancer Effects Evaluation

As mentioned above, chromium (III) is an essential nutrient involved in maintenance of normal metabolism (ATSDR, 2000). EPA (2010) verified an RfD of 1.5E+0 mg/kg-day for chronic oral exposure to chromium (III) by applying an uncertainty factor of 1000 to a NOEL of 1800 g/kg (1.468 mg/kg body weight/day) as an average total ingested dose in a dietary study in which rats were given 600 feedings of chromic oxide baked into bread. No other dose levels were tested. Confidence in the RfD is low. No target organ or critical effect was identified for the toxicity of oral exposure to chromium (III).

The available data do not suggest a significant difference between chronic and subchronic exposure regarding the threshold for the noncancer effects of chromium (III). Therefore, EPA (1997) adopted the verified chronic oral RfD, which at the time was listed on IRIS as 1E+0 mg/kg-day, as the provisional subchronic oral RfD for chromium (III). Subsequently, EPA (2010) recalculated the average daily dose, which led to revising the chronic oral RfD from 1E+0 to 1.5E+0 mg/kg-day. Since the only change between the earlier and the present verified oral RfD reflects a recalculation, it is recommended that the current verified chronic oral RfD of 1.5E+0 mg/kg-day should also be used for subchronic exposure.

EPA (2010) verified an RfD for chronic oral exposure to chromium (VI) of 3E-3 mg/kg-day based on a NOAEL of 25 mg/L (2.5 mg/kg-day) in the drinking water of rats exposed for one year. An overall uncertainty factor of 900 was applied. The uncertainty factor consists of factors of 10 each for inter- and intra-species variation, a factor of 3 to expand from subchronic to chronic exposure, and a modifying factor of 3 to address concerns that relatively low levels may induce GI effects and possibly some forms of cancer in humans. Inclusion of the modifying factor of 3 was a relatively recent (1998) revision to a previously verified derivation. Nonetheless, EPA (2010) notes that confidence in the chronic oral RfD is low.

EPA (1997) presented a provisional subchronic oral RfD of 2E-2 mg/kg-day for chromium (VI) based on the same drinking water study and an uncertainty factor of 200 (not otherwise explained). Presumably, this derivation reflects the earlier IRIS evaluation before inclusion of the modifying factor of 3. However, the concern for GI effects that gave rise to the modifying factor could apply to subchronic as well as chronic exposure. Therefore, it is recommended that a new preliminary subchronic oral RfD should be calculated for chromium (VI). This is done by applying an overall uncertainty factor of 300 to the NOAEL of 2.5 mg/kg-day from the drinking water study. The uncertainty factor of 300 reflects the overall uncertainty factor of 900 used for the chronic RfD without the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD for chromium (VI) so calculated is 8E-3 mg/kg-day.

The data summarized by EPA (2010) are insufficient to identify a mechanism of toxicity or target organ(s) for chronic oral exposure to chromium (VI). Data from other sources, however, are sufficient to identify potential target organs. Goyer (1991) reported that renal tubular necrosis is the major effect in humans from ingestion of chromium compounds. The context, however, suggests that this effect is associated with acute exposure to high doses (e.g., accidental or attempted suicide ingestion); therefore, the kidney may not be an acceptable candidate as a target organ for chronic oral exposure. Furthermore, a review of human and animal studies by EPA (1998a) did not implicate the kidney as a target organ for chronic oral exposure. EPA (1998a), however, reported GI effects in a cohort of 155 Chinese villagers who consumed water from a well located adjacent to a chromium alloy plant. Symptoms included oral ulcers, diarrhea, abdominal pain, indigestion, and leukocytosis with a left shift. The dose rate of chromium (VI) was estimated at 0.57 mg/kg-day, nearly 200 times the oral RfD. It is likely that the GI symptoms reflect the irritant nature of chromium (VI).

EPA (1998a) also reviewed several reproductive studies. An early and inadequately conducted study reported no effects in rats fed a diet containing 0.125 percent K_2CrO_4. A diet containing 0.25 percent resulted in "subnormal condition" (including rough coat) and "subnormal" offspring; a diet containing 0.5 percent resulted in diarrhea and sterility. Assuming rats consume food equivalent to 5 percent of their body weight each day (EPA, 1980), dietary levels of 0.125, 0.25 and 0.5 percent correspond to chromium (VI) dose rates of 17, 33, and 67 mg/kg-day, respectively. Drinking water studies consistently report developmental effects and impaired reproduction in rats and mice ingesting approximately 20 mg chromium (VI)/kg-day. A well controlled study reported slightly decreased erythrocyte size and hemoglobin content in rats and mice of either sex fed diets containing 400 ppm potassium chromate. The effects were interpreted as "suggestive of a potential bone marrow/erythroid response." The 400 ppm diet is equivalent to chromium (VI) dose rates of 6.4 mg/kg-day in rats (EPA, 1998a) and approximately 11 mg/kg-day in mice, assuming that mice consume food equivalent to 10 percent of their body weight/day (EPA, 1980). In addition, cytoplasmic vacuolization of hepatocytes was observed in mice fed diets containing 50, 100 or 400 ppm potassium chromate, but it is not clear if either the incidence or intensity occurred in a dose-related manner. The 50 ppm diet, the lowest associated with hepatocellular vacuolization, is equivalent to a chromium (VI) intake rate of approximately 1.3 mg/kg-day. A subsequent study reported decreased erythrocyte hemoglobin content in female mice fed a diet containing 100 ppm potassium chromate, equivalent to a chromium (VI) dose rate of 6.0 mg/kg-day.

The GI signs reported in humans are clearly an indication of adverse effects; therefore, the GI tract is selected as a target organ. The mechanism of toxicity is assumed to be the irritation induced by tissue contact with chromium (VI). The slight hematologic changes observed in rats and mice and the hepatocellular vacuolization observed in mice do not appear to be sufficiently threatening to be considered adverse. Therefore, target organs selected for chronic oral exposure to chromium (VI) include the GI tract, unborn child (fetus), and reproductive system.

Inhalation (occupational) exposure to chromium may induce respiratory symptoms, changes in lung function and irritation, erosion or perforation of the nasal septum, depending in part on the exposure level (EPA, 2010). No adverse effects were observed in workers exposed to 1E-3 mg/m^3 for 0.2 to 23.6 years (average 2.5 years). EPA (1998b, 2010) reviewed several human and animal studies, and determined that effects observed from inhalation exposure to chromium (VI) are not relevant to exposure to chromium (III). EPA (1998c) concluded that chromium (VI) is the only form of chromium of concern for inhalation exposure.

EPA (2010) developed separate chronic inhalation RfC values for human exposure to chromic acid mists and dissolved chromium (VI) aerosols, and for exposure to chromium (VI) particulates. A verified chronic inhalation RfC of 8E-6 mg/m^3 for chromic acid mists and dissolved chromium (VI) aerosols is based on an adjusted LOAEL of 7.14E-4 mg/m^3 associated with atrophy of the nasal septum in subchronically occupationally exposed humans. An overall uncertainty factor of 90 was used, consisting of factors of 10 for intraspecies variation, 3 to estimate a NOAEL from a LOAEL, and 3 to expand from subchronic to chronic exposure. Confidence in the inhalation RfC is low. The upper respiratory tract is considered the target organ for inhalation exposure to chromic acid mists and dissolved chromium (VI) aerosols. The chronic inhalation RfC is equivalent to a chronic inhalation RfD of 2.3E-6 mg/kg-day.

Inhalation exposure to chromium (VI) particulates is associated with pneumocyte toxicity; i.e., with effects on the lungs themselves. EPA (2010) derived an RfC of 1E-4 mg/m^3 for chronic inhalation exposure to chromium (VI) particulates from an adjusted benchmark concentration of 3.4E-2 mg/m^3 associated with altered enzyme activity in bronchioalveolar lavage fluid from rats exposed to sodium dichromate dust intermittently for up to 90 days. An uncertainty factor of 300 was used, consisting of a factor of 10 to provide additional protection to unusually sensitive individuals, and factors of 3 each to cover for physiologic differences between rats and humans, and to expand from subchronic to chronic exposure. Confidence in the RfC is medium. The RfC is equivalent to a chronic inhalation RfD of 2.9E-5 mg/kg-day. The lung is considered to be the target organ for chronic inhalation exposure to chromium (VI) particulates.

No current EPA-derived subchronic inhalation evaluation exists for chromic acid mists and dissolved chromium (VI) aerosols. However, a preliminary subchronic inhalation RfC can be derived by excluding the uncertainty factor of 3 for expanding from subchronic to chronic exposure. Application of the remaining overall uncertainty factor of 30 to the adjusted LOAEL of 7.14E-4 mg/m^3 yields a preliminary subchronic inhalation RfC of 2E-5 mg/m^3 for chromic acid mists and dissolved chromium (VI) aerosols. The preliminary subchronic inhalation RfC is equivalent to a subchronic inhalation RfD of 6.8E-6 mg/kg-day.

No current EPA-derived subchronic inhalation evaluation exists for chromium (VI) particulates. However, a preliminary subchronic inhalation RfC can be derived by excluding the uncertainty

factor of 3 for expanding from subchronic to chronic exposure. Application of the remaining overall uncertainty factor of 100 to the adjusted LOAEL benchmark concentration of 3.4E-2 mg/m^3 yields a preliminary subchronic inhalation RfC of 3E-4 mg/m^3 for chromium (VI) particulates. The preliminary subchronic inhalation RfC is equivalent to a subchronic inhalation RfD of 9.7E-5 mg/kg-day.

EPA (1998c) noted that exposure to chromic acid mists and dissolved chromium (VI) aerosols is likely to be restricted to occupational settings and that most environmental exposures would involve exposure to chromium (VI) particulates. Therefore, the chronic inhalation RfC and RfD for chromium particulates will be used in this evaluation.

6.0 Carcinogenicity Evaluation

EPA (2010) classified chromium (III) in cancer weight-of-evidence group D – not classified as to carcinogenicity to humans – because of inadequate data. Chromium (VI) is classified in cancer weight-of-evidence group A – known human carcinogen – based on the consistent finding of lung cancer in epidemiologic studies of occupationally exposed workers in chromate production and the chrome pigment industry (EPA, 2010). Conclusions regarding the human data are corroborated by data from animal experiments. There is no evidence that oral exposure to chromium (VI) induces cancer, and EPA (2010) assigned chromium (VI) to Group D for oral exposure.

An inhalation URF of 1.2E-2 per ug/m^3, equivalent to an inhalation SF of 4.2E+1 per mg/kg-day, was based on increased risk of lung cancer deaths in chromate production workers (EPA, 2010). It should be noted that the quantitative assessment is based on the concentration of total chromium to which the workers were exposed, including insoluble (trivalent) and soluble (hexavalent) forms. It was assumed that chromium (VI) constituted not less than one-seventh of the total chromium; i.e., a ratio of 1 part of chromium (VI) to 6 parts of chromium (III). However, the forms of chromium in the air were not identified, and chromium (VI) may have constituted a significantly much greater portion of total airborne chromium. EPA (200) presented the URF of 1.2E-2 per ug/m^3 as a verified potency estimate for chromium (VI), but noted that this estimate may underestimate the potency of exposure to pure chromium (VI) by as much as seven-fold.

7.0 Toxicity Summary

Toxicity values for chromium (III) and chromium (VI) are summarized below. Theoretical noncancer toxicity values are calculated for total chromium assuming a chromium (III)/chromium (VI) ratio of 6 to 1. Note that the URF is based on a mixture of chromium (III) and chromium (VI).

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Chromium (III)										
1.5E+0	1.5E+0	ND	ND	ND	NA	D	ND	D	ND	ND
Chromium (VI)[b]										
8E-3	3E-3	GI, F, Re	3E-4/ 9.7E-5	1E-4/ 2.9E-5	Lu	D	ND	A	1.2E-2	4.2E+1

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Total Chromium[c]										
NA	2.1E-2	GI, F, Re	NA	7E-4/ 2.0E-4	Lu	D	ND	A	1.2E-2	4.2E+1

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: GI = gastrointestinal tract, F = unborn child (fetus), Re = reproductive system, Lu = lung.
[a]sRfDo and cRfDo should be adjusted by the appropriate GAF values described above when used for dermal exposure.
[b]Inhalation RfC/RfD values listed are those for chromium (VI) particulates. Values for chromic acid mists and dissolved chromium (VI) aerosols are not relevant to environmental exposures.
[c]For noncancer effects is based on the assumption that total chromium consists of 1 part of chromium (VI) and 6 parts of chromium (III); note that the URF assumed a mixture of chromium (III) to chromium (VI) at a ratio of approximately 6:1 (EPA 2010; EPA, 2004b).

8.0 References

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NICKEL 7440-02-0)

1.0 Introduction and Physical Properties

Nickel is a naturally occurring **metal** whose concentration ranks 24th in the crust of the earth (ATSDR, 2003), averaging approximately 180 mg/kg (HSDB, 2010). Its primary use is in metal alloys, to which it imparts many desirable characteristics. Nickel compounds are used for nickel plating, to color ceramics, to make some batteries, and as catalysts that increase the rate of various chemical reactions (ATSDR, 2003). Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
58.69[a] (element)	NA	NA	6.5E+1[b]	NA	NA	ND	Note1[c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.
Note 1: Nickel chloride as a typical nickel salt: 6.42E+5 at 20°C.
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
U.S. Environmental Protection Agency (EPA), 2004, *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final*, Office of Superfund Remediation [c]Agency for Toxic Substances and Disease Registry, (ATSDR), 2003, *Update Toxicological Profile for Nickel*, Draft for Public Comment, U.S. Public Health Service, Atlanta, Georgia, September, on line.

2.0 Environmental Fate and Transport

Natural sources of airborne particles that contain nickel include soil, sea, volcanoes, forest fires, and vegetation (ATSDR, 2003; HSDB, 2010). Meteorites that enter the atmosphere contribute free nickel to the air. Anthropogenic releases of nickel, approximately 1.4 to 5 times as great as natural releases, include combustion of fuel oils (including diesel engine emissions) and coal, nickel refining, municipal waste incineration, and steel and nickel alloy production. Nickel exists in the atmosphere as free metal and as various compounds, all in the form of particulates. Small particles may remain suspended in the air for long periods (half-life up to 30 days) and travel great distances. Removal mechanisms are limited to gravitational settling and wet and dry deposition.

Nickel is deposited on soil from the atmosphere (ATSDR, 2003; HSDB, 2010). Other contributions to soil include direct disposal of nickel-containing waste. Nickel in soil is subject to adsorption, precipitation, coprecipitation and complexation, which tend to immobilize it and reduce leaching. Several soil properties, such as calcium and magnesium content and pH, control the extent to which nickel remains immobile or available for leaching because several nickel salts are quite soluble.

Nickel gains access to surface water bodies by direct deposition from the atmosphere, in waste streams from industries that process or use nickel, from domestic waste water treatment plants, and by erosion and runoff from soil (ATSDR, 2003; HSDB, 2010). Nickel in water is subject to the same processes that occur in soil. These processes transfer nickel to particles that are subject to settling; however, large proportions may remain in solution (e.g., 75 percent in one polluted lake).

Empirical data with voles and rabbits living in sludge-amended land, as well as with aquatic invertebrates and vertebrates, suggest that nickel is not biomagnified in either aquatic or terrestrial food chain pathways (ATSDR, 2003). Nickel was identified by the EPA (1995) as a metal of no concern to human health from bioconcentration in fish. Therefore, biotransfer factors are not presented for nickel.

3.0 Toxicokinetics

Absorption of inhaled nickel that reaches the lungs (i.e., particles less than 1 micrometer in diameter) approximates 20 to 35 percent, depending on the specific compound involved (ATSDR, 2003). Uptake from the GI tract depends greatly on the specific compound and the mode of administration. In one study with humans uptake of nickel sulfate given in drinking water was much higher (approximately 27 percent) than when given in food (approximately 0.7 percent). Other studies show that the nature of the food items and their time of consumption relative to the time of administration of nickel have a great effect on the extent of uptake. EPA (2004) cited a 1972 study to support the GAF of 4 percent in humans for nickel in food and water.

Nickel has been shown to penetrate the skin. Studies in humans with radioactive nickel sulfate applied to occluded skin indicate that 55 to 77 percent of the dose is absorbed within 24 hours (ATSDR, 2003). A study in guinea pigs indicated that systemic absorption accounts for 0.005 percent to 0.51 percent of the dermally applied dose of radioactive nickel chloride. The extent to which nickel is sequestered in the dermis and the extent to which it passes to the bloodstream is unclear.

Highest levels of nickel are found in the lungs in occupationally exposed humans and animals exposed by the inhalation route (ATSDR, 2003). Highest levels in animals treated *per os* were found in the kidneys and blood. Significant levels were also found in liver, heart, lung and fat. Highest levels in animals following dermal treatment were found in the skin, blood, kidneys and liver.

Nickel metabolism is limited to ligand exchange reactions by which the cation binds to albumin and other serum proteins (ATSDR, 2003). Dogs lack some of the binding sites present in the serum of other species; therefore, more nickel in dog serum remains free and unbound. Some serum nickel binds to nickeloplasmin, which appears to represent a non-exchangeable pool.

Absorbed nickel is excreted in the urine regardless of the route of exposure (ATSDR, 2003). An excretion half-time of approximately 28 hours has been estimated for humans.

4.0 Dermal Exposure

As noted above, EPA (2004) estimated a GAF for nickel of 4 percent.

Data were not located regarding dermal uptake of nickel in soil and EPA (2004) provides no estimate of the extent of dermal absorption (ABS). EPA (2004) recommends that dermal uptake from soil should not be quantified for chemicals without ABS recommendations, but that these chemicals should be discussed qualitatively as a source of uncertainty. EPA (2004) compiled *in vitro* data from human experiments of dermal uptake of soluble salts of nickel from water. An

average K_p value of 6.4E-5 cm/hour is estimated for nickel from 5 studies reviewed by EPA (2004) and is used in this evaluation.

5.0 Noncancer Effects Evaluation

In a subchronic gavage study with nickel chloride in water, clinical signs of toxicity in rats included lethargy, ataxia, irregular breathing, reduced body temperature, salivation and discolored extremities (EPA, 2010). EPA (2010) presented a verified RfD of 2E-2 mg/kg-day for chronic oral exposure to nickel, based on a NOAEL for decreased organ and body weights in a two-year dietary study with nickel sulfate in rats and an uncertainty factor of 300. Confidence in the oral RfD is medium. The critical effect of prolonged oral exposure to low levels of nickel appears to be reduced body and organ weights without histopathological lesions. EPA (1997) adopted the verified chronic oral RfD of 2E-2 mg/kg-day as the provisional subchronic oral RfD. These oral RfDs are appropriate for oral exposure to soluble salts of nickel or unspeciated nickel compounds.

Inhalation exposure is associated with asthma and pulmonary fibrosis in welders using nickel alloys (ACGIH, 1991). Lung effects were observed in laboratory animals exposed by inhalation. Although the lung is clearly the target organ for inhalation exposure, the data are inadequate for derivation of an inhalation RfC or RfD.

6.0 Carcinogenicity Evaluation

Occupational exposure to nickel is associated with increased risk of nasal, laryngeal and lung cancer (ACGIH, 1991; ATSDR, 2003). Inhalation exposure of rats to nickel subsulfide increased the incidence of lung tumors. EPA (2010) presented a cancer weight-of-evidence Group A classification (human carcinogen) for nickel, and an inhalation URF of 2.4E-4 per $\mu g/m^3$ for nickel refinery dust. The URF is equivalent to 8.4E-1 per mg/kg-day. The quantitative estimate was derived from human occupational studies. EPA (2010) also presented a URF of 4.8E-4 per $\mu g/m^3$, which is equivalent to 1.7E+0 per mg/kg-day, for nickel subsulfide, one of the most predominant constituents in nickel refinery dust. The evaluation for nickel refinery dust is ordinarily chosen when the form of nickel in air is unknown. The Group A classification should apply only to inhalation exposure.

7.0 Toxicity Summary

Toxicity values for nickel are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Nickel, Soluble Salts or Unspeciated										
2E-2	2E-2	BW	ND	ND	NA	Please see below.				
Nickel Refinery Dust										
Please see above						NA	NA	A	2.4E-4	8.4E-1
Nickel Subsulfide										
Please see above.						NA	NA	A	4.8E-4	1.7E+0

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable. Target organ or critical effect abbreviations: BW = reduced body weight. [a]sRfDo and cRfDo should be adjusted by the GAF of 0.04 when applied to dermal exposure.										

9.0 References

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SELENIUM (7782-49-2)

Introduction and Physical Properties

Selenium is an inorganic element ubiquitously distributed in nature that occurs in most rocks and soils at concentrations of 0.1 to 2.0 mg/kg, or 0.05 to 0.09 mg/kg in the earth s crust as a whole (ATSDR, 1996, HSDB, 2010). Selenium is used predominantly in the semiconductor industry and in photoelectric applications. Other uses include glass making, pigments, anti-dandruff shampoos, fungicides and medical imaging. Dietary supplements for livestock are the largest agricultural use. Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
78.96[a] (Element)	NA	NA	5.0E+0[b]	NA	NA	ND	Note 1[a, c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry□s Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable; ND = no data.
Note 1: Element insoluble in water; many selenites and selenates are relatively soluble.
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, last accessed 26 September.
[b]U.S. Environmental Protection Agency (EPA), 2002, *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.
[c]Agency for Toxic Substances and Disease Registry, (ATSDR), 1996, ***Update Toxicological Profile for Selenium***, U.S. Public Health Service, Atlanta, Georgia, August, on line.

Environmental Fate and Transport

Natural atmospheric releases of selenium result from volatilization, as a result of biomethylation of selenium by plants and bacteria, and from volcanic eruptions (ATSDR, 1996; HSDB, 2010). Leaching and weathering of parent bedrock material may result in release to soil. Anthropogenic selenium releases include mining and milling operations, base metal smelting and refining, selenium refining, the burning of coal, oil and solid waste, and the uses described above. Burning of coal and production of copper account for approximately 62 and 26 percent, respectively, of the total anthropogenic selenium released to the atmosphere. One study showed that approximately 53 percent of the selenium contained in coal is emitted to the atmosphere as volatilized selenium compounds or included with particles of fly ash.

The majority of selenium in the atmosphere is expected to exist in the particulate form (HSDB, 2010). Particulate-phase selenium may be physically removed from the air by wet and dry deposition (ATSDR, 1996; HSDB, 2010). The volatile selenium compounds such as selenium dioxide, hydrogen selenide, dimethyl selenide, and dimethyl diselenide are expected to exist in the gaseous state in the atmosphere. Hydrogen selenide released to the air is unstable and will be oxidized to elemental selenium and water. Selenium dioxide released to the air from the combustion of fossil fuels is expected to be reduced to elemental selenium by sulfur dioxide formed during combustion.

The behavior of selenium in soils is affected by redox conditions, pH, hydrous oxide content, clay content, organic materials and the presence of competing anions (ATSDR, 1996; HSDB, 2010). Heavy metal selenides, which are insoluble, predominate in acidic soils and soils with

high amounts of organic matter. Selenium in this form is immobile. Sodium and potassium selenites dominate in neutral, well-drained mineral soils. Selenites are soluble, but can strongly adsorb to soil minerals and organic matter. Iron and manganese oxides adsorb selenite, with iron oxides adsorbing more than manganese. In alkaline, well-oxidized soil environments, selenates predominate. The selenates are very mobile because of their high water solubility and low tendency to sorb to soil particles. Selenite adsorption was observed to decrease with increasing pH in the range 4 to 9, and selenate adsorption was minimal under most pH conditions. In soils, elemental selenium and inorganic selenium compounds can be methylated by microorganisms and subsequently volatilize to the atmosphere. Dimethyl selenide and dimethyl diselenide are the principal methylation products.

Surface water may receive selenium from the atmosphere by wet or dry deposition, from surface runoff or erosion, from subsurface drainage or from various waste streams (ATSDR, 1996; HSDB, 2010). Selenium in surface water forms oxyanions and exhibits anionic chemistry. Volatilization of the ionic forms is not expected to be an important fate process. Which anionic form predominates is determined by the pH and redox potential of the water. Elemental selenium is favored by low pH and reducing conditions. Selenates are stable under alkaline oxidizing conditions. Selenious acid species occur under the intermediate to slightly oxidizing conditions generally encountered in aerobic water. Over the pH range of 3 to 9, the biselenite species predominate; at pH values greater than 9, selenite is the major species. At pH values less than 7 and under mildly reducing conditions, selenites are reduced to elemental selenium. Reducing conditions in surface sediments or ponded laboratory sediment columns promote the progressive reduction of soluble selenate to soluble selenite to insoluble elemental selenium, which tends to accumulate. Reduced selenium adsorbed to sediment will remain relatively immobile unless the sediments are chemically or biologically oxidized. Biological methylation of selenium may occur in aquatic environments; inorganic selenium undergoes biological methylation to dimethyl selenide and dimethyl diselenide, which are subject to volatilization.

Selenium has been identified by the EPA (1995) as a chemical that is not a bioaccumulative chemical of concern in fish; therefore, a BCF is not included for selenium. Other data, however, indicate that selenium has the potential to bioaccumulate and possibly to biomagnify in terrestrial food chain pathways (ATSDR, 1996; HSDB, 2010). Biotransfer factors for selenium are compiled below:

BCF	Bpr	Bpv	Bb	Bm
NA[a]	2.5E-2[b]	2.5E-2[b]	1.5E-2[b]	4.0E-3[b]

BCF = bioconcentration factor, L/kg; Bpr = soil-to-plant (reproductive portions) biotransfer factor, unitless; Bpv = soil-to-plant (vegetative portions) biotransfer factor, unitless; Bb = soil-to-beef biotransfer factor, days/kg; Bm = soil-to-milk biotransfer factor, days/kg; NA = not applicable.
[a]See above.
[b]Baes, C.F., R.D. Sharp, A.L. Sjoreen, R.W. Shor, 1984, ***A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture***, Health and Safety Division, Oak Ridge National Laboratory, ORNL-5786, September.

Data for Evaluating Dermal Exposure

Selenium appears to be readily absorbed from the GI tract, apparently nearly free from homeostatic control, especially the soluble forms, but the form of selenium administered affects absorption efficiency (ATSDR, 1996; HSDB, 2010). Reported absorption efficiencies range

from 30 to 46 percent up to 95 to 97 percent. Reasons for the wide range are unclear. Cats treated with a variety of selenium compounds excreted not more than 20 percent of the dose in the feces, indicating that upwards of 80 percent was absorbed. EPA (2004) recommends that oral toxicity values should not be adjusted to reflect the extent of GI absorption unless GI absorption is significantly less than complete. Therefore, a GAF is not estimated for selenium and the oral toxicity values are used for dermal exposure without adjustment.

The empirical data indicate that selenium is absorbed by the skin, at least by mice. However, data were not located regarding the extent of dermal uptake of selenium from soil, and EPA (2004) provides no estimate of the extent of dermal absorption (ABS). EPA (2004) recommends that dermal uptake from soil should not be quantified for chemicals without ABS recommendations, but that these chemicals should be discussed qualitatively as a source of uncertainty. The EPA (2001) default PC for metals of 1E-3 cm/hour is used for selenium.

Noncancer Effects Evaluation

Selenium is a nutritionally essential trace element that is an integral part of the enzyme glutathione peroxidase and other proteins (Högberg and Alexander, 1986). NRC (1989) recommended dietary allowances for humans range from 10 to 75 μg/day. Chronic ingestion of 5 mg/day (7.1E-2 mg/kg-day, assuming humans weigh 70 kg) induces clinical selenosis in humans, characterized by the characteristic "garlic odor" of excess selenium excretion in the breath and urine, thickened and brittle nails, hair and nail loss and abnormal re-growth, lowered hemoglobin levels, mottled teeth, skin lesions, and CNS abnormalities (peripheral anesthesia, acroparesthesia and pain in the extremities) (EPA, 2010; Högberg and Alexander, 1986). Effects in domestic grazing animals exposed to high levels of selenium include emaciation, lameness, and loss of hair and hooves. EPA (2010) presents a verified RfD of 5E-3 mg/kg-day for chronic oral exposure to selenium compounds based on effects in humans exposed to selenium in high selenium areas. An uncertainty factor of 3 was used. Confidence in the RfD is high. The target organs for oral exposure to selenium are the skin (including the nails and hair), teeth, erythrocyte and CNS.

EPA (1997) adopted the chronic oral RfD of 5E-3 mg/kg-day as sufficiently protective for subchronic oral exposure as well.

Occupational exposure to selenium fume or various selenium compounds is associated with intense ocular and respiratory tract irritation, chemical pneumonia, skin rashes, garlic odor to the breath, metallic taste in the mouth, and various socio-psychological effects (ACGIH, 1991). The data are inadequate for estimation of an inhalation RfC.

Carcinogenicity Evaluation

An impressive body of data indicates that selenium exerts an anticarcinogenic effect (Högberg and Alexander, 1986). In laboratory animals, selenium supplementation decreases the incidence of chemical-induced cancers. In humans, the incidence of lymphomas and cancers of the breast, digestive tract and lung were lower in geographic areas with high soil selenium levels. Occupational data suggest that selenium may protect against lung cancer. Several animal tests with various deficiencies in design and conduct equivocally associated exposure to selenium with increased incidence of cancer. In a well controlled oral experiment, selenium sulfide was associated with an increase in the incidence of liver tumors in rats, and with liver and lung

tumors in mice. On the basis of this study, EPA (2010) classified selenium sulfide a cancer weight-of-evidence Group B2 compound (probable human carcinogen), but declined to derive quantitative risk estimates. Selenium and other selenium compounds were classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA, 2010). Quantitative risk estimates are not derived for Group D substances.

Toxicity Summary

Toxicity values for selenium are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
5E-3	5E-3	S, Te, E, CNS	ND	ND	NA	D	ND	D	ND	ND

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data.
Target organ abbreviations: S = skin; Te = teeth; E = erythrocyte; CNS = central nervous system; NA = not applicable.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

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THALLIUM (7440-28-0)

1.0 Introduction and Physical Properties

Thallium is a naturally occurring metal with an abundance of about 0.3-0.6 ppm in the earth s crust (HSDB, 2010). Thallium compounds are used in a variety of applications, including pharmaceuticals, semiconductors, photoelectric cells, optical systems, ore separation, glass production, and as oxidizing agents in organic synthesis (ATSDR, 1992; HSDB, 2010). An important former use of thallium and its compounds (before 1972) was in pesticide formulations for the control of insects and rodents. Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
204.38[a] (Elemental)	NA	NA	7.1E+1[b,c]	NA	NA	NA	Note 1[a]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.
Note 1: Thallium sulfate as a typical thallium salt: 4.87E+4 mg/L (20˚ C).
[a]Agency for Toxic Substances and Disease Registry, (ATSDR), 1992, ***Toxicological Profile for Thallium***, U.S. Public Health Service, Atlanta, Georgia, July, on line.
[b]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.
[c]pH = 6.8.

2.0 Environmental Fate and Transport

Thallium is released to the atmosphere largely from coal-burning power plants, cement factories, and ferrous and nonferrous smelting operations (ATSDR, 1992; HSDB, 2010). Facilities that produce or use thallium or its compounds account for a much smaller part of the total release.

Thallium in air exists as oxide, hydroxide, sulfate or sulfide particles (ATSDR, 1992; HSDB, 2010). The hydroxide and sulfate are water soluble and may partition to water vapor for eventual removal in precipitation. The less soluble particulate forms are probably removed by dry deposition.

Thallium released to surface water usually occurs as the monovalent ion that forms soluble salts with several anions (ATSDR, 1992; HSDB, 2010). Adsorption to suspended particulates may also occur, but the soluble forms remain in the water column.

Although empirical data are sparse, the high solubility of several thallium salts suggests that mobility in soil could be high and the potential for leaching could be significant (ATSDR, 1992).

Terrestrial plants have been shown to absorb thallium from soil, but there is no evidence that biomagnification occurs. EPA (1995) identifies thallium as a chemical unlikely to be of concern for bioaccumulation in aquatic food chains. For these reasons it is concluded that thallium is unlikely to participate significantly in food-chain pathways and biotransfer factors are not estimated or compiled.

3.0 Toxicokinetics

Data regarding absorption from the respiratory tract following inhalation exposure were not located; however, small particles are probably absorbed to some extent and larger particles are probably removed by mucocilliary clearance and swallowed, where they become available for GI absorption. Thallium compounds appear to be completely absorbed from the GI tract in humans and laboratory animals (ATSDR, 1992). Quantitative data regarding dermal uptake of thallium were not located, although HSDB (2010) states that absorption through the skin occurs readily.

Tissue levels in acutely poisoned humans appear to be highest in the brain in regions dense with neurons, such as the gray matter (HSDB, 2010). Tissue distribution testing in a single human volunteer cancer patient treated orally with a small dose of radiolabeled thallium indicated that highest concentrations were located in scalp hair followed by kidney, heart, bone, spleen and brain (ATSDR, 1992). Distribution in orally treat laboratory animals is rapid and widespread, with higher levels generally associated with the kidney. The rank of levels in other tissues is more variable (ATSDR, 1992), but high levels are ordinarily found in the testis (HSDB, 200).

The biological half-life in rats in one study was 3.3 days, indicating that excretion is efficient (ATSDR, 1992). Excretion in humans may be somewhat slower. Excretion appears to be primarily through the urine in humans, and through the urine and feces. However, the data are somewhat confusing because animal studies indicate that the ratio of fecal to urinary excretion increases with time. This suggests that biliary excretion and enterohepatic recirculation may be operative, which could result in underestimation of fecal excretion in the human study.

4.0 Dermal Exposure

As noted above, GI absorption of thallium is essentially complete. EPA (2004) concluded that a GAF should not be estimated for thallium for the purpose of adjusting oral toxicity values for dermal exposure. Therefore, the oral toxicity values described below are used directly without adjustment for evaluating dermal exposure.

Thallium may be readily absorbed by the skin (HSDB, 2010); however, data were not located regarding the extent of dermal uptake of thallium from soil, and EPA (2004) provides no estimate of the extent of dermal absorption (ABS). EPA (2004) recommends that dermal uptake from soil should not be quantified for chemicals without ABS recommendations, but that these chemicals should be discussed qualitatively as a source of uncertainty. Empirical data regarding the uptake of soluble forms of thallium from water were not located. The EPA (2004) default K_p for inorganic chemicals of 1E-3 cm/hour is selected for thallium.

5.0 Noncancer Effects Evaluation

Thallium is highly toxic. Acute oral exposure to thallium and its compounds may cause death preceded by neurological disturbances and effects on the lungs, heart and liver (ATSDR, 1992). Formerly, thallium was used medicinally to induce alopecia to facilitate treatment of ringworm of the scalp, sometimes with disastrous results. The critical effect of chronic oral exposure to low levels of thallium compounds in animals and humans is alopecia (ACGIH, 1992; EPA, 2010). EPA (2010) verified chronic oral RfDs for several thallium salts (thallium acetate, thallium carbonate, thallium chloride, thallium nitrate and thallium sulfate) based on a study with thallium sulfate. The oral RfD for thallium sulfate of 8E-5 mg/kg-day was based on a NOAEL of 0.25 mg/kg-day in rats treated by gavage with thallium sulfate for 90 days. No adverse effects

were seen in this study. An uncertainty factor of 3000 was used, consisting of factors of 10 each to extrapolate from subchronic to chronic data, for intraspecies extrapolation and to account for interspecies variability, and a factor of 3 to account for lack of reproductive and chronic toxicity data. Confidence in the chronic RfD is low. The skin (alopecia) and liver (altered blood biochemistry) are considered the target organs for oral exposure to thallium, based on other data reviewed by EPA (2010). Effects on the testis and kidney were observed in rats exposed to higher dose rates. The chronic oral RfD for thallium sulfate of 8E-5, when adjusted for differences in molecular weight, is equivalent to a chronic oral RfD for thallium of 6.5E-5 mg/kg-day. This adjustment is based on the assumption that the toxicity of thallium sulfate is due entirely to thallium, rather than to the sulfate moiety. The oral RfD for thallium of 6.5E-5 mg/kg-day will be used in this evaluation.

EPA (1997) provides a provisional subchronic oral RfD for thallium sulfate of 8E-4 mg/kg-day, equivalent to a subchronic oral RfD for thallium of 6.5E-4 mg/kg-day, based on the study discussed above and an uncertainty factor of 300, eliminating the factor of 10 to expand from subchronic to chronic exposure. EPA (1997) provides provisional subchronic chronic oral RfDs for several thallium salts (thallium acetate, thallium carbonate, thallium chloride, thallium nitrate and thallium sulfate) based on the same study with thallium sulfate.

Inhalation exposure data are very limited. An occupational study suggests that neurological effects may develop following prolonged inhalation exposure (ATSDR, 1992). The nervous system appears to be more sensitive than the skin as a target organ for inhalation exposure. The data are inadequate for estimation of an inhalation RfC or RfD.

6.0 Carcinogenicity Evaluation

Several thallium compounds (thallium oxide, thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium sulfate) are classified as cancer weight-of-evidence Group D substances (not classifiable as to carcinogenicity to humans) (EPA, 2010). No weight-of-evidence classification was located for thallium alone, but the Group D classification can be applied to thallium. Quantitative cancer risk estimates are not derived for Group D chemicals.

7.0 Toxicity Summary

Toxicity values for thallium are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
6.5E-4	6.5E-5	S, L	ND	ND	NA	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: L = liver; S = skin.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

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POLYCHLORINATED BIPHENYLS (PCBs)

Introduction and Physical Properties

The nomenclature of the PCBs is somewhat unique, including the terms homologue and congener. The PCB molecule consists of two joined phenyl groups. The carbon atoms are numbered 1 through 6 on one ring and 1′ through 6′ on the other ring. Numbering proceeds consecutively starting with the carbon atom attached to the other ring. The term homologue refers to the degree of chlorination. Since one carbon atom in each phenyl group is bonded to the other ring, five carbon atoms on each phenyl group – a total of 10 carbon atoms – are available for bonding to chlorine atoms. Consequently, there are ten possible *homologues* of PCBs – monochlorinate biphenyls, dichlorinated biphenyls, through decachlorinated biphenyls. Congener refers to the exact placement of each chlorine atom. For example, there are three congeners of monochlorinated biphenyls: 2-PCB, 3-PCB and 4-PCB. The term isomer is often incorrectly used instead of congener with the PCBs. Free rotation around the C-C bond, however, precludes stereoisomerism, which limits the number of theoretically possible congeners.

The PCBs are a class of chemicals generally considered to be **SVOC**s including 209 possible individual congeners (ATSDR, 2000). The PCBs formerly manufactured and used in the U.S. are called Aroclors. The Aroclors are mixtures of several PCB congeners and related compounds. Many individual PCB congeners are common to all or many of the different Aroclors. The Aroclors differ from one another primarily in the relative proportions of the different congeners. ATSDR (2000) provides a summary of the proportion of each homologue in commercial mixtures of the various Aroclors. Of interest to this evaluation is the observation that Aroclor-1016 and Aroclor-1221 contain no homologues with 5 or more chlorines. Most Aroclors occur as oily or viscous liquids at room temperatures, although Aroclor-1260 occurs as a sticky resin. Aroclors were used as dielectric and heat exchange agents in several open and closed systems, but since the middle 1970s, use has been restricted largely to electrical transformers and capacitors.

Analysis of PCBs in environmental media is often performed using EPA Method SW8082. Method SW8082 is usually performed as a "finger printing" procedure and the results are reported as the commercial Aroclor(s) that most closely reflect the fingerprint(s) (ATSDR, 2000). Recently, however, concern has arisen that some individual PCB congeners may behave toxicologically in the same manner as the polychlorinated dibenzo-p-dioxins (PCDD), and that they may have greater toxic potency than is reflected in the toxicity assessment of the commercial Aroclors. This problem may be exacerbated by weathering, which may preferentially remove the less toxic (generally less highly chlorinated) congeners, leading to a relative concentration of the more highly chlorinated congeners. The more highly chlorinated congeners are generally the more persistent and the more toxic (ATSDR, 2000; EPA, 2010). For this reason, Method SW8082 has been expanded so that the analytical results can be reported in terms of selected specific congeners. In addition, EPA Method E1668 is a very high-resolution protocol (highly sensitive and highly selective) that reports individual congeners as well as totals for each of the homologues. Method E1668 is seldom used for environmental media, with the exception of incinerator emissions testing, because of its unusually high cost.

EPA (2000) identified 12 individual PCB congeners with dioxin-like activity. The PCB mixtures and dioxin-like congeners analyzed by the EPA methods mentioned above are compiled below:

PCB Mixture or Congener	CASRN	IUPAC No.	Method 8082	Method 1668
Aroclor-1016	12674-11-2	NA	Yes	No
Aroclor-1221	11104-28-2	NA	Yes	No
Aroclor-1232	11141-16-5	NA	Yes	No
Aroclor-1242	53469-21-9	NA	Yes	No
Aroclor-1248	12672-29-6	NA	Yes	No
Aroclor-1254	11097-69-1	NA	Yes	No
Aroclor-1260	11096-82-5	NA	Yes	No
3,3′,4,4′-Tetrachlorobiphenyl	**32598-13-3**	**77**	**Probably[a]**	**Yes**
3,4,4′,5-Tetrachlorobiphenyl	**70362-50-4**	**81**	**Probably[a]**	**Yes**
2,3,3′,4,4′-Pentachlorobiphenyl	**32598-14-4**	**105**	**Probably[a]**	**Yes**
2,3,4,4′,5-Pentachlorobiphenyl	**74472-37-0**	**114**	**Probably[a]**	**Yes**
2,3′,4,4′,5-Pentachlorobiphenyl	**31508-00-6**	**118**	**Probably[a]**	**Yes**
2′,3,4,4′,5-Pentachlorobiphenyl 2,3′,4,4′,5′-Pentachlorobiphenyl)	**65510-44-3**	**123**	**Probably[a]**	**Yes**
3,3′,4,4′,5-Pentachlorobiphenyl	**57465-28-8**	**126**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5-Hexachlorobiphenyl	**38380-08-4**	**156**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5′-Hexachlorobiphenyl	**69782-90-7**	**157**	**Probably[a]**	**Yes**
2,3′,4,4′,5,5′-Hexachlorobiphenyl	**52663-72-6**	**167**	**Probably[a]**	**Yes**
3,3′,4,4′,5,5′-Hexachlorobiphenyl	**32774-16-6**	**169**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5,5′-Heptachlorobiphenyl	**39635-31-9**	**189**	**Probably[a]**	**Yes**
Total Monochlorobiphenyls	NA	NA	No	Yes
Total Dichlorobiphenyls	NA	NA	No	Yes
Total Trichlorobiphenyls	NA	NA	No	Yes
Total Tetrachlorobiphenyls	NA	NA	No	Yes
Total Pentachlorobiphenyls	NA	NA	No	Yes
Total Hexachlorobiphenyls	NA	NA	No	Yes
Total Heptachlorobiphenyls	NA	NA	No	Yes
Total Octachlorobiphenyls	NA	NA	No	Yes
Total Nonachlorobiphenyls	NA	NA	No	Yes
Total Decachlorobiphenyls	NA	NA	No	Yes

PCB = polychlorinated biphenyl; CASRN = Chemical Abstract Service Registry Number; IUPAC No. = congener-specific identification number assigned by the International Union of Pure and Applied Chemists. The specific congeners are sometimes called PCB-77, PCB-81, etc., and are sometimes *inappropriately* called Aroclor-77, Aroclor-81, etc. Technically, the term Aroclor should be reserved for chemical mixtures, where the name Aroclor is followed by a four-digit identifier code.
Dioxin-like congeners are bolded.
[a]Although the congeners listed have not been tested by Method 8082, it is likely that they could be quantified by the method. Other non-dioxin-like congeners can routinely be analyzed by this method.

Data regarding the physical properties of the individual PCB congeners are very limited. The physical characteristics of the Aroclor mixtures have been much more thoroughly evaluated. Although the Aroclors described below are classified as SVOCs, largely on the basis of MW, their H values suggest that volatilization could be important. Furthermore, the MW values provided are proportional averages based on the composition of the various Aroclor mixtures. Several individual PCB congeners have MW values below the limit of 200 g/mole ordinarily used to distinguish VOCs from SVOCs. For these reasons it is recommended that the Aroclors should be evaluated in exposure pathways (such as vapor intrusion into indoor air) that involve volatilization. Relevant physical properties are compiled below:

MW[a]	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Aroclor-1016 (12674-11-2)										
257.9[b]	5.6[b]	2.9E-4[b] (25°C)	4.96[c]	4.69E-2[d] (25°C)[e]	6.8E-6[c] (25°C)	5.3E-7[b] (25°C)	4.2E-1[b] (25°C)	340.50[f]	475.22[f]	1.8E+4[f]
Aroclor-1221 (11104-28-2)										
200.7[b]	4.7[b]	3.5E-3[b] (25°C)	2.44[c]	5.54E-2[d] (25°C)[e]	7.5E-6[c] (25°C)	8.8E-6[b] (25°C)	5.9E-1[b] (24°C)	571[b]	ND	ND
Aroclor-1232 (11141-16-5)										
232.2[B]	5.1[B]	8.64E-4[c] (25°C)[e]	2.83[c]	5.03E-2[d] (25°C)[e]	7.2E-6[c] (20°C)	5.34E-6[b] (25°C)	4.5E-1[b] (25°C)	581[b]	ND	ND
Aroclor-1242 (53469-21-9)										
266.5[b]	5.6[b]	5.2E-4[b] (25°C)	4.59[c]	4.59E-2[d] (25°C)[e]	6.1E-6[c] (20°C)	5.34E-7[b] (25°C)	2.4E-1[b] (25°C)	345.5[f]	482.20[f]	1.8E+4[f]
Aroclor-1248 (12672-29-6)										
299.5[g]	6.2[g]	2.8E-3[g] (25°C)	5.64[c]	4.24E-2[d] (25°C)[e]	6.6E-6[c] (20°C)	6.5E-7[g] (25°C)	5.7E-2[g] (24°C)	631[g]	ND	ND
Aroclor-1254 (11097-69-1)										
328[b]	6.5[b]	2.0E-3[b] (25°C)	5.0[c]	3.99E-2[d] (25°C)[e]	5.6E-6[c] (20°C)	1.01E-7[b] (25°C)	3.5E-2[b] (24°C)	377.50[f]	512.27[f]	1.9E+4[f]
Aroclor-1260 (11096-82-5)										
375.7[b]	6.8[b]	4.6E-3[b] (25°C)	6.42[c]	3.65E-2[d] (25°C)[e]	5.3E-6[c] (20°C)	5.33E-8[b] (25°C)	8.0E-2[b] (24°C)	402.50[f]	539.37[f]	1.9E+4[f]
Aroclor-1262 (1336-36-3)										
389[b]	ND	ND	ND	3.57E-2[d] (25°C)[e]	4.13E-6[d] (25°C)[e]	ND	5.2E-2[b] (24°C)	680.7[b]	ND	ND
Aroclor-1268 (11100-14-4)										
453[b]	ND	ND	ND	3.22E-2[d] (25°C)[e]	3.73E-6[d] (25°C)[e]	ND	3.0E-1[b] (24°C)	ND	ND	ND

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K); $\Delta H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); I = insoluble; ND = no data.

[a]Average molecular mass for the proportions of individual congeners in the commercial product.

[b]Agency for Toxic Substances and Disease Registry (ATSDR), 2000, ***Update Toxicological Profile for Polychlorinated Biphenyls***, U.S. Department of Health and Human Services, Atlanta, Georgia, November, on line.

[c]Montgomery, J.H., 1996, ***Groundwater Chemicals Desk Reference, Second Edition***, Lewis Publishers, New York.

[d]Calculated as described in Introduction to Toxicological profiles.

[e]Assumed.

[f]U.S. Environmental Protection Agency (EPA), 1997, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings***, prepared by Environmental Quality Management, Inc. for the Office of Emergency and Remedial Response, Washington, DC, September.

[g]Earlier version (1995 Draft for Public Comment) of ATSDR Toxicological profile.

Environmental Fate and Transport

Former PCB releases occurred as a result of their manufacture, use, disposal, and leakage from damaged PCB-containing equipment (ATSDR, 2000). Currently the major source of PCB release to the environment is recycling of PCBs previously introduced into the environment, which involves volatilization from ground surfaces (water, soil) into the atmosphere with subsequent removal from the atmosphere via wet/dry deposition, followed by volatilization (HSDB, 2010). PCBs are also currently released to the environment from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, and improper (or illegal) disposal of PCB materials, such as waste transformer fluid, to open areas.

Major sources to air include emissions from the overhaul, repair or reuse of PCB-containing items, and the recycling of previously released PCBs described above (ATSDR, 2000; HSDB, 2010). The vapor pressures of the PCBs indicate that they would exist primarily in the vapor-phase in the atmosphere; monitoring data have shown that between 87 and 100% of the PCBs in air are in the vapor-phase. Vapor pressure of the PCBs generally decreases with an increase in the extent of chlorination; therefore, the more highly chlorinated PCBs are more likely to be associated with the particulate phase in air than are the lesser chlorinated PCBs. Physical removal of PCBs in the atmosphere is accomplished by wet and dry deposition; dry deposition occurs only for PCBs in the particulate phase.

Major sources to soil include leaks or discharges from PCB-containing items and deposition from the atmosphere (ATSDR, 2000). PCBs sorb tightly to constituents of soil with adsorption generally increasing with the degree of chlorination. Although the lesser chlorinated biphenyls may exhibit some mobility in soil, PCBs generally will not leach significantly in most aqueous soil systems. In the presence of organic solvents, PCBs may leach quite rapidly through soil. Vapor loss of PCBs from soil surfaces appears to be an important loss mechanism with the rate of volatilization decreasing with increasing chlorination. Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of the PCBs. In general, persistence of the PCBs increases with an increase in the degree of chlorination (ATSDR, 2000; HSDB, 2010). Mono-, di- and trichlorinated biphenyls (Aroclors-1221 and -1232) biodegrade relatively rapidly, tetrachlorinated biphenyls (Aroclors-1016 and -1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors-1248, -1254 and -1260) are quite resistant to biodegradation. The position of chlorination within a given homologue also determines the extent of biodegradation. Although biodegradation of the more highly chlorinated congeners may occur very slowly on an environmental basis, no other degradation mechanisms have been shown to be important in soil systems; therefore, biodegradation may be the ultimate degradation process in soil.

Former sources of PCBs to surface water include waste streams and discharges from waste water treatment plants (ATSDR, 2000). Currently, direct deposition, runoff and erosion probably predominate. Removal from water involves sorption to particles and sedimentation, as well as volatilization.

The PCBs are among the compounds that bioaccumulate in food chain pathways and are of special concern for biomagnification from sediment in benthic fish. Biotransfer factors for the PCBs are compiled below:

BSAF	Bpr[a,b]	Bpv[a,b]	Bb[a]	Bm[a]
Aroclor-1016				
See below	2.24E-2	2.24E-2	1.00E-2	3.16E-3
Aroclor-1221				
See below	7.44E-2	7.44E-2	1.26E-3	3.98E-4
Aroclor-1232				
See below	4.37E-2	4.37E-2	3.16E-3	1.00E-3
Aroclor-1242				
See below	2.24E-2	2.24E-2	1.00E-2	3.16E-3
Aroclor-1248				
See below	1.01E-2	1.01E-2	3.98E-2	1.26E-2
Aroclor-1254				
See below	6.78E-3	6.78E-3	7.94E-2	2.51E-2
Aroclor-1260				
See below	4.55E-3	4.55E-3	1.58E-1	5.01E-2
Aroclor-1262				
See below	ND	ND	ND	ND
Aroclor-1268				
See below	ND	ND	ND	ND

BSAF = biota-sediment accumulation factor, unitless; Bpr = soil-to-plant (reproductive portions) biotransfer factor, unitless; Bpv = soil-to-plant (vegetative portions) biotransfer factor, unitless; Bb = soil-to-beef biotransfer factor, days/kg; Bm = soil-to-milk biotransfer factor, days/kg; ND = no data.
[a]Calculated as described in Introduction to the Toxicological profiles.
[b]The methodology for estimating biotransfer of organic chemicals to plants does not differentiate vegetative and reproductive portions.

EPA (1995) reported a biota/sediment absorption factor (BSAF) for total PCBs of 1.85 for trout in the Great Lakes ecosystem. The BSAF of 1.85 is applied to all PCBs in this evaluation.

Data for Evaluating Dermal Exposure

Toxicokinetic data from laboratory animals suggest that the efficiency of GI absorption is roughly inversely related to the degree of chlorination (ATSDR, 2000). The GI absorption of mono- to hexachlorinated biphenyls exceeds 90 percent. Dichlorobiphenyl GI absorption efficiency is approximately 95 percent, but the absorption efficiency of octachlorobiphenyl approximates only 75 percent. GI absorption efficiency of Aroclor-1254 approximates 85.4 percent in ferrets and greater than 90 percent in monkeys. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for the PCBs and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

The PCBs appear to be readily absorbed by the skin when applied as neat compound or mixed with a suitable vehicle (ATSDR, 2000), but efficiency falls when soil is the medium of exchange. The EPA (2004) recommended ABS of 0.14 for PCBs is used in this evaluation. EPA (2004) provides values for K_p, τ, FA and B for 4-chlorobiphenyl and hexachlorobiphenyl. Generally, the more highly chlorinated PCB congeners are the more persistent in the environment; therefore, the values for hexachlorobiphenyl provided below are applied to all PCBs in this evaluation.

Chemical	K_p	τ	FA	B
Hexachlorobiphenyl	4.3E-1	1.13E+1	0.5	3.2

K_p = permeability coefficient (cm/hour); τ= lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).

Noncancer Effects Evaluation

The acute oral toxicity of the PCBs is low to moderate, as indicated by LD_{50} values in laboratory animals ranging from 750 mg/kg (mink) to 4250 mg/kg (rats) (ATSDR, 2000). Mink appear to be unusually sensitive, which is also true for prolonged oral exposure. Death appears to be due to respiratory depression and dehydration from diarrhea.

The best known incident involving oral exposure of humans to PCBs is the "Yusho" incident in Japan, in which persistent chloracne, gastrointestinal irritation and central nervous symptoms followed ingestion of cooking oil contaminated with PCBs (Gaffey, 1983). Further investigation, however, revealed that concentrations of polychlorinated dibenzofurans (PCDF) and polychlorinated quaterphenyls in the cooking oil were similar to those of PCBs, which confounds interpretation of the reported observations and symptoms.

Prolonged oral exposure of laboratory animals to various Aroclors leads to liver damage, signs of chloracne, immunological effects, and neurological impairment, particularly of the young (EPA, 2010). A verified oral RfD for Aroclor-1254 of 2E-5 mg/kg-day for chronic oral exposure is based on a LOAEL of 5E-3 mg/kg-day associated with chloracne and related signs and immunological effects in monkeys treated with the test material in gelatin capsules for over five years. An uncertainty factor of 300 was applied. Confidence in the RfD is medium. The immune system and skin are considered the target organs for prolonged oral exposure to Aroclor-1254.

EPA (1997) derived a provisional RfD for subchronic oral exposure to Aroclor-1254 of 5E-5 by applying an uncertainty factor of 100 to the LOAEL from the monkey study described above.

A verified oral RfD of 7E-5 mg/kg-day for Aroclor-1016 is based on a NOAEL of 7E-3 mg/kg-day in a perinatal and neurobehavioral toxicity study in monkeys (EPA, 2010). An uncertainty factor of 100 was applied to the NOAEL. The uncertainty factor of 100 consists of factors of 3 each to extrapolate from monkeys to humans, to provide greater protection for more sensitive humans, to account for deficiencies in the database, and to expand from a subchronic study to chronic exposure. The LOAEL (2.8E-2 mg/kg-day) was associated with low birth weights. The

fetus is considered to be the most sensitive member of the population. The fetus and nervous system are selected as "target organs" for prolonged oral exposure to Aroclor-1016. Confidence in the oral RfD is medium.

There is no EPA-derived subchronic oral RfD for exposure to Aroclor-1016. However, a preliminary RfD can be derived from the NOAEL of 7E-3 mg/kg-day in the monkey study described above by application of an uncertainty factor of 30. The uncertainty factor of 30 includes all the factors described above except the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 2.3E-4 mg/kg-day, which is expressed as 2E-4 mg/kg-day when rounded to one significant figure.

The similarities in the nature of the adverse effects and the thresholds for these effects for various Aroclors suggest that the adverse effects and thresholds across all the Aroclors may be similar. That this may be true is supported by the fact that many of the same PCB congeners are present in the different Aroclors, which differ from one another primarily only in the relative proportions of the individual congeners. Therefore, it is reasonable to apply the RfDs described above to any of the Aroclor mixtures, or to total PCBs. The chronic and subchronic oral RfDs developed for Aroclor-1016 rather than those developed for Aroclor-1254 are selected as surrogates for evaluating the adverse effects of the other Aroclors or total PCBs. The RfDs developed for Aroclor-1016 are preferred because they include less uncertainty than the RfDs developed for Aroclor-1254. Therefore, the verified chronic oral RfD of 7E-5 mg/kg-day and the preliminary subchronic oral RfD of 2E-4 mg/kg-day developed for Aroclor-1016 are applied to the Aroclors other than Aroclor-1254 or to total PCBs.

Occupational exposure to PCBs, which involved both inhalation and dermal exposure, was associated with upper respiratory tract and ocular irritation, loss of appetite, liver enlargement and increased serum concentrations of liver enzymes, skin irritation, rashes and chloracne, and, in heavily exposed female workers, decreased birth weight of their infants (ATSDR, 2000). Concurrent exposure to PCB contaminants, such as polychlorinated dibenzofurans, confounds the interpretation of the occupational exposure studies. Rats, mice, rabbits and guinea pigs intermittently exposed to Aroclor-1254 vapors exhibit moderate liver degeneration, decreased body weight gain and slight renal tubular degeneration; however, the accuracy of the reported exposure concentration is in doubt. Neither verified nor provisional chronic inhalation RfC values are available.

Carcinogenicity Evaluation

EPA (2010) declined to evaluate the carcinogenicity of the individual Aroclor mixtures. Instead, EPA (2010) classified PCBs in cancer weight-of-evidence Group B2 (probable human carcinogen) based on adequate evidence for liver tumors in laboratory animals and inadequate data in humans. EPA (2010) established a three-tiered approach for estimating the cancer potency of exposure to PCBs.

<u>High-Risk Tier</u> A SF of 2.0E+0 per mg/kg-day is verified as an upper-bound potency factor for exposure to PCBs in the high-risk tier (EPA, 2010). The high-risk tier includes ingestion in the food chain, incidental ingestion of soil or sediment, inhalation of dust or aerosol, or dermal contact with soil or sediment if a dermal absorption factor is applied. The high-risk SF of 2.0E+0 per mg/kg-day is used for mixtures containing any congeners considered to be persistent

or acting in a dioxin-like manner, and for any early life exposures (e.g., residential scenario, which includes both adults and children). In this context, persistent congeners are those with five or more chlorines. The 12 PCB congeners considered to act in a dioxin-like manner were identified above. The SF of 2.0E+0 per mg/kg-day is equivalent to a URF (for dust or aerosol inhalation) of 5.7E-4 per $\mu g/m^3$.

The high-risk tier SF for central tendency (CT) assessment is 1.0E+0 per mg/kg-day. The SF of 1.0E+0 per mg/kg-day is equivalent to a URF (for dust or aerosol inhalation) of 2.9E-4 per $\mu g/m^3$.

Low-Risk Tier EPA (2010) verified an upper-bound SF of 4E-1 per mg/kg-day for the low-risk tier, which includes ingestion of water-soluble congeners (assumed to be PCBs detected in drinking water), inhalation of evaporated congeners, and dermal exposure if no absorption factor is applied. The SF of 4E-1 per mg/kg-day is equivalent to a URF for vapor inhalation of 1E-4 per $\mu g/m^3$. A SF of 3E-1 per mg/kg-day is recommended for the low-risk CT evaluation. The SF of 3E-1 per mg/kg-day is equivalent to a URF (for vapor inhalation) of 9E-5 per $\mu g/m^3$.

Lowest-Risk Tier EPA (2010) verified an upper-bound SF of 7E-2 per mg/kg-day for the lowest-risk tier, which is restricted to data sets for which homologue-or congener-specific data verify that congeners containing 5 or more chlorines constitute less than 0.5 percent of the total PCBs. The SF of 7E-2 per mg/kg-day is equivalent to a URF for inhalation of 2E-5 per $\mu g/m^3$. These values are used in for Aroclor 1016. An SF of 4E-2 per mg/kg-day is recommended for the low-risk CT evaluation. The SF of 4E-2 per mg/kg-day is equivalent to a URF for vapor inhalation of 1E-5 per $\mu g/m^3$.

An alternative method of PCB cancer risk assessment was proposed by EPA (2000, 2010) for sites where congener-specific analytical results are available. This method involves application of toxicity equivalency factors (TEF) adopted by EPA (2000) to the 12 dioxin-like congeners identified above. TEFs are factors that express the toxicity of the dioxin-like congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD or "dioxin"), the reference compound. EPA (1997) presented a provisional oral and inhalation SF for 2,3,7,8-TCDD of 1.5E+5 per mg/kg-day. As explained in the toxicological profile for polychlorinated dibenzo-p-dioxins and dibenzofurans, the inhalation SF is more accurately expressed as 1.1E+5 per mg/kg-day, which is equivalent to 3.3E+1 per $\mu g/m^3$, or 3.3E+4 per mg/m^3. Application of the EPA (2000) TEFs to the dioxin-like PCB congeners yields the cancer potency factors presented below:

Dioxin-Like PCB Congener	IUPAC No.	TEF	SFo	URFi	SFi
3,3′,4,4′-Tetrachlorobiphenyl	77	0.0001	1.5E+1	3.3E+0	1.1E+1
3,4,4′,5-Tetrachlorobiphenyl	81	0.0001	1.5E+1	3.3E+0	1.1E+1
2,3,3′,4,4′-Pentachlorobiphenyl	105	0.0001	1.5E+1	3.3E+0	1.1E+1
2,3,4,4′,5-Pentachlorobiphenyl	114	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3′,4,4′,5-Pentachlorobiphenyl	118	0.0001	1.5E+1	3.3E+0	1.1E+1
2′,3,4,4′,5-Pentachlorobiphenyl 2,3′,4,4′,5′-Pentachlorobiphenyl)	123	0.0001	1.5E+1	3.3E+0	1.1E+1
3,3′,4,4′,5-Pentachlorobiphenyl	126	0.1	1.5E+4	3.3E+3	1.1E+4
2,3,3′,4,4′,5-Hexachlorobiphenyl	156	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3,3′,4,4′,5′-Hexachlorobiphenyl	157	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3′,4,4′,5,5′-Hexachlorobiphenyl	167	0.00001	1.5E+0	3.3E-1	1.1E+0
3,3′,4,4′,5,5′-Hexachlorobiphenyl	169	0.01	1.5E+3	3.3E+2	1.1E+3

Dioxin-Like PCB Congener	IUPAC No.	TEF	SFo	URFi	SFi
2,3,3′,4,4′,5,5′-Heptachlorobiphenyl	189	0.0001	1.5E+1	3.3E+0	1.1E+1

IUPAC No. = unique sequentially numbered congener identifier proposed by the International Union of Pure and Applied Chemists; TEF = toxicity equivalency factor (EPA, 2000); SFo = oral slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per milligram per cubic meter); SFi = inhalation slope factor (risk per milligram per kilogram-day).

Toxicity Summary

Cancer potency factors for the dioxin-like congeners are summarized above. Toxicity values for PCBs other than the dioxin-like congeners, are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Aroclor-1016[b]										
2E-4	7E-5	F	ND	ND	NA	Please see below.				
Aroclor-1254										
5E-5	2E-5	Is, S	ND	ND	NA	Please see below.				
PCBs, High-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote “b.”						B2	2.0E+0	B2	5.7E-1[c]	2.0E+0[c]
PCBs, High-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote “b.”						B2	1.0E+0	B2	2.9E-1[c]	1.0E+0[c]
PCBs, Low-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote “b.”						B2	4E-1	B2	1E-1[d]	4E-1[d]
PCBs, Low-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote “b.”						B2	3E-1	B2	9E-2[d]	3E-1[d]
PCBs, Lowest-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote “b.”						B2	7E-2	B2	2E-2	7E-2
PCBs, Lowest-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote “b.”						B2	4E-2	B2	1E-2	4E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per milligram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data.
Target organ abbreviations: F = unborn child; Is = immune system; S = skin; NA = not applicable.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.
[b]sRfDo and cRfDo derived for Aroclor-1016 should be applied to all other Aroclor mixtures except Aroclor-1254. They should also be applied to total PCBs, but not to specific PCB homologues congeners.

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
[c]Reasonable maximum exposure evaluation of inhalation pathways including dusts or aerosols. [d]Reasonable maximum exposure evaluation of inhalation pathways limited to vapors, excluding dusts or aerosols.										

PCB Risk Assessment

The approach to risk assessment of PCB-contaminated sites depends on the analytical data available. Typically, PCB analysis is limited to Method 8082 for the seven commonly encountered Aroclors. When this is the case, the hazard associated with noncancer effects is evaluated with the oral RfDs provided in the table above. Cancer risk is evaluated with the upper bound (reasonable maximum exposure) SFo and URFi or SFi from the "High-Risk" or "Low-Risk" scenarios, depending on the Aroclors identified, and the receptors and exposure pathways evaluated.

If homologue-specific analytical results are available in addition to results for the seven common Aroclors, it will be necessary to decide which is the better data set. This decision will depend, at least in part, on the samples included in each data set. Generally, the data set that better represents contamination across the area of investigation or a given exposure unit should be used in the risk assessment. If the same samples were analyzed for both Aroclors and specific homologues so that representativeness is not an issue, EPA (2010) generally considers that the homologue data are preferable because they are based on greater sensitivity (lower reporting limits) and are more likely to reflect selective removal of the more labile and mobile congeners due to weathering. Homologue-specific analytical results in the absence of data for the seven common Aroclors can be evaluated directly in the risk assessment.

The homologues should not be evaluated individually for noncancer hazard using the oral RfDs provided above. However, their sum (total PCBs) can be evaluated with the oral RfD for Aroclor-1016, assuming that the total PCBs reflect the characteristics of a mixture not unlike one or more of the Aroclors. If this assumption is not reasonable, the reason should be clearly stated, total PCBs should not be evaluated for noncancer hazard, and the uncertainty imparted to the risk assessment (probably a non-conservative bias) should be discussed. For example, it might be that only the most highly chlorinated homologues persist and are identified from an "old" release.

Cancer risk is estimated for total PCBs rather than the individual homologues. If the penta-, hexa-, hepta-, octa-, nona- and deca-chlorinated homologues together constitute not more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "Lowest-Risk" scenario should be used. If the penta-, hexa-, hepta-, nona- and deca-chlorinated homologues together constitute more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "High-Risk" or "Low-Risk" scenario should be used, depending on the receptors and exposure pathways evaluated.

It is unlikely that congener-specific data will be the only PCB data available. Should that be the case, however, the noncancer hazard can be estimated for total PCBs as described in the previous

paragraph. For cancer risk assessment, the dioxin-like congeners should be assessed as explained below. The non-dioxin-like congeners should be summed. If the penta-, hexa-, hepta-, nona- and deca-chlorinated congeners together constitute not more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "Lowest-Risk" scenario should be used. If the penta-, hexa-, hepta-, nona- and deca-chlorinated congeners together constitute more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "High-Risk" or "Low-Risk" scenario should be used, depending on the receptors and exposure pathways evaluated. The cancer risks from the non-dioxin-like and the dioxin-like congeners (see next paragraph) should be summed.

It is more likely that limited congener-specific data will be available *in addition to* data for the Aroclors or homologues. (Limited in the number of congeners analyzed and/or the number of samples analyzed for individual congeners.) For cancer risk assessment, EPA (2010) recommends evaluating the Aroclors or homologues as described above, after reducing their exposure-point concentrations by the contribution from the dioxin-like congeners. In most cases the concentrations of the dioxin-like congeners will be so much lower than the concentrations of the Aroclors or homologues that their subtraction would make little difference. The cancer risk for the dioxin-like congeners is assessed by using the TEF-based SFo and URFi or SFi values presented above. The two sets of risk estimates are then summed.

The newer trends in PCB analysis and risk assessment introduce additional sources of uncertainty to the assessment. PCBs tend to be somewhat ubiquitous because of their once-common use, potential for widespread dispersion, and persistence in environmental media (ATSDR, 2000). Method 1668 is a high-resolution method that is likely to identify very low "anthropogenic background" levels of PCBs that may not be reflected in the results of Aroclor analysis using Method 8082. This may impart an overly conservative bias to the risk assessment. Another source of uncertainty may arise from analyzing only a limited number or proportion of PCB-bearing samples for individual congeners. The limited analysis could impart either a conservative or non-conservative bias to the risk assessment, depending on the relative level of contamination in the samples selected for analysis of individual congeners.

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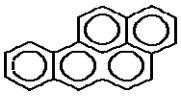
Semivolatile Organic Compounds

POLYNUCLEAR AROMATIC HYDROCARBONS a.k.a.
POLYCYCLIC AROMATIC HYDROCARBONS a.k.a.
***POLYAROMATIC HYDROCARBONS (PAH) (*130498-29-2)**



Benzo(a)Pyrene
(A representative and the most studied member of this class of compounds.)

1.0 Introduction and Physical Properties

The PAHs regularly observed in environmental media and addressed in this profile include acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, naphthalene, phenanthrene and pyrene. All exist as solids at room temperature. Classification as **SVOC** or **VOC** is made in the following table. PAHs are the products of incomplete combustion of fossil fuels or other organic matter, hence include both natural and anthropogenic sources (ATSDR, 1995a; HSDB, 2010). Several are also components of crude oil. Naphthalene is used in the synthesis of phthalic anhydride, the insecticide carbaryl, leather tanning agents and surface active agents, and is a component of diesel and other fuels (ATSDR, 1995b). Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Acenaphthene (83-32-9) **(VOC)**										
154.21[a]	3.92[a]	1.55E-4[b] (25°C)	3.85[b]	4.21E-2[b] (25°C)	7.69E-6[b] (25°C)	3.29E-6[a] (25°C)	4.24E+0[b] (20-25°C)	550.54[c]	803.15[c]	1.22E+4[c]
Acenaphthylene (208-96-8) **(VOC)**										
152.20[a]	4.07[a]	1.13E-5[a] (25°C)	3.42[a]	6.67E-2[d] (25°C)[e]	7.72E-6[d] (25°C)[e]	1.20E-6[a] (25°C)	3.93E+0[a] (25°C)	543.2[a]	797[f]	1.1E+4[g]
Anthracene (120-12-7) **VOC)**										
178.23[a]	4.45[a]	6.51E-5[b] (25°C)	4.47[b]	3.24E-2[b] (25°C)	7.74E-6[b] (25°C)	3.51E-9[a] (25°C)	4.34E-2[b] (20-25°C)	615.18[c]	873.00[c]	1.31E+4[c]
Benzo(a)anthracene (56-55-3) **(SVOC)**										
228.3[h]	5.66[h]	3.34E-6[b] (25°C)	5.60[b]	5.10E-2[b] (25°C)	9.00E-6[b] (25°C)	2.50E-9[a] (25°C)	9.40E-3[b] (20-25°C)	708.15[c]	1004.8[c]	1.60E+4[c]
Benzo(a)pyrene (50-32-8) **(SVOC)**										
252.3[a]	6.10[h]	1.13E-6[b] (25°C)	6.01[b]	4.30E-2[b] (25°C)	9.00E-6[b] (25°C)	7.22E-12[a] (25°C)	1.62E-3[b] (20-25°C)	715.90[c]	969.27[c]	1.90+4[c]
Benzo(b)fluoranthene (205-99-2) **(VOC)**										
252.3[h]	6.12[h]	1.11E-4[b] (25°C)	6.09[b]	2.26E-2[b] (25°C)	5.56E-6[b] (25°C)	6.58E-10[a] (20°C)	1.50E-3[b] (20-25°C)	715.90[c]	969.27[c]	1.70+4[c]
Benzo(k)fluoranthene (207-08-9) **(SVOC)**										
252.32[a]	6.84[a]	8.29E-7[b] (25°C)	6.09[b]	2.26E-2[b] (25°C)	5.56E-6[b] (25°C)	1.28E-12[a] (25°C)	8.00E-4[b] (20-25°C)	753.15[c]	1019.7[c]	1.80E+4[c]
Benzo(g,h,i)perylene (191-24-2) **(SVOC)**										
276.34[a]	6.63[a]	2.66E-7[a] (20°C)	4.98[a]	4.48E-2[d] (25°C)[e]	5.19E-6[d] (25°C)[e]	1.3E-13[a] (25°C)	2.6E-4[a] (25°C)	823[a]	1097[f]	1.96E+4[g]
Chrysene (218-01-9) **(VOC)**										
228.3[h]	5.66[h]	9.46E-5[b] (25°C)	5.60[b]	2.48E-2[b] (25°C)	6.21E-6[b] (25°C)	8.20E-12[a] (25°C)	1.60E-3[b] (20-25°C)	714.15[c]	979.0[c]	1.65E+4[c]
Dibenz(a,h)anthracene (53-70-3) **(SVOC)**										
278.4[h]	6.84[h]	1.47E-8[b] (25°C)	6.58[b]	2.02E-2[b] (25°C)	5.18E-6[b] (25°C)	1.32E-13[a] (20°C)	2.49E-3[b] (20-25°C)	743.24[c]	990.41[c]	3.00E+4[c]
Fluoranthene (206-44-0) **(VOC)**										
202.3[h]	4.95[h]	1.61E-5[b] (25°C)	5.03[b]	3.02E-2[b] (25°C)	6.35E-6[b] (25°C)	1.21E-8[a] (25°C)	2.06E-1[b] (20-25°C)	655.95[c]	905.0[c]	1.38E+4[c]

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Fluorene (86-73-7) **(VOC)**										
166.21[a]	4.18[i]	6.37E-5[b] (25°C)	4.14[b]	3.63E-2[b] (25°C)	7.88E-6[b] (25°C)	4.21E-7[a] (20°C)	1.98E+0[b] (20-25°C)	570.44[c]	870.0[c]	1.27E+4[c]
Indeno(1,2,3-cd)pyrene (193-39-5) **(SVOC)**										
276.3[h]	6.58[h]	1.60E-6[b] (25°C)	6.54[b]	1.90E-2[b] (25°C)	5.66E-6[b] (25°C)	1.71E-13[a] (25°C)	2.20E-5[b] (20-25°C)	809.15[c]	1078.2[c]	1.90E+4[c]
Naphthalene (91-20-3) **(VOC)**										
128.2[h]	3.30[h]	4.83E-4[b] (25°C)	3.30[b]	5.90E-2[b] (25°C)	7.50E-6[b] (25°C)	1.12E-4[a] (25°C)	3.10E+1[b] (20-25°C)	491.14[c]	748.40[c]	1.04E+4[c]
Phenanthrene (85-01-8) **(VOC)**										
178.2[h]	4.46[h]	1.24E-4[a] (25°C)	4.36[a]	6.00E-2[d] (25°C)[e]	6.95E-6[d] (25°C)[e]	8.95E-7[a] (25°C)	1.29E+0[a] (25°C)	613.2[a]	869.2[j]	1.42E+4[a]
Pyrene (129-00-0) **(VOC)**										
202.26[a]	4.88[a]	1.1E-5[b] (25°C)	5.02[b]	2.72E-2[b] (25°C)	7.24E-6[b] (25°C)	1.17E-7[a] (25°C)	1.35E-1[b] (20-25°C)	667.95[c]	936.0[c]	1.44E+4[c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 23 April.
[b]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.
[c]U.S. Environmental Protection Agency (EPA), 2000, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings, Revised***, Office of Emergency and Remedial Response, Washington, DC, December.
[d]Calculated as described in Introduction to Toxicological profiles.
[e]Assumed.
[f]Estimated from Equation 12-4 in Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[g]Estimated from Equation 13-5 in Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[h]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)***, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.
[i]Syracuse Research Corporation (SRC), 2004, ***CHEMFATE Search Results***, on line, accessed 23 April.
[j]National Institute of Standards and Technology (NIST), 2004, Standard Reference Data Program, Online Data Bases, accessed 23 April.

2.0 Environmental Fate and Transport

The PAHs are ubiquitous products of incomplete combustion; natural sources include volcanoes and forest fires (ATSDR, 1995a, b; HSDB, 2010). There is some evidence for biosynthesis by plants, bacteria and algae. Some of the PAHs occur naturally in fossil fuels. Anthropogenic releases to the environment, primarily to the atmosphere, greatly outweigh the natural sources and include any processes that involve incomplete combustion of fossil fuels and organic matter, including wood-burning for home heat (the predominant source), cigarette smoke, internal combustion engine exhaust, and fuel oil emissions. Industrial sources include coal mining, processing and storing, wood treatment (creosote), manufactured gas plants (coal tar), power generation, production of coal tar, coke and asphalt, petroleum cracking and industrial and municipal incineration. Other sources include various crude oils, fresh and used motor oils, gasolines, charcoal-broiled foods, processed foods, various oils, margarine, butter and fats, fruits, vegetables, and cereals, roasted coffee and tea. Indoor sources include unvented kerosene heaters and gas cooking and heating appliances. Naphthalene is released during its manufacture and processes that involve its use (e.g., vaporization from moth balls).

Although PAHs generally occur in mixtures, certain chemicals tend to predominate in different sources so that the PAH pattern observed in a given medium may provide a clue to the source. The following compilation relates individual PAHs and sources:

Source[a]	Individual PAHs
Residential wood burning	Acenaphthylene
Auto emissions	Benzo(g,h,i)perylene, pyrene
Diesel exhaust particulates	Fluoranthene, phenanthrene, pyrene
Diesel exhaust vapors	Phenanthrene, anthracene
Diesel total emissions	Acenaphthene, fluorene, phenanthrene

Source[a]	Individual PAHs
Fly ash & bottom ash from U.S. municipal waste incinerators	Phenanthrene
Fly ash from U.K. municipal waste incinerators	Benzo(g,h,i)perylene
Particulate emissions from municipal waste incinerator	Benzo(a)fluoranthene, benzo(g,h,i)perylene, chrysene, fluoranthene, indeno(1,2,3-cd)pyrene, phenanthrene
Municipal/medical waste incinerator	Benzo(a)anthracene, benzo(g,h,i)perylene
Rotary kiln incinerator charged with polyethylene, no afterburner	Fluoranthene, phenanthrene, pyrene (see next entry)
Rotary kiln incinerator charged with polyethylene with afterburner	Benzo(a)anthracene, phenanthrene (total PAH emissions reduced 100-fold compared with no afterburner)
Coal tar pitch emissions	Phenanthrene and pyrene 20 to 80 X > benzo(a)pyrene, benzo(g,h,i)perylene
Natural gas home appliances – fine particulate emissions	Chrysene, fluoranthene, pyrene, triphenylene
Groundwater near coal & oil gasification plant	Acenaphthylene, acenaphthene, fluorene, phenanthrene, fluoranthene, pyrene, chrysene
Groundwater near wood treatment facilities	Benzo(a)pyrene, phenanthrene
[a]Agency for Toxic Substances and Disease Registry, (ATSDR), 1995, ***Update Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)***, U.S. Public Health Service, Atlanta, Georgia, August, on line.	

Data compiled for soils are not included in the table above because the lists are long and the variation from one site to another is great, so that the information is not useful for identifying sources. Similarly, sediment is a sink for PAHs from all sources including atmospheric deposition from far distant locations, and the patterns observed in sediment do not necessarily reflect a nearby source (ATSDR, 1995a). Generally, PAHs associated with combustion exhibit highly condensed ring structures and little alkylation (i.e., benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, benzo(e)pyrene, chrysene, fluoranthene, pyrene).

Crude petroleum and products made from crude petroleum (e.g., asphalts, fuels, oils) contain PAHs, but the patterns observed depend on the location of the source (ATSDR, 1995a; Potter and Simmons, 1998). Phenanthrene and alkylated forms of PAHs, particularly the methylnaphthalenes, often predominate in petroleum products.

The partitioning and fate of the PAHs in environmental media depend largely on VP (tendency to exist in air as a vapor), H (indicator of partitioning between air and water) and K_{oc} (indicator of affinity to bind to organic matter in soil and sediment). ATSDR (1995a) noted that H and K_{oc} are roughly directly related to MW, and that VP is roughly inversely related to MW. Therefore, they grouped the PAHs into the following categories to facilitate understanding their behavior in the environment:

- Low MW (152 to 178 g/mole) – acenaphthene, acenaphthylene, anthracene, fluorene, naphthalene, phenanthrene

- Medium MW (approximately 202 g/mole) – fluoranthene, pyrene

- High MW (228 to 278 g/mole) – benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene.

PAHs exist in the atmosphere as vapors or adsorbed to particulates, the proportion depending on the vapor pressure of the individual chemical and atmospheric conditions such as temperature and humidity (ATSDR, 1995a, b; HSDB, 2010). Generally, the two- and three-ring compounds (low and medium MW except pyrene) exist predominantly as vapors; the four-ring compounds exist in both the vapor and particulate phase, and compounds with 5 or more rings (benzo[a]pyrene, benzo[g,h,i]perylene exist predominantly as particulates. Increasing atmospheric temperature and relative humidity favors existence in the vapor phase. PAHs may travel short or long distances before removal from the air. Vapor forms are subject to chemical oxidation processes in the air, which reduces the distance they may travel. Residence time and transport distance is inversely related to the size of the particles on which the PAHs are adsorbed. Small particles may have residence times of weeks, permitting transport for hundreds or upwards of a thousand miles. Wet and dry deposition accounts for removal of the particulates.

The predominant sources of PAHs in surface water are deposition from the atmosphere, industrial and sewage effluent and oil spills (ATSDR, 1995a, b). Runoff and erosion can also contribute PAHs to surface water bodies. Volatilization is a significant fate process for PAHs with H values of 1E-5 atm-m^3/mole or greater (generally the low and medium MW compounds). Volatilization is a very limited removal process for most of the high MW compounds. Volatilization is enhanced by high temperature, turbulence and high wind. Sorption to benthic or suspended sediment and biodegradation are competing removal processes. Generally, volatilization and biodegradation are the predominant removal processes for the low MW compounds, and volatilization and sorption are the predominant removal processes for medium and high MW compounds. Naphthalene is relatively water soluble and may remain largely in solution. PAHs in sediment may biodegrade, recycle back to the water column (lower MW compounds) or accumulate in the lower trophic levels of living organisms.

Deposition from the atmosphere is the principal source of PAHs in soil (ATSDR, 1995a, b). Other sources include industrial activities, disposal of sewage sludge, and leaching from coal storage sites. Most PAHs sorb to soil constituents because of their low solubility and high affinity for organic matter. Volatilization is an important removal process for the low molecular weight compounds. Some of the low molecular weight compounds, particularly naphthalene, may leach fairly rapidly to groundwater.

The high lipophilicity of many PAHs evidenced by high log K_{ow} values suggest that the PAHs might cross biologic membranes and participate significantly in food-chain pathways involving fish, fruit and vegetable, and meat and milk consumption. This, however, is not the case. For example, the PAHs are efficiently metabolized and excreted by fin fish, greatly reducing the likelihood of bioconcentration from water or bioaccumulation from sediment (ATSDR, 1995a). Consequently, there are often great discrepancies between modeled BCF values and empirical BCF values, where care was taken to identify the parent compound rather than PAH metabolites in fish tissue. This distinction is necessary because aromatic fragments from PAH metabolism are readily assimilated as normal endogenous components of biological tissue. PAH concentrations in aquatic food-chain systems usually decrease with increasing trophic level, reflecting the generally greater efficiency of metabolism in the higher trophic organisms. Approximate tissue-to-sediment PAH concentration ratios were reported to be 0.6 to 1.2 for amphipods, 0.1 for clams, and 0.05 for fin fish and shrimp.

It has been reported that terrestrial plants can accumulate PAHs from soil by uptake through the roots (ATSDR, 1995a). More careful investigation, however, reveals that root uptake is quite low, and there is no evidence of bioconcentration or biomagnification. For example, PAH ratios in vegetation to soil in one survey ranged from 0.001 to 0.18 for total PAHs, and from 0.002 to 0.33 for benzo(a)pyrene. It is likely that most of the PAH contamination on the vegetation resulted from atmospheric deposition. Increased concentrations in vegetation have not been observed when soil PAH concentrations were greatly increased by amendment with sewage sludge. Data from carrots (*Daucus carotu*) showed that PAHs adhere to the outer skin of the root but show little tendency to penetrate to deeper layers, and even less tendency to translocate to aerial parts.

The inability of plants to bioconcentrate PAHs from soil reduces concern for bioconcentration in agricultural products such as meat and milk. It should be noted, however, that measurable concentrations of PAHs may accumulate on the aerial parts of plants because of deposition from the atmosphere (ATSDR, 1995a). Also, food-producing animals ingest a substantial amount of soil while grazing or consuming mechanically harvested forage crops. Mammals, however, efficiently metabolize the PAHs to polar compounds that are readily excreted, reducing concern for bioconcentration.

In summary, the PAHs are highly lipophilic, which implies significance in food-chain exposure pathways. Empirical data, however, show that fish and mammals efficiently metabolize and eliminate these compounds. Furthermore, bioconcentration in plants because of uptake from soil is not significant. Therefore, it is unlikely that the PAHs would participate significantly in food-chain pathways, and biotransfer factors are not estimated.

3.0 Toxicokinetics

The PAHs are absorbed by all routes of exposure, but the rate and extent depends on the compound, the species of animal and the vehicle (or nature of the particulates) (ATSDR, 1995a). PAHs are readily absorbed during inhalation exposure; however, intratracheal instillation studies indicate that particle size is the most important determiner of the extent of pulmonary uptake.

Toxicokinetic studies of several PAHs summarized by ATSDR (1995a) provide limited quantitative information regarding the extent of GI absorption. Limitations arise largely because biliary excretion complicates quantification of uptake from the GI tract. A study of benzo(a)pyrene in rats suggests that GI absorption ranges from 38 to 58 percent. A study in rats reported absorption efficiency for anthracene ranging from 53 to 74 percent. GI absorption of pyrene, chrysene and dibenzo(a,h)anthracene is described as high. Administration of the test compound in oil or in a high-fat diet appears to increase the extent of GI absorption.

Empirical data with pure compound dissolved or suspended in various vehicles suggest that dermal uptake of benzo(a)pyrene is extensive (ATSDR, 1995a). One occupational study reported that approximately 75 percent of systemically absorbed pyrene entered the body through dermal uptake rather than through inhalation exposure. Combining PAHs with soil appears to significantly reduce the extent of dermal uptake compared with oleaginous or acetone vehicles. Anecdotal evidence from using cloth diapers stored in contact with naphthalene indicates that naphthalene is absorbed by the skin, but quantitative data are not available (ATSDR, 1995b).

Distribution of absorbed PAHs is generally widespread, with highest levels located initially in lipid-rich tissues (ATSDR, 1995a). Highest levels of metabolites (radioactivity following administration of radiolabeled compounds) are located in the liver and GI tract, even after inhalation exposure, probably reflecting extensive metabolism in the liver followed by biliary excretion. Ciliary clearance and deglutition probably contribute to levels associated with the GI tract. Concentrations of radioactivity following administration of radiolabeled compound reveal fetal levels approximately 2- to 10-fold lower than maternal levels, although this depends on the specific compound administered.

Metabolism of the PAHs, particularly benzo(a)pyrene, has been extensively studied (ATSDR, 1995a). Metabolism proceeds rapidly, yielding products that are more water soluble and readily excreted than the parent compound. All tissues have the ability to metabolize the PAHs, potentially to carcinogenic intermediates, which probably accounts for the observation that cancers occur at the point of contact. There is considerable variability in tissue metabolic activity; however, the liver is probably the most active in most cases.

ATSDR (1995a) distinguishes between "alternant" and "nonalternant" PAHs, based on the nature of the electron density associated with the molecule, which influences how that compound is metabolized to its ultimate carcinogen. Alternant PAHs (e.g., benzo[a]pyrene, benzo[a]anthracene, chrysene, dibenz[a,h]anthracene) exhibit a uniformly distributed electron density. Nonalternant PAHs (e.g., fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene) behave more like two separate molecules because of uneven electron distribution.

Alternant PAHs (based on data for benzo[a]pyrene) are initially metabolized by the microsomal cytochrome P-450 system to several arene oxides (ATSDR, 1995a). These may re-arrange spontaneously to phenols or undergo hydration to form the corresponding trans-dihydrodiols. Further oxidation of the dihydrodiols results in formation of quinones, phenol diols and dihydrodiol epoxides. Phenols may be formed by direct insertion of oxygen into a ring. Many of the metabolites resulting from the reactions described above are subject to conjugation with various substrates followed rapidly by excretion. The dihydrodiol epoxides that form in the "bay" region (the three-sided concave region formed by the fusion of three benzene rings) are most likely the ultimate carcinogens that covalently bind to macromolecules such as DNA, resulting in alkylation or other adducts that yield genetic errors.

Metabolism of the nonalternant PAHs differs from the alternant PAHs in that more extensive oxidation to hydroxy-epoxy-diols may be important to achieve genotoxicity (ATSDR, 1995a). Dihydrodiol epoxide formation in the bay region is associated with carcinogenicity of the PAHs generally; however, some of the nonalternant PAHs yield reactive metabolites that deviate from the classical bay region model.

PAH metabolites are readily excreted, largely through the bile, although some metabolites are excreted by the kidney (ATSDR, 1995a). The extent of elimination is species specific. In one inhalation study rats excreted metabolites much more efficiently than dogs or monkeys.

4.0 Dermal Exposure

EPA (2004) recommends that oral toxicity values for the PAHs should not be adjusted when applied to dermal exposure because GI absorption probably exceeds 50 percent. Therefore, no GAF is estimated and the oral toxicity values described below should be used for dermal exposure without adjustment.

Empirical data with pure compound dissolved or suspended in various vehicles suggest that dermal uptake of benzo(a)pyrene is extensive (ATSDR, 1995a). Anecdotal evidence from using cloth diapers stored in contact with naphthalene indicates that naphthalene is absorbed by the skin, but quantitative data are not available (ATSDR, 1995b). EPA (2004) reviewed empirical data regarding dermal uptake of benzo(a)pyrene and recommended an ABS of 0.13 for the PAHs. However, EPA (2004) also notes that VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels. Therefore, the ABS of 0.13 is applied to all the PAHs identified above as SVOCs. Dermal uptake of the PAHs identified as VOCs is not quantified.

Values for t*, K_p, τ, FA and B are provided or estimated as follows:

Chemical	t*	K_p	τ	FA	B
Acenaphthene[a]	1.84	8.39E-2	7.67E-1	1	0.4
Acenaphthylene[a]	1.79	1.08E-1	7.47E-1	1	0.5
Anthracene[a]	4.06	1.38E-1	1.05E+0	1	0.7
Benzo(a)anthracene[b]	8.53	4.7E-1	2.03E+0	1.0	2.8
Benzo(a)pyrene[b]	11.67	7.0E-1	2.69E+0	1.0	4.3
Benzo(b)fluoranthene[b]	12.03	7.0E-1	2.77E+0	1.0	4.3
Benzo(k)fluoranthene[a]	12.40	2.00E+0	2.72E+0	0.4	12
Benzo(g,h,i)perylene[a]	8.89	1.07E+0	3.70E+0	0.7	6.8
Chrysene[b]	8.53	4.7E-1	2.03E+0	1.0	2.8
Dibenz(a,h)anthracene[b]	17.57	1.5E+0	3.88E+0	0.6	9.7
Fluoranthene[b]	5.68	2.2E-1	1.45E+0	1.0	1.2
Fluorene[a]	2.15	1.07E-1	8.95E-1	1	0.5
Indeno(1,2,3-cd)pyrene[b]	16.83	1.0E+0	3.78E+0	0.6	6.7
Naphthalene[b]	1.34	4.7E-2	5.6E-1	1	0.2
Phenanthrene[b]	4.11	1.4E-1	1.06E+0	1	0.7
Pyrene[a]	3.42	1.94E-1	1.43E+0	1	1.1

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Estimated as described in Introduction to Toxicological profiles.
[b]Provided by U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)***, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

5.0 Noncancer Effects Evaluation

The PAHs are generally divided into two EPA cancer weight-of-evidence groups: Group D – not classifiable as to carcinogenicity to humans, and Group B2 – probable human carcinogens. The Group D PAHs have not been evaluated for carcinogenicity by toxicity testing; therefore, cancer SFs or URFs cannot be estimated for these compounds (EPA, 1986, 2010). Risk evaluation of these compounds is limited to noncancer effects. It is theoretically possible that cancer risk could be the "driver" for some of these compounds. However, the mechanism of carcinogenicity of the PAHs is fairly well understood to be correlated with molecular morphology and the

propensity to form certain active metabolites (ATSDR, 1995a). The Group D PAHs either do not fit the morphologic mold or have been shown empirically to be unlikely to cause cancer, reducing greatly the uncertainty that significant cancer risk is being overlooked.

Cancer SFs and URFs are available for the Group B2 compounds, but noncancer RfDs or RfCs are not. Therefore, risk evaluation of these compounds is limited to cancer risk. These compounds have the morphologic requirements for carcinogenicity; therefore, it has been thought that cancer risk is the driver, and that noncancer effects are relatively insignificant, although empirical data were lacking until recently.

Recent data support this assumption. De Jong et al. (1999) reported a study in which male rats were treated by gavage with benzo(a)pyrene 5 days per week for 35 days at dose rates of 0 (control), 3, 10, 30 or 90 mg/kg. Significantly reduced rate of body weight gain and altered organ weights were observed in the 90 mg/kg group. Fore stomach lesions were found in the 30 and 90 mg/kg groups. Decreased thymus weights and hematological evidence of erythrocyte toxicity were observed in a dose-related manner in rats treated with 10 mg/kg and above. Subtle alterations in measures of immune function were also observed in these groups, establishing 10 mg/kg as the LOAEL and 3 mg/kg as the NOAEL for this study. The 3 mg/kg dose is equivalent to a NOAEL of 2.1 mg/kg-day when adjusted for continuous exposure. Application of an uncertainty factor of 1000 (factor of 10 to expand from subchronic to chronic exposure, and factors of 10 each to provide additional protection for intra- and interspecies variation) allows development of a preliminary oral RfD of 2E-3 mg/kg-day. Uncertainty surrounding the preliminary oral RfD is very high because the data base for the noncancer effects of benzo(a)pyrene is essentially limited to one study and several toxicological endpoints (e.g., developmental, reproductive, neurological) were not investigated.

The sole purpose for developing this oral RfD is to evaluate the potential for noncancer effects to be the driver for the Group B2 PAHs. The RfD was not developed with sufficient rigor to be used in the risk assessment of the noncancer effects of benzo(a)pyrene. The oral SF for benzo(a)pyrene is 7.3 per mg/kg-day (please see below), from which it is estimated that the oral RfD is equivalent to a cancer risk of 1.5E-2. This cancer risk is orders of magnitude above the EPA (1990) risk management range of 1E-6 to 1E-4, strengthening the position that noncancer effects are unlikely to be the driver for the Group B2 PAHs.

Data regarding the toxicity of acute oral exposure to the PAHs are generally scarce. Prolonged oral exposure to the Group D PAHs is associated with a number of renal, hematologic and other effects, depending on the compound.

Subchronic (90 day) gavage treatment of mice with acenaphthene is associated with histopathologic evidence of liver hypertrophy. A verified RfD of 6E-2 mg/kg-day for chronic oral exposure was derived from the NOAEL of 175 mg/kg-day and an uncertainty factor of 3000 (EPA, 2010). The LOAEL in this study was 350 mg/kg-day. Confidence in the RfD is low. The liver is considered the target organ for prolonged oral exposure to acenaphthene. EPA (1997) derived a provisional subchronic oral RfD for acenaphthene of 6E-1 mg/kg-day from the same mouse study using an uncertainty factor of 300.

A verified RfD of 3E-1 mg/kg-day for chronic oral exposure to anthracene was derived from a NOEL of 1000 mg/kg-day, the highest dose tested, in a 90-day gavage study in mice (EPA, 2010). An uncertainty factor of 3000 was applied. Confidence in the RfD is low. The data are inadequate to identify a target organ for prolonged oral exposure to anthracene. EPA (1997) derived a provisional subchronic oral RfD for anthracene of 3E+0 mg/kg-day from the same mouse study using an uncertainty factor of 300.

Subchronic exposure to fluoranthene induces liver and kidney effects and hematologic alterations in orally treated mice (EPA, 2010). A verified RfD of 4E-2 mg/kg-day for chronic oral exposure was derived from a NOAEL of 125 mg/kg-day in a 13-week gavage study. The LOAEL was 250 mg/kg-day in this study. An uncertainty factor of 3000 was applied. Confidence in the oral RfD is low. The kidney, liver and blood cells are chosen as the target organs for prolonged oral exposure to fluoranthene. EPA (1997) derived a provisional subchronic oral RfD for fluoranthene of 4E-1 mg/kg-day from the same mouse study using an uncertainty factor of 300.

Subchronic exposure to fluorene induces hemolytic anemia in orally treated mice (EPA, 2010). A verified RfD of 4E-2 mg/kg-day for chronic oral exposure was derived from a NOAEL of 125 mg/kg-day in a 13-week gavage study. The LOAEL was 250 mg/kg-day in this study. An uncertainty factor of 3000 was applied. Confidence in the oral RfD is low. The erythrocyte is the target organ for prolonged oral exposure to fluorene. EPA (1997) derived a provisional subchronic oral RfD for fluorene of 4E-1 mg/kg-day from the same mouse study using an uncertainty factor of 300.

Single-dose LD_{50} values for naphthalene include 533 to 710 mg/kg for mice and 2200 to 2400 mg/kg for rats, establishing the mouse as more sensitive to the lethal effects of acute oral exposure (ATSDR, 1995b). Decreased terminal body weights, accompanied by a remarkable absence of hematological and histopathological effects, were observed in rats treated by gavage for 13 weeks (EPA, 2010). The LOAEL in this study was 142 mg/kg-day associated with greater than 10 percent reduction in terminal body weights. The NOAEL was 71 mg/kg-day. The high dose rate, 286 mg/kg-day was a FEL associated with increased mortality. Application of an uncertainty factor of 3000 (10 to extrapolate from rats to humans, 10 to protect sensitive humans, 10 to extrapolate from subchronic to chronic exposure, and 3 for database deficiencies) to the NOAEL of 71 mg/kg-day yields the verified chronic oral RfD of 2E-2 mg/kg-day. Confidence in the RfD is low. A preliminary subchronic oral RfD can be derived for naphthalene by applying an uncertainty factor of 300 to the NOAEL of 71 mg/kg-day described above. The uncertainty factor of 300 reflects the chronic uncertainty factor of 3000 without the factor of 10 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 2E-1 mg/kg-day.

The key study described above is not sufficient to identify target organs for prolonged oral exposure to naphthalene. Adults, children and neonates exposed to moth balls exhibit hemolytic anemia, evidence of liver disease and neurological deficits (EPA, 1993a, 1998). The liver effects and neurological deficits may be secondary to hemolytic anemia and reduced oxygen-carrying capacity of the blood. Hemolytic anemia and cataract formation have been seen also in orally exposed humans (EPA, 1998). The data suggest that the erythrocyte may be the most sensitive tissue in humans. Among common species of laboratory mammals, hemolytic anemia is seen

only in dogs (EPA, 1998). Cataracts are seen in several laboratory mammals, but only at relatively high doses. The key study identifies reduced body weight as the critical effect in rats. Rats, however, do not exhibit hemolytic anemia, and exhibit cataracts only at very high doses, suggesting that rats may not be a totally acceptable model for the toxicity of naphthalene to humans. Therefore, based on the effects observed in humans, the erythrocyte and eye are selected as target organs for prolonged oral exposure to naphthalene. Reduced body weight is also included as a critical effect because this was the only endpoint observed in rats in the key study.

EPA (1993a, 2010) reported a 2-year study in which mice were exposed to naphthalene vapors for 6 hours/day on 5 days/week. Inflammation of the nasal and olfactory epithelium was the most consistently observed sign; granulomatous lesions in the lungs were also observed. There was equivocal evidence of hematologic involvement. The lowest exposure concentration, 10 ppm, was a LOAEL for the nasal effects, which are considered the critical effects of inhalation exposure. The LOAEL is equivalent to a human equivalent concentration of 9.3 mg/m^3 (EPA, 2010). Application of an uncertainty factor of 3000 yields a verified chronic inhalation RfC of 3E-3 mg/m^3, which is equivalent to a chronic inhalation RfD of 8.6E-4 mg/kg-day. The nasal and olfactory epithelia are the target organs for inhalation exposure to naphthalene. Confidence in the RfC is medium. The chronic inhalation RfC of 3E-3 mg/m^3, equivalent to an inhalation RfD of 8.6E-4 mg/kg-day, is adopted as sufficiently protective for subchronic inhalation exposure as well.

Subchronic exposure to pyrene induces mild renal tubular degeneration and reduced kidney weight in orally treated mice (EPA, 2010). A verified RfD of 3E-2 mg/kg-day for chronic oral exposure was derived from a NOAEL of 75 mg/kg-day in a 13-week gavage study. The LOAEL was 125 mg/kg-day in this study. An uncertainty factor of 3000 was applied. Confidence in the oral RfD is low. The kidney tubule is chosen as the target organ for chronic oral exposure to pyrene. EPA (1997) derived a provisional subchronic oral RfD for pyrene of 3E-1 mg/kg-day from the same mouse study using an uncertainty factor of 300.

Data regarding prolonged oral exposure are not available for several of the Group D PAHs, which hinders estimation of an RfD or RfC and compromises evaluation of the potential for noncancer effects. Therefore, surrogates are used to develop toxicity values for the noncancer effects.

Generally surrogates are chosen on the basis of the following hierarchy:

- Toxicological similarity (effects and dose-response relationship).
- Toxicokinetic similarity, assuming that likeness in absorption, distribution and especially products of biotransformation suggests toxicological similarity.
- Structural similarity, assuming that likeness in structure suggests similarity in toxicokinetics.

Selection of defensible surrogates for the PAHs is compromised because toxicological and toxicokinetic data are virtually non-existent (except for benzo[a]pyrene), and the structural

similarities often are not very convincing, which imparts a great deal of uncertainty to the effort. Therefore, the most defensible approach for some of the PAHs is to select the most conservative surrogate; i.e., the PAH with the smallest verified oral RfD, which happens to be pyrene.

Data regarding the effects of chronic or subchronic exposure to acenaphthylene were not located in the available literature. Acenaphthene is adopted as a reasonable surrogate for acenaphthylene based on structural similarity, since the surrogate differs from the principal chemical only in the presence of two hydrogen atoms and the absence of a double bond. Therefore, the verified chronic oral RfD of 6E-2 mg/kg-day for acenaphthene is adopted as the RfD for chronic oral exposure to acenaphthylene. The liver, which is the target organ for acenaphthene, is adopted for oral exposure to acenaphthylene. Similarly, the provisional subchronic oral RfD of 6E-1 mg/kg-day for acenaphthene is adopted as the RfD for subchronic oral exposure to acenaphthylene.

Data regarding the effects of chronic or subchronic exposure to benzo(g,h,i)perylene were not located in the available literature. Pyrene is adopted as a reasonable surrogate for benzo(g,h,i)perylene based somewhat on structural similarity, but more on the selection of a conservative approach as justified above. Therefore, the verified oral RfD of 3E-2 mg/kg-day for pyrene is adopted as the oral RfD for chronic exposure to benzo(g,h,i)perylene. Similarly, the provisional subchronic oral RfD of 3E-1 mg/kg-day for pyrene is adopted as the RfD for subchronic oral exposure to benzo(g,h,i)perylene. The kidney tubule, which is the target organ for pyrene, is adopted for oral exposure to benzo(g,h,i)perylene.

Relevant data regarding chronic or subchronic exposure to phenanthrene were not located. Potential surrogates based on similarity in chemical structure include anthracene and pyrene. Pyrene is selected as the surrogate only because it is the more conservative choice. Therefore, the verified oral RfD of 3E-2 mg/kg-day for pyrene is adopted as the oral RfD for chronic exposure to phenanthrene. Similarly, the provisional subchronic oral RfD of 3E-1 mg/kg-day for pyrene is adopted as the RfD for subchronic oral exposure to phenanthrene. The kidney tubule, which is the target organ for pyrene, is adopted for oral exposure to phenanthrene.

Data regarding inhalation exposure sufficient for development of inhalation RfCs were not located for any of the PAHs with the exception of naphthalene as noted above.

6.0 Carcinogenicity Evaluation

Acenaphthylene, anthracene, benzo(g,h,i)perylene, fluoranthene, fluorene, phenanthrene and pyrene are classified in EPA cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) because of a lack of human data and inadequate animal data (EPA, 2010). Data regarding the carcinogenicity of acenaphthene were not located. Benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, , chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene are classified in EPA weight-of-evidence Group B2 (probable human carcinogens) (EPA, 1997, 2010). Benzo(a)pyrene is the most extensively studied member of the class, inducing tumors in tissues at the point of contact of practically all laboratory species tested by all routes of exposure.

Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans, the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic

substances in these mixtures (ATSDR, 1995a). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke and coke oven emissions accounted for only 0.1-8% of the total mutagenic activity in *Salmonella* of the unfractionated complex mixture (Lewtas, 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounts for the majority of the mutagenicity of coke oven emissions and cigarette smoke. Furthermore, coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR, 1995a).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups is based largely on the results of animal studies with large doses of purified compound (EPA, 2010). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Although the carcinogenicity of benzo(a)pyrene in animals managed in an unnatural manner in laboratory conditions has been well established, the potential for carcinogenicity to humans in environmental settings involving exposure to low concentrations remains unclear.

EPA (200) verified a SF for oral exposure to benzo(a)pyrene of 7.3E+0 per mg/kg-day, based on several dietary studies in mice and rats. Recent reevaluations of the carcinogenicity and mutagenicity of the Group B2 PAHs suggest that there are large differences between individual PAHs in cancer potency (Krewski et al., 1989). Based on the available cancer and mutagenicity data, and assuming that there is a constant relative potency between different potential carcinogens across different bioassay systems and that the PAHs under consideration have similar dose-response curves, EPA (1993b) adopted relative potency values for several PAHs. These values and the corresponding oral SFs, based on a relative potency for benzo(a)pyrene of 1.0, are presented below:

Relative Potency Estimates for PAHs				
			Inhalation	
PAH	Relative Potency	Oral Slope Factor (per mg/kg-day)	Unit Risk Factor (per µg/m^3)	Slope Factor (per mg/kg-day)
Benzo(a)pyrene	1.0	7.3E+0	8.8E-4	3.1E+0
Benzo(a)anthracene	0.1	7.3E-1	8.8E-5	3.1E-1
Benzo(b)fluoranthene	0.1	7.3E-1	8.8E-5	3.1E-1
Benzo(k)fluoranthene	0.01	7.3E-2	8.8E-6	3.1E-2
Chrysene	0.001	7.3E-3	8.8E-7	3.1E-3
Dibenz(a,h)anthracene	1.0	7.3E+0	8.8E-4	3.1E+0
Indeno(1,2,3-cd)pyrene	0.1	7.3E-1	8.8E-5	3.1E-1

Although the EPA has not verified SFs for Group B2 PAHs other than benzo(a)pyrene, the SFs above represent reasonable estimates based on the data available. The relative potency approach employed here meets criteria considered to be desirable for this type of analysis (Lewtas, 1988). For example, the chemicals compared have similar chemical structures and would be expected to have similar toxicokinetic fate in mammalian systems. In addition, the available data suggest that the Group B2 PAHs have a similar mechanism of action, inducing frameshift mutations in *Salmonella* and tumor initiation in the mouse skin painting assay. Similar noncancer effects (minor changes in the blood, liver, kidneys) of the Group D PAHs support the hypothesis of a common mechanism of toxicity. Finally, the same endpoints of toxicity, i.e., potency in various

cancer assays, and related data, were used to derive the relative potency values (Krewski et al., 1989). The oral SF for benzo(a)pyrene of 7.3E+0 per mg/kg-day, and the SFs presented above for the other Group B2 PAHs are adopted for the purposes of this evaluation.

An EPA (1994) evaluation of the inhalation cancer data suggests adoption of an inhalation SF for benzo(a)pyrene of 3.1E+0 per mg/kg-day, based on the incidence of upper respiratory and digestive tract tumors in hamsters. Applying the relative potency estimates presented above yield the inhalation URFs and SFs for the other Group B2 PAHs presented above.

EPA (2010) classified naphthalene in EPA cancer weight-of-evidence group C – possible human carcinogen – based on benign respiratory tumors and one carcinoma in female mice exposed to naphthalene by inhalation. Cancer potency factors are not available because the data are not sufficient.

7.0 Toxicity Summary

Toxicity values for the PAHs are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Acenaphthene										
6E-1	6E-2	L	ND	ND	NA	ND	NA	ND	NA	NA
Acenaphthylene										
6E-1	6E-2	L	ND	ND	NA	D	NA	D	NA	NA
Anthracene										
3E+0	3E-1	ND	ND	ND	NA	D	NA	D	NA	NA
Benzo(a)anthracene										
ND	ND	NA	ND	ND	NA	B2	7.3E-1	B2	8.8E-5	3.1E-1
Benzo(a)pyrene										
ND	ND	NA	ND	ND	NA	B2	7.3E+0	B2	8.8E-4	3.1E+0
Benzo(b)fluoranthene										
ND	ND	NA	ND	ND	NA	B2	7.3E-1	B2	8.8E-5	3.1E-1
Benzo(k)fluoranthene										
ND	ND	NA	ND	ND	NA	B2	7.3E-2	B2	8.8E-6	3.1E-2
Benzo(g,h,i)perylene										
3E-1	3E-2	K	ND	ND	NA	D	NA	D	NA	NA
Chrysene										
ND	ND	NA	ND	ND	NA	B2	7.3E-3	B2	8.8E-7	3.1E-3
Dibenz(a,h)anthracene										
ND	ND	NA	ND	ND	NA	B2	7.3E+0	B2	8.8E-4	3.1E+0
Fluoranthene										
4E-1	4E-2	L,K,B	ND	ND	NA	D	NA	D	NA	NA
Fluorene										
4E-1	4E-2	E	ND	ND	NA	D	NA	D	NA	NA
Indeno(1,2,3-cd)pyrene										
ND	ND	NA	ND	ND	NA	B2	7.3E-1	B2	8.8E-5	3.1E-1
Naphthalene										
2E-1	2E-2	E, Ey, BW	3E-3/ 8.6E-4	3E-3/ 8.6E-4	Ne, Oe	C	ND	C	ND	ND
Phenanthrene										
3E-1	3E-2	K	ND	ND	NA	D	NA	D	NA	NA
Pyrene										
3E-1	3E-2	K	ND	ND	NA	D	NA	D	NA	NA

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: B = blood cells; BW = reduced body weight; E = erythrocyte; Ey = eye; L = liver; K = kidney; Ne = nasal epithelium; Oe = olfactory epithelium.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

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VOLATILE ORGANIC COMPOUNDS

BENZENE (71-43-2)

1.0 Introduction and Physical Properties

Benzene is a naturally occurring **VOC**, present in low levels in many plants and animals, as well as a synthetic compound derived from petroleum (ATSDR, 1997). It occurs as a colorless or slightly yellow liquid, or as a solid at temperatures below approximately 5.5 EC (HSDB, 2010). Benzene is widely used as a solvent and in the manufacturing of other organic compounds such as styrene, phenol, detergents, pesticides, explosives, polychlorinated biphenyls, flavors, perfumes, paints and coatings, nylon intermediates and photographic chemicals, among others. It is also used in food processing and leather tanning and occurs as a component of gasoline. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
78.1[a]	2.13[a]	5.56E-3[b] (25°C)	1.77[b]	8.80E-2[b] (25°C)	9.80E-6[b] (25°C)	1.25E-1[c] (25°C)	1.75E+3[b] (20-25°C)	353.24[d]	562.16[d]	7.34E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K); $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004a, *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)*, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

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[c]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 21 April.

[d]U.S. Environmental Protection Agency (EPA), 2003, *Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings*, Office of Emergency and Remedial Response, Washington, DC, 19 June.

2.0 Environmental Fate and Transport

Benzene enters the atmosphere primarily from fugitive emissions and automobile exhaust because of its presence in gasoline (ATSDR, 1997; HSDB, 2010). Other important sources are emissions associated with its production and use as a solvent, as an industrial intermediate in the production of the materials mentioned above, and coke oven blast furnaces and coke by-product recovery plants. In addition, there are discharges into water from industrial effluents and losses during spills. Sources of release to soil include land disposal of benzene-containing wastes, industrial discharge, and leaks from underground storage tanks and associated plumbing.

Benzene released to the atmosphere will exist almost exclusively in the vapor phase (ATSDR, 1997; HSDB, 2010). It is not subject to direct photolysis but it will react with photochemically produced hydroxyl radicals with a half-life of approximately 13 days. The reaction time in polluted atmospheres containing nitrogen oxides or sulfur dioxide is accelerated with the half-life being reported as 4 to 6 hours. Products of photooxidation include phenol, nitrophenols, nitrobenzene, formic acid, and peroxyacetyl nitrate. Benzene is fairly soluble in water and is removed from the atmosphere in rain.

Benzene released to soil will be subject to rapid volatilization near the surface; that which does not evaporate will be highly to very highly mobile in the soil and may leach to groundwater (ATSDR, 1997; HSDB, 2010). Benzene may be subject to biodegradation under aerobic conditions. Microbial transformation proceeds through formation of cis-dihydrodiols to catechols, which lead to destruction of the aromatic ring.

Benzene may be subject to photooxidation and biodegradation in shallow, aerobic groundwaters, but probably not significantly under deeper or anaerobic conditions (ATSDR, 1997; HSDB, 2010). Benzene released to surface water will be subject to rapid volatilization. It is not expected to sorb to sediment or to hydrolyze significantly. Biodegradation is unlikely to be a significant fate process in sediment.

Benzene is not expected to participate significantly in food-chain pathways (ATSDR, 1997; EPA, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Benzene is rapidly and readily absorbed during inhalation exposures of humans or laboratory animals to vapors (ATSDR, 1997). However, the extent of uptake and retention decrease with increasing concentration or time of exposure, reflecting saturation of the metabolic processes that remove the substance from the blood. Excretion data from laboratory animals show that absorption is rapid and essentially complete following oral administration. Dermal uptake of liquid benzene through intact skin has been demonstrated in humans and may be toxicologically significant. The extent of dermal uptake of benzene vapor has not been quantified in humans, but experiments in laboratory animals suggest this exposure route also may be toxicologically significant.

Benzene is widely distributed following any route of exposure (ATSDR, 1997). Highest levels were located in adipose and other tissues high in fat such as the brain following inhalation exposure. Highest levels following oral administration were located in liver, kidney and bone marrow.

Quantitative aspects of the metabolism of benzene are somewhat species-, exposure route- and dose-dependent (ATSDR, 1997), but certain relevant generalizations pertain. The first metabolic step appears to be cytochrome P-450-catalyzed oxidation of benzene to benzene epoxide in the liver, which rearranges to phenol. Phenol is transformed to hydroxyquinone, which is further oxidized to benzoquinone, considered to be a major toxicant in bone marrow. Alternatively, phenol may be further oxidized to catechol and possibly to trihydroxybenzene. Phenol and all its derivatives except benzoquinone have been shown to undergo detoxication by conjugation with glucuronide or sulfate, followed by excretion.

A second general pathway involves the further oxidation of benzene oxide to benzene dihydrodiol, which leads to ring-opening and the formation of muconic dialdehyde, a known hematotoxin, primarily in the liver (ATSDR, 1997).

Absorbed benzene is excreted through the lungs as unchanged compound, and through the urinary tract as conjugated metabolites (ATSDR, 1997). Urinary excretion predominates.

Nothing in this review of toxicokinetics suggests that laboratory animals would not serve as appropriate models for the toxicity of benzene to humans.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for benzene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

As mentioned above, benzene in the liquid and vapor form are absorbed through the skin (ATSDR, 1997). Combining benzene with soil slows dermal uptake. EPA (2004) notes that VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels. Therefore, dermal uptake of benzene from soil is not evaluated. EPA (2003) provides t*, K_p, τ, B and FA values as follows:

Chemical	t*	K_p	τ	FA	B
Benzene	0.70	1.5E-2	0.29	1	0.1

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).

5.0 Noncancer Effects Evaluation

Short-term exposure to benzene induces central nervous system (CNS) effects such as drowsiness, dizziness and headaches; long-term exposure may induce anemia (ATSDR, 1997). Oral dosing in animals induces hematopoietic and immune effects. EPA (2010) verified a chronic oral RfD of 4E-3 mg/kg-day, based on hematological effects (reduced lymphocyte count) in a cross-sectional occupational study of 44 workers exposed to benzene. The workers were exposed for 0.7 to 16 years, with a mean of 6.3 years. Because the mean fell below 7 years, the minimal length considered to be chronic, the study is judged to be subchronic in duration. The control group consisted of an equal number of age- and gender-matched unexposed workers. The exposed workers were divided into two equal groups of 22: those exposed to airborne concentrations below and those exposed to airborne concentrations above the median concentration for all workers of 99 mg/m^3. An exposure-adjusted benchmark concentration (BMC) for minimal hematologic effects of 8.2 mg/m^3 was estimated. Assuming that uptake from the respiratory tract is approximately 50 percent of that from the GI tract allowed application of inhalation-to-oral extrapolation to estimate a benchmark dose (BMD) of 1.2 mg/kg-day. Application of an uncertainty factor of 300 to the BMD yielded the verified chronic oral RfD of 4E-3 mg/kg-day. The uncertainty factor of 300 consists of factors of 3 each for extrapolation from a BMC for minimal adverse effects to a BMC for no adverse effects, to expand from subchronic to chronic exposure, and to reflect deficiencies in the data base; a factor of 10 was used to provide additional protection for more sensitive human subpopulations. The blood cells (specifically inhibited hematopoiesis) are the target for prolonged oral exposure to benzene. Confidence in the chronic oral RfD is medium.

A preliminary subchronic oral RfD can be developed from the BMD described above. The subchronic oral RfD is derived by applying an uncertainty factor of 100 to the BMD of 1.2

mg/kg-day. The uncertainty factor of 100 reflects the uncertainty factor of 300 developed by EPA (2010) without the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 1.2E-2 mg/kg-day, which is rounded to 1E-2 mg/kg-day.

A verified chronic inhalation RfC of 3E-2 mg/m^3 for benzene was based on the exposure-adjusted BMC of 8.2 mg/m^3 in the occupational exposure study from which the oral RfD values were derived (EPA, 2010). Application of the uncertainty factor of 300 (as described for the verified chronic oral RfD) to the BMC of 8.2 mg/m^3 yielded the chronic RfC of 3E-2 mg/m^3. The RfC is equivalent to an inhalation RfD of 8.6E-3 mg/kg-day. Confidence in the chronic inhalation RfC is medium. The blood cells (specifically inhibited hematopoiesis) are the target for prolonged inhalation exposure to benzene. A preliminary subchronic inhalation RfC can be derived by application of an uncertainty factor of 100 to the BMC of 8.2 mg/m^3 in the same manner as was done to derive the subchronic oral RfD. The subchronic inhalation RfC so derived is 8.2E-2 mg/m^3, rounded to 8E-2 mg/m^3. The preliminary subchronic inhalation RfC is equivalent to an inhalation RfD of 2.3E-2 mg/kg-day.

6.0 Carcinogenicity Evaluation

Benzene is classified as an EPA cancer weight-of-evidence Group A chemical – a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies (EPA, 2010). Epidemiologic studies and case studies provide clear evidence of a causal association between exposure to benzene and acute nonlymphocytic leukemia, and also provide supportive evidence for chronic nonlymphocytic leukemia and chronic lymphocytic leukemia. Other neoplastic conditions in humans that are associated with benzene exposure include hematologic neoplasms, blood disorders such as preleukemia and aplastic anemia, Hodgkin's lymphoma, and myelodysplastic syndrome. Experimental animal data add to the weight of evidence that exposure to benzene increases the risk of cancer in multiple species at multiple organ sites (hematopoietic, oral and nasal, liver, forestomach, preputial gland, lung, ovary, and mammary gland). A verified URF range for inhalation exposure of 2.2E-6 to 7.8E-6 per µg/m^3 was based on the incidence of leukemia in humans exposed to benzene while employed in the Pliofilm industry (EPA, 2010). A low-dose linearity model utilizing maximum likelihood estimates was used to estimate the URF. The range reflects differences in the exposure assumptions estimated from the study. EPA (2010) concluded that neither any specific URF value nor either end of the range is more defensible than the other. Therefore, the upper end of the range, 7.8E-6 per µg/m^3, is conservatively selected for this evaluation. The URF of 7.8E-6 per µg/m^3 is equivalent to an inhalation SF of 2.7E-2 per mg/kg/day.

The occupational data that serve as the basis for the inhalation URF also serve as the basis for a verified oral SF for benzene (EPA, 2010). The inhalation URF range was mathematically converted to an equivalent oral SF range by applying standard inhalation rate and body weight assumptions, and by adjusting for route-specific differences in absorption as described for the chronic oral RfD derivation. The oral SF range so derived is 1.5E-2 to 5.5E-2 per mg/kg-day. The upper end of the range, 5.5E-2 per mg/kg-day, is conservatively selected as the oral SF for this evaluation.

7.0 Toxicity Summary

Toxicity values for benzene are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1E-2	4E-3	Bc	8E-2/ 2.3E-2	3E-2/ 8.6E-3	Bc	A	5.5E-2	A	7.8E-6	2.7E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day).
Target organ abbreviations: Bc = blood cells.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

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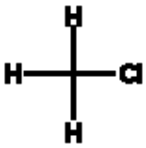
CHLOROMETHANE (METHYL CHLORIDE) (74-87-3)



1.0 Introduction and Physical Properties

Chloromethane is a **VOC** that exists as a colorless gas at ambient temperatures (HSDB, 2010). It is used largely as a methylating agent in the production of silicones, tetramethyl lead, methyl cellulose, methylene chloride, methyl mercaptan, plastics, pesticides, pharmaceuticals, dyes, perfumes, ethers, resins, agricultural chemicals, quaternary amines and butyl rubber (ACGIH, 1991; ATSDR, 1998; HSDB, 2010). It is also used as an extractant for fats, oils and resins, as a blowing agent and as a refrigerant. It was formerly used as a propellant in various aerosol products. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
50.5[a]	0.91[a]	8.82E-3[b] (25□C)	1.15[b]	1.39E-1[c] (25□C)[d]	1.49E-5[e] (20□C)	5.66E+0[b] (25□C)	5.32E+3[b] (25 □C)	247[f]	416[f]	5.15E+3[f]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).
[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.
[b]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
[c]Calculated as described in Introduction to Toxicological profiles.
[d]Assumed.
[e]Montgomery, J.H., 1996, ***Groundwater Chemicals Desk Reference, Second Edition***, Lewis Publishers, New York, pp. 664-666.
[f]National Institute of Standards and Technology (NIST), 2010, ***NIST Chemistry WebBook***, on line.

2.0 Environmental Fate and Transport

Chloromethane is one of the few halogenated organic chemicals for which natural sources are significant. Natural sources include volcanoes, volatilization from plants, and forest fires (HSDB, 2010). It is produced in seawater by the reaction of methyl iodide with chloride ions. Methyl iodide is produced photosynthetically by several marine organisms, and there may be other oceanic sources as well. The mean air flux of 1.3E-6 g/cm^2-year for the eastern Pacific Ocean, when extrapolated to global waters, provides an adequate source to explain the atmospheric reservoir of chloromethane. Chloromethane is released from brush and forest fires. The estimated 0.6 g of chloromethane released per kg of vegetation burned is believed to make a significant impact on the global burden of the compound.

The production and many uses of chloromethane may result in its release to the environment through various waste streams (HSDB, 2010). It is also released in tobacco smoke and turbine exhaust. Just as forest fires contribute to natural sources, wood burning, field burning, and backyard burning also contribute to atmospheric burdens. Chloromethane is formed in the chlorination of drinking water and sewage effluent, and is found in the effluent of some publically owned treatment works.

Most chloromethane is released to the atmosphere where it is expected to exist solely as a gas (HSDB, 2010). Gas-phase chloromethane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to

be 310 days, which provides opportunity for widespread dispersion. The dominant process for removal from the troposphere is upward diffusion, although washout by rain may also be important. From the tropopause to about 30 km, both upward diffusion and reaction with hydroxyl radicals will be of approximately equal importance, and above 30 km in the stratosphere, diffusion, reaction with hydroxyl radicals, and photodissociation will have approximately equal importance. The surface half-life resulting from upward diffusion is approximately 80 days.

Based on its very low estimated K_{oc} value, chloromethane is expected to have very high mobility in soil (HSDB, 2010). Its relatively high water solubility suggests the compound released in sufficient quantity in the liquid phase or dissolved in water could leach readily to groundwater. Volatilization from moist soil surfaces is expected to be an important removal process for small quantities released to the surface. Volatilization from dry soil surfaces may also be important. Field and laboratory studies demonstrate that several halogenated aliphatics may biodegrade slowly under anaerobic conditions, but not under aerobic conditions. Biodegradation is not expected to be a significant removal process.

Chloromethane is not expected to adsorb to suspended solids and sediment in surface water (HSDB, 2010). Volatilization from water surfaces is expected to be a significant removal mechanism; volatilization half-lives for a model river and model lake are 46 minutes and 3 days, respectively. Field and laboratory studies demonstrate that several halogenated aliphatics may biodegrade slowly under anaerobic conditions, but not under aerobic conditions. Biodegradation is not expected to be a significant removal process. Hydrolysis may occur, but very slowly, with a half-life of approximately 2 years (ATSDR, 1998). Hydrolysis will not be a significant removal process for chloromethane in surface water, but may be the only significant removal process for the compound in groundwater, where it has been shown to persist.

Chloromethane is water soluble, volatile and labile, and is not expected to participate significantly in food-chain pathways (ATSDR, 1998; HSDB, 2010). Therefore, biotransfer factors are not estimated for this compound.

3.0 Toxicokinetics

Studies regarding the toxicokinetics of chloromethane are limited to inhalation exposure. Chloromethane is readily absorbed by the respiratory system and equilibrium conditions are reached in approximately one hour in exposed humans and laboratory animals (ATSDR, 1998). Data regarding the extent of respiratory absorption were not located. Although data were not located for GI absorption, it is expected that GI absorption would be rapid and extensive because the compound is a low MW organic substance that is readily absorbed by the respiratory system. HSDB (2010) stated that dermal absorption can occur, but no other information was provided.

Radioactivity is rapidly and extensively distributed throughout the body following inhalation exposure to radiolabeled chloromethane, with highest levels of radioactivity measured in liver, kidney, testis and lung (ATSDR, 1998). Approximately 20 percent of the radiolabel in liver and testis was found to have been metabolically incorporated into the cellular structure, reflecting uptake and utilization from the one-carbon atom pool of the body.

The primary and predominant metabolic pathway of chloromethane involves enzymatic conjugation with glutathione to form S-methylglutathione (ATSDR, 1998). Further metabolism involves deamination to yield S-methylcysteine and other sulfur-containing conjugation derivatives, which are excreted in the urine. Another metabolic pathway involves dehalogenation of chloromethane to form formaldehyde (a toxicant in its own right) and formic acid. These intermediates contribute to the one-carbon atom pool that is available for incorporation into the tissues or for mineralization to carbon dioxide followed by excretion through the lungs.

Studies have shown that adult humans, unlike laboratory animals, may be divided into two groups – fast or slow metabolizers – depending on the activity in their erythrocytes of glutathione-S-transferase, which is responsible for the first conjugation reaction (ATSDR, 1998). Fast metabolizers clear chloromethane from the body more rapidly than slow metabolizers. Whether the unborn, new born or young children exhibit this characteristic is unknown. The implications of glutathione-S-transferase activity regarding population differences in toxic potency are unclear. On the one hand, it may appear that fast metabolizers should be more tolerant of chloromethane because of their enhanced ability to clear the compound from the body. On the other hand, experiments in laboratory animals suggest that a metabolite of S-methylglutathione may be the ultimate toxicant, in which case fast metabolizers may be more sensitive to chloromethane toxicity.

Chloromethane residues are rapidly cleared from the body following the cessation of inhalation exposure (ATSDR, 1998). Clearance in laboratory animals and humans is biphasic, consistent with a linear 2-compartment model. The half-lives for both phases were measured in minutes, indicating the rapidity with which clearance occurs. The exception is that the β-phase in some humans (slow metabolizers) approximated 1.5 hours.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Chloromethane is assumed to be absorbed readily from the GI tract; therefore, no GAF is developed.

Data regarding the dermal uptake of chloromethane were not located, except that HSDB (2010) stated that dermal absorption could occur. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of chloromethane from soil is not quantified. EPA (2004a) provides t*, K_p, τ, FA and B values as follows:

Chemical	t*	K_p	τ	FA	B
Chloromethane	0.49	3.3E-3	0.20	1	0
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Data regarding the toxicity of oral exposure to chloromethane consist of the statement that liver effects were not observed in rats given a single oral dose of 420 mg/kg (ATSDR, 1998; EPA, no date). EPA (no date) derived a provisional chronic oral RfD of 4E-3 mg/kg-day based on a LOAEL of 69.32 mg/m^3 for subtle neurobehavioral effects from an occupational (inhalation) study. Subsequently, EPA (2010) reviewed the data and determined that ingestion exposure is not applicable because the compound exists as a gas at ambient temperatures. They also noted that the oral exposure data are insufficient to develop an oral RfD. Therefore, the EPA (no date) derivation is considered to be not applicable and no oral RfD is offered herein.

EPA (2010) verified a chronic inhalation RfC of 9E-2 mg/m^3 based on a NOAEL of 50 ppm (103.2 mg/m^3) from an 11-day continuous exposure study in mice. The NOAEL corresponds to a human equivalent concentration (HEC) of 94.6 mg/m^3. The LOAEL in this study, 100 ppm (206.4 mg/m^3 or a HEC of 189.2 mg/m^3) was associated with slight to moderate degenerative changes in the granule cells of the cerebellum. EPA (2010) applied an uncertainty factor of 1000 to the NOAEL HEC of 94.6 mg/m^3 to derive the RfC of 9E-2 mg/m^3. The uncertainty factor consists of factors of 10 each to provide additional protection for unusually sensitive humans and to expand from a short-term to lifetime exposure, and factors of 3 each to extrapolate from mice to humans and to address the uncertainty regarding the potential for CNS effects in the developing young. Confidence in the verified chronic inhalation RfC is medium. The verified inhalation RfC is equivalent to an inhalation RfD of 2.6E-2 mg/kg-day. The CNS is the target organ for prolonged inhalation exposure to chloromethane.

EPA (1996) developed a provisional chronic RfC based on subtle neurobehavioral effects from an occupational (inhalation) study. They adopted the provisional chronic RfC for subchronic application, noting no significant differences in response between chronic and subchronic exposure. However, given the EPA (2010) reevaluation and verification of a more conservative chronic RfC, it is deemed prudent to adopt the verified chronic RfC rather than the provisional chronic RfC as sufficiently protective for subchronic exposure.

6.0 Carcinogenicity Evaluation

Previous EPA (1997, 1998) evaluations classified chloromethane in EPA weight-of-cancer Group C (possible human carcinogen), and developed an oral SF and an inhalation URF. More recently, EPA (2010) reevaluated the data, noting that chloromethane is a naturally occurring compound, and concluded that it is more appropriately classified in Group D (not classifiable as to its human carcinogenicity). Applying more recent cancer assessment guidelines, EPA (2010) concluded that the available data suggest that chloromethane would be classified as an agent whose carcinogenic potential *cannot be determined*. Previously developed potency factors are considered to be not applicable; no potency factors are offered herein for chloromethane.

7.0 Toxicity Summary

Toxicity values for chloromethane are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
			9E-2/	9E-2/						

Noncancer Effects						Carcinogenicity				
Oral Exposure			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
ND	ND	NA	2.6E-2	2.6E-2	CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: CNS = central nervous system.

8.0 References

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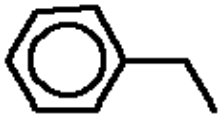
U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

ETHYLBENZENE (PHENYLETHANE) (100-41-4)



1.0 Introduction and Physical Properties

Ethylbenzene, also known as phenylethane, is a **VOC** that exists in the pure state as a colorless liquid at ambient temperatures (HSDB, 2010). It is used primarily as a precursor in the manufacture of styrene (ATSDR, 1999). It is also used as a solvent and diluent, and as an intermediate in the manufacturing of cellulose acetate, rubber and other organic compounds (ATSDR, 1999; HSDB, 2010). It is also a constituent of asphalt, naphtha, and automotive and aviation fuels. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
106.2[a]	3.15[a]	7.88E-3[b] (25°C)	2.56[b]	7.50E-2[b] (25°C)	7.80E-6[b] (25°C)	1.26E-2[c] (25°C)	1.69E+2[b] (20-25°C)	409.34[d]	617.20[d]	8.50E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

[b]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.

[c]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***User's Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, June 19.

2.0 Environmental Fate and Transport

Ethylbenzene enters the atmosphere primarily from fugitive emissions and exhaust connected with its use in gasoline (ATSDR, 1999; HSDB, 2010). More localized sources to the air and other environmental media include fugitive emissions, waste water and spills from its production and industrial use, leaking underground storage tanks, land disposal of ethylbenzene-containing waste, and municipal and hazardous waste combustion.

Ethylbenzene exists in the air in the vapor phase (ATSDR, 1999; HSDB, 2010). It is somewhat water soluble and may be removed by precipitation; however, it is likely to reenter the air by volatilization. Ethylbenzene is transformed in air by various oxidative processes to yield ethylphenols, benzaldehyde, acetophenone, and m- and p-nitroethylbenzene. Reaction with photolytically generated hydroxyl radicals is probably the most significant reaction, with a half-life estimated to be 55 hours, suggesting that ethylbenzene will not persist in the air or travel long distances.

Ethylbenzene released to soil will volatilize rapidly (ATSDR, 1999; HSDB, 2010). That which infiltrates is expected to be quite mobile in soil and may leach to groundwater because sorption to soil particles is not sufficiently strong to retard movement. Biodegradation in soil in aerobic conditions involves oxidation of the ethyl side chain, ring hydroxylation and ring cleavage, and may be a significant source of removal. Anaerobic degradation is expected to be insignificant.

Ethylbenzene released to surface water will volatilize rapidly to air (ATSDR, 1999; HSDB, 2010). Biodegradation in aerobic conditions will significantly reduce water-borne

concentrations. Photolysis and hydrolysis are unlikely to be significant. Ethylbenzene partitions weakly to sediment.

Ethylbenzene is not expected to participate significantly in food-chain pathways (ATSDR, 1999; EPA, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Ethylbenzene is rapidly absorbed during inhalation exposure, with retention near or exceeding 50 percent of the inhaled concentration (ATSDR, 1999). The compound is similarly rapidly absorbed following oral administration. Excretion data in rabbits suggest that a minimum of 72 to 92 of an oral dose is absorbed. Liquid ethylbenzene applied to the skin of humans is rapidly absorbed, but ethylbenzene vapor is poorly absorbed.

Ethylbenzene appears to be efficiently and widely distributed throughout the body following inhalation exposure (ATSDR, 1999). The same is expected following oral and dermal exposure, but data are lacking.

Metabolism in humans of absorbed ethylbenzene following either inhalation or oral exposure predominantly involves successive oxidation of the ethyl side-chain through 1-phenylethanol, acetophenone, ω-hydroxyacetophenone, and 1-phenyl-1,2-ethanediol to form mandelic acid and phenylglyoxylic acid (ATSDR, 1999). Small quantities of the first 4 intermediates are conjugated and excreted in the urine. Mandelic acid and phenylglyoxylic acid are excreted without conjugation and comprise the majority of excreted metabolites. A minor metabolic pathway involves epoxide formation and hydroxylation of the phenyl ring with or without oxidation of the ethyl group to yield small quantities of metabolites that are conjugated and excreted. Experiments with laboratory animals reveal species differences in the predominance of certain metabolites, but the pathways described above appear to hold. There is no evidence that the metabolites are more toxic than the parent compound.

Excretion of metabolites, primarily through the urine, is rapid and extensive in humans and laboratory animals (ATSDR, 1999). Pulmonary excretion of carbon dioxide is a minor pathway.

Nothing in this review of toxicokinetics suggests that laboratory animals would not serve as appropriate models for the toxicity of ethylbenzene to humans.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. GI absorption of ethylbenzene is extensive as discussed in Section 3.0. Therefore, no GAF is developed for ethylbenzene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data were not located for dermal uptake of ethylbenzene from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of ethylbenzene from soil is not evaluated. Values for t^*, K_p, τ, FA and B are provided by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
Ethylbenzene	1.01	4.9E-2	0.42	1.0	0.2
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

An oral LD_{50} in rats of 5.46 g/kg (HSDB, 2010) suggests that the acute toxicity of ingested ethylbenzene is low. Subchronic to chronic oral exposure of laboratory animals to ethylbenzene induces mild liver and kidney lesions (EPA, 2010). EPA (2010) presents a verified chronic oral RfD for ethylbenzene of 1E-1 mg/kg-day based on a NOEL for liver and kidney lesions in female rats of 136 mg/kg-day in a six-month gavage study. The LOAEL in this study was 408 mg/kg-day. An uncertainty factor of 1000 was applied to the NOEL to derive the chronic oral RfD. The uncertainty factor consists of factors of 10 each to extrapolate from animals to humans, to provide additional protection for more sensitive humans, and to expand from subchronic to chronic exposure. Confidence in the oral RfD is low. The liver and kidney are the target organs for prolonged oral exposure to ethylbenzene.

No EPA-derived subchronic oral RfD is available for ethylbenzene. A preliminary subchronic oral RfD can be made, however, by applying an uncertainty factor of 100 to the NOEL of 136 mg/kg-day in the 6-month rat study described above. The uncertainty factor of 100 reflects the chronic uncertainty factor of 1000 without the factor of 10 for expansion from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 1E+0 mg/kg-day.

Acute inhalation exposure induces irritation of the mucous membranes in animals and humans (ACGIH, 1991). Prolonged inhalation exposure induces liver enzymes and results in elevated liver and kidney weights; prolonged exposure to relatively high levels is associated with mild histopathological lesions of the liver, kidney and testes (ACGIH, 1991; EPA, 2010). EPA (2010) considered that the effects on the liver at the lower exposure concentrations are adaptive rather than adverse. EPA (2010) presented a verified chronic inhalation RfC of 1E+0 mg/m^3 derived from a human equivalent adjusted NOAEL of 434 mg/m^3 from studies on developmental effects in rats and rabbits. An uncertainty factor of 300 was used. The human equivalent adjusted LOAEL of 4340 mg/m^3 was associated with equivocal evidence of reduced fetal size in rabbits, an increased incidence of extra ribs in rat fetuses, and increased liver, kidney and spleen weights in the rats. The inhalation RfC is equivalent to 2.9E-1 mg/kg-day. Confidence in the RfC is low. The chronic inhalation RfC is adopted as being sufficiently conservative for subchronic as well as chronic inhalation exposure.

The key study from which the inhalation RfC is derived does not clearly identify target organ(s) for inhalation exposure to ethylbenzene. The entire inhalation exposure data base, however, suggests that the liver, kidneys and fetus may be the most significant target organs for prolonged inhalation exposure.

6.0 Carcinogenicity Evaluation

Ethylbenzene is currently verified as a cancer weight-of-evidence Group D compound (not classifiable as carcinogenicity to humans) (EPA, 2010) based on an absence of human or animal cancer studies. Cancer potency factors are not estimated for Group D compounds.

7.0 Toxicity Summary

Toxicity values for ethylbenzene are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1E+0	1E-1	L, K	1E+0/ 2.9E-1	1E+0/ 2.9E-1	L, K, F	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: L = liver; K = kidney; F = fetus.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1999, ***Update Toxicological Profile for Ethylbenzene***, U.S. Department of Health and Human Services, Atlanta, Georgia, July, on line.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, Cincinnati, OH, p. 581-584.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*** Final, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

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DICHLOROPROPENES

1,3-Dichloropropene

1.0 Introduction and Physical Properties

This profile addresses 1,1-dichloropropene, 1,2-dichloropropene, 1,3-dichloropropene, and 2,3-dichloropropene. All are **VOCs** and all are liquid at ambient temperatures (ATSDR, 2006; EPA, 2002; HSDB, 2010)

Various mixtures and formulations of dichloropropenes and 1,2-dichloropropane (78-87-5) are used as soil fumigants and nematocides (HSDB, 2010; Meister, 1999). It is generally applied pre-planting by injection equipment at depths of 12 to 18 inches. Compacting the treated soil, covering the treated soil with tarps, or irrigating with water are often done immediately after treatment to retard volatilization loss.

Most soil fumigant mixtures contain predominantly 1,3-dichloropropene (542-75-6), with lesser amounts of 1,2-dichloropropene (563-54-2) and traces of 1,1-dichloropropene (563-58-6) and 2,3-dichloropropene (78-88-6) as well (ATSDR, 2006; HSDB, 2010). The use of 1,3-dichloropropene as a pesticide increased after the EPA phased out the use of ethylene dibromide, and its use is likely to increase further with the planned EPA phase-out of methyl bromide. 1,3-Dichloropropene is also used in much smaller quantities in organic synthesis and as a solvent; small quantities of 2,3-dichloropropene are used in organic synthesis.

Little is known about the physical properties of 1,1-dichloropropene or 2,3-dichloropropene. 1,2-Dichloropropene occurs as a minor constituent in Telone II, one of the more common dichloropropene formulations used as a soil fumigant. There are 2 isomers of 1,3-dichloropropene (542-75-6): cis- (or Z-) 1,3-dichloropropene (10061-01-5) and trans- (or E-) 1,3-dichloropropene (10061-02-6). Many preparations and formulations of 1,3-dichloropropene contain approximately equivalent proportions of the two isomers (HSDB, 2010). Relevant physical properties of the dichloropropenes are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
1,1-Dichloropropene (563-58-6)										
110.97[a]	2.53[a,b]	2.16E-2[a,b] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	1.19E-1[a,e] (20°C)	1.18E+3[a,b] (25°C)[d]	349.65[f]	551.66[g]	7.15E+3[h]
1,2-Dichloropropene (563-54-2)										
110.97[i]	2.53[a,b]	4.91E-3[a,e] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	3.16E-2[a,e] (25°C)	2.7E+3[i,e] (25°C)	348[i]	549[g]	7.11E+3[h]
1,3-Dichloropropene (both isomers: 542-75-6)										
111[j]	2.00[i]	3.55E-3[a,b] (25°C)	1.91[a,b]	6.26E-2[k] (25°C)	1.00E-5[k] (25°C)	4.47E-2[a,e] (25°C)	2.80E+3[k] (20-25°C)	381.15[l]	587.38[l]	7.90E+3[l]
2,3-Dichloropropene (78-88-6)										
110.97[a]	2.42[a,b]	4.16E-3[a,e] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	8.05E-2[i] (25°C)	2.15E+3[a,e] (25°C)	367.2[i]	551.6[i]	8.44E+3[i]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$

[a]U.S. Environmental Protection Agency (EPA), 2004a, ***EPI Suite Version 3.12*** Software for estimating physical properties, online, 30 November.
[b]Estimated.
[c]Calculated as described in Introduction to Toxicological profiles.
[d]Assumed.
[e]Measured.
[f]Agency for Toxic Substances and Disease Registry (ATSDR), 2006, ***Toxicological Profile for Dichloropropenes***, Draft for Public Comment, U.S. Department of Health and Human Services, Atlanta, GA, September, on line.
[g]Estimated by Equation 12-4 of Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[h]Estimated by Equation 13-5 of Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[i]Hazardous Substances Data Bank (HSDB), 2010, National Library of Medicine, on line.
[j]U.S. Environmental U.S. Environmental Protection Agency (EPA), 2004b, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.
[k]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.
[l]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

Considerable discrepancy was noted in the VP values for 1,2-dichloropropene. HSDB (2010) offered a value of 90.8 mm Hg (1.19E-1 atm) at 20 °C from a 1981 report, and EPA (2004a) offered a value of 24 mm Hg (3.16E-2 atm) at 25 °C from an experimental data base (not specified). The reason for the discrepancy is not apparent. The value of 24 mm Hg (3.16E-2 atm) at 25 °C from EPA (2004a) was selected as the more rigorously reviewed source.

There was also discrepancy between the log K_{ow} values for 1,3-dichloropropene. EPA (2004a) offered a value of 1.60, but the source is unclear. HSDB (2010) offered a value of 2.00, and EPA (2004a) cited experimental values of 2.03 and 2.06. The value of 2.00 from HSDB (2010) was selected as better documented and more consistent with the experimental values compiled by EPA (2004a).

Considerable discrepancy was noted in values for H for 1,3-dichloropropene. EPA (2002) offered a value for H' (unitless) of 7.26E-1, equivalent to 1.77E-2 atm-m^3/mole, from an unclear source. HSDB (2010) offered 9.76E-4 atm-m^3/mole, but the source is unclear. EPA (2004a) offered 3.55E-3 atm-m^3/mole from an experimental database. The latter value is chosen because it is surrounded with less uncertainty than the other values. Similar discrepancy was noted in values for K_{oc}. EPA (2002) offered a value for K_{oc} of 45.7 L/kg from an unclear source. HSDB (2010) offered 290 L/kg estimated by a regression equation from the K_{ow}. EPA (2004a) offered 80.77 L/kg from an updated regression equation. The value of 80.77 L/kg (log K_{oc} = 1.91) from EPA (2002a) is selected as fraught with less uncertainty than the EPA (2002) value and estimated by a more current model than the HSDB (2010) value.

2.0 Environmental Fate and Transport

Dichloropropenes may be released in wastewater or as fugitive emissions during their production and use as chemical intermediates or soil fumigants; however releases of 1,1-, 1,2-, and 3,3-

dichloropropene are probably low because no uses were located for these isomers, although they may appear at low levels in other dichloropropene mixtures and formulations. The dichloropropenes may be inadvertently formed by the chlorination of water supplies carrying organic matter (ATSDR, 2006).

Dichloropropenes released to soil are likely to be highly mobile because of their low K_{oc} values (HSDB, 2010). Although the dichloropropenes may leach to groundwater, hydrolysis and biodegradation may attenuate the leaching process (ATSDR, 2006). Also, dichloropropenes will adsorb much more readily from the vapor phase than from the dissolved phase. Volatilization from moist soil surfaces is expected to be an important removal process for the dichloropropenes. Volatilization from dry soil surfaces may also be important.

Dichloropropenes released to water are not expected to adsorb to suspended solids and sediment (HSDB, 2010). Volatilization from water surfaces is likely to be the major removal mechanism. Volatilization half-lives for a model river and model lake have been estimated at 1 and 101 hours (specific isomer[s] not specified), respectively. Hydrolysis may also be significant; a hydrolysis half-life of 11 days was estimated for the dichloropropenes at a temperature range of 15 to 25 °C. Biodegradation is also expected to be significant (ATSDR, 2006).

The dichloropropenes are expected to exist entirely in the vapor phase in the ambient atmosphere (HSDB, 2010). Vapor-phase dichloropropene is subject to reaction with photochemically-produced hydroxyl radicals; a half-life of 3 days (specific isomer[s] not specified) was estimated for this reaction. The dichloropropenes are not expected to directly photolyze in the presence of sunlight. Wet deposition may also be a significant removal mechanism (ATSDR, 2006) (1,3-dichloropropene has been detected in rainwater), but volatilization is likely to rapidly return the dichloropropenes to the atmosphere.

Their relatively low log K_{ow}, high water solubility and their potential for metabolism suggests that the dichloropropenes are unlikely to participate significantly in food-chain pathways (HSDB, 2010). 1,3-Dichloropropene was identified by the EPA (1995) as not being of concern for human health through the aquatic food chain. Therefore, biotransfer factors are not compiled or estimated.

3.0 Toxicokinetics

Data for 1,3- and 2,3-dichloropropene indicate that the dichloropropenes are readily absorbed following inhalation or oral exposure (ATSDR, 2006). Excretion data from rats indicate that 62 to 82 percent of inhaled 1,3-dichloropropene and 35 to 40 percent of inhaled 2,3-dichloropropene are absorbed. The higher absorption percentages were estimated at lower concentrations. Higher concentrations were shown to decrease respiratory rate and saturate elimination mechanisms, which effectively reduced respiratory absorption.

Excretion data for 1,3- and 2,3-dichloropropene in rats indicate that well upwards of 90 percent of an ingested dose is absorbed from the GI tract. Excretion data from human volunteers indicate that dermal uptake occurs, but reliable quantitative data were not located.

The dichloropropenes appear to be widely distributed following absorption. After 6-hour inhalation exposure to radiolabeled 2,3-dichloropropene, tissue levels of radioactivity from

highest to lowest were found in the urinary bladder, nasal turbinates, kidneys, small intestine, liver, trachea and larynx (ATSDR, 2006). Approximately 9 percent of the absorbed radioactivity was accounted for in the body tissues. Following gavage treatment with radiolabeled 1,3-dichloropropene, highest levels of radioactivity were found in the forestomach and urinary bladder; lower levels were located in a wide variety of tissues. Tissue levels fall rapidly after exposure is terminated; there appears to be no tendency for the body to accumulate the parent compound or its metabolites. It is noteworthy that the forestomach is the target organ of prolonged oral exposure, the nasal turbinates are the target of prolonged inhalation exposure, and the urinary bladder is a target organ for the carcinogenicity of 1,3-dichloropropene.

Data from rats indicate that 1,3-dichloropropene is metabolized by three primary pathways. Most important is dechlorination and conjugation with glutathione at carbon-3, followed by conversion to a mecapturic acid and acetylation to form the N-acetyl-cysteine metabolite, which is excreted (ATSDR, 2006). Excretion data from occupationally exposed humans indicate that this pathway predominates in humans as well. A minor pathway involves hydrolysis and dechlorination at carbon-3 to form 1-chloroallyl alcohol, which undergoes further oxidation and oxidative dechlorination at carbon-1 and breakage of the chain to form carbon dioxide and acetyl-CoA, which may be incorporated into body tissues. Another minor pathway involves epoxide formation followed by dechlorination and additional oxidation to form glycidaldehyde and methylglyoxal.

Data from rats indicate that 2,3-dichloropropene is subject to the same three metabolic pathways as observed for the 1,3-isomer (ATSDR, 2006). Conjugation with glutathione predominantes. The other two minor pathways (epoxide formation and hydrolysis with dechlorination) yield metabolites that have been shown to be mutagenic, which apparently accounts for the carcinogenicity of the 1,3-isomer.

Excretion of dichloropropene metabolites is primarily in the urine following inhalation exposure of rats (ATSDR, 2006). Fecal excretion is also significant, more so for 2,3-dichloropropene than for 1,3-dichloropropene. Approximately 3 percent of the absorbed amount of 2,3-dichloropropene was excreted as carbon dioxide. Similar patterns are observed after oral exposure of rats; urinary excretion predominates. cis-1,3-Dichloropropene is metabolized and excreted in the urine as mercapturic acid metabolites more efficiently than the trans-isomer. trans-1,3-Dichloropropene is metabolized to carbon dioxide more efficiently than the cis-isomer.

The limited toxicokinetic data suggest that there is more variation between the dichloropropene isomers than between species. The differences appear to be quantitative rather than qualitative, supporting the assumption that the toxicity of the isomers may be similar.

4.0 Dermal Exposure

EPA (2004b) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. As noted above, the dichloropropenes are extensively absorbed from the GI tract. Therefore, no GAF is developed for the dichloropropenes and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

It appears from the previous section that the dichloropropenes are absorbed through the skin, but data are not available regarding the extent of dermal uptake from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004b). Therefore, dermal uptake of the dichloropropenes from soil is not quantified.

As noted above, the value selected for log K_{ow} for 1,3-dichloropropene differs slightly from that provided by EPA (2004b). Therefore, values for t*, K_p, τ, FA and B tabulated below were not taken directly from EPA (2004b). Instead, they were recalculated from MW and the accepted log K_{ow} given above using the EPA (2004b) method. Values for t*, K_p, τ, FA and B for the other isomers were also estimated from the MW and log K_{ow} values given above and the EPA (2004b) method, as follows:

Chemical	t*	K_p	τ	FA	B
1,1-Dichloropropene	1.05	1.77E-2	0.44	1	0[a]
1,2-Dichloropropene	1.05	1.77E-2	0.44	1	0[a]
1,3-Dichloropropene	1.05	7.92E-3	0.44	1	0[a]
2,3-Dichloropropene	1.05	1.50E-2	0.44	1	0[a]

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Sufficiently small to be rounded to zero.

5.0 Noncancer Effects Evaluation

Toxicity data were not located for 1,1-dichloropropene. Acute toxicity data for 1,2-dichloropropene include an oral LD_{50} value of 2 g/kg in rats and a dermal LD_{50} of 8750 mg/kg in rabbits (HSDB, 2010), indicating that the acute toxicity of the 1,2-isomer is relatively low. Descriptions of agonal signs or necropsy findings were not located. Data regarding prolonged exposure to 1,2-dichloropropene were not located.

Oral LD_{50} values for 1,3-dichloropropene include 94 to 713 mg/kg in rats and 215 to 640 mg/kg in mice (ATSDR, 2006; HSDB, 2010). The wide range of values within species probably reflects differences in the preparations and formulations tested. Necropsy of the decedents from one study revealed liver and kidney damage. EPA (2010) verified a chronic oral RfD for 1,3-dichloropropene of 3E-2 mg/kg-day based on chronic stomach irritation in rats treated with a microencapsulated formulation of Telone II (96 percent 1,3-dichloropropene) in the diet. In the key study, male and female rats were fed diets that provided dose rates of 0 (control), 2.5, 12.5 or 25 mg/kg-day for 24 months. Decreased body weight gain was observed in a dose-related manner that reached statistical significance in rats of both sexes in the high dose group. The incidence of hyperplasia of the epithelium of the forestomach showed a statistically significant increase in rats in the 12.5 and 25 mg/kg-day groups. Hyperplasia of the forestomach was interpreted as a response to chronic stomach irritation, which was identified as the critical effect of prolonged ingestion exposure. The 95 percent lower confidence limit on the maximum likelihood estimate of the dose corresponding to a 10 percent response rate ($BMDL_{10}$) was estimated as 3.4 mg/kg-day. Application of an uncertainty factor of 100 (factors of 10 each for intra- and inter-species variation) to the $BMDL_{10}$ yields the verified chronic oral RfD of 3E-2 mg/kg-day. Confidence in the verified chronic oral RfD is high. Stomach irritation is considered the critical effect of prolonged oral exposure.

EPA (1997) provides a provisional subchronic oral RfD for 1,3-dichloropropene of 3E-3 mg/kg-day based on a NOEL for increased organ weights of 3 mg/kg-day in rats in a 90-day dietary study. A LOAEL was not identified. An uncertainty factor of 1000 was used. It is likely that the increase in organ weights reported by EPA (1997) may reflect increased organ-to-body weight ratios, which would be expected and consistent with the observation of decreased overall body weight gain in the chronic study described in the previous paragraph. The NOEL of 3 mg/kg-day in the 90-day study also is consistent with the effect levels observed in the chronic study described in the previous paragraph. It is not reasonable, however, that a subchronic oral RfD should be lower than a chronic oral RfD. Therefore, the verified chronic oral RfD of 3E-2 mg/kg-day is adopted as sufficiently protective for subchronic as well as chronic exposure.

Occupational exposure to high levels of 1,3-dichloropropene in air is associated with skin, eye and respiratory tract irritation (ACGIH, 1991). EPA (2010) presents a verified inhalation RfC for 1,3-dichloropropene of 2E-2 mg/m^3 based on a human equivalent adjusted 95 percent lower confidence limit on the maximum likelihood estimate of the concentration corresponding to a 10 percent response rate ($BMCL_{10}$) of 0.72 mg/m^3 in an intermittent exposure inhalation study in mice. In the key study rats and mice were exposed to technical grade 1,3-dichloropropene at concentrations of 0 (control), 5 ppm (22.7 mg/m^3), 20 ppm (90.8 mg/m^3) or 60 ppm (272 mg/m^3) 6 hours/day, 5 days/week for two years. The only treatment-related effects in rats were histopathological changes in the nasal epithelium in the high-concentration group after 24 months, but not after 6 or 12 months of exposure. Effects in mice included masses in the lungs in males in the high-dose group, and slight hypertrophy and hyperplasia of the nasal epithelium in the two lower concentration groups. Hyperplasia of the urinary bladder epithelium was also observed in female mice in the high-concentration group. Hypertrophy and hyperplasia in the nasal epithelium of mice was identified as the critical effect of inhalation exposure. An uncertainty factor of 30 was applied to the human equivalent adjusted $BMCL_{10}$ of 0.72 mg/m^3 to derive the verified chronic inhalation RfC of 2E-2 mg/m^3. The chronic inhalation RfC is equivalent to an inhalation RfD of 5.7E-3 mg/kg-day.

EPA (1997) adopted the verified chronic inhalation RfC of 2E-2 mg/m^3 (5.7E-3 mg/kg-day) as the provisional subchronic inhalation RfC. Hypertrophy and hyperplasia of the nasal epithelium is the critical effect of prolonged inhalation exposure to 1,3-dichloropropene.

Toxicity date for 2,3-dichloropropene are largely limited to an oral LD_{50} values in rats of 285 to 320 mg/kg and a dermal LD_{50} in rabbits of 1580 mg/kg (ATSDR, 2006; HSDB, 2010).

Toxicity information sufficient for RfD or RfC estimation is not available for 1,1-, 1,2- or 2,3-dichloropropene. 1,3-Dichloropropene is selected as a surrogate for 1,1-, 1,2- and 2,3-dichloropropene because of the similarity in chemical structure. Therefore, the noncancer oral and inhalation toxicity values described for 1,3-dichloropropene are applied also to 1,1-, 1,2- and 2,3-dichloropropene.

6.0 Carcinogenicity Evaluation

In an earlier assessment, EPA (2010) verified 1,3-dichloropropene as a cancer weight-of-evidence B2 compound – probable human carcinogen – on the basis of increased incidences of various tumor types in rats and mice in three oral studies and an inhalation study. Also, 1,3-dichloropropene yields positive results in tests for mutagenic activity and is structurally similar

to chemicals known to produce similar types of tumors in rodents. Consistent with more recent EPA (2005) cancer assessment guidelines, EPA (2010) characterized 1,3-dichloropropene as likely to be a human carcinogen.

EPA (2010) developed and verified oral cancer SFs of 1E-1 per mg/kg-day from the incidence of urinary bladder tumors in female mice in a two-year gavage study, 5E-2 per mg/kg-day for liver tumors in male rats in the same study, and 5E-2 per mg/kg-day for liver tumors in male rats in a two-year dietary study. EPA (2010) recommended the SF of 1E-1 per mg/kg-day because it is the most conservative estimate and there was less uncertainty regarding the actual dose delivered.

EPA (2010) developed and verified an inhalation URF of 4E-6 per $\mu g/m^3$ based on the incidence of lung tumors in male mice in a two-year inhalation study. The URF is equivalent to an inhalation SF of 1.4E-2 per mg/kg-day.

The toxicokinetic data summarized above and the EPA (2010) cancer evaluation strongly indicate that the mode of action for carcinogenicity arises from genotoxicity. EPA (2005) proposed that the oral and inhalation cancer potency factors developed for genotoxic chemicals should be modified with the age dependent adjustment factors (ADAF) of 10 for children under 2 years of age, 3 for children 2 to 16 years of age, and 1 for adults and children over 16 years of age.

7.0 Toxicity Summary

Toxicity values for 1,3-dichloropropene are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
3E-2	3E-2	St	2E-2/ 5.7E-3	2E-2/ 5.7E-3	NE	See above	1E-1	See above	4E-6	1.4E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: St = stomach; NE = nasal epithelium.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

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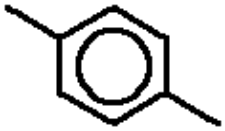
XYLENES (DIMETHYLBENZENE), TOTAL (1330-20-7)



p-Xylene

1.0 Introduction and Physical Properties

The xylenes, also known as dimethylbenzenes, exist as three isomers: 2-xylene (1,2-dimethylbenzene, ortho- or o-xylene) (95-47-6), 3-xylene (1,3-dimethylbenzene, meta- or m-xylene) (108-38-3) and 4-xylene (1,4-dimethylbenzene, para- or p-xylene) (106-42-3) (HSDB, 2010). The o-, m-, and p-xylene nomenclature will be used herein to be consistent with the manner in which xylene concentrations in environmental media are usually reported by the analytical laboratories.

The xylenes are **VOCs** derived from coal tar, coal gas, and petroleum distillation (ATSDR, 1995; HSDB, 2010). They occur as clear liquids in the pure state at most ambient temperatures, although 4-xylene occurs as colorless plates or prisms at temperatures below 56EF (13.33EC). Natural sources include petroleum, forest fires and volatiles from plants.

Xylenes are used in the manufacture of other organic compounds, dyes, insecticides and pharmaceuticals, and as solvents (ATSDR, 1995; HSDB, 2010). They are components of aviation gasoline, asphalt and naphtha, and automobile exhaust. Technical xylene consists of approximately 44 percent m-xylene, 20 percent o-xylene, 20 percent p-xylene, and 15 percent ethylbenzene. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
o-Xylene (95-47-6)										
106.16[a]	3.12[a]	5.20E-3[b] (25°C)	2.56[b]	8.70E-2[b] (25°C)	1.00E-5[b] (25°C)	8.70E-3[a] (25°C)	1.78E+2[b] (20-25°C)	417.60[c]	630.30[c]	8.66E+3[c]
m-Xylene (108-38-3)										
106.2[d]	3.20[d]	7.34E-3[b] (25°C)	2.61[b]	7.00E-2[b] (25°C)	7.80E-6[b] (25°C)	1.09E-2[a] (25°C)	1.16E+2[b] (20-25°C)	412.27[c]	617.05[c]	8.52E+3[c]
p-Xylene (106-42-3)										
106.16[a]	3.15[a]	7.66E-3[b] (25°C)	2.59[b]	7.69E-2[b] (25°C)	8.44E-6[b] (25°C)	1.16E-2[a] (25°C)	1.85E+2[b] (20-25°C)	411.52[c]	616.20[c]	8.53E+3[c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
[b]U.S. U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, Washington, D.C., 9355.4-24, December.
[c]U.S. Environmental Protection Agency (EPA), 2000, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings, Revised***, Office of Emergency and Remedial Response, Washington, DC, December.
[d]U.S. Environmental Protection Agency (EPA), 2004a, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)***, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

It is recommended that the physical properties for m-xylene should be applied to mixed xylene isomers or data where the specific isomer is not identified, because m-xylene is the major component of the technical mixture and is likely to predominate following xylene releases.

2.0 Environmental Fate and Transport

Naturally occurring sources of xylenes are petroleum, forest fires and volatiles from plants (ATSDR, 1995; HSDB, 2010). The production and use of xylenes in petroleum products and as chemical solvents and intermediates may result in their release to the environment as fugitives and through various waste streams. Outgassing from landfills and automobile exhaust are other significant sources. Spills, leaking underground storage tanks and associated plumbing, and leaching from landfills may release xylenes to soil and groundwater.

Xylenes will exist solely in the vapor phase in the ambient atmosphere, where they are subject to degradation by reaction with photochemically-produced hydroxyl radicals as the only significant removal process (ATSDR, 1995; HSDB, 2010). The atmospheric lifetime of xylenes is about 1 to 2 days. Products of photooxidation include a wide variety of ring hydroxylation and ring cleavage products.

Volatilization is expected to be the major removal mechanism for xylenes released to surface soil, but, depending on the size and rate of release, the majority of the release may infiltrate to subsurface soil (ATSDR, 1995). Photooxidation may degrade a significant part of the small amount that remains at the surface. Biodegradation is probably the only significant subsurface removal mechanism, but it is expected to be slow. A wide range of experimental K_{oc} values have been reported in soil samples with differing pH and organic carbon content (HSDB, 2010), suggesting a wide range in the extent to which the xylenes adsorb to soil particles. Generally, however, xylene is fairly mobile and may readily leach to groundwater.

Volatilization is probably the most significant removal mechanism for xylenes in surface water (ATSDR, 1995). Biodegradation may occur in surface water, groundwater and landfill leachate, although quite slowly. Oxidative reactions are expected to be insignificant. Xylenes are expected to adsorb somewhat to suspended solids and sediment in water (HSDB, 2010).

Xylenes are not expected to participate significantly in food-chain pathways (ATSDR, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Xylenes are rapidly and extensively absorbed during inhalation and following oral exposure (ATSDR, 1995). In humans, approximately 50 to 73 percent of the inhaled concentration is retained. In laboratory animals, approximately 90 percent of an ingested dose is absorbed. Liquid xylene is absorbed through the skin of humans, but dermal uptake of xylene vapors approximates only 1 percent of inhalation uptake. A study in rats showed that dermal uptake of xylene adsorbed to soil is much slower than dermal uptake of the neat liquid.

Absorbed xylene is largely bound to serum protein in the blood and is distributed rapidly and widely throughout the body, but particularly to highly vascular tissues and those high in lipid content (e.g., blood, brain, liver) (ATSDR, 1995). Levels in fetal tissues approximate 2 percent of those measured in maternal brain.

The predominant metabolic pathway in humans and animals for all xylene isomers is oxidation of one of the methyl groups to the corresponding alcohol, with further oxidation to the corresponding methylbenzoic acid (ATSDR, 1995). The methylbenzoic acids conjugate with

glycine to form the corresponding methylhippuric acids, which are the major urinary metabolites. Much lesser quantities of the alcohols and o-methylbenzoic acid are also excreted in the urine. Methylbenzaldehyde, proposed as the ultimate toxicant because it inactivates enzymes involved in electron transport, has been identified in laboratory animals but not in humans. Metabolism in laboratory animals differs from that in humans largely in the nature of the conjugates that are formed. Ring hydroxylation to form phenolic compounds represents a minor metabolic pathway is both humans and animals.

The xylenes are rapidly excreted from the body, largely in the form of urinary metabolites (ATSDR, 1955) as described above. Approximately 5 percent of an oral dose is eliminated in the breath as unchanged compound. Residues are slightly more persistent in adiposa and muscle tissue than other tissues of the body, but no organ or tissue accumulates xylene residues.

4.0 Dermal Exposure

EPA (2003) recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Xylenes are nearly completely absorbed from the GI tract; therefore, no GAF is developed for the xylenes and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Xylene in soil is absorbed by the skin, albeit very slowly, but quantitative data were not located. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of xylenes from soil is not evaluated. Values for t*, K_p, τ, FA and B are provided by EPA (2003) for m-xylene as follows:

Chemical	t*	K_p	τ	FA	B
m-Xylene	1.01	5.3E-2	0.42	1.0	0.2
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

The dermal uptake parameters presented by EPA (2004) for m-xylene are applied to all the xylene isomers as well as to total xylenes.

5.0 Noncancer Effects Evaluation

Oral LD_{50} values for xylenes include 3.5 to 8.6 g/kg in rats and 1.6 to 5.6 g/kg in mice (HSDB, 2010), suggesting that the acute toxicity is low, but that mice may be slightly more sensitive than rats to the acute effects of ingested xylenes. Prolonged oral exposure of animals to xylenes is associated with reduced body weight and survival without histopathological alterations in the internal organs (EPA, 2010). EPA (2010) confirmed a previously verified chronic oral RfD for total xylenes of 2E-1 mg/kg-day based on a NOAEL of 250 mg/kg, equivalent to a continuous dose rate of 179 mg/kg-day, for decreased body weight and survival in male rats in chronic gavage studies with mixed xylenes. The LOAEL was 500 mg/kg, equivalent to 357 mg/kg-day. Mice were also included in the study. The LOAEL in mice was 1000 mg/kg, equivalent to 714 mg/kg-day, associated with hyperactivity, which was consistently observed at 5 to 30 minutes after treatment. The mice did not exhibit the effects on body weight or survival that was observed with the rats, establishing the rat as the more sensitive species to prolonged oral

exposure. An uncertainty factor of 1000 was applied to the rat NOAEL to derive the verified chronic oral RfD. Confidence in the chronic oral RfD is medium.

EPA (1997) presented provisional chronic oral RfDs for o-xylene and m-xylene of 2E+0 mg/kg-day based on an earlier IRIS evaluation of the same rat and mouse gavage study. Given the EPA (2010) re-evaluation of this study and the lowering of the chronic oral RfD by an order of magnitude, it seems prudent to adopt the verified chronic oral RfD of 2E-1 mg/kg-day for use with mixed xylenes and all xylene isomers. The critical effects of prolonged oral exposure to xylenes are reduced body weight and reduced survival. The verified chronic oral RfD is adopted as sufficiently protective for subchronic oral exposure to mixed xylenes and all xylene isomers.

Occupational exposure to xylenes induces CNS effects and GI disturbances (ACGIH, 1991). Other effects attributed to occupational exposure to xylene (blood dyscrasias, and heart, liver and kidney damage) may arise from concurrent exposure to other chemicals. EPA (2010) verified a chronic inhalation RfC of 1E-1 mg/m^3 based on CNS effects. In the key study, male rats were intermittently exposed to m-xylene at airborne concentrations of 0, 50 or 100 ppm for three months. Endpoints evaluated included spontaneous motor activity and motor coordination as measured by performance on a rotorod. Body and organ weights and limited blood chemistry and hematological parameters were also evaluated, but minor alterations in these endpoints were not attributed to exposure to m-xylene. The study identified 50 ppm as the NOAEL and 100 ppm as the LOAEL for impaired rotorod performance. The NOAEL is equivalent to an adjusted human equivalent concentration of 39 mg/m^3. Application of an uncertainty factor of 300 to the NOAEL yields the verified chronic inhalation RfC of 1E-1 mg/m^3, which is equivalent to a chronic inhalation RfD of 2.9E-2 mg/kg-day. The uncertainty factor consists of a factor of 10 to provide greater protection for more sensitive individuals, and factors of 3 each to extrapolate from animals to humans, to expand from subchronic to chronic exposure, and to account for deficiencies in the data base. The CNS is considered to be the target organ for prolonged inhalation exposure to xylenes. Confidence in the chronic inhalation RfC is medium.

No EPA-derived subchronic inhalation RfC is available for exposure to xylene. However, a preliminary subchronic inhalation RfC can be derived by applying an uncertainty factor of 100 to the NOAEL of 39 mg/m^3 in the subchronic rotorod study in rats described above. The uncertainty factor of 100 reflects the chronic uncertainty factor of 300 without the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic inhalation RfC so derived is 4E-1 mg/m^3, which is equivalent to a subchronic inhalation RfD of 1.1E-1 mg/kg-day.

6.0 Carcinogenicity Evaluation

Xylene was previously characterized as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans). Using a more descriptive characterization, EPA (2010) concluded that the cancer *data are inadequate for an assessment of the carcinogenic potential of xylenes*. Adequate human data on the carcinogenicity of the xylenes are not available, and the available animal data are inconclusive as to the ability of xylenes to cause a carcinogenic response. Evaluations of the genotoxic effects of xylenes have consistently given negative results. Potency factors are not available.

7.0 Toxicity Summary

Toxicity values for the xylenes are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2E-1	2E-1	BW, Su	4E-1/ 1.1E-1	1E-1/ 2.9E-2	CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: BW = reduced body weight; Su = reduced survival; CNS = central nervous system.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

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APPENDIX D

RISK CHARACTERIZATION SPREADSHEETS

Table D-1

Groundskeeper, Current and Future Scenarios, Exposure to Soil [a]
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Inorganics										
Beryllium	1.14E+01	3.97E-06	1.11E-05	NA	5.55E-03	NA	NA	NA	NA	NA
Thallium	2.95E+00	1.03E-06	2.89E-06	NA	4.44E-02	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1016	1.58E-01	5.52E-08	1.55E-07	3.86E-09	2.21E-03	4.42E-09	5.10E-08	1.43E-07	3.57E-09	2.04E-03
Semivolatile Organic Compounds										
Benzo(a)pyrene	1.70E-01	5.94E-08	1.66E-07	4.34E-07	NA	4.42E-09	5.10E-08	1.43E-07	3.72E-07	NA
Benzo(b)fluoranthene	1.60E-01	5.59E-08	1.57E-07	4.08E-08	NA	4.16E-09	4.80E-08	1.34E-07	3.50E-08	NA
Total ILCR or HI				**4.78E-07**	**5.22E-02**				**4.11E-07**	**2.04E-03**

[a] This receptor is exposed to both surface soil and total soil; however all COPCs come from surface soil so the EPC is the same for both.

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter

Table D-1

Groundskeeper, Current and Future Scenarios, Exposure to Soil [a]
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Inorganics							
Beryllium	1.14E-06	7.93E-08	2.22E-07	6.66E-07	3.90E-02	**6.66E-07**	**4.45E-02**
Thallium	2.95E-07	2.06E-08	5.77E-08	NA	NA	**NA**	**4.44E-02**
Polychlorinated biphenyls (PCB)							
Aroclor 1016	1.58E-08	1.10E-09	3.09E-09	7.73E-11	NA	**7.51E-09**	**4.25E-03**
Semivolatile Organic Compounds							
Benzo(a)pyrene	1.70E-08	1.19E-09	3.33E-09	3.68E-09	NA	**8.09E-07**	**NA**
Benzo(b)fluoranthene	1.60E-08	1.12E-09	3.13E-09	3.47E-10	NA	**7.62E-08**	**NA**
Total ILCR or HI				**6.70E-07**	**3.90E-02**	**1.56E-06**	**9.32E-02**

[a] This receptor is exposed to both surface soil and total soil; however all COPCs come from surface soil so the EPC is the same for both.

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter

Table D-2

Groundskeeper, Future Scenario, Exposure to Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics												
Selenium	1.95E-02	6.81E-05	1.91E-04	NA	3.82E-02	1.95E-08	2.25E-07	6.30E-07	NA	1.26E-04	**NA**	**3.83E-02**
Semivolatile Organic Compounds												
Naphthalene	7.40E-03	2.59E-05	7.24E-05	NA	3.62E-03	7.19E-07	8.30E-06	2.32E-05	NA	1.16E-03	**NA**	**4.78E-03**
Volatile Organic Compounds												
Benzene	3.36E-01	1.17E-03	3.29E-03	6.46E-05	8.22E-01	7.79E-06	8.99E-05	2.52E-04	4.94E-06	6.29E-02	**6.95E-05**	**8.85E-01**
Chloromethane	4.49E-02	1.57E-04	4.39E-04	NA	NA	2.07E-07	2.39E-06	6.70E-06	NA	NA	**NA**	**NA**
trans-1,3-Dichloropropene	1.70E-03	5.94E-06	1.66E-05	5.94E-07	5.54E-04	1.36E-08	1.56E-07	4.38E-07	1.56E-08	1.46E-05	**6.10E-07**	**5.69E-04**
Ethylbenzene	5.71E-02	2.00E-04	5.59E-04	NA	5.59E-03	5.01E-06	5.78E-05	1.62E-04	NA	1.62E-03	**NA**	**7.21E-03**
Xylenes, total	6.30E-01	2.20E-03	6.16E-03	NA	3.08E-02	5.98E-05	6.90E-04	1.93E-03	NA	9.66E-03	**NA**	**4.05E-02**
Total ILCR or HI				**6.52E-05**	**9.01E-01**				**4.96E-06**	**7.55E-02**	**7.01E-05**	**9.76E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram

Table D-3

Groundskeeper, Future Scenario, Exposure to Groundwater, Overburden Wells
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics												
Nickel	7.42E-02	2.59E-04	7.26E-04	NA	3.63E-02	1.48E-08	1.71E-07	4.79E-07	NA	5.99E-04	**NA**	**3.69E-02**
Total ILCR or HI				**NA**	**3.63E-02**				**NA**	**5.99E-04**	**NA**	**3.69E-02**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram

Table D-4

Indoor Worker, Future Scenario, Exposure to Surface Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion					
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics							
Beryllium	1.14E+01	1.98E-06	5.55E-06	NA	2.78E-03	**NA**	**2.78E-03**
Thallium	2.95E+00	5.16E-07	1.44E-06	NA	2.22E-02	**NA**	**2.22E-02**
Polychlorinated biphenyls (PCB)							
Aroclor 1016	1.58E-01	2.76E-08	7.73E-08	1.93E-09	1.10E-03	**1.93E-09**	**1.10E-03**
Semivolatile Organic Compounds							
Benzo(a)pyrene	1.70E-01	2.97E-08	8.32E-08	2.17E-07	NA	**2.17E-07**	**NA**
Benzo(b)fluoranthene	1.60E-01	2.80E-08	7.83E-08	2.04E-08	NA	**2.04E-08**	**NA**
Total ILCR or HI						**2.39E-07**	**2.61E-02**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram

Table D-5

Indoor Worker, Future Scenario, Exposure to Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics												
Selenium	1.95E-02	6.81E-05	1.91E-04	NA	3.82E-02	1.95E-08	2.25E-07	6.30E-07	NA	1.26E-04	**NA**	**3.83E-02**
Semivolatile Organic Compounds												
Naphthalene	7.40E-03	2.59E-05	7.24E-05	NA	3.62E-03	7.19E-07	8.30E-06	2.32E-05	NA	1.16E-03	**NA**	**4.78E-03**
Volatile Organic Compounds												
Benzene	3.36E-01	1.17E-03	3.29E-03	6.46E-05	8.22E-01	7.79E-06	8.99E-05	2.52E-04	4.94E-06	6.29E-02	**6.95E-05**	**8.85E-01**
Chloromethane	4.49E-02	1.57E-04	4.39E-04	NA	NA	2.07E-07	2.39E-06	6.70E-06	NA	NA	**NA**	**NA**
trans-1,3-Dichloropropene	1.70E-03	5.94E-06	1.66E-05	5.94E-07	5.54E-04	1.36E-08	1.56E-07	4.38E-07	1.56E-08	1.46E-05	**6.10E-07**	**5.69E-04**
Ethylbenzene	5.71E-02	2.00E-04	5.59E-04	NA	5.59E-03	5.01E-06	5.78E-05	1.62E-04	NA	1.62E-03	**NA**	**7.21E-03**
Xylenes, total	6.30E-01	2.20E-03	6.16E-03	NA	3.08E-02	5.98E-05	6.90E-04	1.93E-03	NA	9.66E-03	**NA**	**4.05E-02**
Total ILCR or HI				**6.52E-05**	**9.01E-01**				**4.96E-06**	**7.55E-02**	**7.01E-05**	**9.76E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram

Table D-6

Indoor Worker, Future Scenario, Exposure to Overburden Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics												
Nickel	7.42E-02	2.59E-04	7.26E-04	NA	3.63E-02	1.48E-08	1.71E-07	4.79E-07	NA	5.99E-04	**NA**	**3.69E-02**
Total ILCR or HI				**NA**	**3.63E-02**				**NA**	**5.99E-04**	**NA**	**3.69E-02**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

Table D-7

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Inorganics										
Beryllium	1.14E+01	2.62E-07	3.65E-05	NA	1.83E-02	NA	NA	NA	NA	NA
Thallium	2.95E+00	6.81E-08	9.50E-06	NA	1.46E-01	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1016	1.58E-01	3.64E-09	5.09E-07	2.55E-10	7.27E-03	6.64E-09	1.53E-09	2.14E-07	1.07E-10	3.05E-03
Semivolatile Organic Compounds										
Benzo(a)pyrene	1.70E-01	3.92E-09	5.47E-07	2.86E-08	NA	6.63E-09	1.53E-09	2.13E-07	1.12E-08	NA
Benzo(b)fluoranthene	1.60E-01	3.69E-09	5.15E-07	2.69E-09	NA	6.24E-09	1.44E-09	2.01E-07	1.05E-09	NA
Total ILCR or HI				**3.16E-08**	**1.72E-01**				**1.23E-08**	**3.05E-03**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter

Table D-7

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Inorganics							
Beryllium	3.97E-06	5.55E-09	7.75E-07	4.66E-08	1.36E-01	**4.66E-08**	**1.54E-01**
Thallium	1.03E-06	1.44E-09	2.02E-07	NA	NA	**NA**	**1.46E-01**
Polychlorinated biphenyls (PCB)							
Aroclor 1016	5.53E-08	7.73E-11	1.08E-08	5.41E-12	NA	**3.68E-10**	**1.03E-02**
Semivolatile Organic Compounds							
Benzo(a)pyrene	5.95E-08	8.32E-11	1.16E-08	2.58E-10	NA	**4.00E-08**	**NA**
Benzo(b)fluoranthene	5.60E-08	7.83E-11	1.09E-08	2.43E-11	NA	**3.77E-09**	**NA**
Total ILCR or HI				**4.69E-08**	**1.36E-01**	**9.08E-08**	**3.11E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter

Table D-8

Construction Worker, Current and Future Scenarios, Exposure to Sediment
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
	EPC	Dose	Dose			Absorbed	Dose	Dose			**Total**	**Total**
Chemicals of Potential Concern	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ	**ILCR**	**HI**
Inorganics												
Arsenic	9.03E+00	2.08E-07	2.91E-05	3.12E-07	9.69E-02	8.12E-08	1.87E-08	2.62E-06	2.81E-08	8.72E-03	**3.40E-07**	**1.06E-01**
Chromium	1.10E+01	2.53E-07	3.54E-05	NA	1.69E-03	NA	NA	NA	NA	NA	**NA**	**1.69E-03**
Total ILCR or HI				**3.12E-07**	**9.86E-02**				**2.81E-08**	**8.72E-03**	**3.40E-07**	**1.07E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-9

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Adult Dose mg/kg-day	Child Dose mg/kg-day	Resident ILCR	Adult Dose Absorbed mg/cm^2-day	Adult Dose mg/kg-day	Child Dose Absorbed mg/cm^2-day	Child Dose mg/kg-day	Resident ILCR
Inorganics									
Beryllium	1.14E+01	4.80E-06	1.12E-05	NA	NA	NA	NA	NA	NA
Thallium	2.95E+00	1.25E-06	2.91E-06	NA	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)									
Aroclor 1016	1.58E-01	6.68E-08	1.56E-07	1.56E-08	1.55E-09	4.15E-08	4.42E-09	6.79E-08	7.65E-09
Semivolatile Organic Compounds									
Benzo(a)pyrene	1.70E-01	7.19E-08	1.68E-07	1.75E-06	1.55E-09	4.14E-08	4.42E-09	6.78E-08	7.97E-07
Benzo(b)fluoranthene	1.60E-01	6.76E-08	1.58E-07	1.65E-07	1.46E-09	3.90E-08	4.16E-09	6.38E-08	7.50E-08
Total ILCR				**1.93E-06**					**8.80E-07**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter

Table D-9

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Adult Dose mg/kg-day	Inhalation Child Dose mg/kg-day	Inhalation Resident ILCR	**All Pathways Total ILCR**
Inorganics					
Beryllium	2.24E-08	2.10E-09	1.22E-09	2.79E-08	**2.79E-08**
Thallium	5.81E-09	5.46E-10	3.18E-10	NA	**NA**
Polychlorinated biphenyls (PCB)					
Aroclor 1016	3.11E-10	2.92E-11	1.71E-11	3.24E-12	**2.32E-08**
Semivolatile Organic Compounds					
Benzo(a)pyrene	3.35E-10	3.15E-11	1.83E-11	1.54E-10	**2.55E-06**
Benzo(b)fluoranthene	3.15E-10	2.96E-11	1.73E-11	1.45E-11	**2.40E-07**
Total ILCR				**2.81E-08**	**2.84E-06**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter

Table D-10

On-Site Child Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Concentration	Inhalation		**All**
	EPC	Child Dose	Child Resident	Dose Absorbed	Child Dose	Child Resident	in Air C_a	Child Dose	Child Resident	**Pathways Total**
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	**HI**
Inorganics										
Beryllium	1.14E+01	1.31E-04	6.53E-02	NA	NA	NA	2.24E-08	1.43E-08	2.51E-03	**6.78E-02**
Thallium	2.95E+00	3.40E-05	5.22E-01	NA	NA	NA	5.81E-09	3.72E-09	NA	**5.22E-01**
Polychlorinated biphenyls (PCB)										
Aroclor 1016	1.58E-01	1.82E-06	2.60E-02	4.42E-09	7.92E-07	1.13E-02	3.11E-10	1.99E-10	NA	**3.73E-02**
Semivolatile Organic Compounds										
Benzo(a)pyrene	1.70E-01	1.96E-06	NA	4.42E-09	7.91E-07	NA	3.35E-10	2.14E-10	NA	**NA**
Benzo(b)fluoranthene	1.60E-01	1.84E-06	NA	4.16E-09	7.45E-07	NA	3.15E-10	2.01E-10	NA	**NA**
Total HI			**6.14E-01**			**1.13E-02**			**2.51E-03**	**6.28E-01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter

Table D-11

On-Site Adult Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Concentration	Inhalation		All
	EPC	Adult Dose	Adult Resident	Dose Absorbed	Adult Dose	Adult Resident	in Air C_a	Adult Dose	Adult Resident	**Pathways Total**
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	**HI**
Inorganics										
Beryllium	1.14E+01	1.40E-05	7.00E-03	NA	NA	NA	2.24E-08	6.12E-09	1.07E-03	**8.07E-03**
Thallium	2.95E+00	3.64E-06	5.60E-02	NA	NA	NA	5.81E-09	1.59E-09	NA	**5.60E-02**
Polychlorinated biphenyls (PCB)										
Aroclor 1016	1.58E-01	1.95E-07	2.78E-03	1.55E-09	1.21E-07	1.73E-03	3.11E-10	8.53E-11	NA	**4.51E-03**
Semivolatile Organic Compounds										
Benzo(a)pyrene	1.70E-01	2.10E-07	NA	1.55E-09	1.21E-07	NA	3.35E-10	9.17E-11	NA	**NA**
Benzo(b)fluoranthene	1.60E-01	1.97E-07	NA	1.46E-09	1.14E-07	NA	3.15E-10	8.63E-11	NA	**NA**
Total HI			**6.58E-02**			**1.73E-03**			**1.07E-03**	**6.86E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter

Table D-12

On-Site Resident, Future Scenario, Cancer Risk from Exposure to Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					Inhalation of VOCs				All
		Adult	Child		Dose	Adult	Dose	Child		Concentration	Adult	Child		Pathways
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	ILCR	in Air mg/m^3	Dose mg/kg-day	Dose mg/kg-day	ILCR	**Total ILCR**
Inorganics														
Selenium	1.95E-02	1.83E-04	1.07E-04	NA	3.90E-09	3.66E-07	6.49E-09	2.35E-07	NA	NA	NA	NA	NA	**NA**
Semivolatile Organic Compounds														
Naphthalene	7.40E-03	6.95E-05	4.05E-05	NA	3.22E-07	3.02E-05	4.15E-07	1.50E-05	NA	3.70E-03	1.45E-05	8.43E-06	2.75E-06	**2.75E-06**
Volatile Organic Compounds														
Benzene	3.36E-01	3.16E-03	1.84E-03	2.75E-04	4.13E-06	3.88E-04	4.74E-06	1.71E-04	3.08E-05	1.68E-01	6.57E-04	3.83E-04	2.81E-05	**3.34E-04**
Chloromethane	4.49E-02	4.22E-04	2.46E-04	NA	8.89E-08	8.35E-06	1.09E-07	3.93E-06	NA	2.25E-02	8.78E-05	5.12E-05	NA	**NA**
trans-1,3-Dichloropropene	1.70E-03	1.60E-05	9.32E-06	2.53E-06	6.06E-09	5.69E-07	7.82E-09	2.83E-07	8.52E-08	8.50E-04	3.33E-06	1.94E-06	7.37E-08	**2.69E-06**
Ethylbenzene	5.71E-02	5.36E-04	3.13E-04	NA	2.24E-06	2.11E-04	2.89E-06	1.05E-04	NA	2.86E-02	1.12E-04	6.51E-05	NA	**NA**
Xylenes, total	6.30E-01	5.92E-03	3.45E-03	NA	2.67E-05	2.51E-03	3.45E-05	1.25E-03	NA	3.15E-01	1.23E-03	7.18E-04	NA	**NA**
Total ILCR				**2.77E-04**					**3.08E-05**				**3.09E-05**	**3.39E-04**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-13

Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			All
		Child		Dose	Child		Concentration	Child		Pathways
	EPC	Dose		Absorbed	Dose		in Air	Dose		Total
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	HI
Inorganics										
Selenium	1.95E-02	1.25E-03	2.49E-01	6.49E-09	2.74E-06	5.48E-04	NA	NA	NA	**2.50E-01**
Semivolatile Organic Compounds										
Naphthalene	7.40E-03	4.73E-04	2.37E-02	4.15E-07	1.75E-04	8.76E-03	3.70E-03	9.84E-05	8.46E-08	**3.24E-02**
Volatile Organic Compounds										
Benzene	3.36E-01	2.15E-02	5.37E+00	4.74E-06	2.00E-03	5.00E-01	1.68E-01	4.47E-03	3.84E-05	**5.87E+00**
Chloromethane	4.49E-02	2.87E-03	NA	1.09E-07	4.58E-05	NA	2.25E-02	5.97E-04	1.55E-05	**1.55E-05**
trans-1,3-Dichloropropene	1.70E-03	1.09E-04	3.62E-03	7.82E-09	3.30E-06	1.10E-04	8.50E-04	2.26E-05	1.29E-07	**3.73E-03**
Ethylbenzene	5.71E-02	3.65E-03	3.65E-02	2.89E-06	1.22E-03	1.22E-02	2.86E-02	7.59E-04	2.20E-04	**4.89E-02**
Xylenes, total	6.30E-01	4.03E-02	2.01E-01	3.45E-05	1.46E-02	7.28E-02	3.15E-01	8.38E-03	2.43E-04	**2.74E-01**
Total HI			**5.88E+00**			**5.94E-01**			**5.17E-04**	**6.48E+00**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-14

Adult On-Stie Resident, Future Scenario, Noncancer Hazard from Exposure to Bedrock Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			All
		Noncancer		Dose	Adult		Concentration	Adult		Pathways
	EPC	Dose		Absorbed	Dose		in Air	Dose		Total
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	HI
Inorganics										
Selenium	1.95E-02	5.34E-04	1.07E-01	3.90E-09	1.07E-06	2.14E-04	NA	NA	NA	**1.07E-01**
Semivolatile Organic Compounds										
Naphthalene	7.40E-03	2.03E-04	1.01E-02	3.22E-07	8.81E-05	4.41E-03	3.70E-03	4.22E-05	3.63E-08	**1.45E-02**
Volatile Organic Compounds										
Benzene	3.36E-01	9.21E-03	2.30E+00	4.13E-06	1.13E-03	2.83E-01	1.68E-01	1.92E-03	1.65E-05	**2.58E+00**
Chloromethane	4.49E-02	1.23E-03	NA	8.89E-08	2.44E-05	NA	2.25E-02	2.56E-04	6.66E-06	**6.66E-06**
trans-1,3-Dichloropropene	1.70E-03	4.66E-05	1.55E-03	6.06E-09	1.66E-06	5.54E-05	8.50E-04	9.70E-06	5.53E-08	**1.61E-03**
Ethylbenzene	5.71E-02	1.56E-03	1.56E-02	2.24E-06	6.14E-04	6.14E-03	2.86E-02	3.26E-04	9.45E-05	**2.19E-02**
Xylenes, total	6.30E-01	1.73E-02	8.63E-02	2.67E-05	7.33E-03	3.66E-02	3.15E-01	3.59E-03	1.04E-04	**1.23E-01**
Total HI			**2.52E+00**			**3.30E-01**			**2.22E-04**	**2.85E+00**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-15

On-Site Resident, Future Scenario, Cancer Risk from Exposure to Overburden Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					All
		Adult	Child		Dose	Adult	Dose	Child		Pathways
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	ILCR	**Total ILCR**
Inorganics										
Nickel	7.42E-02	6.97E-04	4.07E-04	NA	2.97E-09	2.79E-07	4.94E-09	1.79E-07	NA	**NA**
Total ILCR				**NA**					**NA**	**NA**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

Table D-16

Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			All
		Noncancer		Dose	Noncancer		**Pathways**
	EPC	Dose		Absorbed	Dose		**Total**
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm²-day	mg/kg-day	HQ	**HI**
Inorganics							
Nickel	7.42E-02	4.74E-03	2.37E-01	4.94E-09	2.08E-06	2.61E-03	**2.40E-01**
Total HI			**2.37E-01**			**2.61E-03**	**2.40E-01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm² - milligram per square meter

Table D-17

Adult On-Stie Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Groundwater
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			**All**
		Noncancer		Dose	Adult		**Pathways**
	EPC	Dose		Absorbed	Dose		**Total**
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm²-day	mg/kg-day	HQ	**HI**
Inorganics							
Nickel	7.42E-02	2.03E-03	1.02E-01	2.97E-09	8.13E-07	1.02E-03	**1.03E-01**
Total HI			**1.02E-01**			**1.02E-03**	**1.03E-01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm² - milligram per square meter

Table D-18

On-Site Resident, Future Scenario, Exposure to Sediment Cancer Risk
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					All
		Adult	Child		Dose	Adult	Dose	Child		Pathways
Chemicals of Potential Concern	EPC mg/kg	Dose mg/kg-day	Dose mg/kg-day	Resident ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	Resident ILCR	**Total ILCR**
Inorganics										
Arsenic	9.03E+00	4.24E-07	9.89E-07	2.12E-06	1.90E-09	7.54E-09	5.42E-09	1.23E-08	2.98E-08	**2.15E-06**
Chromium	1.10E+01	5.16E-07	1.20E-06	NA	NA	NA	NA	NA	NA	**NA**
Total ILCR or HI				**2.12E-06**					**2.98E-08**	**2.15E-06**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-19

On-Site Child Resident, Future Scenario, Exposure to Sediment Noncancer Risk
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			All
		Child		Dose	Child		**Pathways**
	EPC	Dose	Child	Absorbed	Dose	Child	**Total**
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	**HI**
Inorganics							
Arsenic	9.03E+00	1.15E-05	3.85E-02	5.42E-09	1.44E-07	4.80E-04	**3.90E-02**
Chromium	1.10E+01	1.41E-05	6.69E-04	NA	NA	NA	**6.69E-04**
Total ILCR or HI			**3.91E-02**			**4.80E-04**	**3.96E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-20

On-Site Adult Resident, Future Scenario, Exposure to Sediment Noncancer Risk
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			All
		Adult		Dose	Adult		Pathways
	EPC	Dose	Adult	Absorbed	Dose	Adult	Total
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	HI
Inorganics							
Arsenic	9.03E+00	1.24E-06	4.12E-03	1.90E-09	2.20E-08	7.33E-05	**4.20E-03**
Chromium	1.10E+01	1.51E-06	7.17E-05	NA	NA	NA	**7.17E-05**
Total ILCR or HI			**4.19E-03**			**7.33E-05**	**4.27E-03**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-21

Adult Hunter, Future Scenario, Exposure to Surface Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact				
	EPC	Cancer Dose	Noncancer Dose	Adult Hunter	Adult Hunter	Dose Absorbed	Cancer Dose	Noncancer Dose	Adult Hunter	Adult Hunter
Chemicals of Potential Concern	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ
Inorganics										
Beryllium	1.14E+01	2.67E-07	6.22E-07	NA	3.11E-04	NA	NA	NA	NA	NA
Thallium	2.95E+00	6.93E-08	1.62E-07	NA	2.49E-03	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1016	1.58E-01	3.71E-09	8.66E-09	2.60E-10	1.24E-04	4.42E-09	3.43E-09	8.00E-09	2.40E-10	1.14E-04
Semivolatile Organic Compounds										
Benzo(a)pyrene	1.70E-01	3.99E-09	9.32E-09	2.91E-08	NA	4.42E-09	3.43E-09	7.99E-09	2.50E-08	NA
Benzo(b)fluoranthene	1.60E-01	3.76E-09	8.77E-09	2.74E-09	NA	4.16E-09	3.22E-09	7.52E-09	2.35E-09	NA
Total HI				**3.21E-08**	**2.92E-03**				**2.76E-08**	**1.14E-04**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter

Table D-21

Adult Hunter, Future Scenario, Exposure to Surface Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

	Concentration in Forage C_p	Concentration in Venison C_v	Consumption of Venison				**All Pathways**	
			Cancer Dose	Noncancer Dose			**Total**	**Total**
Chemicals of Potential Concern	mg/kg	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	**ILCR**	**HI**
Inorganics								
Beryllium	NA	NA	NA	NA	NA	NA	**NA**	**3.11E-04**
Thallium	1.48E-03	3.08E-07	2.35E-11	5.49E-11	NA	8.44E-07	**NA**	**2.49E-03**
Polychlorinated biphenyls (PCB)								
Aroclor 1016	4.42E-03	2.31E-07	1.76E-11	4.11E-11	1.23E-12	5.88E-07	**5.01E-10**	**2.39E-04**
Semivolatile Organic Compounds								
Benzo(a)pyrene	NA	NA	NA	NA	NA	NA	**5.41E-08**	**NA**
Benzo(b)fluoranthene	NA	NA	NA	NA	NA	NA	**5.10E-09**	**NA**
Total HI					**1.23E-12**	**1.43E-06**	**5.97E-08**	**3.04E-03**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter

Table D-22

Hunter's Child, Future Scenario, Exposure to Surface Soil
Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	EPC mg/kg	Concentration in Forage C_p mg/kg	Concentration in Venison C_v mg/kg	Consumption of Venison: Cancer Dose mg/kg-day	Consumption of Venison: Noncancer Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics									
Beryllium	1.14E+01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Thallium	2.95E+00	1.48E-03	3.08E-07	8.44E-12	9.85E-11	NA	1.51E-06	**NA**	**1.51E-06**
Polychlorinated biphenyls (PCB)									
Aroclor 1016	1.58E-01	4.42E-03	2.31E-07	6.33E-12	7.38E-11	4.43E-13	1.05E-06	**4.43E-13**	**1.05E-06**
Semivolatile Organic Compounds									
Benzo(a)pyrene	1.70E-01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Benzo(b)fluoranthene	1.60E-01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Total HI						**4.43E-13**	**2.57E-06**	**4.43E-13**	**2.57E-06**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram

RESPONSE TO COMMENTS

Responses to Comments
Baseline Human Health Risk Assessment (BHHRA) Assessment
for Powerhouse 2 Ash Pits
Plum Brook Ordnance Works, Sandusky, Ohio,
Dated June 21, 2010

Comments by Janusz Byczkowski, Risk Assessor, Ohio Environmental Protection Agency, received August 9, 2010. Note that additional Comment Nos. 5 through 7 were made by the reviewer with respect to the screening level human health risk assessment (SLERA). Responses to these additional comments are attached to the final SLERA report.

Comment 1: **<u>Section 2.4.2, Page 2-3, Line 26, Section 8.0, Page 8-2, Line 10, Table 2-4 to 2-9</u>. This document states: *"…Groundwater RBSCs used in the BHHRA are derived from the Oak Ridge National Laboratory-EPA regional screening levels (RSL) table…"* The RSL are no longer provided by the *Oak Ridge National Laboratory*. The RSLs are provided at the U.S. EPA's "Mid Atlantic Risk Assessment" Website. The initial Website used to be developed with DOE's ORNL involvement, under an Interagency Agreement, but currently it serves as an official update of the harmonized EPA Region 3 RBC, Region 6 HHMSSL and the Region 9 PRG Tables. A few editorial corrections are recommended before releasing this document to the public. Please correctly attribute the RSL table to the U.S EPA.**

Response 1: The references will be revised to "EPA" instead of "ORNL-EPA," although the Oak Ridge National Laboratory (ORNL) is still integrally involved in the revision of RSL values. As the reviewer states, the RSL website was developed as an update of various Region 3, Region 6 and Region 9 values. The RSLs are a regionally lead effort, which receives only guidance from Superfund/Headquarters. The website itself is no longer hosted by ORNL, but is now hosted by EPA Region 3. However, updated RSLs are still calculated by ORNL, under the direction of the EPA Regions. ORNL still also provides assistance such as how the interpretation of new guidance may affect the RSLs. Note that the calculator link (http://epa-prgs.ornl.gov/cgi-bin/chemicals/csl_search) on the RSL website hosted by Region 3 links back to ORNL

Comment 2: **<u>Section 1.4, Page 1-4, Line 27, and Section 3.1.8, Page 3-16, Line 23</u>. This document states: *"…Parts of the facility may be used for training by the National Guard…"* and then: *"…Because they would likely not represent an upper bound for nonresidential exposure, these receptors are not evaluated…"* It is not clear, which exposure scenario could be relevant to the National Guardsmen. In the Section 7.0 there is no discussion on how the cumulative health hazards/cancer risks to guardsmen would compare with those, whose ILCRs and HIs were actually calculated. Please discuss exposure scenario pertinent to the National Guard's trainee and provide some evaluation of risk/hazards relative to the other receptors, whose ILCRs and HIs were calculated.**

Response 2: The exposure pathways encountered by the National Guard Reserve soldier would likely not differ from those of a groundskeeper and the two receptors would be similarly attired (shoes/boots, pants, shirt). Text will be added to Section 3.1.3.8 that qualitatively compares a 25-year daily groundskeeper exposure scenario (250 days per year, 8 hours per day) to a National Guard Trainee who does all of his training at the LBA assuming and subsequently remains in the Guard as a 20-year Reserve soldier, again spending his entire National Guard Reserve career at the LBA. Please note that the National Guard scenario has been mentioned and described as a potential receptor that would not represent a reasonable maximum exposure non-residential scenario in each of the BHHRA work plans and reports submitted over the past decade.

Comment 3: **In the Equation 3-19, the variable "l_v" is not defined. Please add a definition of "l_v" below the equation.**

Response 3: The definition provided for the first "IR_v" actually applies to "I_v". The first term "IR_v" will be revised as "I_v".

Comment 4: **<u>Tables 2-3 to 2-9 and A-2; also SLERA Table 2-9</u>. Some chemicals, whose reporting limits (RL) exceed screening levels (RBSC), have not been selected as chemicals of potential concern (COPC), and thus, they are not included in baseline risk assessment – for example: *"Indeno(1,2,3-cd)pyrene"* in Table 2-4; or *"Cadmium"* in Table 2-7; etc. All chemicals whose RL>RBSC should not be just eliminated from the data set used for baseline risk assessment. Instead, it should be assumed that each of them may be present at a concentration equal to ½ RL, and thus, it should be processed as COPC. Similarly in SLERA, the chemicals whose RL>ESV should be included as COPECs, assuming their concentration = ½ of RL. Please check the data tables for cases of RL>RBSC and/or ESV, and evaluate those that exceed screening levels as COPC in the BHHRA and/or as COPEC in SLERA, assuming that their concentration = ½ of RL.**

Response 4: The data evaluation protocols used in the BHHRA and SLERA are consistent with that which has been performed for PBOW human health and ecological risk assessments for the past decade. These protocols are described in the approved site-specific risk assessment work plans (Shaw, 2010a). Further, these approaches are clearly consistent with EPA (1989) guidance which emphasizes the use of positive data and recognizes that the state of the practice is such that achieving RLs that are lower than screening values is not possible for certain chemicals. This guidance even recommends that individual samples with elevated RLs be excluded from the quantitative risk assessment.

The focus of the BHHRA and SLERA is quantitative risk assessment. Please note that the MDCs are used for screening, and that this is the most conservative value that is reasonable to consider in quantitative evaluation as

an exposure point concentration (EPC). The ProUCL statistical program, used to estimate EPCs, weighs nondetects differently and will typically not recommend a value that exceeds the MDC. The method that has been used in the BHHRA and SLERA, as described the work plan, ensures that the EPC will not exceed the MDC. Therefore, the use of the MDC as the screening value reflects the "worst case" of the quantitative portion of the risk assessment.

The reviewer cites two examples in the BHHRA, indeno(1,2,3-c,d)pyrene in surface soil and cadmium in overburden groundwater, that the reviewer suggests should be evaluated based on the surrogate value for a nondetect rather than the MDC. These two chemicals are discussed in greater detail in the following paragraphs to exemplify that the use of the MDC to is the appropriate technical approach during screening.

Indeno(1,2,3-c,d)pyrene was reported in only 1 of 20 surface soil samples. This sample, PH2SO01, was collected in 1996 as part of the limited site investigation. Of the 13 PAHs detected in AP2 surface soil, the MDCs for 12 were detected in this sample. Clearly, it appears that this sample represents higher concentrations of PAHs than any of the other samples. The fact that the lone detection of indeno(1,2,3-c,d)pyrene was detected in this higher-PAH sample provides evidence that the value is appropriate to represent the true maximum concentration of the data set; it is extremely unlikely that a concentration higher than the detection but below the RL is present at one of the nondetect sample locations, given the evident higher concentrations in sample PH2SO01 for the full suite of PAHs, and not just indeno(1,2,3-c,d)pyrene. Thus, indeno(1,2,3-c,d)pyrene was appropriately screened out during the COPC screening step.

The example of cadmium cited by the reviewer was collected from AP2-MW02 in May 2009 during the wet season, which was the most turbid of the wells sampled in May 2009 (Shaw, 2010b). Duplicate samples were collected and both filtered and unfiltered inorganics were run. In both cases, cadmium was reported as detected in the unfiltered samples (1.1J and 1.0J µg/L), but not in the filtered samples. Even though the RLs exceed the RBSCs, the analytical method used is shown to consistently detect cadmium in the unfiltered samples at concentrations less than the RBSC of 1.8 µg/L. Because the analysis can clearly and reproducibly detect cadmium at less than the one-half the RL, there is no technical justification for assuming a surrogate value instead of the MDC. Incidentally, the analytical results also suggest that the cadmium reported in the unfiltered samples were associated with suspended particulates. May 2009 results could not be compared to November 2009 results for LBA-MW02 because it was found to be highly turbid in November 2009 and did not produce enough water to sample for inorganics.

References Used in Responses

Shaw Environmental, Inc. (Shaw), 2010a, ***Baseline Human Health Risk Assessment Work Plan, Garage Maintenance Area (Locomotive Building Area), Plum Brook Ordnance Works, Sandusky, Ohio***, May.

Shaw Environmental, Inc. (Shaw), 2010b, ***Site Characterization Report, Garage Maintenance Area (Locomotive Building Area), Final, Plum Brook Ordnance Works, Sandusky, Ohio***, September.

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