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October 30, 2013

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Subject: *Submittal of the Final Baseline Human Health Risk Assessment (BHHRA) for Ash Pit No. 3 and the BHHRA and Screening-Level Ecological Risk Assessment Addenda for Coal Yard No. 3 Former Plum Brook Ordnance Works, Sandusky, Ohio Contract No. W912QR-08-D-0013: Shaw Project Number 132457*

Dear Ms. Coleman:

In accordance with the requirements of Delivery Order No. DX02 of Contract No. W912QR-08-D-0013 awarded to Shaw Environmental & Infrastructure, Inc., a CB&I company, we are pleased to submit the Final Baseline Human Health Risk Assessment (BHHRA) for Ash Pit No. 3 (AP3), and the final Addenda to the AP3 BHHRA and Screening-Level Ecological Risk Assessment for Coal Yard No. 3 at the Former Plum Brook Ordnance Works (PBOW) located in Sandusky, Ohio. This report was prepared consistent with other PBOW risk assessment reports, U.S. Environmental Protection Agency guidance, and the AP3 risk assessment work plan.

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

Should you have any questions or require additional information regarding this submittal, please do not hesitate to contact me at (865) 694-7496.

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Project Description: Ash Pit No. 3 BHHRA
Former Plum Brook Ordnance Works, Sandusky, Ohio

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Final

**Ash Pit No. 3
Baseline Human Health Risk Assessment
FUDS Project No. G05OH001821**

**Former Plum Brook Ordnance Works
Sandusky, Ohio**

Prepared for:

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List of Acronyms

ABS	dermal absorption factor
AF	soil-to-skin adherence factor
AP3	Ash Pit 3
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/hour	centimeters per hour
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
FUDS	Formerly Used Defense Sites
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
ICI	International Consultants Incorporated
IEUBK	Integrated Exposure Uptake Biokinetic

List of Acronyms (Continued)

ILCR	incremental lifetime cancer risk
IRIS	Integrated Risk Information System
IT	IT Corporation
kg	kilogram
kg/day	kilograms per day
K_p	permeability coefficient
LMS	linearized multistage
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
$\mu g/m^3$	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
$\mu g/L$	micrograms per liter
mg/L	milligrams per liter
NASA	National Aeronautics and Space Administration
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
OEPA	Ohio Environmental Protection Agency
PAH	polycyclic aromatic hydrocarbon
PBOW	Plum Brook Ordnance Works
PDT	Project Delivery Team
PEF	particulate emission factor
PRG	preliminary remediation goal
RBSC	risk-based screening concentration
RfC	reference concentration

List of Acronyms (Continued)

RfD	reference dose
RI	remedial investigation
RME	reasonable maximum exposure
RSL	regional screening level
SF	cancer slope factor
Shaw	Shaw Environmental & Infrastructure, 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
WWTP3	Waste Water Treatment Plant No. 3

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 Ash Pit 3 (AP3) 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 & Infrastructure, Inc (a CBI company).

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-dinitrotoluene, 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, 3,230 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. Each power station consisted of a main powerhouse, a coal storage area, and an aboveground fuel storage tank. The fuel storage tank was surrounded by a berm to contain any potential spills or leaks. Each powerhouse building consisted of a boiler house, compressor room, electrical room, filter room, and locker room.

Each building 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 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.” AP3 was associated with Powerhouse 3.

AP3 is located approximately 700 feet southwest of the intersection of Maintenance and Ransom Roads and is west of the former NASA K-Site Test Facility research building (former Powerhouse 3). The pit is partially surrounded by thick vegetation, including a mixture of mature and smaller trees with a dense understory. Boundaries of the pit appear to be the same as the original boundaries, with the exception of an indentation to the west on the eastern boundary. During its operation, the K-Site supplied noncontact cooling water to AP3. The original impoundment was intact as of 1999, holding water supplied by NASA’s K-Site Test Facility. A pipe at the northeastern corner of the pit supplied the cooling water. Operations at the K-Site were officially abandoned in 2007. Water discharged from this former ponded area via an east-west-trending drainage ditch that eventually discharges into Pipe Creek. Since the K-Site ceased operations, the apparent major source of water to AP3 has been eliminated. A pair of eagles was found to be nesting near AP3.

Approach. The BHHRA evaluated exposure to chemicals in surface soil, subsurface soil, overburden groundwater, surface water, and sediment for cancer risks and noncancer hazards. Only validated analytical data were used in the BHHRA. Validated analytical data from samples other than groundwater include those collected during 2008 and 2009, as reported in the 2010 site characterization report, and those collected in 1999 and reported in the limited site investigation report. Groundwater data includes those analytical results from samples collected in 2011 and 2012, as reported in the site characterization report addendum. It is noted that the Ohio Department of Natural Resources guidance prohibits operating large machinery within a radius of 660 feet of the eagle’s nests. Therefore, the presence of this nest resulted in modifications to the groundwater sampling protocol.

The standard steps of risk assessment, including data analysis, exposure assessment, toxicity assessment, risk characterization, and uncertainty analysis, were executed in the AP3 BHHRA. 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. This screening

included a risk-based screening and, for inorganics in soil, a background screening. This background screening protocol, which is based on PBOW Project Delivery Team agreement, differs somewhat from the current Ohio Environmental Protection Agency guidance. Use of this method for the screening of background ensures consistency between all of the PBOW project sites.

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 each AP3 environmental medium except for surface water, indicating that surface water does not contribute significantly to risks. The receptors listed below were evaluated for exposure to the COPCs and their associated hazards and risks, with media evaluated for each receptor shown in parentheses:

- Current groundskeeper (surface soil)
- Future groundskeeper (combined surface and subsurface soil [referred to as “total soil”], overburden groundwater)
- Indoor worker (surface soil, subsurface soil [air pathway only], overburden groundwater)
- Current/future construction worker (total soil, surface water, sediment)
- Hypothetical future site resident (total soil, surface water, sediment, overburden groundwater)
- Future hunter (surface soil)
- Future hunter’s child (surface soil [venison pathway only]).

Although no COPCs were found in surface water, this medium is still regarded as evaluated.

Note that there is currently no groundskeeper at AP3, but the current groundskeeper is included because current land use does not prohibit the presence of a groundskeeper or other on-site workers. Even though hunting is not currently permitted at AP3, hunting is permitted in other areas within PBOW; therefore, future use of AP3 for hunting is evaluated in this BHHRA. No construction is planned for AP3, 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. Because AP3 is a low-lying former impoundment and seasonally inundated, the site would need to be covered with a few feet of soil before construction could commence;

therefore, exposure to a future receptor would likely involve minimal exposure to current AP3 soil.

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) goal of 1 and 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.” Cancer risk results were also compared to the PBOW Project Delivery Team target incremental lifetime cancer risk (ILCR) goal of $1E-5$ (i.e., 1 additional incidence of cancer per 100,000 individuals exposed). A target cancer risk goal of $1E-5$ is also used by the Ohio Environmental Protection Agency. This value was selected by the PBOW Project Delivery Team as a basis to consider remedial action, as it is the logarithmic midpoint of the NCP risk management range. Use of this $1E-5$ goal represents a departure from the Army’s practice of consistently using a cancer risk exceeding a value of $1E-4$ (the upper end of the NCP risk management range) to trigger remedial action considerations.

The overall HI and ILCR values are summarized in the following bullets; exceedances of PBOW cancer risk goal ($ILCR > 1E-5$) are shown as bold, and exceedances of the noncancer hazard goal ($HI > 1$) or the NCP risk management range ($1E-6$ to $1E-4$) are shown as bold italics. Please note that initial evaluations resulted in ILCR values that exceeded the PBOW cancer risk goal. However, these cancer risks that resulted in ILCR values were mostly associated with arsenic, which is evidently unrelated to former site activities. Therefore, ILCR values were recalculated excluding the contributions of arsenic in soil and/or groundwater for the receptors with initial ILCR values greater than $1E-5$.

- Current groundskeeper: ILCR = ***$2E-5$*** ($1E-6$ excluding background-related arsenic in surface soil); HI = 0.2
- Future groundskeeper: **ILCR = $6E-5$** ($1E-6$ excluding background-related arsenic in total soil); HI = 0.1
- Future groundskeeper excluding groundwater: ILCR = ***$2E-5$*** ($9E-7$ excluding background-related arsenic in total soil); HI = 0.1
- Future indoor worker: ILCR = ***$5E-5$*** ; HI = 0.7
- Future indoor worker excluding groundwater: ILCR = $8E-6$; HI = 0.1
- Construction worker: ILCR = $1E-6$; HI = 0.4

- Hypothetical future resident: *ILCR = 2E-4; child HI = 5; adult HI = 2*
- Hypothetical future resident excluding groundwater: *ILCR = 6E-5* (5E-6 excluding background-related arsenic in soil); *child HI = 1; adult HI = 0.1*
- Future hunter: *ILCR = 1E-6; HI = 0.009*
- Future hunter's child: *HI = 0.000001*; none of the carcinogenic COPCs are bioaccumulative; cancer risks are assumed to be de minimis.

The ILCR value for each of the AP3 exposure scenarios is within (or less than) the NCP risk management range and the PBOW cancer risk goal of 1E-5, if non-site-related arsenic in soil and groundwater is excluded from the evaluations.

The HI values of each of the AP3 receptors, except the future adult and child resident, meet the noncancer target HI of 1. Arsenic and manganese in overburden groundwater are responsible for the elevated HI values for the adult and child resident. The sources of these inorganics in groundwater are evidently unrelated to former site operations, and they appear to be naturally occurring.

No construction is currently planned at AP3, and no groundskeeping of any sort appears to be occurring at AP3. Because AP3 is in a low-lying former impoundment and frequently inundated, additional soil would need to be added before construction could occur at the site. Therefore, potential exposure to current AP3 surface or subsurface soil is likely minimal and the associated risks/hazards for any receptor in this BHHRA that assumes future development (i.e., each receptor except the hunter and hunter's child) are likely exaggerated.

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 Ash Pit No. 3 (AP3), located at the former Plum Brook Ordnance Works (PBOW), Sandusky, Erie County, Ohio. This work is being conducted by Shaw Environmental & Infrastructure (Shaw) (a CB&I company) for the U.S. Army Corps of Engineers (USACE) under the Defense Environmental Restoration Program-Formerly Used Defense Sites (FUDS), 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 Area A (TNTA) and TNT Area C (TNTC) soil (IT Corporation [IT], 2001a), the BHHRA for groundwater at PBOW (Shaw, 2006) and, most specifically, the AP3 BHHRA work plan (Shaw, 2009).

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 AP3.

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-dinitrotoluene (DNT), and pentolite (International Consultants Incorporated [ICI], 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 TNTA, TNT Area B (TNTB), and 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, 3,230 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, also known as the Magazine Area, which consists of 2,800 acres. The Department of the Army reacquired the 3,230 acres in 1954. 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. The Army continued cleanup efforts until 1963.

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 covered with fill material. 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 excavated nitroaromatic-filled flumes (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 (ICI, 1995).

1.3 Ash Pit No. 3 Description and History

As noted in Section 1.2, 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. Coal Yard No. 3, which is located immediately to the south of Powerhouse No. 3 and immediately east of Ash Pit No. 3, is approximately 1 acre in size. Coal Yard No. 3 is evaluated separately (please see the Addendum for Coal Yard No. 3, which is appended to this report following the AP3 BHHRA). Each powerhouse building 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 powerhouses generated steam that 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” (ICI, 1995). AP3 was associated with Powerhouse 3. The location of AP3 is shown on Figure 1-2.

AP3 is located approximately 700 feet southwest of the intersection of Maintenance and Ransom Roads and is west of the former NASA K-Site Test Facility research building (former Powerhouse 3) (Figure 1-2). The pit is partially surrounded by dense vegetation, including both mature and smaller trees. Boundaries of the pit appear to be the same as the original boundaries, with the exception of an indentation to the west on the eastern boundary (USACE, 2000). During NASA’s operation of K-Site they released noncontact cooling water to AP3. In 1999 the original impoundment was still intact and held used K-Site cooling water (USACE, 2000). A pipe at the northeastern corner of the pit discharged the K-Site cooling water into AP3. It is possible that this is the original pipe that once supplied the ash slurry to the pit during PBOW operations. Operations at the K-Site were officially abandoned in 2007, and the former Powerhouse 3 building is scheduled by NASA for demolition in 2013. Abandoned railroad tracks running in a north-south direction are located immediately east of AP3. Water was discharged from this former ponded area via an east-west-trending drainage ditch that eventually discharges into Pipe Creek (USACE, 2000). Since the K-Site ceased operations, the apparent major source of water to

AP3 has been eliminated. The presence or lack of standing water associated with various site visits and recent aerial photographs are described in further detail in Section 3.1.1.5.

A pair of bald eagles (*Haliaeetus leucocephalus*) was found to be nesting near AP3. Bald eagles are a threatened species in the state of Ohio. It is noted that the Ohio Department of Natural Resources guidance prohibits operating large machinery within a radius of 660 feet of an active eagle nest. Therefore, the presence of this nest resulted in modifications to the groundwater sampling protocol (Section 2.1).

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. Over 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 discontinuous and variably saturated groundwater system exists within the unconsolidated material atop the bedrock under much of the site.

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 uses of AP3 are considered to be industrial or residential for the purpose of developing receptor and exposure scenarios. There are no current NASA activities at AP3, but because PBOW is under NASA control, the potential for NASA activities at AP3 exists. Even though hunting is not currently permitted at AP3, hunting is permitted in other areas within

PBOW; therefore, future use of AP3 for hunting is evaluated in this BHHRA. It is conservatively assumed for purposes of this BHHRA that groundwater may be developed as a source of potable water in the future. Section 3.1.3 presents a discussion of receptors and exposure scenarios.

1.5 Protocol for the Baseline Human Health Risk Assessment

The BHHRA was performed consistent with the AP3 BHHRA work plan (Shaw, 2009). The AP3 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), 2010a, *ProUCL Version 4.1 Technical Guide*, Draft, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, EPA/600/R-07/041, May.
- U.S. Environmental Protection Agency (EPA), 2010b, *ProUCL Version 4.1 User Guide*, Draft, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, EPA/600/R-07/038, May.
- U.S. Environmental Protection Agency (EPA), 2011, *ProUCL Version 4.1*, 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>.

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. It is noted that the background screening protocol differs from the current OEPA (2009c) guidance as explained in Section 2.4.3.
- **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 AP3. Risks associated with exposure to all appropriate AP3 media 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 directly relevant to the risk assessment conclusions for AP3 that are 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 AP3 environmental medium sampled, a discussion of data quality, a description of the methodology used for identification of the COPCs, and a summary of the COPCs for each 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, overburden groundwater, surface water, and sediment.

Note that groundwater sampling activities were modified because of a pair of nesting bald eagles located nearby; Ohio Department of Natural Resources guidance prohibits operating large machinery within a radius of 660 feet of an active eagle nest. Therefore, monitoring wells were installed using direct-push technique during August 2011, after eagle nesting activities were completed. The wells were sampled in December 2011 using low-flow sampling. During the May 2012 sampling event, precautions were taken to minimize disturbance to the nesting eagles, as this was within the nesting period. As part of these precautions, the wells were sampled using Snap SamplersTM as described in the site characterization report addendum (Shaw, 2013).

The sample summary table identifies each sample used in the BHHRA and the associated analytical suite. Samples included those collected as part of the limited site investigation for AP3 (USACE, 2000) and the remedial investigation (RI) samples (Shaw, 2010; 2013). All AP3 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 site characterization report (Shaw, 2010), the site characterization report addendum (Shaw, 2013), and the limited site investigation report (USACE, 2000). 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 likely occur after surface and subsurface soil had been excavated and/or mixed. 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 was done for AP3 soil.

Surface soil is defined as samples collected from within the interval of 0 to 1 foot below ground surface (bgs), and subsurface soil is defined as samples collected from depths greater than 1 foot bgs per the RI work plan (Shaw, 2008a). The limited site investigation (USACE, 2000) historical surface soil samples were collected from a depth of either 0 to 0.5 or 1.0 foot bgs. As possible, the RI subsurface soil samples are generally collected at depths of 3 to 5 and 8 to 10 feet bgs. Where refusal or the water table was encountered before a depth of 10 feet bgs, samples are collected from the deepest 2-foot interval above the water table. Because the water table at AP3 is shallow, all subsurface soil samples were collected from within an aggregate depth range of 0.8 to 5 feet (Table 2-1).

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 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 - Quality control 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. The analytical data evaluated in the BHHRA are included in Appendix A.

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 if 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 ($\mu\text{g/L}$) 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 EPA (2012a) regional screening level (RSL) table "tap water" values, and RBSCs for soil are derived from "residential soil" RSL values. This is a change in the source of the RBSCs for PBOW BHHRA work plans begun prior to March 2009 based on discussion between USACE and OEPA (2009d), and this change is consistent with recent 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; EPA, 2012a). 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. 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 AP3 sediment and surface water are also evaluated in the BHHRA. The AP3 surface water includes standing water in the far northeast corner of the site and the standing water in the drainage ditch located west of AP3. 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 or 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 AP3 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 and also because the drainage ditch does not contain nearly enough water for drinking water use. 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 sediment and surface water RBSCs on RSLs rather than PRGs, just as soil and groundwater RBSCs are currently based on RSLs. 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 Project Delivery Team (PDT) agreement for screening surface water and sediment (OEPA, 1999), which considered site-specific conditions, is used for PBOW FUDS sites in place of the current OEPA (2009b) guidance. The current OEPA guidance 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. Use of this agreed PBOW PDT protocol ensures consistency in the evaluation of PBOW FUDS sites. 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 (2012b) 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 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 the Office of Drinking Water action level of 15 µg/L can be used to conservatively screen for a potential average concentration of 15 µg/L, this concentration 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 be protective of 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 AP3 total soil, the MDC for lead (20.4 mg/kg) is less than both the RBSC (400 mg/kg) and the criterion for average concentration (250 mg/kg), and lead was not detected in AP3 overburden groundwater samples. Therefore, the IEUBK model was not run for AP3 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, 2012b). Secondary drinking water regulations are nonpromulgated 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, 2012b).

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 is high that a given chemical is present, 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 are 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 in the vicinity of PBOW (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 (Section 2.4.3.1) and is performed after the risk characterization (Chapter 5.0). The results of the statistical data testing are discussed in the uncertainty analysis (Chapter 6.0). No suitable background data set exists for overburden groundwater, 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) (Table 2-2), and the background data set and derivation of BSCs for PBOW bedrock groundwater are described in the 2004 groundwater report (Shaw, 2005) (Table 2-3). It is noted that the method agreed upon for the development of BSCs, as recorded in the May 10, 2000 PBOW PDT (2000) meeting minutes, differs from that shown in current OEPA (2009c) guidance. Use of this method for the development of BSCs and as part of the COPC screening process ensures consistency between all of the PBOW FUDS project sites.

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 (PBOW PDT, 2000). 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 presented in the 2004 groundwater report (Shaw, 2005), and the background data set for soil is presented 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).

2.4.3.3 Treatment of Organic Compounds

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), total soil (Table 2-6), 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 reporting limits
- Arithmetic mean of site concentrations

- Appropriate BSC
- Appropriate RBSC
- Selection/exclusion of chemical as a COPC
- 95th percent upper confidence limit on the arithmetic mean (UCL) (for COPCs only)
- 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.

The COPCs for each of the AP3 media are identified as follows:

- **Surface Soil** – Arsenic, thallium, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene
- **Subsurface Soil** – Thallium
- **Total Soil** – Arsenic, thallium, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene
- **Overburden Groundwater** – Arsenic, chromium, cobalt, iron, manganese, sulfate
- **Sediment** – Arsenic, chromium, benzo(a)pyrene, benzo(b)fluoranthene
- **Surface Water** – None.

As discussed in Section 2.2, the total soil COPCs include all surface soil and subsurface soil COPCs.

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 AP3 environmental media as portrayed by the CSEM in Section 3.1. Note that these environmental media include soil, groundwater, surface water, and sediment.

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, 1997). 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 (e.g., Shaw [2008b]), 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; however, the groundwater underlying AP3 is not 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 hypothetical future on-site residential (or on-site worker) use as tap water is initially regarded as plausible for purposes of this BHHRA. Groundwater quality and potential use is discussed further in the risk characterization (Chapter 5.0) and uncertainty analysis (Chapter 6.0). It is important to note that the site-specific risk assessment, including the evaluation of future land uses and groundwater use, was performed to satisfy administrative requirements, including those described by FUDS regulations (USACE, 2004).

3.1.1 Physical Setting

AP3 is just west of the former NASA K-Site Test Facility research building (former Powerhouse 3) (Figure 1-2). Operations at the K-Site were officially abandoned in 2007, and the former Powerhouse 3 building is scheduled for demolition in 2013. AP3 is partially surrounded by thick, shrubby vegetation, as well as successional forest consisting of both mature and smaller trees.

The following sections describe the physical setting of AP3, including the climate and meteorology of the Sandusky region (Section 3.1.1.1) and the geology (Section 3.1.1.2), soil (3.1.1.3), hydrology (Section 3.1.1.4), and surface water characteristics (Section 3.1.1.5) of the area and of AP3 in particular. A general description of AP3 is provided in Section 1.3.

3.1.1.1 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).

3.1.1.2 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).

No bedrock monitoring wells were installed at AP3, because the analytical results of overburden groundwater indicated no site-related contamination in the upper unit . However, at Waste Water Treatment Plant No. 3 (WWTP3), which is located approximately 300 feet north of AP3, petroleum was observed at depth in the rock cores of all three bedrock monitoring wells (Shaw, 2010). One of these WWTP3 wells, WWTP3-BEDGW-002, also exhibited high levels of hydrogen sulfide (>200 parts per million), which supports the assumption that bedrock groundwater in the general vicinity of AP3 is of naturally poor quality.

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). None of the AP3 wells were advanced through the shale; thus, definitive information concerning the thickness of the shale at AP3 is not available. However, the shale layer at WWTP3, located just north of AP3, was found to be 1.3 feet thick. This indicates that the shale layer at AP3 is likely to be similarly thin.

3.1.1.3 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). The overburden in the vicinity of AP3 is approximately 28 feet, based on boring logs (Shaw, 2013).

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.

3.1.1.4 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. Data collected during the more recent

investigations (Shaw, 2005; IT, 1997, 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.

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. The general flow direction in the overburden water-bearing zone is to the north in the immediate vicinity of AP3. A hydrogeological study conducted by the U.S. Geological Survey (1992) 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.

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 localized groundwater flow direction toward the southeast in the vicinity of AP3.

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). AP3 is likely in Zone 2.

3.1.1.5 Surface Water

AP3 had formerly been filled with water during former PBOW Powerhouse 3 and NASA K-Site operations. Water is still present at times in AP3. A drainage ditch extends from AP3 to the west. A few inches of standing water were present in this ditch during field reconnaissance and the RI sampling event, but no flow was evident.

3.1.2 Contaminant Sources, Release Mechanisms, and Migration Pathways

Contaminant sources, release mechanisms, and migration pathways are summarized on Figure 3-1. Briefly, AP3 received a coal ash slurry via a pipeline from Powerhouse 3, as described in Section 1.3. AP3 also received ash from the incinerators at WWTP 3. Depending on specific location, this coal ash layer is present at AP3 from the surface to a depth of up to 2.3 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. Surface water from the ditch located west of AP3 may have been impacted via surface drainage by contaminants originating from AP3. Also, standing water in the northeast corner of AP3 may have been impacted by contaminants leaching from AP3 soils and sediment.

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 AP3 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 are listed below:

- Current groundskeeper
- Future groundskeeper
- Indoor worker (future)
- Construction worker (current/future)
- Hypothetical on-site resident (future)
- Hunter (future)
- Hunter's child (future).

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. The construction worker may be viable under current or future land use. However, the evaluation for this receptor is exactly the same under current and future land use.

Most BHHRA 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 upper 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 (ET) 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 (Section 3.1.3.5).

Receptors and the associated exposure pathways are presented in Sections 3.1.3.1 through 3.1.3.8. 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 (typically assumed to be 1 to 10 feet bgs) will be spread as surface soil (0 to 1 foot bgs). Groundwater use is also evaluated for the groundskeeper in the future site-use scenario, as discussed in Section 3.1, in which groundwater could theoretically be developed as a source of drinking water. It is assumed that any contact with surface water or sediment associated with AP3 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.

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 previously, 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 (m³) per

8-hour workday or 2.5 cubic meters per hour (m³/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 (cm²) and a soil AF of 0.2 milligrams per square centimeter (mg/cm²) 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 cm² 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 (Section 3.3).

3.1.3.3 Future Indoor Worker

This hypothetical future receptor scenario was used 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 with 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 m³/8-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 (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 cm² 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 (Section 3.3.4).

3.1.3.4 Current/Future 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 AP3, but this site 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. Hypothetical exposure to groundwater by the construction worker is also possible under a future scenario; however, if on-site groundwater were developed as a tap water source, other potential future groundwater receptors such as the future 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 associated with AP3 during projects such as installation of underground utilities or rerouting of surface flow. Sediment and especially surface water are relatively scarce at AP3, and exposure associated with these media for the AP3 construction worker would be correspondingly minimal to negligible. However, for the sake of consistency and in accordance with the work plan (Shaw, 2009), the construction worker exposure to surface water and sediment is being evaluated with the same protocol used for other PBOW sites. It is understood that AP3-specific issues associated with these media may lead to the risk characterization results being revisited, if appropriate, in the uncertainty analysis. 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 m³/8-hour workday (2.5 m³/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/cm² and an exposed body surface area of 3,300 cm² 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 cm², 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.

As mentioned previously, the exposure assumptions used in the evaluation of surface water and sediment exposure and included in the work plan (Shaw, 2009) are selected to be consistent with other PBOW sites. Therefore, these assumptions (e.g., body surface area available for exposure) are particularly conservative for AP3, where the presence of sediment and especially surface water, and the corresponding exposure, are minimal. As stated previously, this exposure assessment protocol is assumed for the AP3 surface water and sediment evaluation with the understanding that the risk characterization results based on the exposure assumptions can be revisited in the uncertainty analysis.

The construction worker scenario 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 Hypothetical Future On-Site Resident

The hypothetical future on-site residential scenario is used to evaluate long-term exposure to site soil, surface water, sediment, and groundwater under the future land-use scenario. Residential land use is plausible because property abutting the PBOW facility is residential. 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 hypothetical future 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; 2012c).

The hypothetical future 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 hypothetical future resident is also assumed to be exposed to VOCs 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 can be addressed in the uncertainty analysis in cases where the subsurface soil-to-indoor air pathway significantly affects risk and hazard estimates. This pathway did not contribute significantly to risk at AP3, as no VOCs were identified as COPCs in subsurface soil.

It is assumed that, under hypothetical future residential land use, the overburden/shale water unit will be developed as a source of potable water (Section 3.1). The hypothetical 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 hypothetical future resident could be exposed to contaminants in AP3 surface water and sediment. 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 hypothetical future 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 $0.83 \text{ m}^3/\text{hr}$ (EPA, 1991a). A body surface area of $5,700 \text{ cm}^2$, representing the hands, forearms, head, and lower legs, is assumed to be available for dermal exposure to soil (EPA, 2004a). A soil AF of $0.07 \text{ mg}/\text{cm}^2$ 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 hypothetical future 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 $10 \text{ m}^3/\text{day}$ or $0.417 \text{ m}^3/\text{hr}$ (EPA, 2004d). An average body surface area of $2,800 \text{ cm}^2$ 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 $0.2 \text{ mg}/\text{cm}^2$ 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 hypothetical future resident would visit the unnamed tributary to Plum Brook in the vicinity of AP3 surface water 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 $5,700 \text{ cm}^2$, and the AF of $0.07 \text{ mg}/\text{cm}^2$ are also applied to sediment exposure in the adult. Similarly, the resident child soil ingestion rate of 200 mg/day, skin surface area of $2,800 \text{ cm}^2$, and soil AF of $0.2 \text{ mg}/\text{cm}^2$ are applied to sediment exposure for this receptor. Water within AP3 is typically less than 1 foot deep, and water within the drainage ditch west of AP3 is generally only a few inches deep and only intermittently present. The shallowness of the surface water would limit the surface area of the body that would typically be exposed. It is assumed that an adult body surface area of $7,000 \text{ cm}^2$ 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 $2,800 \text{ cm}^2$, 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 (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.

A hypothetical future adolescent resident may be the most likely individual to have regular exposure to sediment and surface water associated with AP3. It is not expected that adults would regularly visit the AP3 surface water areas, as these areas do 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 these areas 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 BHHRA's performed for PBOW sites in the past.

With respect to groundwater exposure, it is assumed that a hypothetical future adult resident ingests 2 L/day of tap water (EPA, 1991a) and that the young child drinks 1 L/day (EPA, 2012c). 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 cm² and the total surface area for the young child is assumed to be 6,600 cm² (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), and the adult is assumed to shower for 12 minutes per day (0.2 hour/day) (EPA, 1997a). Inhalation rates of 0.833 m³/hr for the adult (EPA, 1991a) and 0.416 m³/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 Future Hunter

This scenario is created to evaluate the potential for contaminants in soil to affect food chain pathways. AP3 provides habitat for deer and other wildlife. Even though hunting is not currently permitted at AP3, deer hunting is permitted in other areas within PBOW; therefore, future use of AP3 for hunting is evaluated in this BHHRA.

Many kinds of game animals may be hunted and consumed (e.g., squirrel, pheasant and other upland birds, turkey, or 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 cm², 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/cm² (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 the AP3 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 Future 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. AP3 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 previously. Because they would likely not represent an upper bound for nonresidential exposure, these receptors are not evaluated. The unnamed tributary to Plum Brook is too small to support game fish, as is the AP3 drainage ditch. 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 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 (2010a,b; 2011) ProUCL (Version 4.1) 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 (Section 2.5). One-half the reporting limit is used as the ProUCL input concentration for nondetects as a conservative estimate of the method detection limit, because method detection limits are not available for the historical data (USACE, 1999). 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 nondetect 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, 2011) includes mathematical manipulations under the default “with NDs” mode 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 AP3 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 (e.g., normal, lognormal, gamma, or not discernable) that provides the best fit, number of nondetects, size of the data set, and skewness. If the recommended value(s) under the default mode equals or exceeds the MDC, ProUCL is rerun using the full data set mode, under the assumption that the COPC is present in nondetects at one-half the reporting limit. 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 the ProUCL protocol described in the preceding paragraphs 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. Note that EPA (1989a) guidance states that an estimate of average rather than the MDC should be used to represent the EPC under chronic exposure and that use of the maximum is typically not reasonable. Therefore, data sets which use the MDC as the EPC are generally biased high. 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 sections for estimating COPC concentrations in air include only those relevant and used specifically for AP3 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):

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

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 ($1\text{E-}3$ kg per g).

Plausible values for D include $2\text{E-}4$ grams per cubic meter (g/m^3) for agricultural activity (DOE, 1989), $6\text{E-}4$ g/m^3 for construction work (DOE, 1983), and $1\text{E-}4$ g/m^3 for other activity (National Council on Radiation Protection and Measurements, 1984). The value for D of $1\text{E-}4$ g/m^3 for other activity is used for the groundskeeper. It is assumed that construction activities requiring intensive contact with soil, for which $D = 6\text{E-}4$ 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

$D = 1E-4 \text{ g/m}^3$. Therefore, a time-weighted average dust loading factor for construction work of $3.5E-4 \text{ g/m}^3$ 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:

$$PEF = Q/C \times \frac{3600}{0.036 \times (1 - V) \times (U_m / U_t)^3 \times F(x)} \quad \text{Eq. 3.2}$$

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 [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:

$$C_a = \frac{C_s}{PEF} \quad \text{Eq. 3.3}$$

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 $1\text{E-}5$ 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 EPA (1991b) Human Health Evaluation Manual (HHEM) 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

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

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 AP3; therefore, the fraction of total browse consumed from AP3 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 AP3 area is relatively small in comparison to the home range of a white-tailed deer. The total acreage of the AP3 study area is about 1.5 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 AP3 is approximately 1 percent of this land area. Although the use of FI_r equal to 0.01 or lower is justified, an FI_r value of 0.03 is used in the BHHRA to be consistent with other small sites evaluated at PBOW.

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, and future 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 future indoor worker and future 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/m³)
- FI_a = fraction of exposure attributed to site media (unitless)
- IR_a = inhalation rate (m³/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, future resident, future indoor worker, hunter) or sediment (construction worker, future 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, future resident) is estimated from the following equation:

$$I_w = \frac{(C_w)(IR_w)(FI_w)(EF_w)(ED_w)}{(BW)(AT)}$$

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/cm²-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 (cm²)
- 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, future on-site resident, hunter) or sediment (construction worker, future 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/cm²-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/cm²-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, future resident) or surface water (construction worker, future 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 ($\text{mg}/\text{cm}^2\text{-day}$, calculated)
- C_w = concentration of COPC in water (mg/L)
- K_p = permeability coefficient (centimeters per hour [cm/hour])
- ET_w = time of exposure (hours/day)
- CF = conversion factor ($1\text{E-}3$ 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 ET is less than or greater than the estimated time to reach steady state. Non-steady-state exposures occur when either the ET 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 ($\tau > ET$, 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 ($\text{mg}/\text{cm}^2\text{-day}$, 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 ($1\text{E-}3$ 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 ($\tau < ET$), 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/cm²-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/cm³)
 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

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

where:

- K_p = permeability coefficient (cm/hour, calculated)
 $\text{log } K_{ow}$ = 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)
- C_v = concentration of COPC in venison (mg/kg)
- IR_v = 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, 2013). 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. 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. Because human studies typically have limitations (e.g., uncertainties regarding exposure concentrations, durations, lack of experimental control, small sample sizes, and representativeness of the exposed population), most SFs are derived from animal data. Uncertainties associated with animal studies are further mentioned in the uncertainties analysis.

The SF is expressed as risk per mg/kg-day, shown mathematically as $(\text{mg/kg-day})^{-1}$. 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, 2013) 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 $(\text{micrograms per cubic meter } [\mu\text{g}/\text{m}^3])^{-1}$. 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 $(\text{mg/kg-day})^{-1}$. 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 $\mu\text{g}/\text{m}^3$) by 70 kg and by 1,000 micrograms per milligram, 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, 2013) expresses the inhalation noncancer reference value as a reference concentration (RfC) in units of mg/m³. Because the noncancer hazard characterization described in the work plan (Shaw, 2009) requires a reference value expressed as mg/kg-day, the RfC is converted to an inhalation RfD. Because the inhalation RfC is based on continuous exposure of an adult human (assumed to inhale 20 m³ of air per day and to weigh 70 kg), the mathematical conversion consists of multiplying the RfC (mg/m³) by 20 m³/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, 2013) 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 (2013) 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, in order of hierarchy:

- Oral absorption efficiency data compiled by the National Center for Environmental Assessment for the Superfund Health Risk Technical Support Center of EPA, as listed in EPA (2004a).
- 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 evaluated for suitability for use in deriving dermal toxicity values from oral toxicity values. Some of the GAF values are also listed on the RSL table (EPA, 2012a). 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 COPC-specific toxicity profiles, including sources of the toxicity and GAF values, 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, and target organs 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 designed to be health protective and tend to overestimate rather than underestimate risk (EPA, 1989a; Burmaster and Harris, 1993; Cogliano, 1997). 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):

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

Eq. 5.1

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.

Because all of the risks associated with AP3 COPCs are less than 1E-2, only Equation 5.1 was used in this BHHRA. 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

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

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 following equation:

Eq. 5.4

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

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 goal of 1E-5, the logarithmic midpoint of the NCP risk management range, was selected by the PBOW PDT as a basis to consider remedial action. This 1E-5 goal is also recognized by the State of Ohio as a goal for cancer risk (OEPA, 2009b). Use of this 1E-5 goal represents a departure from the Army’s practice of using a cancer risk exceeding a value of 1E-4 (the upper end of the NCP risk management range) to trigger remedial action considerations. Total ILCR values are rounded to one significant figure, consistent with EPA (1989a) guidance.

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 5.5 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

$$HI = HQ_1 + HQ_2 + \dots HQ_i$$

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 (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

$$\text{Total Target Organ HI}_a = HI_{p1-a} + HI_{p2-a} + \dots HI_{pi-a}$$

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. Total HI values (including those specific to target organs) are rounded to one significant figure or to the nearest whole number if greater than 1, consistent with EPA (1989a) guidance.

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. Sections 5.3.1 through 5.3.7 describe the risk characterization results for each receptor associated with AP3. 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. Analytical results used as input for the BHHRA are included as Appendix A.

5.3.1 Current Groundskeeper

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 $2E-5$. This is within the $1E-6$ to $1E-4$ NCP risk management range but exceeds the PBOW cancer risk goal of $1E-5$.

Approximately 95 percent of this value is associated with arsenic in surface soil. Arsenic was identified as a COPC in surface soil because the concentration (44.1 mg/kg) at one location (AP3-SB08) slightly exceeded the BSC (36.5 mg/kg). Because the ILCR for this receptor exceeds the PBOW cancer risk goal, a WRS statistical test was performed that compares the arsenic concentrations in the AP3 surface soil data set to that of PBOW background. A WRS test result with a p-value less than 0.05 is regarded as indicating that two data sets are different. The p-value for this AP3 surface soil versus the PBOW background data set is 0.27 (Appendix E). Because this value is not less than 0.05, the AP3 surface soil data set is not regarded as significantly different from background. Therefore, the ILCR associated with arsenic is not regarded as being related to former PBOW operations. If the cancer risk contributions of arsenic in surface soil are excluded based on non-site relatedness, the resulting ILCR for this receptor is $1E-6$; this value is less than the PBOW cancer risk goal and equals the low end of the NCP cancer risk management range.

The total HI for the current groundskeeper (Table 5-1) is 0.2. This is 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 overburden groundwater via direct contact (ingestion and dermal exposure).

The total ILCR for the future groundskeeper, assuming hypothetical use of overburden groundwater, is $6E-5$ (Table 5-1). This value is within the NCP risk management range but exceeds the PBOW target cancer risk goal of $1E-5$. Approximately 70 percent of this value is associated with exposure to arsenic (ILCR= $4.2E-5$) in groundwater.

Arsenic was detected in two of the six overburden groundwater samples, both of which were collected in May 2012 with Snap Samplers. Each of the detections was “J” qualified as less than the reporting limit. Arsenic in AP3 groundwater is not regarded as resultant from former site operations for the following reasons:

- The two detected concentrations (5.2J $\mu\text{g/L}$ [AP3-MW2] and 9.9 $\mu\text{g/L}$ [AP3-MW01]) are similar to the range of detected concentrations in PBOW background groundwater (3.3 to 7.4 $\mu\text{g/L}$).
- Arsenic has not been identified as being part of any production processes or waste streams at PBOW.
- Arsenic was not found to be present in the overlying soils at concentrations significantly greater than PBOW background (see discussion below).

It is also noted that both detected concentrations were less than the maximum concentration limit of 10 $\mu\text{g/L}$ for arsenic in drinking water.

If the potential cancer effects associated with arsenic in groundwater are excluded because of non-site relatedness, the resulting total ILCR is $2\text{E-}5$ (Table 5-1). This is within the $1\text{E-}6$ to $1\text{E-}4$ NCP risk management range but exceeds the PBOW cancer risk goal of $1\text{E-}5$. Approximately 80 percent of this value is associated with arsenic in total soil. Arsenic was identified as a COPC in total soil because the concentration (44.1 mg/kg) in surface soil sample AP3-SB08 slightly exceeded the BSC (36.5 mg/kg). Because the ILCR for this receptor exceeds the PBOW cancer risk goal, a WRS statistical test was performed that compares the arsenic concentrations in the AP3 total soil data set to that of PBOW background. The p-value for this AP3 total soil data set versus the PBOW background data set is 0.097 (Appendix E). Because this value exceeds 0.05, the AP3 total soil arsenic data set is not regarded as significantly different from background soil arsenic concentrations. Therefore, the ILCR associated with arsenic in total soil is not regarded as being related to former PBOW operations.

If the contribution of arsenic to the ILCR in surface soil is excluded, the resulting ILCR for this receptor is $9\text{E-}7$. This value is less than the NCP cancer risk management range and the PBOW cancer risk goal.

The total HI for the future groundskeeper, assuming hypothetical use of overburden groundwater, is 0.7 (Table 5-1). This is less than the target HI goal of 1, indicating that noncancer health effects are regarded as unlikely to occur under this scenario. Approximately 84 percent of this value is associated with background-related inorganics in groundwater, chiefly arsenic (discussed above) and manganese (Section 5.3.5). If groundwater exposure pathways are appropriately excluded for this receptor because the noncancer hazards are associated with naturally occurring constituents, the HI (0.1) is even lower (Table 5-2).

5.3.3 Future 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 overburden groundwater via direct contact (ingestion and dermal exposure). The total ILCR for the future indoor worker, assuming hypothetical use of overburden groundwater, is $5E-5$ (Table 5-1). This value is within the NCP risk management range but exceeds the PBOW target cancer risk goal of $1E-5$. Approximately 84 percent of this value is associated with exposure to arsenic ($ILCR=4.2E-5$) in groundwater.

As described in Section 5.3.2, the presence of arsenic in AP3 overburden groundwater appears to be unrelated to former PBOW operations. If the potential cancer effects associated with arsenic in groundwater are thus excluded, the resulting total ILCR is $8E-6$ (Table 5-2). This is within the $1E-6$ to $1E-4$ NCP risk management range and less than the PBOW cancer risk goal of $1E-5$. Approximately 80 percent of this soil-based value is associated with arsenic in surface soil. Arsenic was identified as a COPC in surface soil because the concentration (44.1 mg/kg) in surface soil sample AP3-SB08 slightly exceeded the BSC (36.5 mg/kg). As described in Section 5.3.2, arsenic in surface soil is not significantly different from background.

If the contribution of arsenic to the ILCR in surface soil is excluded, the resulting ILCR for this receptor is $3E-7$ (Table 5-2). This value is less than the NCP cancer risk management range and the PBOW cancer risk goal.

The total HI for the future indoor worker, assuming hypothetical use of overburden groundwater, is 0.7 (Table 5-1). This is less than the target HI goal of 1 , indicating that noncancer health effects are regarded as unlikely to occur under this scenario. Approximately 89 percent of this value is associated with background-related inorganics in groundwater, chiefly arsenic (discussed above) and manganese (Section 5.3.5). If the groundwater exposure pathways are appropriately excluded for this receptor because the noncancer hazards are associated with naturally occurring constituents, the HI (0.07) is even lower (Table 5-2).

5.3.4 Construction Worker

The construction worker is assumed to be exposed to COPCs in total soil, both through direct contact and via inhalation of suspended particulates, and to sediment and surface water, via direct contact. The resulting ILCR of the AP3 construction worker is estimated as $1E-6$ (Table 5-1). This equals the low end of the $1E-6$ to $1E-4$ NCP risk management range and is less than the PBOW cancer risk goal 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 Hypothetical Future On-Site Resident

The exposure assumptions for the hypothetical future 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 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 (Section 3.1.3.5). The results of each are described below.

The total ILCR for the hypothetical future resident, assuming overburden groundwater exposure, is $2E-4$ (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk goal. Approximately 76 percent of this value is associated with exposure to overburden groundwater, all of which is associated with exposure to arsenic ($ILCR=1.8E-4$) in groundwater. As described in Section 5.3.2, arsenic does not appear to be associated with former PBOW operations.

If the contributions of the hypothetical groundwater exposure pathways are appropriately excluded because of the non-site relatedness of arsenic in groundwater, the resulting ILCR for this receptor is $6E-5$. This value is within the NCP risk management range but exceeds the PBOW cancer risk goal. Approximately 97 percent of this ILCR value is associated with exposure to total soil, and approximately 92 percent of the total ILCR value (minus groundwater) is associated with arsenic in soil. As described in Section 5.3.2, arsenic concentrations in total soil are not significantly greater than PBOW background soil. The ILCR (i.e., excluding groundwater) for this receptor without the contributions of background-related arsenic in soil is $5E-6$, which is less than the PBOW cancer risk goal.

The total HI for the hypothetical future child resident, assuming overburden groundwater exposure, is 5 (Table 5-1). This exceeds the target HI value of 1, which would seem to indicate that noncancer health effects cannot be regarded as unlikely to occur. Approximately 76 percent of this HI is associated with exposure to overburden groundwater, and approximately 87 percent of the groundwater HI is associated with arsenic ($HQ=1.7$) and manganese ($HQ=1.6$) in groundwater. As described in Section 5.3.2, arsenic concentrations in overburden groundwater do not appear to be related to former PBOW activities. Manganese was not selected as a COPC in total soil, which indicates that no site-related soil contamination was identified that may have

consequently leached to the underlying groundwater. Therefore, manganese in AP3 groundwater does not appear to have originated from any site-related activities. Also, please note that groundwater manganese concentrations exceeding the BSC (636 µg/L) were found only in well AP3-MW01 (1,760 and 779 µg/L), but not in the four samples from the other wells (52 to 472 µg/L).

If the groundwater pathway is appropriately excluded because the chemicals responsible for the elevated HI are unrelated to former U.S. Department of Defense activities, then the total HI for the hypothetical future child resident exposed to AP3 total soil, sediment, and surface water is 1 (Table 5-2). This value equals the target HI goal of 1, indicating that adverse noncancer health effects are unlikely.

The total HI for the hypothetical future adult resident, assuming overburden groundwater exposure, is 2 (Table 5-1). This exceeds the target HI value of 1, which indicates that noncancer health effects are not regarded as unlikely to occur. As discussed above for the hypothetical future child resident, most of the contribution to this HI is associated with arsenic and manganese in overburden groundwater, which are evidently unrelated to former PBOW activities. Thus, the contribution of the groundwater exposure may be appropriately excluded to evaluate for potential Department of Defense-related hazards. The resultant total HI for the hypothetical future adult resident exposed to AP3 total soil, sediment, and surface water is 0.1 (Table 5-2), which is less than the target HI goal of 1.

5.3.6 Adult Hunter

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

The total HI for the adult hunter (Table 5-1) is 0.009. 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 hunter's child (Table 5-1) exposed to surface soil via the ingestion of venison from deer which grazed on site cannot be quantified because none of the carcinogenic COPCs in surface soil bioaccumulate in food. Cancer risks associated with PBOW to this receptor are regarded as de minimis and less than the 1E-6 to 1E-4 NCP risk management range and the PBOW cancer risk goal of 1E-5.

The total HI for the future hunter's child is 1E-6 (Table 5-1). 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 PBOW risk goal) and that of scenario “B” may be $5E-5$ (exceeds the PBOW risk goal). 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 sets of uncertainties that are specific to AP3.

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 Sections 6.2.1 through 6.2.8 provide 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 AP3 area, where ash waste was deposited. The well locations were selected from within and adjacent to AP3, including downgradient areas. Surface water and sediment sample locations were selected within AP3 and along the drainage ditch emanating from AP3 to the west. The sample locations appear to be representative of the site, and no information suggests that the selection of these locations has introduced an identifiable bias.

6.2.2 Laboratory Analysis

State-of-the-practice SW-846 laboratory methods were used for analysis of the RI samples (Shaw, 2010; 2013). For metals in groundwater, this included Method 6010b, which utilizes inductively coupled plasma spectrometry. This method is subject to electronic interferences, particularly at concentrations less than the reporting limit. Site investigation samples, also used in this BHHRA, were collected in 1999 (USACE, 2000). No laboratory analytical data quality issues were identified that affected the BHHRA results.

6.2.3 Exposure-Point Concentration Estimates

Uncertainty is introduced in the statistical approach used to calculate the EPCs. As stated in the HHEM (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 4.1 software (EPA, 2011) 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 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.

Sediment 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 a high bias to the risk values.

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 AP3, the use of this receptor likely overestimates risks and hazards to a current site worker. The assumed future use of the hunter and the hunter's child are reasonable, as other areas within PBOW may currently be legally hunted by permit.

Unrestricted land use, including hypothetical future residential use, is a reasonable assumption for the future at AP3, given the rural residential use of property adjacent to the PBOW facility. However, AP3 is a low-lying former impoundment and is inundated throughout much of the year. Therefore, AP3 would have to be covered with several feet of soil before an industrial or residential building could be constructed at the site. This would essentially eliminate future exposure of the resident or long-term site worker (i.e., groundskeeper or indoor worker) to current AP3 surface or subsurface soil. Because of the apparent shallowness of the overburden groundwater as evidenced by the inundation, a basement could not likely be constructed. Therefore, exposure of a future construction worker to total soil would be minimized.

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 associated 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 not on assumptions made specifically for AP3. The assumption that overburden groundwater underlying AP3 could be used as a potable source is conservative, as it is unknown whether the groundwater in the vicinity of AP3 would provide ample yield for residential use. This question concerning yield is based on the limited yield in the overburden/shale unit underlying most of PBOW. However, water yield in the three AP3 wells was adequate for low-flow sampling, which indicates that use of AP3 groundwater may be plausible.

6.2.6 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.

This BHHRA used an RfD for thallium that was withdrawn from IRIS (EPA, 2013) in September 2010. This RfD was withdrawn because the thallium studies were judged to be of poor quality. To account for this poor quality, EPA formerly included a UF of 3,000, which is the maximum that EPA uses for verified RfDs. Inclusion of an RfD for thallium with this high UF is likely to introduce a conservative bias. The fact that EPA has withdrawn the RfD for thallium should be considered in any environmental decision related to the potential effects of thallium on human health.

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., former Group B2 (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.

6.2.7 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.

6.2.8 Evaluation of Selected COPCs for Site Relatedness

As described in Section 2.4.3, a comparison of naturally occurring metals is performed in two steps. The first is a comparison to BSCs; a chemical whose MDC does not exceed the BSC is assumed to be related to background and thus eliminated from further evaluation. The second step is performed for selected metals and organic compounds during the risk characterization step, after exposure calculations are performed which include the potential background-related contributions as part of the overall ILCR and HI results.

For inorganics, this risk characterization background evaluation step typically includes a statistical comparison of the site data set to the background data set using the WRS statistical test as appropriate (Section 2.4.3.2). The WRS output and box-and-whisker plots are provided in Appendix E. Note that no suitable background data sets exist for overburden groundwater, sediment, or surface water.

The WRS tests were run for the two metal COPCs in AP3 surface soil and total soil. The concentrations of neither arsenic ($p=0.27$) nor thallium ($p=0.90$) in surface soil were found to differ significantly from PBOW background concentrations. Similarly, the WRS results for AP3 total soil indicate that the concentrations of neither arsenic ($p=0.097$) nor thallium ($p=0.28$) differ significantly from those found in PBOW background soil.

With respect to groundwater, there is no background data set used to screen out naturally occurring inorganics in overburden groundwater. Thus, carrying inorganics forward into the risk characterization is a conservative decision. The concentrations of inorganics found in AP3 overburden groundwater were generally comparable to the background data set used for the limestone bedrock groundwater. None of the inorganic groundwater COPCs have been identified with any former PBOW waste streams. The inorganics with the highest cancer risk (arsenic) and noncancer hazard (arsenic and manganese [soil only]) are evidently unrelated to former PBOW

site operations. Manganese was not identified as an AP3 soil COPC, and AP3 soil concentrations of arsenic were shown to be statistically similar to those of the PBOW background data set. Therefore, the arsenic and manganese in AP3 groundwater apparently do not result from contamination in soil. These observations provide further evidence that the inclusion of groundwater inorganics in the risk characterization biases the risk/hazard estimates high.

7.0 Summary and Conclusions

7.1 Summary

The BHHRA was conducted to evaluate cancer risk and noncancer hazards associated with AP3 surface soil, subsurface soil, overburden groundwater, surface water, and sediment. It is noted that the BHHRA, including the evaluation of future uses and groundwater use, was conducted to meet administrative requirements, including FUDS regulations (USACE, 2004). 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)
- Future indoor worker (surface soil, subsurface soil [inhalation pathway only], groundwater)
- Current/future construction worker (total soil, surface water, sediment)
- Hypothetical future resident (total soil, surface water, sediment, overburden groundwater,)
- Future adult hunter (surface soil, including venison pathway)
- Future hunter's child (surface soil [venison pathway only]).

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 PBOW cancer risk goal ($ILCR > 1E-5$) are shown as bold and exceedances of the noncancer hazard goal ($HI > 1$) or the NCP risk management range ($1E-6$ to $1E-4$) are shown as bold italics:

- Current groundskeeper: $ILCR = \mathbf{2E-5}$ ($1E-6$ excluding background-related arsenic); $HI = 0.2$
- Future groundskeeper: $\mathbf{ILCR = 6E-5}$ ($1E-6$ excluding background-related arsenic); $HI = 0.1$
- Future groundskeeper excluding groundwater: $ILCR = \mathbf{2E-5}$ ($9E-7$ excluding background-related arsenic in soil); $HI = 0.1$

- Future indoor worker: ILCR = **5E-5**; HI = 0.7
- Future indoor worker excluding groundwater: ILCR = 8E-6; HI = 0.1
- Construction worker: ILCR = 1E-6; HI = 0.4
- Hypothetical future resident: **ILCR = 2E-4; child HI = 5; adult HI = 2**
- Hypothetical future resident excluding groundwater: ILCR = **6E-5** (5E-6 excluding background-related arsenic in soil); child HI = 1; adult HI = 0.1
- Future hunter: ILCR = 1E-6; HI = 0.009
- Future hunter's child: HI = 0.000001; none of the carcinogenic COPCs is bioaccumulative; cancer risks are assumed to be de minimis.

No construction is currently planned at AP3, and no groundskeeping of any sort appears to be occurring at AP3. Therefore, the current groundskeeper, which assumes a full-time, 25-year employee who works exclusively at the AP3 for 250 days per year, represents an extreme overestimate of exposure to any current receptor. It is also noted that because AP3 is a low-lying former impoundment and is frequently inundated, several feet of fill would have to be placed on the current surface before any construction could reasonably occur at the site. Therefore, potential exposure to current AP3 surface or subsurface soil is likely minimal for any receptor, even under unrestricted future use. Therefore, cancer risks and noncancer hazard estimates associated with soil in this BHHRA that assume future development are likely exaggerated.

7.2 Conclusions

The ILCR values for each of the AP3 exposure scenarios are within (or less than) the NCP risk management range and the PBOW cancer risk goal of 1E-5, except the hypothetical future resident who is assumed to be exposed to groundwater. As presented in Section 5.3.5, arsenic, which is responsible for the ILCR associated with groundwater, is evidently unrelated to former PBOW activities. Therefore, the future groundskeeper, future indoor worker, and future resident were re-evaluated without the contribution of arsenic in groundwater. Of these, the future groundskeeper and future resident (even excluding groundwater) exceed the PBOW ILCR goal, as does the current groundskeeper. Nearly all of this ILCR is associated with arsenic in soil, which was observed at concentrations that are not statistically significantly greater than those found in the PBOW background soil data set. If the contributions of arsenic in soil are excluded from the ILCR based on a lack of site relatedness, then the ILCR value for each of the receptors is less than the PBOW ILCR goal.

The HI values of each of the AP3 receptors, except the future adult and child resident assumed to use overburden groundwater, meet the noncancer target HI of 1. Arsenic and manganese in overburden groundwater are responsible for the elevated HI values for the adult and child resident. As presented in Section 5.3.5, the sources of these inorganics in groundwater are evidently unrelated to former site operations, and they appear to be naturally occurring. If the contributions of these naturally occurring inorganics in groundwater are appropriately excluded from the adult and child resident HI estimates, all receptors meet the HI goal of 1.

Because AP3 is in a low area and frequently inundated, additional soil would need to be added to the site before construction could commence, minimizing the potential for exposure to current site soil. Therefore, cancer risks and noncancer hazard estimates associated with soil in this BHHRA that assume future development (i.e., each receptor except the hunter and hunter's child) are likely exaggerated.

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TABLES

Table 2-1

Summary of Samples Evaluated
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

Location	Sample Number	Sample Type	Sample Date	Depth (ft)	Analyses
SOIL					
Surface Soil					
ASH PIT 3-SB01	AP0032	REG	19-Aug-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB02	AP0036	REG	19-Aug-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB07	AP0051	REG	21-Aug-09	0 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB03	AP0039	REG	19-Aug-09	0.2 - 1.7	Exp, Metals, PCB, SVOC
ASH PIT 3-SB06	AP0048	REG	20-Aug-09	0.2 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB04	AP0042	REG	21-Aug-09	0.3 - 0.8	Exp, Metals, PCB, SVOC
ASH PIT 3-SB05	AP0045	REG	21-Aug-09	0.3 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB08	AP0054	REG	21-Aug-09	0.3 - 1	Exp, Metals, PCB, SVOC
ASH PIT 3-SB08	AP0056	FD	21-Aug-09	0.3 - 1	Exp, Metals, PCB, SVOC
Subsurface Soil					
ASH PIT 3-SB04	AP0043	REG	21-Aug-09	0.8 - 1.5	Exp, Metals, PCB, SVOC
ASH PIT 3-SB05	AP0046	REG	21-Aug-09	1 - 2	Exp, Metals, PCB, SVOC
ASH PIT 3-SB06	AP0049	REG	20-Aug-09	1 - 2	Exp, Metals, PCB, SVOC
ASH PIT 3-SB07	AP0052	REG	21-Aug-09	1 - 2	Exp, Metals, PCB, SVOC
ASH PIT 3-SB07	AP0059	FD	21-Aug-09	1 - 2	Exp, Metals, PCB, SVOC
ASH PIT 3-SB08	AP0055	REG	21-Aug-09	1 - 2	Exp, Metals, PCB, SVOC
ASH PIT 3-SB02	AP0037	REG	19-Aug-09	1.2 - 2.3	Exp, Metals, PCB, SVOC
ASH PIT 3-SB06	AP0061A	REG	20-Aug-09	2 - 2.7	Exp, Metals, PCB, SVOC
ASH PIT 3-SB01	AP0033	REG	19-Aug-09	3 - 5	Exp, Metals, PCB, SVOC
OVERBURDEN GROUNDWATER					
AP3-MW01	AP3083	REG	12/19/2011	1.3 - 1.3	Exp, Field Tests, Gen Chem, Metals (f & uf), SVOC, VOC
AP3-MW01	AP3084	FD	12/19/2011	1.3 - 1.3	Exp, Gen Chem, Metals (f & uf), SVOC, VOC
AP3-MW01	AP3088	REG	5/30/2012	0 - 0	Exp, Gen Chem, Metals, VOC
AP3-MW02	AP3086	REG	12/16/2011	10.02 - 10.19	Exp, Field Tests, Gen Chem, Metals, SVOC, VOC
AP3-MW02	AP3091	REG	5/30/2012	0 - 0	Exp, Gen Chem, Metals, VOC
AP3-MW03	AP3087	REG	12/20/2011	7.22 - 7.41	Exp, Field Tests, Gen Chem, Metals (f & uf), SVOC, VOC
AP3-MW03	AP3092	REG	5/30/2012	0 - 0	Exp, Gen Chem, Metals, VOC
SEDIMENT					
AP3-SD01	AP1009	REG	23-May-09	0 - 0.5	Exp, Metals, PCB, SVOC
AP3-SD02	AP1010	REG	23-May-09	0 - 0.5	Exp, PCB
AP3-SD03	AP1011	REG	23-May-09	0 - 0.5	Exp, PCB
PBOW99-SDA302	PBOW99SDA302	REG	11-Jun-99	0 - 0	Metals, SVOC
PBOW99-SDA303	PBOW99SDA303	REG	11-Jun-99	0 - 0	Metals, SVOC

Table 2-1

**Summary of Samples Evaluated
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 2)

Location	Sample Number	Sample Type	Sample Date	Depth (ft)	Analyses
PBOW99-SDA303	PBOW99SDA303DUP	FD	11-Jun-99	0 - 0	Metals, SVOC
SURFACE WATER					
AP3-SW01	AP2009	REG	23-May-09	NA	Exp
AP3-SW02	AP2010	REG	23-May-09	NA	Exp
AP3-SW03	AP2011A	REG	27-May-09	NA	Exp
PBOW99-SWA301	PBOW99SWA301	REG	11-Jun-99	NA	Metals, SVOC
PBOW99-SWA302	PBOW99SWA302	REG	11-Jun-99	NA	Metals, SVOC
PBOW99-SWA303	PBOW99SWA303	REG	11-Jun-99	NA	Metals, SVOC
PBOW99-SWA303	PBOW99SWA303DUP	FD	11-Jun-99	NA	Metals, SVOC

ft bgs - Feet below ground surface.

FD - Field duplicate; averaged with the regular sample at the same location to create one result.

REG - Regular sample.

Exp - Explosives.

f & uf - Metals analysis performed on filtered and unfiltered samples, respectively.

Gen Chem - General chemistry.

Metals - Metals analysis performed on unfiltered samples unless otherwise noted.

PCB - Polychlorinated biphenyls.

SVOC - Semivolatile organic compounds.

VOC - Volatile organic compounds.

Table 2-2

**Background Screening Concentrations of Inorganics in Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works
Sandusky, Ohio**

Chemical Name (mg/kg)	Frequency of Detection	Range of Detected Concentrations	Range of Reporting Limits ^a	Statistical Distribution	Arithmetic Mean	95% UTL ^b	Background Screening Concentration ^c
Aluminum	12 / 12	3520 - 15500	22.6 - 26.5	L	8.43E+03	2.69E+04	15500
Antimony	9 / 25	5.9 - 9.3	5.4 - 8.0	NP	4.68E+00	NA	9.30
Arsenic	23 / 26	2.1 - 36.5	1.1 - 24.7	L	1.08E+01	7.10E+01	36.5
Barium	9 / 12	35.6 - 826	22.6 - 26.5	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	566 - 663	L	1.13E+04	2.18E+05	52300
Chromium	25 / 26	4.4 - 29	1.1 - 12.3	NP	1.34E+01	NA	29.0
Cobalt	9 / 12	9.6 - 116	5.7 - 61.7	L	2.26E+01	2.48E+02	116
Copper	23 / 26	2.3 - 56.2	2.2 - 3.3	L	1.70E+01	1.47E+02	56.2
Iron	12 / 12	5880 - 234000	11.3 - 123	L	4.01E+04	3.58E+05	234000
Lead	26 / 26	1.9 - 48.6	0.34 - 7.4	L	1.28E+01	5.13E+01	48.6
Magnesium	12 / 12	629 - 10400	566 - 663	L	3.26E+03	3.08E+04	10400
Manganese	26 / 26	21 - 13300	1.7 - 18.5	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	4.5 - 5.3	L	2.28E+01	7.79E+01	55.1
Potassium	11 / 12	579 - 3390	566 - 663	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	5.7 - 61.7	L	2.48E+01	8.31E+01	40.9
Zinc	26 / 26	6.6 - 655	0.57 - 12.3	L	7.30E+01	3.22E+02	322

L - Lognormal; mg/kg - milligrams per kilogram; NP - nonparametric; NA - not applicable; not available.

^a A single background sample had to be diluted such that the reporting limits of this sample (BCG-SB01, 6990) were elevated 10 or 20 times higher than they would have been if not diluted. This affects the maximum reporting limit shown for arsenic, chromium, cobalt, iron, lead, manganese, and vanadium. Reporting limits for these analytes in all other samples were much lower, approximately by an order of magnitude or more in each case.

^b 95% UTL - 95% upper tolerance limit calculated as described in IT Corporation (IT), 1998, *Site Investigation of Acid Areas*, Plum Brook Ordnance Works, Sandusky, Ohio, August.

^c 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. This approach was agreed upon for all future Plum Brook Ordnance Works (PBO) risk assessments by the PBO Project Delivery Team (PDT) during the May 10, 2000 PDT meeting.

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: IT, 2001, *TNT Areas A and C Remedial Investigation, Volume 2 Baseline Human Health Risk Assessment, Final, Former Plum Brook Ordnance Works, Sandusky, Ohio*, November, and reports referenced therein, including IT (1998).

Table 2-3

**Background Screening Concentrations of Inorganics in Groundwater
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemical	Detection Frequency	Percent hits	Range of Values, µg/L				Mean µg/L	Standard Deviation	UTL ^a µg/L	BSC ^b µg/L
			Detected Concentrations		Reporting Limits					
			Minimum	Maximum	Minimum	Maximum				
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

^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 xylene

Source: Shaw Environmental, Inc. (Shaw), 20052004 Data Summary and Evaluation Report, Final, Plum Brook Ordnance Works, Sandusky, Ohio, April.

Table 2-4

**Statistical Summary and Selection of COPCs in Surface Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 2)

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg						Mean mg/kg	BSC ^a mg/kg	RBSC ^b mg/kg	COPC? ^{c,d}	95% UCL ^e mg/kg	EPC ^f mg/kg
			Detected Concentrations			Reporting Limits								
			Minimum	VQ	Maximum	VQ	Minimum	Maximum						
Inorganics														
Aluminum	8 / 8	100	4.76E+03		9.35E+03		1.30E+01	2.90E+01	7.69E+03	1.55E+04	7700	N (b)		---
Antimony	2 / 8	25	1.20E+00	J	2.34E+00	/UJ	3.80E+00	9.30E+00	2.81E+00	9.30E+00	3.1	N (a)		---
Arsenic	8 / 8	100	4.90E+00		4.41E+01		5.00E-01	1.20E+00	1.65E+01	3.65E+01	0.39	Y	2.88E+01	2.88E+01
Barium	8 / 8	100	3.85E+01		2.03E+02		1.30E+01	2.90E+01	9.32E+01	8.26E+02	1500	N (a)		---
Beryllium	8 / 8	100	4.10E-01	J	2.10E+00		3.10E-01	7.40E-01	1.19E+00	1.00E+00	16	N (a)		---
Cadmium	3 / 8	38	3.00E-01	J	4.40E-01		2.50E-01	3.10E+00	5.26E-01	NA	7	N (a)		---
Calcium	8 / 8	100	2.06E+03		1.63E+04		3.10E+02	7.40E+02	1.16E+04	5.23E+04	Nutrient	N (c)		---
Chromium	8 / 8	100	6.90E+00		1.66E+01		1.30E+00	3.00E+00	1.20E+01	2.90E+01	0.29	N (b)		---
Cobalt	8 / 8	100	4.50E+00		8.90E+00		3.10E+00	7.40E+00	6.32E+00	1.16E+02	2.3	N (b)		---
Copper	8 / 8	100	1.16E+01		1.24E+02		1.60E+00	3.70E+00	3.72E+01	5.62E+01	310	N (a)		---
Iron	8 / 8	100	1.24E+04	J	6.83E+04		6.30E+00	1.60E+01	2.69E+04	2.34E+05	5500	N (b)		---
Lead	8 / 8	100	3.90E+00	J	2.04E+01		6.30E+00	1.60E+01	1.10E+01	4.86E+01	400	N (a)		---
Magnesium	8 / 8	100	3.43E+02	J	5.00E+03	J	3.10E+02	7.40E+02	2.20E+03	1.04E+04	Nutrient	N (c)		---
Manganese	8 / 8	100	5.45E+01	J	3.53E+02	J	1.10E+00	2.30E+00	2.25E+02	3.51E+03	180	N (b)		---
Mercury	6 / 8	75	3.60E-02	J	1.50E-01		1.10E-01	2.40E-01	8.49E-02	8.50E-02	2.3	N (a)		---
Nickel	8 / 8	100	1.15E+01		2.29E+01		2.50E+00	5.90E+00	1.57E+01	5.51E+01	150	N (a)		---
Potassium	8 / 8	100	5.24E+02	J	1.29E+03	J	6.30E+02	1.50E+03	9.34E+02	3.39E+03	Nutrient	N (c)		---
Selenium	8 / 8	100	5.90E-01	J	2.90E+00	J	6.30E+00	1.60E+01	1.74E+00	2.00E+00	39	N (a)		---
Sodium	8 / 8	100	1.68E+02	J	5.70E+02	J	6.30E+02	1.50E+03	4.09E+02	NA	Nutrient	N (c)		---
Thallium	1 / 8	13	3.00E+00	U	3.00E+00	U	6.30E-01	6.50E+00	1.85E+00	1.30E+00	0.078	Y	2.69E+00	2.69E+00
Vanadium	9 / 9	100	1.07E+01		2.89E+01		3.00E+00	7.80E+00	2.04E+01	4.09E+01	39	N (a)		---
Zinc	9 / 9	100	1.59E+01	J	7.87E+01		1.20E+00	2.90E+00	4.91E+01	3.22E+02	2300	N (a)		---
Polychlorinated biphenyls (PCB)														
Aroclor 1260	2 / 8	25	1.50E-02	J	4.30E-02	J	2.20E-02	5.00E-02	2.08E-02		0.22	N (a)		---
Explosives														
Dinitrotoluene, 2,6-	3 / 8	38	5.62E-02	J	6.62E-02	JJ	1.60E-01	1.90E-01	7.67E-02		0.71	N (a)		---
Semivolatile Organic Compounds														
Acenaphthylene	1 / 8	13	5.16E-02	J	5.16E-02	J	2.10E-01	4.95E-01	1.71E-01		340	g	N (a)	---
Benzo(a)anthracene	1 / 8	13	1.56E-01	J	1.56E-01	J	2.10E-01	4.95E-01	1.84E-01		0.15	Y	2.20E-01	1.56E-01
Benzo(a)pyrene	1 / 8	13	1.68E-01	J	1.68E-01	J	2.10E-01	4.95E-01	1.85E-01		0.015	Y	2.21E-01	1.68E-01
Benzo(b)fluoranthene	1 / 8	13	2.68E-01	J	2.68E-01	J	2.10E-01	4.95E-01	1.98E-01		0.15	Y	2.38E-01	2.38E-01
Benzo(ghi)perylene	1 / 8	13	8.82E-02	J	8.82E-02	J	2.10E-01	4.95E-01	1.75E-01		170	h	N (a)	---
Benzo(k)fluoranthene	1 / 8	13	9.72E-02	J	9.72E-02	J	2.10E-01	4.95E-01	1.76E-01		1.5	N (a)		---
Chrysene	1 / 8	13	1.90E-01	J	1.90E-01	J	2.10E-01	4.95E-01	1.88E-01		15	N (a)		---
Fluoranthene	1 / 8	13	4.01E-01	J	4.01E-01	J	2.10E-01	4.95E-01	2.14E-01		230	N (a)		---
Indeno(1,2,3-cd)pyrene	1 / 8	13	9.14E-02	J	9.14E-02	J	2.10E-01	4.95E-01	1.75E-01		0.15	N (a)		---
Phenanthrene	1 / 8	13	1.07E-01	J	1.07E-01	J	2.10E-01	4.95E-01	1.77E-01		170	h	N (a)	---
Pyrene	1 / 8	13	2.73E-01	J	2.73E-01	J	2.10E-01	4.95E-01	1.98E-01		170	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.

Table 2-4

**Statistical Summary and Selection of COPCs in Surface Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 2)

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 EPA Regional Screening Level Table (November 2012) residential soil 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 95% UCL (Upper confidence limit) determined using ProUCL software (U.S. Environmental Protection Agency (EPA), 2011, ProUCL Version 4.1.01, 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.

^g RBSC based on acenaphthene.

^h RBSC based on pyrene.

Table 2-5

**Statistical Summary and Selection of COPCs in Subsurface Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 2)

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg				Mean mg/kg	BSC ^a mg/kg	RBSC ^b mg/kg	COPC? ^{c,d}	
			Detected Concentrations		Reporting Limits						
			Minimum	VQ Maximum	VQ	Minimum	Maximum				
Inorganics											
Aluminum	8 / 8	100	4.87E+03	1.14E+04		1.20E+01	2.70E+01	8.28E+03	1.55E+04	7.70E+03	N (b)
Antimony	5 / 8	63	5.00E-01	J 2.26E+00	JU	3.50E+00	1.20E+01	2.11E+00	9.30E+00	3.10E+00	N (a)
Arsenic	8 / 8	100	5.00E+00	3.27E+01		4.70E-01	1.60E+00	1.69E+01	3.65E+01	3.90E-01	N (b)
Barium	8 / 8	100	4.12E+01	1.13E+02		1.20E+01	2.70E+01	7.24E+01	8.26E+02	1.50E+03	N (a)
Beryllium	8 / 8	100	4.00E-01	1.50E+00		2.90E-01	6.70E-01	7.05E-01	1.00E+00	1.60E+01	N (a)
Cadmium	6 / 8	75	1.40E-01	J 2.10E-01	J	2.30E-01	4.00E+00	4.21E-01	NA	7.00E+00	N (a)
Calcium	8 / 8	100	3.20E+03	5.28E+04		3.00E+02	6.70E+02	3.52E+04	5.23E+04	Nutrient	N (c)
Chromium	8 / 8	100	7.70E+00	1.49E+01	J	1.20E+00	2.60E+00	1.17E+01	2.90E+01	2.90E-01	N (b)
Cobalt	8 / 8	100	5.80E+00	1.22E+01		2.90E+00	6.70E+00	7.43E+00	1.16E+02	2.30E+00	N (b)
Copper	8 / 8	100	1.50E+01	2.65E+01		1.50E+00	3.40E+00	1.91E+01	5.62E+01	3.10E+02	N (a)
Iron	8 / 8	100	1.12E+04	6.78E+04		5.80E+00	2.00E+01	2.50E+04	2.34E+05	5.50E+03	N (b)
Lead	8 / 8	100	3.40E+00	J 1.16E+01	JJ	5.90E+00	2.00E+01	8.44E+00	4.86E+01	4.00E+02	N (a)
Magnesium	8 / 8	100	6.10E+02	1.75E+04		2.90E+02	6.70E+02	1.19E+04	1.04E+04	Nutrient	N (c)
Manganese	8 / 8	100	8.17E+01	1.27E+03		1.80E+00	8.90E+00	4.43E+02	3.51E+03	1.80E+02	N (b)
Mercury	7 / 8	88	1.40E-02	J 3.70E-02	J	9.00E-02	2.20E-01	3.44E-02	8.50E-02	2.30E+00	N (a)
Nickel	8 / 8	100	1.34E+01	3.19E+01		2.30E+00	5.40E+00	1.87E+01	5.51E+01	1.50E+02	N (a)
Potassium	8 / 8	100	1.29E+03	2.11E+03		5.80E+02	1.30E+03	1.64E+03	3.39E+03	Nutrient	N (c)
Selenium	8 / 8	100	6.60E-01	J 2.20E+00	J	5.90E+00	2.00E+01	1.31E+00	2.00E+00	3.90E+01	N (a)
Sodium	8 / 8	100	2.24E+02	J 6.40E+02	J	5.80E+02	1.30E+03	3.69E+02	NA	Nutrient	N (c)
Thallium	2 / 8	25	5.00E-01	J 2.90E+00		5.80E-01	6.50E+00	1.14E+00	1.30E+00	7.80E-02	Y
Vanadium	7 / 7	100	1.33E+01	2.43E+01		2.90E+00	9.90E+00	1.83E+01	4.09E+01	3.90E+01	N (a)
Zinc	7 / 7	100	3.05E+01	J 5.14E+01		1.20E+00	2.70E+00	4.08E+01	3.22E+02	2.30E+03	N (a)
Explosives											
Dinitrotoluene, 2,6-	1 / 8	13	5.12E-02	J 5.12E-02	J	1.40E-01	1.80E-01	7.52E-02		7.10E-01	N (a)

Note: Exposure point concentrations (EPC) are not provided. The subsurface soil and surface soil data sets are combined for all AP1 soil pathways as "total soil" to evaluate soil exposure for future receptors. EPCs for these receptors are based on total soil. See Table 2.13.

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.

NA - Not available.

RBSC - Risk-based screening concentration.

U - Nondetect; sample results from field duplicates may combine a nondetect with a detect.

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 EPA Regional Screening Level Table (November 2012) residential soil values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.

Table 2-5

**Statistical Summary and Selection of COPCs in Subsurface Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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^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.

Table 2-6

**Statistical Summary and Selection of COPCs in Total Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 2)

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg						Mean mg/kg	BSC ^a mg/kg	RBSC ^b mg/kg	COPC? ^{c,d}	95% UCL ^e mg/kg	EPC ^f mg/kg	
			Detected Concentrations			Reporting Limits									
			Minimum	VQ	Maximum	VQ	Minimum	Maximum							
Inorganics															
Aluminum	16 / 16	100	4.76E+03		1.14E+04			1.20E+01	2.90E+01	7.99E+03	1.55E+04	7700	N (b)	---	
Antimony	7 / 16	44	5.00E-01	J	2.34E+00	/UJ		3.50E+00	1.20E+01	2.46E+00	9.30E+00	3.1	N (a)	---	
Arsenic	16 / 16	100	4.90E+00		4.41E+01			4.70E-01	1.60E+00	1.67E+01	3.65E+01	0.39	Y	2.28E+01 2.28E+01	
Barium	16 / 16	100	3.85E+01		2.03E+02			1.20E+01	2.90E+01	8.28E+01	8.26E+02	1500	N (a)	---	
Beryllium	16 / 16	100	4.00E-01		2.10E+00			2.90E-01	7.40E-01	9.47E-01	1.00E+00	16	N (a)	---	
Cadmium	9 / 16	56	1.40E-01	J	4.40E-01			2.30E-01	4.00E+00	4.74E-01	NA	7	N (a)	---	
Calcium	16 / 16	100	2.06E+03		5.28E+04			3.00E+02	7.40E+02	2.34E+04	5.23E+04	Nutrient	N (c)	---	
Chromium	16 / 16	100	6.90E+00		1.66E+01			1.20E+00	3.00E+00	1.19E+01	2.90E+01	0.29	N (b)	---	
Cobalt	16 / 16	100	4.50E+00		1.22E+01			2.90E+00	7.40E+00	6.87E+00	1.16E+02	2.3	N (b)	---	
Copper	16 / 16	100	1.16E+01		1.24E+02			1.50E+00	3.70E+00	2.81E+01	5.62E+01	310	N (a)	---	
Iron	16 / 16	100	1.12E+04		6.83E+04			5.80E+00	2.00E+01	2.60E+04	2.34E+05	5500	N (b)	---	
Lead	16 / 16	100	3.40E+00	J	2.04E+01			5.90E+00	2.00E+01	9.72E+00	4.86E+01	400	N (a)	---	
Magnesium	16 / 16	100	3.43E+02	J	1.75E+04			2.90E+02	7.40E+02	7.05E+03	1.04E+04	Nutrient	N (c)	---	
Manganese	16 / 16	100	5.45E+01		1.27E+03			1.10E+00	8.90E+00	3.34E+02	3.51E+03	180	N (b)	---	
Mercury	13 / 16	81	1.40E-02	J	1.50E-01			9.00E-02	2.40E-01	5.96E-02	8.50E-02	2.3	N (a)	---	
Nickel	16 / 16	100	1.15E+01		3.19E+01			2.30E+00	5.90E+00	1.72E+01	5.51E+01	150	N (a)	---	
Potassium	16 / 16	100	5.24E+02	J	2.11E+03			5.80E+02	1.50E+03	1.29E+03	3.39E+03	Nutrient	N (c)	---	
Selenium	16 / 16	100	5.90E-01	J	2.90E+00	J		5.90E+00	2.00E+01	1.52E+00	2.00E+00	39	N (a)	---	
Sodium	16 / 16	100	1.68E+02	J	6.40E+02	J		5.80E+02	1.50E+03	3.89E+02	NA	Nutrient	N (c)	---	
Thallium	3 / 16	19	5.00E-01	J	3.00E+00			5.80E-01	6.50E+00	1.50E+00	1.30E+00	0.078	Y	1.47E+00 1.47E+00	
Vanadium	16 / 16	100	1.07E+01		2.89E+01			2.90E+00	9.90E+00	1.95E+01	4.09E+01	39	N (a)	---	
Zinc	16 / 16	100	1.59E+01	J	7.87E+01			1.20E+00	2.90E+00	4.55E+01	3.22E+02	2300	N (a)	---	
Polychlorinated biphenyls (PCB)															
Aroclor 1260	2 / 16	13	1.50E-02	J	4.30E-02	J		2.10E-02	5.00E-02	1.69E-02		0.22	N (a)	---	
Explosives															
Dinitrotoluene, 2,6-	4 / 16	25	5.12E-02	J	6.62E-02	JJ		1.40E-01	1.90E-01	7.59E-02		0.71	N (a)	---	
Semivolatile Organic Compounds															
Acenaphthylene	1 / 16	6	5.16E-02	J	5.16E-02	J		2.00E-01	4.95E-01	1.49E-01		340	g	N (a)	---
Benzo(a)anthracene	1 / 16	6	1.56E-01	J	1.56E-01	J		2.00E-01	4.95E-01	1.56E-01		0.15	Y	1.81E-01 1.56E-01	
Benzo(a)pyrene	1 / 16	6	1.68E-01	J	1.68E-01	J		2.00E-01	4.95E-01	1.56E-01		0.015	Y	1.82E-01 1.68E-01	
Benzo(b)fluoranthene	1 / 16	6	2.68E-01		2.68E-01			2.00E-01	4.95E-01	1.63E-01		0.15	Y	1.91E-01 1.91E-01	
Benzo(ghi)perylene	1 / 16	6	8.82E-02	J	8.82E-02	J		2.00E-01	4.95E-01	1.51E-01		170	h	N (a)	---
Benzo(k)fluoranthene	1 / 16	6	9.72E-02	J	9.72E-02	J		2.00E-01	4.95E-01	1.52E-01		1.5	N (a)	---	
Chrysene	1 / 16	6	1.90E-01	J	1.90E-01	J		2.00E-01	4.95E-01	1.58E-01		15	N (a)	---	
Fluoranthene	1 / 16	6	4.01E-01		4.01E-01			2.00E-01	4.95E-01	1.71E-01		230	N (a)	---	
Indeno(1,2,3-cd)pyrene	1 / 16	6	9.14E-02	J	9.14E-02	J		2.00E-01	4.95E-01	1.52E-01		0.15	N (a)	---	
Phenanthrene	1 / 16	6	1.07E-01	J	1.07E-01	J		2.00E-01	4.95E-01	1.53E-01		170	h	N (a)	---
Pyrene	1 / 16	6	2.73E-01		2.73E-01			2.00E-01	4.95E-01	1.63E-01		170	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.

Table 2-6

**Statistical Summary and Selection of COPCs in Total Soil
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 2)

^a IT Corporation (IT), 1998, *Site Investigation of Acid Areas, Plum Brook Ordnance Works, Sandusky, Ohio*, August.

^b Risk-based screening concentrations based on EPA Regional Screening Level Table (November 2012) residential soil 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 95% UCL (Upper confidence limit) determined using ProUCL software (U.S. Environmental Protection Agency (EPA), 2011, ProUCL Version 4.1.01, 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.

^g RBSC based on acenaphthene.

^h RBSC based on pyrene.

Table 2-7

**Statistical Summary and Selection of COPCs in Overburden Groundwater
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemical	Detection Frequency	Percent hits	Range of values, µg/L				Reporting Limits		Arithmetic Mean µg/L	RBSC ^b µg/L	COPC? ^{c,d}	95% UCL ^e µg/L	EPC [†] µg/L	EPC mg/L
			Detected Concentrations		Reporting Limits									
			Minimum	VQ	Maximum	VQ	Minimum	Maximum						
Inorganics - Unfiltered														
Aluminum	4 / 6	67	3.49E+01	J	8.89E+01	J	2.00E+01	2.40E+01	4.72E+01	1600	N (a)	---	---	
Arsenic	3 / 6	50	5.00E+00		9.90E+00	J	1.00E+00	1.00E+00	3.85E+00	0.045	Y	8.02E+00	8.02E+00	8.02E-03
Barium	6 / 6	100	4.01E+01	J	2.20E+02	J	1.00E+00	4.00E+00	9.96E+01	290	N (a)	---	---	
Calcium	6 / 6	100	1.16E+05		1.61E+05	J	5.00E+01	5.00E+01	1.35E+05	Nutrient	N (b)	---	---	
Chromium	1 / 6	17	1.30E+00	J	1.30E+00	J	1.00E+00	1.00E+00	1.05E+00	0.031	Y	NA	1.30E+00	1.30E-03
Cobalt	3 / 6	50	1.15E+00	J	2.10E+00	J	1.00E+00	1.00E+00	1.31E+00	0.47	Y	1.75E+00	1.75E+00	1.75E-03
Iron	4 / 6	67	6.41E+01	J	2.24E+03		2.30E+01	3.40E+01	6.07E+02	1100	Y	1.35E+03	1.35E+03	1.35E+00
Magnesium	6 / 6	100	2.68E+04	J	7.16E+04	J	5.00E+01	5.00E+01	5.28E+04	Nutrient	N (b)	---	---	
Manganese	6 / 6	100	5.20E+01		1.77E+03		1.00E+00	1.00E+00	5.82E+02	32	Y	1.11E+03	1.11E+03	1.11E+00
Nickel	1 / 6	17	2.40E+00	J	2.40E+00	J	1.00E+00	1.00E+00	1.23E+00	30	N (a)	---	---	
Potassium	6 / 6	100	7.61E+02	J	1.11E+04		5.00E+01	5.00E+01	4.91E+03	Nutrient	N (b)	---	---	
Sodium	6 / 6	100	1.98E+04		1.54E+05		7.50E+02	3.40E+03	6.04E+04	Nutrient	N (b)	---	---	
Vanadium	2 / 6	33	2.10E+00	J	2.10E+00	J	1.00E+00	1.00E+00	1.37E+00	7.8	N (a)	---	---	
Zinc	5 / 6	83	7.60E+00	J	1.84E+01	J	1.00E+00	1.00E+00	1.05E+01	470	N (a)	---	---	
Explosives														
Nitrotoluene, 2-	1 / 6	17	1.80E-01	J	1.80E-01	J	7.80E-02	1.60E-01	1.23E-01	0.27	N (a)	---	---	
Volatile Organics														
Acetone	1 / 6	17	1.76E+01	J	1.76E+01	J	1.00E+01	1.00E+01	1.13E+01	1200	N (a)	---	---	
Chloromethane	1 / 6	17	1.60E+00	J	1.60E+00	J	5.00E-01	5.00E-01	6.83E-01	19	N (a)	---	---	
Dichloroethane, 1,1-	1 / 6	17	1.65E+00		1.65E+00		2.50E-01	2.50E-01	4.83E-01	2.4	N (a)	---	---	
Trichloroethane, 1,1,1-	1 / 6	17	2.65E-01	J	2.65E-01	J	2.00E-01	2.00E-01	2.11E-01	750	N (a)	---	---	
General Chemistry														
Chloride	3 / 3	100	1.75E+03	J	2.70E+04		1.00E+03	5.00E+03	1.33E+04	Nutrient	N (b)	---	---	
Nitrate-Nitrite	3 / 3	100	5.10E+01	J	5.30E+02		5.00E+01	5.00E+01	3.34E+02	1,000,000 ^f	N (a)	---	---	
Sulfate	3 / 3	100	1.65E+05	J	5.38E+05		2.00E+03	1.00E+04	3.36E+05	250,000 ^g	Y	NA	5.38E+05	5.38E+02

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 EPA Regional Screening Level Table (November 2012) tap water values and 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) - Essential nutrient.

^c Y - Chemical is chosen as COPC.

^d 95% UCL (Upper confidence limit) determined using ProUCL Version 4.1.01 (U.S. Environmental Protection Agency (EPA), 2011, Office of Research and Development, Las Vegas, Nevada, and Technology Support Center, Atlanta, GA, May, on line at http://www.epa.gov/esd/tsc/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 Because no Regional Screening Level exists for nitrite-nitrate, 0.1 X the nitrite-nitrate MCL (of 10,000 mg/L) is used for screening.

^g US Environmental Protection Agency (EPA), 2012, *2012 Edition of the Drinking Water Standards and Health Advisories*, Office of Water, April, EPA 822-R-12-001.

Table 2-8

**Statistical Summary and Selection of COPCs in Sediment
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg				Mean mg/kg	RBSC ^a mg/kg	COPC? ^{b,c}	EPC ^d mg/kg		
			Detected Concentrations		Reporting Limits							
			Minimum	VQ	Maximum	VQ	Minimum	Maximum				
Inorganics												
Aluminum	3 / 3	100	2.48E+03	J	7.75E+03		1.80E+01	3.31E+01	4.61E+03	7.70E+04	N (a)	---
Antimony	1 / 3	33	5.40E-01	J	5.40E-01	J	1.50E+00	5.40E+00	7.13E-01	3.10E+01	N (a)	---
Arsenic	3 / 3	100	3.70E+00		6.40E+00		7.20E-01	1.70E+00	5.33E+00	3.90E-01	Y	6.40E+00
Barium	2 / 3	67	2.66E+01		4.78E+01		1.80E+01	3.31E+01	2.99E+01	1.50E+04	N (a)	---
Beryllium	1 / 3	33	2.60E-01	J	2.60E-01	J	4.50E-01	8.30E-01	3.51E-01	1.60E+02	N (a)	---
Cadmium	1 / 3	33	3.00E-01	J	3.00E-01	J	3.05E-01	3.60E-01	2.06E-01	7.00E+01	N (a)	---
Calcium	3 / 3	100	5.09E+03		1.81E+04		4.50E+02	8.28E+02	1.09E+04	Nutrient	N (b)	---
Chromium	3 / 3	100	5.40E+00		1.08E+01		7.55E-01	9.00E-01	7.53E+00	2.90E-01	Y	1.08E+01
Cobalt	1 / 3	33	4.10E+00	J	4.10E+00	J	4.50E+00	8.30E+00	4.01E+00	2.30E+01	N (a)	---
Copper	3 / 3	100	1.44E+01		1.26E+02		2.30E+00	4.10E+00	7.15E+01	3.10E+03	N (a)	---
Iron	3 / 3	100	6.71E+03		1.30E+04	J	9.00E+00	1.66E+01	1.08E+04	5.50E+04	N (a)	---
Lead	3 / 3	100	6.70E+00		1.19E+01		4.55E-01	9.00E+00	9.10E+00	4.00E+02	N (a)	---
Magnesium	3 / 3	100	2.57E+03		5.67E+03		4.50E+02	8.28E+02	4.20E+03	Nutrient	N (b)	---
Manganese	3 / 3	100	1.30E+02	J	3.18E+02		1.40E+00	2.50E+00	1.96E+02	1.80E+03	N (a)	---
Mercury	1 / 3	33	2.70E-02	J	2.70E-02	J	1.40E-01	1.70E-01	6.23E-02	2.30E+01	N (a)	---
Nickel	3 / 3	100	6.55E+00		1.25E+01		3.60E+00	6.60E+00	9.85E+00	1.50E+03	N (a)	---
Potassium	2 / 3	67	3.37E+02	J	1.27E+03		7.59E+02	1.80E+03	6.62E+02	Nutrient	N (b)	---
Selenium	1 / 2	50	1.10E+00		1.10E+00		7.55E-01	8.30E-01	7.39E-01	3.90E+02	N (a)	---
Vanadium	3 / 3	100	6.55E+00	/U	1.93E+01		4.50E+00	8.30E+00	1.24E+01	3.90E+02	N (a)	---
Zinc	3 / 3	100	2.63E+01		5.28E+01	J	1.80E+00	3.30E+00	4.25E+01	2.30E+04	N (a)	---
Semivolatiles Organic Compounds												
Benzo(a)anthracene	1 / 3	33	1.43E-01	J	1.43E-01	J	3.10E-01	5.50E-01	2.23E-01	1.50E-01	N (a)	---
Benzo(a)pyrene	1 / 3	33	1.58E-01	J	1.58E-01	J	3.10E-01	5.50E-01	2.28E-01	1.50E-02	Y	1.58E-01
Benzo(b)fluoranthene	1 / 3	33	2.40E-01	J	2.40E-01	J	3.10E-01	5.50E-01	2.55E-01	1.50E-01	Y	2.40E-01
Benzo(ghi)perylene	1 / 3	33	1.01E-01	J	1.01E-01	J	3.10E-01	5.50E-01	2.09E-01	1.70E+02	N (a)	---
Benzo(k)fluoranthene	1 / 3	33	8.05E-02	J	8.05E-02	J	3.10E-01	5.50E-01	2.02E-01	1.50E+00	N (a)	---
Chrysene	1 / 3	33	1.59E-01	J	1.59E-01	J	3.10E-01	5.50E-01	2.28E-01	1.50E+01	N (a)	---
Fluoranthene	1 / 3	33	2.53E-01	J	2.53E-01	J	3.10E-01	5.50E-01	2.59E-01	2.30E+03	N (a)	---
Indeno(1,2,3-cd)pyrene	1 / 3	33	1.11E-01	J	1.11E-01	J	3.10E-01	5.50E-01	2.12E-01	1.50E-01	N (a)	---
Pyrene	1 / 3	33	2.06E-01	J	2.06E-01	J	3.10E-01	5.50E-01	2.44E-01	1.70E+02	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.

Table 2-8

**Statistical Summary and Selection of COPCs in Sediment
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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- ^a Risk-based screening concentrations based on EPA Regional Screening Level Table (November 2012) residential soil and 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 Exposure-point concentration (EPC) used in risk assessment equal to maximum detected concentration.
- ^e RBSC based on pyrene.

Table 2-9

**Statistical Summary and Selection of COPCs in Surface Water
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemical	Detection Frequency	Percent hits	Range of values, µg/L				Arithmetic Mean µg/L	RBSC ^a µg/L	COPC? ^{b,c}	
			Detected Concentration		Reporting Limits					
			Minimum	VQ	Maximum	VQ				Minimum
Metals										
Calcium	3 / 3	100	24450		27900	5000	5000	26683	Nutrient	N (b)
Magnesium	3 / 3	100	8990		9470	5000	5000	9290	Nutrient	N (b)
Manganese	3 / 3	100	41.65	J	195	15	15	130	320	N (a)
Sodium	2 / 3	67	9740		10350	5000	5000	7530	Nutrient	N (b)

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 EPA Regional Screening Level Table (November 2012) tap water values and 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.

Table 3-1

**Receptor/Exposure Scenarios
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 3)

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	Inhalation ^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^e			
Surface Soil	None	Soil	Incidental Ingestion
			Dermal Contact ^a
	Dust Emissions; Volatilization	Indoor Air	Inhalation ^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
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 3)

Source Medium	Model	Exposure Medium	Exposure Route
Construction Worker – Current/Future			
Total Soil	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	Inhalation ^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	Dermal Contact
	Volatilization from Water	Ambient Air	Inhalation ^a
Sediment	None	Sediment	Incidental Ingestion
			Dermal Contact

Table 3-1

**Receptor/Exposure Scenarios
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 3 of 3)

Source Medium	Model	Exposure Medium	Exposure Route
Hunter – Current/Future			
Surface Soil	None	Soil	Incidental Ingestion Dermal Contact
	Dust Emissions, Volatilization	Ambient Air	Inhalation ^a
	Biouptake	Venison	Venison Consumption
Subsurface Soil	Not Quantified ^e		
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
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 5)

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 (F _{l_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
Inhalation of VOCs in Indoor Air from Subsurface Soil					
Fraction exposed to contaminated medium (F _{l_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 (F _{l_{so}}), unitless	1 ^c	1 ^c	0.9 ^f	1 ^c	1 ^c

Table 3-2

**Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 5)

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
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
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

Table 3-2

**Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 3 of 5)

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Drinking Water Ingestion of Groundwater					
Fraction exposed to contaminated medium (FI_{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 (FI_{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
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

Table 3-2

**Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 4 of 5)

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
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
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

Table 3-2

**Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 5 of 5)

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
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 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.

csv – Chemical-specific value.

NA – Pathway not applicable for receptor.

Table 3-3

Physical Properties of COPCs^a
Ash Pit 3 Baseline Human Health Risk Assessment
Former 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)	<i>tau</i> (hour/event)	<i>t</i> [*] (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
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
Chromium, Total	NA	NA	NA	NA	NA	NA	NA	1.00E-03	NA	NA	NA	NA	52	NA
Cobalt	NA	NA	NA	NA	4.50E+01	NA	NA	4.00E-04	NA	NA	NA	NA	58.93	NA
Iron	NA	NA	NA	NA	2.50E+01	NA	NA	1.00E-03	NA	NA	NA	NA	55.845	NA
Manganese	NA	NA	NA	NA	6.50E+01	NA	NA	1.00E-03	NA	NA	NA	NA	54.94	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
Semivolatile Organic Compounds														
Benzo(a)anthracene	NA	NA	NA	5.10E-02	3.98E+05	5.66E+00	0.13	4.70E-01	2.03	8.53	1	2.8	228.3	3.34E-06
Benzo(a)pyrene	NA	NA	NA	4.30E-02	1.02E+06	6.10E+00	0.13	7.00E-01	2.69	11.67	1	4.3	252.3	4.57E-07
Benzo(b)fluoranthene	NA	NA	NA	2.26E-02	1.23E+06	6.12E+00	0.13	7.00E-01	2.77	12.03	1	4.3	252.3	6.57E-07
Dibenz(a,h)anthracene	NA	NA	NA	2.02E-02	4.74E+05	6.75E+00	0.13	1.50E+00	3.88	17.57	0.6	9.7	278.4	1.41E-07
Indeno(1,2,3-cd)pyrene	NA	NA	NA	1.90E-02	3.47E+06	6.58E+00	0.13	1.00E+00	3.78	16.83	0.6	6.7	276.3	1.60E-06
General Chemistry														
Sulfate	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

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-m³ - Atmospheres per cubic meter.

^a See Appendix C for references for these values.

Table 4-1

Summary of Toxicity Assessment^a
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

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	1.50E+00	1.50E+00	A	4.30E-03	1.50E+01
Chromium III	0.013	D	NA	NA	D	NA	NA
Chromium VI	0.025	D	NA	NA	A	1.20E-02	4.20E+01
Chromium, Total	0.013	D	NA	NA	A	1.20E-02	4.20E+01
Cobalt	1	ND	NA	NA	B	9.00E-04	3.20E+01
Iron	1	ND	NA	NA	ND	NA	NA
Manganese	0.04	D	NA	NA	D	NA	NA
Thallium	1	D	NA	NA	D	NA	NA
Semivolatile Organic Compounds							
Benzo(a)anthracene	1	B2	7.30E-01	7.30E-01	B2	8.80E-05	3.10E-01
Benzo(a)pyrene	1	B2	7.30E+00	7.30E+00	B2	8.80E-04	3.10E+00
Benzo(b)fluoranthene	1	B2	7.30E-01	7.30E-01	B2	8.80E-05	3.10E-01
Dibenz(a,h)anthracene	1	B2	7.30E+00	7.30E+00	B2	8.80E-04	3.10E+00
Indeno(1,2,3-cd)pyrene	1	B2	7.30E-01	7.30E-01	B2	8.80E-05	3.10E-01
General Chemistry							
Sulfate	NA	NA	NA	NA	NA	NA	NA

Table 4-1

Summary of Toxicity Assessment^a
Ash Pit 3 Baseline Human Health Risk Assessment
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 2 of 2)

Chemical of Potential Concern	GAF	Oral		Dermal Reference Dose mg/kg-day	Reference Concentration mg/m ³	Inhalation	
		Reference Dose mg/kg-day	Target Organs			Reference Dose mg/kg-day	Target Organs
Metals							
Arsenic	1	3.00E-04	S	3.00E-04	NA	NA	NA
Chromium III	0.013	1.50E+00	ND	2.00E-02	NA	NA	NA
Chromium VI	0.025	3.00E-03	ND	7.50E-05	1.00E-04	2.90E-05	Lung
Chromium, Total	0.013	2.10E-02	ND	2.70E-04	7.00E-04	2.03E-04	Lung
Cobalt	1	3.00E-04	Thyroid	3.00E-04	6.00E-06	1.70E-06	RT
Iron	1	7.00E-01	GI	NA	ND	NA	NA
Manganese	0.04	4.70E-02	CNS	1.90E-03	5.00E-05	1.40E-05	CNS
Thallium	1	6.50E-05	S,L	6.50E-05	ND	NA	NA
Semivolatile Organic Compounds							
Benzo(a)anthracene	1	ND	NA	NA	ND	NA	NA
Benzo(a)pyrene	1	ND	NA	NA	ND	NA	NA
Benzo(b)fluoranthene	1	ND	NA	NA	ND	NA	NA
Dibenz(a,h)anthracene	1	ND	NA	NA	ND	NA	NA
Indeno(1,2,3-cd)pyrene	1	ND	NA	NA	ND	NA	NA
General Chemistry							
Sulfate	NA	NA	NA	NA	NA	NA	NA

mg/kg-day - Milligram per kilogram - day.

mg/m³ - Milligram per cubic meter.

GAF - Gastrointestinal absorption factor.

NA - Not available or not applicable.

ND - No data.

^a See Appendix C which provides references for these values.

Target Organs: S - skin; GI - gastrointestinal; CNS - central nervous system; L - liver;

Weight of Evidence (WOE) 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 carcinogenic potential.

D - Inadequate evidence to assess carcinogenic potential.

Table 5-1

**Summary of Risk for All Receptors
Ash Pit 3 Baseline Human Health Risk Assessment
Former 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
<i>Exposure Media</i>													
Surface Soil	2.21E-05	0.15	NE	NE	7.82E-06	0.067	NE	NE	NE	1.28E-06	0.0086	NA	0.0000014
Total Soil	1.76E-05	0.11	1.07E-06	0.3	NE	NE	5.63E-05	0.13	1.2	NE	NE	NE	NE
Sediment	NE	NE	2.84E-07	0.1	NE	NE	1.74E-06	0.003	0.03	NE	NE	NE	NE
Surface Water	NE	NE	NA	NA	NE	NE	NA	NA	NA	NE	NE	NE	NE
Groundwater	4.22E-05	0.59	NE	NE	4.22E-05	0.59	1.79E-04	1.6	3.8	NE	NE	NE	NE
Total ILCR or HI	6.E-05	0.7	1.E-06	0.4	5.E-05	0.7	2.E-04	2	5	1.E-06	0.009	NA	0.000001

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:

^aTotal ILCR and total HI values for the groundskeeper reflect the respective totals for the future groundskeeper. The total ILCR and HI values for the current groundskeeper are simply those shown for surface soil. The rounded current groundskeeper ILCR is 2E-5 and the rounded HI is 0.2

Table 5-2

**Summary of Risk for Receptors Excluding Exposure to Groundwater^a
 Ash Pit 3 Baseline Human Health Risk Assessment
 Former Plum Brook Ordnance Works, Sandusky, Ohio**

	Future Groundskeeper ^a		Indoor Worker		Resident		
	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total Adult - HI	Total Child - HI
<i>Exposure Media</i>							
Surface Soil	2.21E-05	0.15	7.82E-06	0.067	NE	NE	NE
Total Soil	1.76E-05	0.11	NE	NE	5.63E-05	0.13	1.2
Sediment	NE	NE	NE	NE	1.74E-06	0.003	0.03
Surface Water	NE	NE	NE	NE	NA	NA	NA
Total ILCR or HI	2.E-05	0.1	8.E-06	0.1	6.E-05	0.1	1
Total ILCR Excluding Arsenic in Soil^b	9.E-07	--	3.E-07	--	5.E-06	--	--

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.

Notes:

^a The receptors are those evaluated for groundwater exposure as presented in Table 5-1. Groundwater exposure is excluded from this table because the risks and hazards are associated with groundwater exposure (Table 5-1) are resultant from the presence of inorganics that are evidently not related to former PBOW operations. Notably these are arsenic (cancer and noncancer) and manganese (noncancer).

^b Even without the contribution of groundwater exposure, the total ILCR values for the future groundskeeper and the resident exceed the PBOW ILCR goal of 1E-5. The risk driving COPC is arsenic, which was shown to be present in AP3 soil at concentrations that are not statistically greater than in the PBOW background data set and are thus interpreted as not being site related. If the contribution of arsenic in soil is excluded from the cancer risk calculations for these receptors, the resulting ILCR is less than the PBOW ILCR goal.

FIGURES

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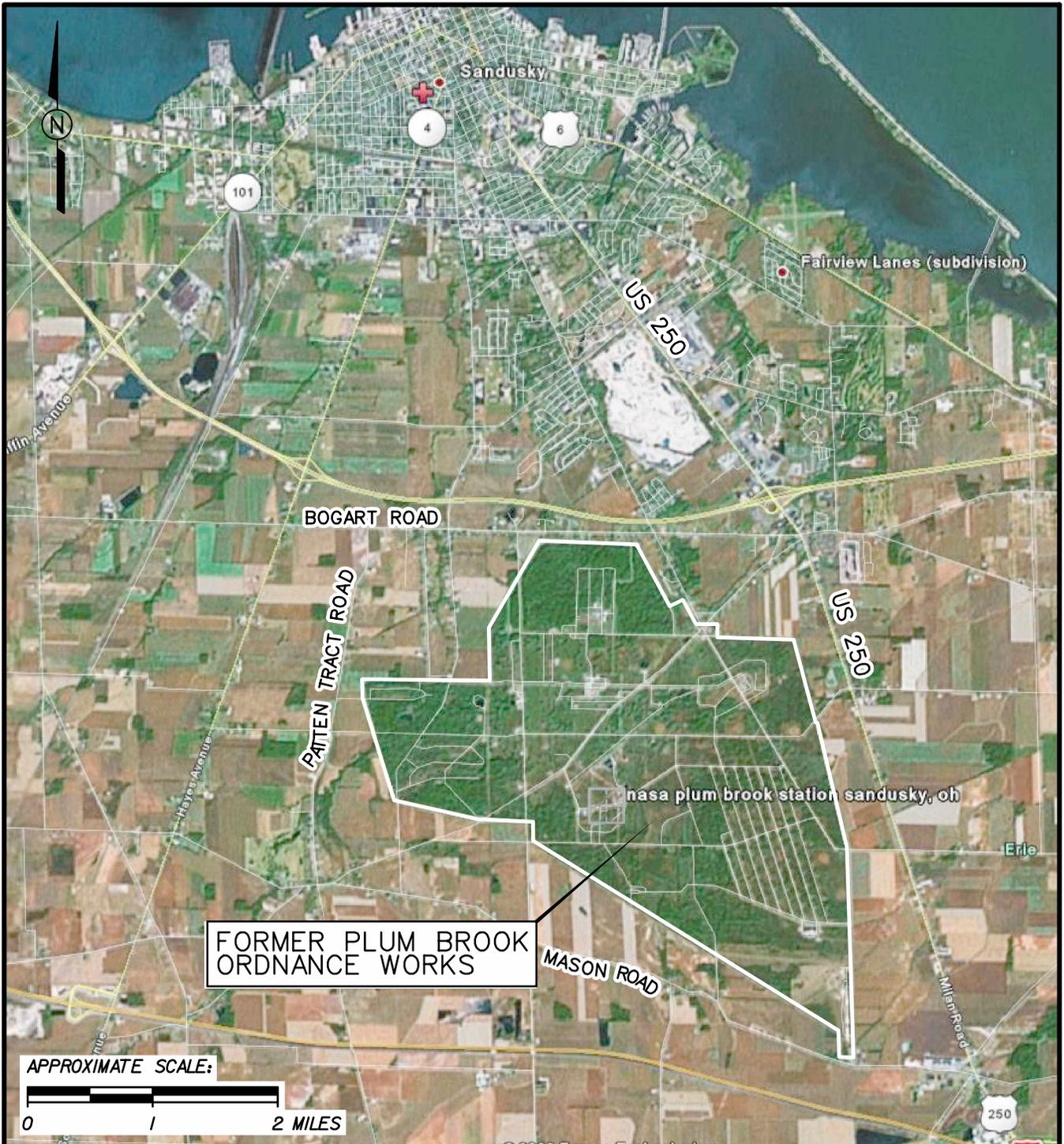
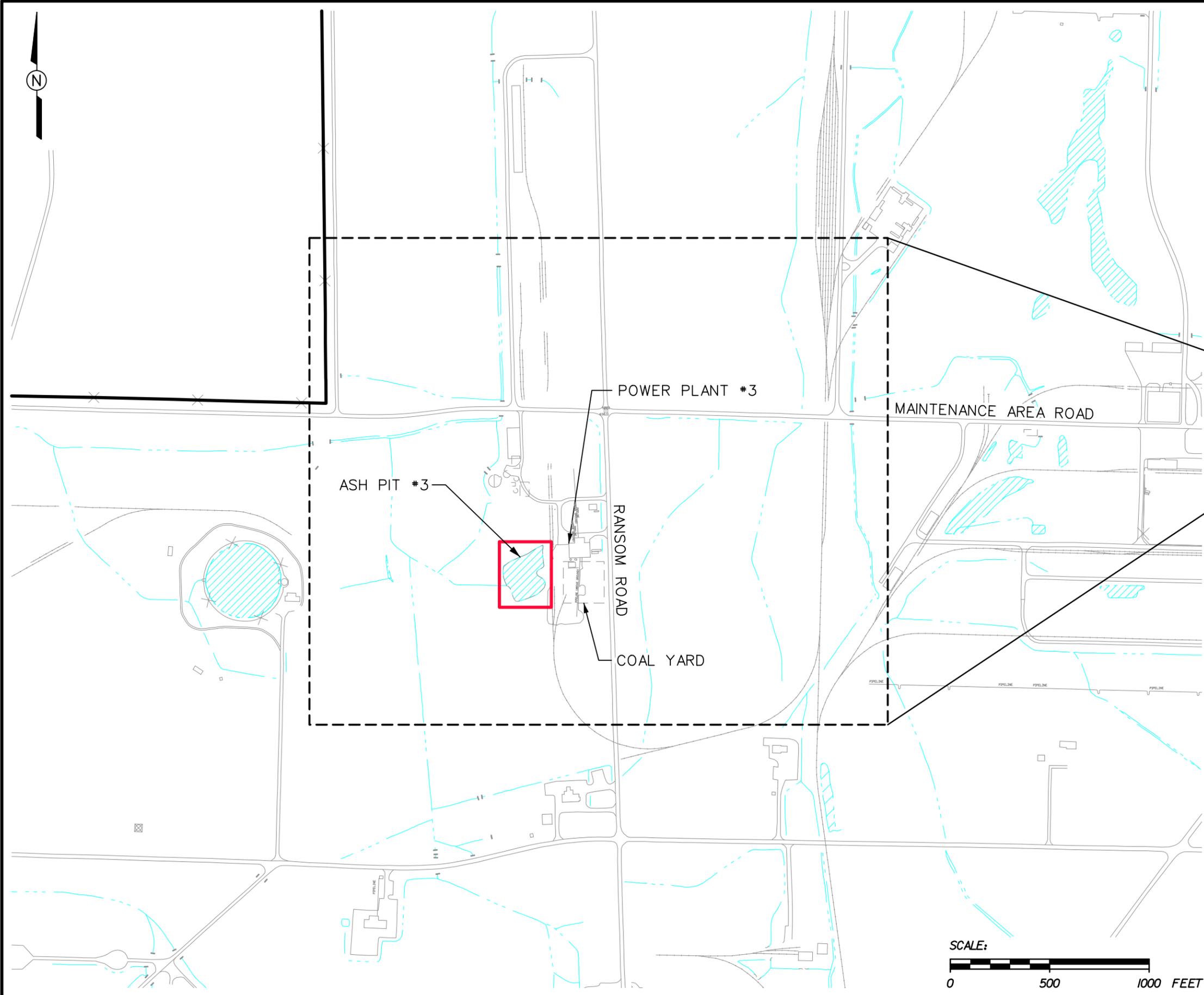


FIGURE 1-1
PBOW VICINITY MAP

ASH PIT 3 BASELINE HUMAN HEALTH
RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

 Shaw Environmental & Infrastructure, Inc.
(A CB&I Company)

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LEGEND:

- AREA OF CONCERN
- POND
- CREEK, DITCH, CONVEYANCE
- FORMER RAILROAD
- ROAD
- APPROXIMATE LOCATION OF FORMER STRUCTURES

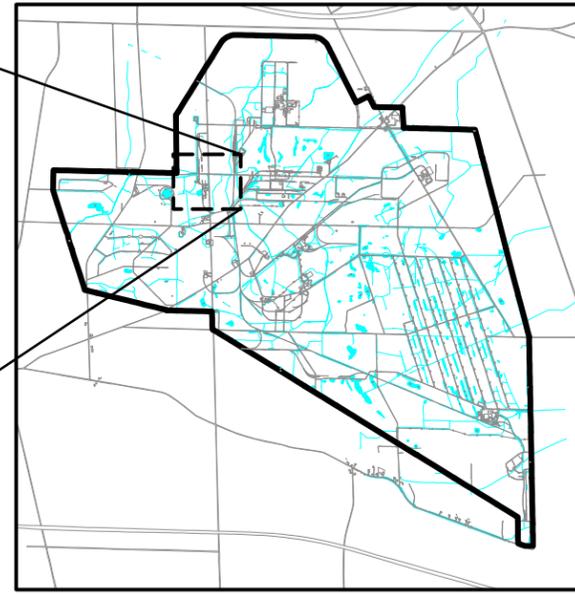
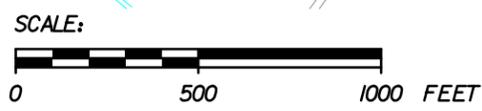


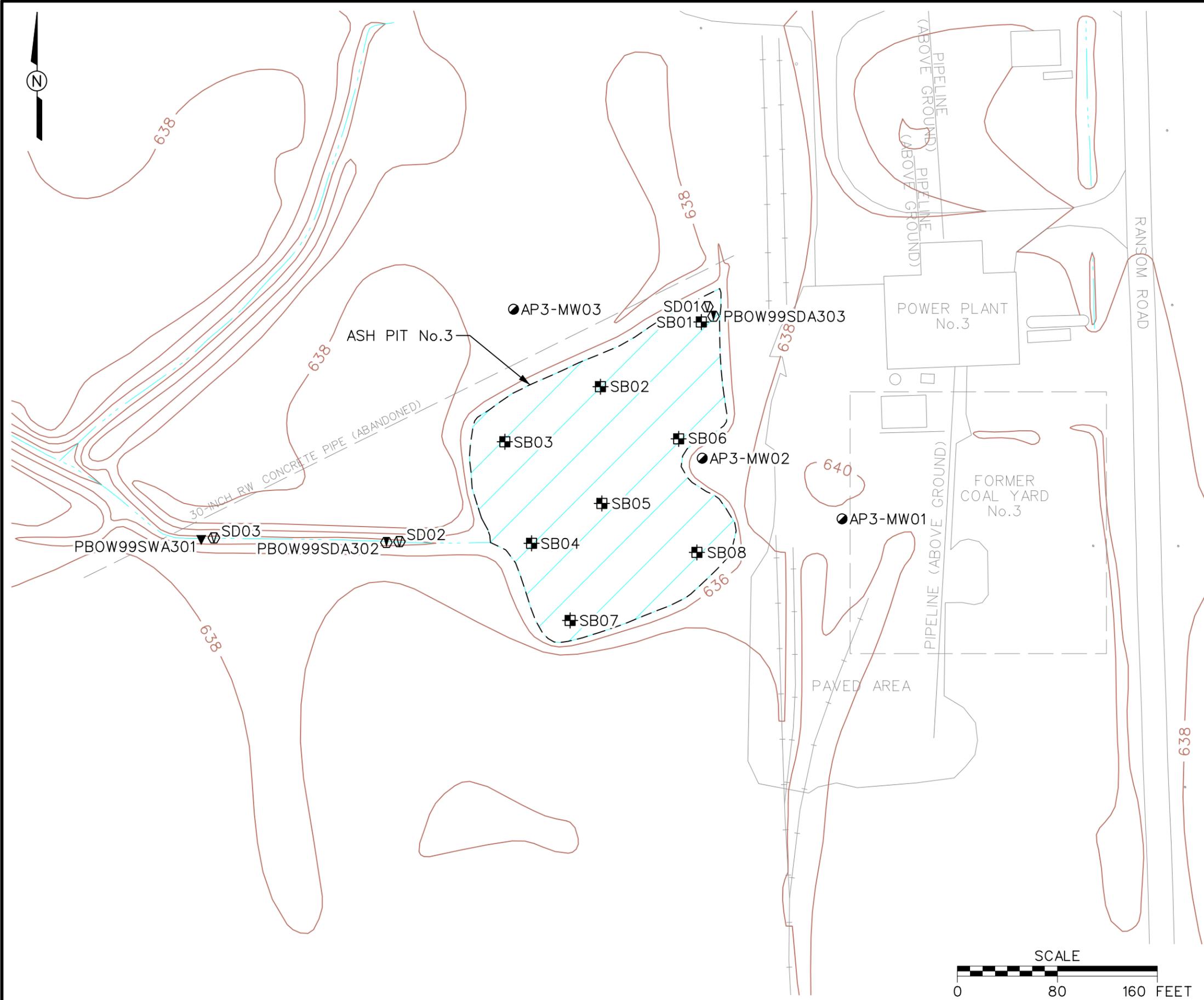
FIGURE 1-2
LOCATION OF ASH PIT 3 AT
PBOW

*ASH PIT 3 BASELINE HUMAN HEALTH
RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO*



Shaw Environmental & Infrastructure, Inc.
(A CB&I Company)

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LEGEND:

- OVERBURDEN MONITORING WELL
- ⊕ SOIL BORING
- ∇ SURFACE WATER/SEDIMENT SAMPLE
- ∇ SI SEDIMENT/SURFACE WATER SAMPLE (USACE, 2000)
- ▼ SI SURFACE WATER SAMPLE (USACE, 2000)
- UTILITY POLE
- ▨ POND
- CREEK, DITCH, CONVEYANCE
- FORMER RAILROAD
- ROAD
- 640— TOPOGRAPHIC CONTOUR (2 FT. INTERVAL)

NOTES:

1. SITE INVESTIGATION (SI) SAMPLES COLLECTED IN 1999 (USACE, 2000, LIMITED SITE INVESTIGATION, FINAL REPORT, FOR THE FORMER PLUM BROOK ORDNANCE WORKS ASH PITS NO. 1 AND 3, SANDUSKY, OHIO, JULY).
2. SOIL SAMPLES OTHER THAN THE SI SAMPLES WERE COLLECTED IN 2008 AND 2009 AS PART OF THE REMEDIAL INVESTIGATION.
3. GROUNDWATER SAMPLES WERE COLLECTED IN 2011 AND 2012 AS PART OF THE REMEDIAL INVESTIGATION.
4. FORMER COAL YARD NO.3 PERIMETER IS BASED ON HISTORICAL AERIAL PHOTOGRAPHY AND IS APPROXIMATE.

FIGURE 2-1
ASH PIT 3 SITE MAP WITH
SAMPLE LOCATIONS

ASH PIT 3 BASELINE HUMAN HEALTH RISK ASSESSMENT
 FORMER PLUM BROOK ORDNANCE WORKS
 NASA PLUM BROOK STATION
 SANDUSKY, OHIO

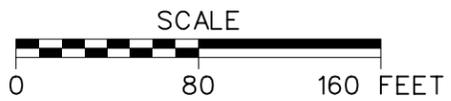
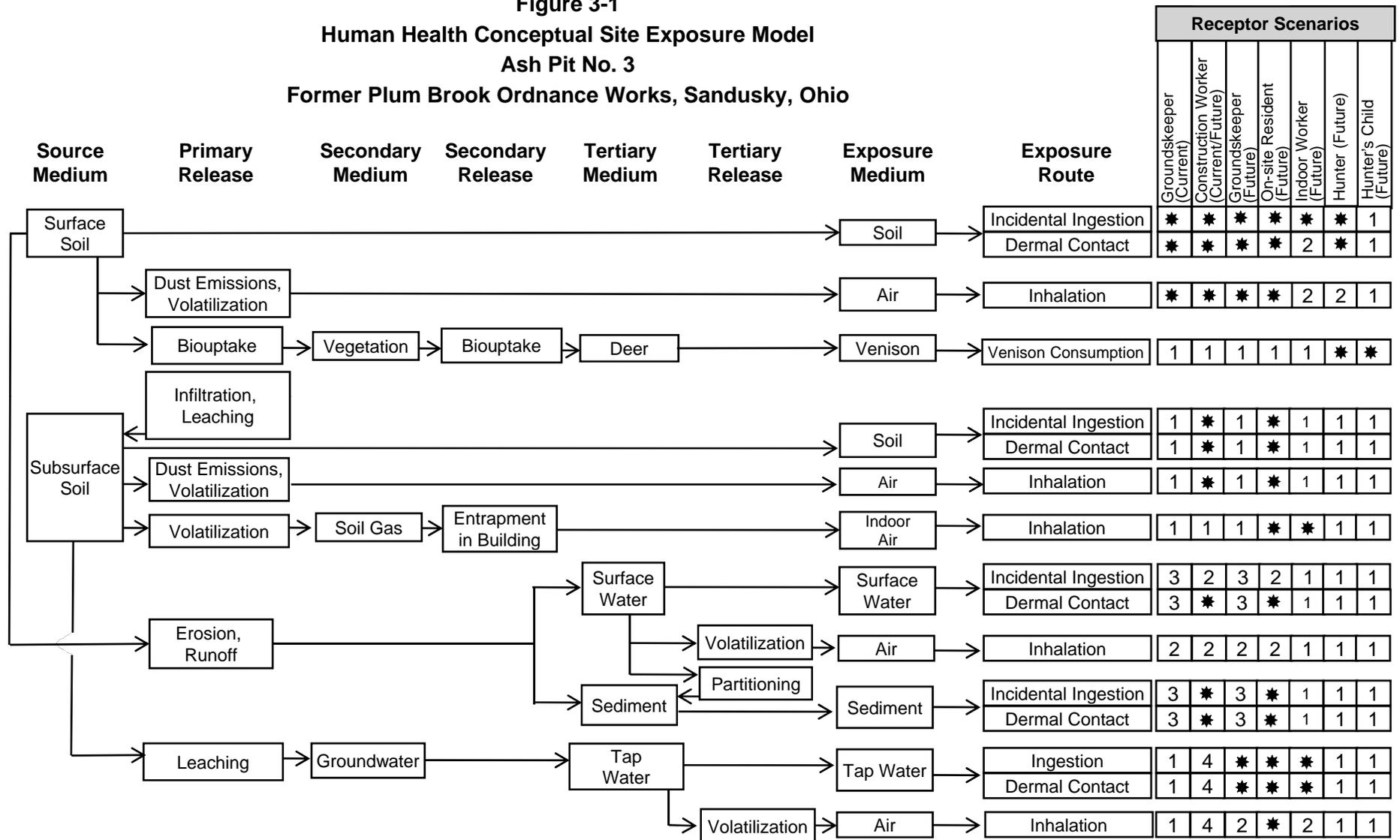


Figure 3-1
Human Health Conceptual Site Exposure Model
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio



* = 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.

APPENDIX A

ANALYTICAL RESULTS EVALUATED IN THE BASELINE HUMAN HEALTH RISK ASSESSMENT

Table A-1

Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 6)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB01 AP0032 19-Aug-09 REG 0-1							ASH PIT 3-SB02 AP0036 19-Aug-09 REG 0-1					ASH PIT 3-SB03 AP0039 19-Aug-09 REG 0.2-1.7				
	Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U	0.19	0.19	0.047	U	U	
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.18	0.18	0.098	U	U	0.16	0.16	0.087	U	U	0.19	0.19	0.1	U	U	
Dinitrobenzene, 1,3-	mg/kg	0.18	0.18	0.048	U	U	0.16	0.16	0.042	U	U	0.19	0.19	0.05	U	U	
Dinitrotoluene, 2,4-	mg/kg	0.18	0.18	0.054	U	U	0.16	0.16	0.048	U	U	0.19	0.19	0.057	U	U	
Dinitrotoluene, 2,6-	mg/kg	0.0563	0.18	0.048	J	J	0.16	0.16	0.042	U	U	0.19	0.19	0.05	U	U	
HMX	mg/kg	0.18	0.18	0.077	U	U	0.16	0.16	0.069	U	U	0.19	0.19	0.082	U	U	
Nitrobenzene	mg/kg	0.18	0.18	0.059	U	U	0.16	0.16	0.053	U	U	0.19	0.19	0.063	U	U	
Nitrotoluene, 2-	mg/kg	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U	0.19	0.19	0.047	U	U	
Nitrotoluene, 3-	mg/kg	0.18	0.18	0.075	U	U	0.16	0.16	0.067	U	U	0.19	0.19	0.08	U	U	
Nitrotoluene, 4-	mg/kg	0.18	0.18	0.056	U	U	0.16	0.16	0.049	U	U	0.19	0.19	0.059	U	U	
RDX	mg/kg	0.18	0.18	0.076	U	U	0.16	0.16	0.068	U	U	0.19	0.19	0.081	U	U	
Tetryl	mg/kg	0.18	0.18	0.046	U	U	0.16	0.16	0.041	U	U	0.19	0.19	0.048	U	U	
Trinitrobenzene, 1,3,5-	mg/kg	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U	0.19	0.19	0.047	U	U	
Trinitrotoluene, 2,4,6-	mg/kg	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U	0.19	0.19	0.047	U	U	
% Solids	Percent	67.3	0				77.2	0				38.9	0				
Aluminum	mg/kg	5060	14	0.79	J	J	7580	13	0.69	J	J	8820	25	1.4	J	J	
Antimony	mg/kg	4.3	4.3	0.32	U	U	3.8	3.8	0.28	U	U	7.6	7.6	0.57	U	U	
Arsenic	mg/kg	7.7	0.58	0.26			4.9	0.5	0.23			12.4	1	0.45			
Barium	mg/kg	59	14	0.36			48.1	13	0.31			90.7	25	0.63			
Beryllium	mg/kg	0.69	0.36	0.072			0.57	0.31	0.063			1.7	0.63	0.13			
Cadmium	mg/kg	0.33	0.29	0.072			0.25	0.25	0.063	U	U	0.5	0.5	0.13	U	U	
Calcium	mg/kg	8460	360	7.2	J	J	11700	310	6.3	J	J	11200	630	13	J	J	
Chromium	mg/kg	8.8	1.4	0.24	J	J	11	1.3	0.2	J	J	14.3	2.6	0.4	J	J	
Cobalt	mg/kg	4.5	3.6	0.06	J	J	5.7	3.1	0.052	J	J	6.7	6.3	0.1	J	J	
Copper	mg/kg	124	1.8	0.15			11.6	1.6	0.13			14.2	3.2	0.26			
Iron	mg/kg	12400	7.2	1.7	J	J	13300	6.3	1.4	J	J	20100	13	2.9	J	J	
Lead	mg/kg	16.5	7.2	0.14			9.7	6.3	0.13			3.9	13	0.25	B	J	
Magnesium	mg/kg	2640	360	7.2	J	J	5000	310	6.3	J	J	1210	630	13	J	J	
Manganese	mg/kg	99.2	1.1	0.036	J	J	353	1.9	0.063	J	J	148	1.9	0.063	J	J	
Mercury	mg/kg	0.15	0.12	0.017			0.047	0.11	0.016	B	J	0.036	0.21	0.031	B	J	
Nickel	mg/kg	13	2.9	0.17			13.6	2.5	0.14			15.1	5	0.29			
Potassium	mg/kg	524	720	7.2	B	J	818	630	6.3	J	J	1290	1300	13	B	J	
Selenium	mg/kg	1.1	7.2	0.22	B	J	0.59	6.3	0.19	B	J	1.9	13	0.39	B	J	
Silver	mg/kg	0.72	0.72	0.087	U	U	0.63	0.63	0.075	U	U	1.3	1.3	0.15	U	U	
Sodium	mg/kg	238	720	36	B	J	196	630	31	B	J	570	1300	63	B	J	
Thallium	mg/kg	2.9	2.9	1	U	U	0.63	0.63	0.21	U	U	6.5	6.5	4.3	U	U	
Vanadium	mg/kg	14.1	3.6	0.048			19.3	3.1	0.042			23.2	6.3	0.083			
Zinc	mg/kg	70.5	1.4	0.27	J	J	40.4	1.3	0.24	J	J	15.9	2.5	0.48	J	J	
Aroclor 1016	mg/kg	0.025	0.025	0.012	U	U	0.022	0.022	0.011	U	U	0.043	0.043	0.022	U	U	
Aroclor 1221	mg/kg	0.025	0.025	0.02	U	U	0.022	0.022	0.017	U	U	0.043	0.043	0.035	U	U	
Aroclor 1232	mg/kg	0.025	0.025	0.02	U	U	0.022	0.022	0.017	U	U	0.043	0.043	0.035	U	U	
Aroclor 1242	mg/kg	0.025	0.025	0.012	U	U	0.022	0.022	0.011	U	U	0.043	0.043	0.022	U	U	
Aroclor 1248	mg/kg	0.025	0.025	0.012	U	U	0.022	0.022	0.011	U	U	0.043	0.043	0.022	U	U	
Aroclor 1254	mg/kg	0.025	0.025	0.012	U	U	0.022	0.022	0.011	U	U	0.043	0.043	0.022	U	U	
Aroclor 1260	mg/kg	0.015	0.025	0.012	J	J	0.022	0.022	0.011	U	U	0.043	0.043	0.022	U	U	
3-Methylphenol and 4-Methylphenol	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Acenaphthene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Acenaphthylene	mg/kg	0.0516	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Anthracene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzo(a)anthracene	mg/kg	0.156	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzo(a)pyrene	mg/kg	0.168	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzo(b)fluoranthene	mg/kg	0.268	0.25	0.049			0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzo(g,h,i)perylene	mg/kg	0.0882	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzo(k)fluoranthene	mg/kg	0.0972	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Benzoic acid	mg/kg	1.2	1.2	0.49	U	U	1.1	1.1	0.43	U	U	2.1	2.1	0.85	U	U	
Benzyl alcohol	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Bis(2-chloroethoxy)methane	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Bis(2-chloroethyl)ether	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Bis(2-chloroisopropyl)ether	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Bis(2-ethylhexyl)phthalate	mg/kg	0.49	0.49	0.25	U	U	0.43	0.43	0.21	U	U	0.85	0.85	0.43	U	U	
Bromophenyl phenyl ether, 4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Butyl benzyl phthalate	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U	
Carbazole	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	
Chloro-3-methylphenol, 4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U	

Table A-1

**Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 6)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB01 AP0032 19-Aug-09 REG 0-1						ASH PIT 3-SB02 AP0036 19-Aug-09 REG 0-1						ASH PIT 3-SB03 AP0039 19-Aug-09 REG 0.2-1.7					
	Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Chloroaniline, 4-	mg/kg	0.25	0.25	0.098	U	U	0.21	0.21	0.086	U	U	0.43	0.43	0.17	U	U		
Chloronaphthalene, 2-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Chlorophenol, 2-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Chlorophenyl phenyl ether, 4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Chrysene	mg/kg	0.19	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Dibenz(a,h)anthracene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Dibenzofuran	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Dichlorobenzene, 1,2-	mg/kg	0.25	0.25	0.064	U	U	0.21	0.21	0.056	U	U	0.43	0.43	0.11	U	U		
Dichlorobenzene, 1,3-	mg/kg	0.25	0.25	0.064	U	U	0.21	0.21	0.056	U	U	0.43	0.43	0.11	U	U		
Dichlorobenzene, 1,4-	mg/kg	0.25	0.25	0.059	U	U	0.21	0.21	0.051	U	U	0.43	0.43	0.1	U	U		
Dichlorobenzidine, 3,3'-	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Dichlorophenol, 2,4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Diethyl phthalate	mg/kg	0.49	0.49	0.25	U	U	0.43	0.43	0.21	U	U	0.85	0.85	0.43	U	U		
Dimethyl phthalate	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Dimethylphenol, 2,4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Di-n-butyl phthalate	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Dinitro-2-methylphenol, 4,6-	mg/kg	0.49	0.49	0.16	U	U	0.43	0.43	0.14	U	U	0.85	0.85	0.27	U	U		
Dinitrophenol, 2,4-	mg/kg	1.2	1.2	0.49	U	U	1.1	1.1	0.43	U	U	2.1	2.1	0.85	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Di-n-octyl phthalate	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Fluoranthene	mg/kg	0.401	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Fluorene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Hexachlorobenzene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Hexachlorobutadiene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Hexachlorocyclopentadiene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Hexachloroethane	mg/kg	0.25	0.25	0.059	U	U	0.21	0.21	0.051	U	U	0.43	0.43	0.1	U	U		
Indeno(1,2,3-cd)pyrene	mg/kg	0.0914	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Isophorone	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Methylnaphthalene, 2-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Methylphenol, 2-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Naphthalene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Nitroaniline, 2-	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Nitroaniline, 3-	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Nitroaniline, 4-	mg/kg	0.49	0.49	0.098	U	U	0.43	0.43	0.086	U	U	0.85	0.85	0.17	U	U		
Nitrobenzene	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Nitrophenol, 2-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Nitrophenol, 4-	mg/kg	1.2	1.2	0.49	U	U	1.1	1.1	0.43	U	U	2.1	2.1	0.85	U	U		
n-Nitroso-di-n-propylamine	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
n-Nitrosodiphenylamine	mg/kg	0.25	0.25	0.098	U	U	0.21	0.21	0.086	U	U	0.43	0.43	0.17	U	U		
Pentachlorophenol	mg/kg	1.2	1.2	0.49	U	U	1.1	1.1	0.43	U	U	2.1	2.1	0.85	U	U		
Phenanthrene	mg/kg	0.107	0.25	0.049	J	J	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Phenol	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Pyrene	mg/kg	0.273	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Trichlorobenzene, 1,2,4-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Trichlorophenol, 2,4,5-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		
Trichlorophenol, 2,4,6-	mg/kg	0.25	0.25	0.049	U	U	0.21	0.21	0.043	U	U	0.43	0.43	0.085	U	U		

Table A-1

Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

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LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB04 AP0042 21-Aug-09 REG 0.3-0.8						ASH PIT 3-SB05 AP0045 21-Aug-09 REG 0.3-1						ASH PIT 3-SB06 AP0048 20-Aug-09 REG 0.2-1					
	Parameter	Units	Result	RL	MDL	LO	VQ	Result	RL	MDL	LO	VQ	Result	RL	MDL	LO	VQ	
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.17	0.17	0.044	U	U	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U		
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.17	0.17	0.095	U	U	0.18	0.18	0.098	U	U	0.16	0.16	0.087	U	U		
Dinitrobenzene, 1,3-	mg/kg	0.17	0.17	0.046	U	U	0.18	0.18	0.048	U	U	0.16	0.16	0.042	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.17	0.17	0.052	U	U	0.18	0.18	0.054	U	U	0.16	0.16	0.048	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.17	0.17	0.046	U	U	0.0562	0.18	0.048	J	J	0.16	0.16	0.042	U	U		
HMX	mg/kg	0.17	0.17	0.075	U	U	0.18	0.18	0.077	U	U	0.16	0.16	0.068	U	U		
Nitrobenzene	mg/kg	0.17	0.17	0.058	U	U	0.18	0.18	0.059	U	U	0.16	0.16	0.052	U	U		
Nitrotoluene, 2-	mg/kg	0.17	0.17	0.044	U	U	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U		
Nitrotoluene, 3-	mg/kg	0.17	0.17	0.073	U	U	0.18	0.18	0.075	U	U	0.16	0.16	0.067	U	U		
Nitrotoluene, 4-	mg/kg	0.17	0.17	0.054	U	U	0.18	0.18	0.056	U	U	0.16	0.16	0.049	U	U		
RDX	mg/kg	0.17	0.17	0.074	U	U	0.18	0.18	0.076	U	U	0.16	0.16	0.067	U	U		
Tetryl	mg/kg	0.17	0.17	0.045	U	U	0.18	0.18	0.046	U	U	0.16	0.16	0.04	U	U		
Trinitrobenzene, 1,3,5-	mg/kg	0.17	0.17	0.044	U	U	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U		
Trinitrotoluene, 2,4,6-	mg/kg	0.17	0.17	0.044	U	U	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U		
% Solids	Percent	33.6		0			37.9		0			63.6		0				
Aluminum	mg/kg	8240	29	1.6			9350	26	1.4			8770	16	0.86				
Antimony	mg/kg	8.8	8.8	0.66	U	U	7.8	7.8	0.58	U	U	1.2	9.3	0.7	B	J		
Arsenic	mg/kg	29	1.2	0.53			13.7	1	0.47			12.7	1.2	0.56				
Barium	mg/kg	127	29	0.74			82.2	26	0.65			203	16	0.39				
Beryllium	mg/kg	2.1	0.74	0.15			1.5	0.65	0.13			0.74	0.39	0.078				
Cadmium	mg/kg	0.3	0.59	0.15	B	J	0.52	0.52	0.13	U	U	3.1	3.1	1.6	U	U		
Calcium	mg/kg	15900	740	15			12800	650	13			2060	390	7.8				
Chromium	mg/kg	15.5	3	0.48			13.6	2.6	0.42			9.6	1.6	0.24				
Cobalt	mg/kg	8.9	7.4	0.12			7.4	6.5	0.11			4.5	3.9	0.065				
Copper	mg/kg	43.6	3.7	0.31			19.3	3.2	0.27			21.6	1.9	0.16				
Iron	mg/kg	24000	15	3.4			22800	13	3			68300	16	3.6				
Lead	mg/kg	20.4	15	0.29			12.5	13	0.26	B	J	4.5	16	0.31	B	J		
Magnesium	mg/kg	2300	740	15			1120	650	13			343	390	7.8	B	J		
Manganese	mg/kg	322	2.2	0.074			339	1.9	0.065			54.5	2.3	0.078				
Mercury	mg/kg	0.13	0.24	0.035	B	J	0.22	0.22	0.032	U	U	0.13	0.13	0.019	U	U		
Nickel	mg/kg	22.9	5.9	0.34			17.2	5.2	0.3			11.5	3.1	0.18				
Potassium	mg/kg	1060	1500	15	B	J	1110	1300	13	B	J	923	780	7.8				
Selenium	mg/kg	2.9	15	0.46	B	J	1.7	13	0.4	B	J	1.4	16	0.48	B	J		
Silver	mg/kg	1.5	1.5	0.18	U	U	1.3	1.3	0.16	U	U	0.78	0.78	0.093	U	U		
Sodium	mg/kg	510	1500	74	B	J	531	1300	65	B	J	493	780	39	B	J		
Thallium	mg/kg	6	6	2	U	U	5.2	5.2	2.2	U	U	3	1.6	0.53				
Vanadium	mg/kg	28.4	7.4	0.097			23.7	6.5	0.085			15.1	7.8	0.1				
Zinc	mg/kg	68.3	2.9	0.56			66.3	2.6	0.49			17.2	1.6	0.3				
Aroclor 1016	mg/kg	0.05	0.05	0.025	U	U	0.044	0.044	0.022	U	U	0.027	0.027	0.013	U	U		
Aroclor 1221	mg/kg	0.05	0.05	0.04	U	U	0.044	0.044	0.035	U	U	0.027	0.027	0.021	U	U		
Aroclor 1232	mg/kg	0.05	0.05	0.04	U	U	0.044	0.044	0.035	U	U	0.027	0.027	0.021	U	U		
Aroclor 1242	mg/kg	0.05	0.05	0.025	U	U	0.044	0.044	0.022	U	U	0.027	0.027	0.013	U	U		
Aroclor 1248	mg/kg	0.05	0.05	0.025	U	U	0.044	0.044	0.022	U	U	0.027	0.027	0.013	U	U		
Aroclor 1254	mg/kg	0.05	0.05	0.025	U	U	0.044	0.044	0.022	U	U	0.027	0.027	0.013	U	U		
Aroclor 1260	mg/kg	0.043	0.05	0.025	J	J	0.044	0.044	0.022	U	U	0.027	0.027	0.013	U	U		
3-Methylphenol and 4-Methylphenol	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Acenaphthene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Acenaphthylene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Anthracene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzo(a)anthracene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzo(a)pyrene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzo(b)fluoranthene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzo(ghi)perylene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzo(k)fluoranthene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Benzoic acid	mg/kg	2.5	2.5	0.99	U	U	2.2	2.2	0.87	U	U	1.3	1.3	0.52	U	U		
Benzyl alcohol	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Bis(2-chloroethoxy)methane	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Bis(2-chloroethyl)ether	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Bis(2-chloroisopropyl)ether	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Bis(2-ethylhexyl)phthalate	mg/kg	0.99	0.99	0.49	U	U	0.87	0.87	0.43	U	U	0.52	0.52	0.26	U	U		
Bromophenyl phenyl ether, 4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Butyl benzyl phthalate	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Carbazole	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Chloro-3-methylphenol, 4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		

Table A-1

**Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 4 of 6)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB04 AP0042 21-Aug-09 REG 0.3-0.8						ASH PIT 3-SB05 AP0045 21-Aug-09 REG 0.3-1						ASH PIT 3-SB06 AP0048 20-Aug-09 REG 0.2-1					
	Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Chloroaniline, 4-	mg/kg	0.49	0.49		0.2	U	0.43	0.43	0.17	U	U	0.26	0.26	0.1	U	U		
Chloronaphthalene, 2-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Chlorophenol, 2-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Chlorophenyl phenyl ether, 4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Chrysene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Dibenz(a,h)anthracene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Dibenzofuran	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Dichlorobenzene, 1,2-	mg/kg	0.49	0.49	0.13	U	U	0.43	0.43	0.11	U	U	0.26	0.26	0.068	U	U		
Dichlorobenzene, 1,3-	mg/kg	0.49	0.49	0.13	U	U	0.43	0.43	0.11	U	U	0.26	0.26	0.068	U	U		
Dichlorobenzene, 1,4-	mg/kg	0.49	0.49	0.12	U	U	0.43	0.43	0.1	U	U	0.26	0.26	0.063	U	U		
Dichlorobenzidine, 3,3'-	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Dichlorophenol, 2,4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Diethyl phthalate	mg/kg	0.99	0.99	0.49	U	U	0.87	0.87	0.43	U	U	0.52	0.52	0.26	U	U		
Dimethyl phthalate	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Dimethylphenol, 2,4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Di-n-butyl phthalate	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Dinitro-2-methylphenol, 4,6-	mg/kg	0.99	0.99	0.32	U	U	0.87	0.87	0.28	U	U	0.52	0.52	0.17	U	U		
Dinitrophenol, 2,4-	mg/kg	2.5	2.5	0.99	U	U	2.2	2.2	0.87	U	U	1.3	1.3	0.52	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Di-n-octyl phthalate	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Fluoranthene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Fluorene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Hexachlorobenzene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Hexachlorobutadiene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Hexachlorocyclopentadiene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Hexachloroethane	mg/kg	0.49	0.49	0.12	U	U	0.43	0.43	0.1	U	U	0.26	0.26	0.063	U	U		
Indeno(1,2,3-cd)pyrene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Isophorone	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Methylnaphthalene, 2-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Methylphenol, 2-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Naphthalene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Nitroaniline, 2-	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Nitroaniline, 3-	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Nitroaniline, 4-	mg/kg	0.99	0.99	0.2	U	U	0.87	0.87	0.17	U	U	0.52	0.52	0.1	U	U		
Nitrobenzene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Nitrophenol, 2-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Nitrophenol, 4-	mg/kg	2.5	2.5	0.99	U	U	2.2	2.2	0.87	U	U	1.3	1.3	0.52	U	U		
n-Nitroso-di-n-propylamine	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
n-Nitrosodiphenylamine	mg/kg	0.49	0.49	0.2	U	U	0.43	0.43	0.17	U	U	0.26	0.26	0.1	U	U		
Pentachlorophenol	mg/kg	2.5	2.5	0.99	U	U	2.2	2.2	0.87	U	U	1.3	1.3	0.52	U	U		
Phenanthrene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Phenol	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Pyrene	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Trichlorobenzene, 1,2,4-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Trichlorophenol, 2,4,5-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		
Trichlorophenol, 2,4,6-	mg/kg	0.49	0.49	0.099	U	U	0.43	0.43	0.087	U	U	0.26	0.26	0.052	U	U		

Table A-1

**Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 5 of 6)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB07 AP0051 21-Aug-09 REG 0-1						ASH PIT 3-SB08 AP0054 21-Aug-09 REG 0.3-1						ASH PIT 3-SB08 AP0056 21-Aug-09 FD 0.3-1					
	Parameter	Units	Result	RL	MDL	LO	VQ	Result	RL	MDL	LO	VQ	Result	RL	MDL	LO	VQ	
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.19	0.19	0.048	U	U	0.16	0.16	0.039	U	U	0.18	0.18	0.046	U	U		
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.19	0.19	0.1	U	U	0.16	0.16	0.086	U	U	0.18	0.18	0.1	U	U		
Dinitrobenzene, 1,3-	mg/kg	0.19	0.19	0.051	U	U	0.16	0.16	0.042	U	U	0.18	0.18	0.049	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.19	0.19	0.058	U	U	0.16	0.16	0.047	U	U	0.18	0.18	0.055	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.19	0.19	0.051	U	U	0.0547	0.16	0.042	J	J	0.0776	0.18	0.049	J	J		
HMX	mg/kg	0.19	0.19	0.083	U	U	0.16	0.16	0.068	U	U	0.18	0.18	0.079	U	U		
Nitrobenzene	mg/kg	0.19	0.19	0.063	U	U	0.16	0.16	0.052	U	U	0.18	0.18	0.061	U	U		
Nitrotoluene, 2-	mg/kg	0.19	0.19	0.048	U	U	0.16	0.16	0.039	U	U	0.18	0.18	0.046	U	U		
Nitrotoluene, 3-	mg/kg	0.19	0.19	0.081	U	U	0.16	0.16	0.066	U	U	0.18	0.18	0.077	U	U		
Nitrotoluene, 4-	mg/kg	0.19	0.19	0.06	U	U	0.16	0.16	0.049	U	U	0.18	0.18	0.057	U	U		
RDX	mg/kg	0.19	0.19	0.082	U	U	0.16	0.16	0.067	U	U	0.18	0.18	0.078	U	U		
Tetryl	mg/kg	0.19	0.19	0.049	U	U	0.16	0.16	0.04	U	U	0.18	0.18	0.047	U	U		
Trinitrobenzene, 1,3,5-	mg/kg	0.19	0.19	0.048	U	U	0.16	0.16	0.039	U	U	0.18	0.18	0.046	U	U		
Trinitrotoluene, 2,4,6-	mg/kg	0.19	0.19	0.048	U	U	0.16	0.16	0.039	U	U	0.18	0.18	0.046	U	U		
% Solids	Percent	53.1		0			37.6		0			30.8		0				
Aluminum	mg/kg	4760		18	1		7540		25	1.4		10400		32	1.8			
Antimony	mg/kg	5.5		5.5	0.42	U	7.6		7.6	0.57	U	8.88		9.7	0.73	B		
Arsenic	mg/kg	7.7		0.74	0.33		27.3		1	0.46		60.8		1.3	0.58	J		
Barium	mg/kg	38.5		18	0.46		83.5		25	0.63		110		32	0.81			
Beryllium	mg/kg	0.41		0.46	0.092	B	1.6		0.63	0.13		2		0.81	0.16	J		
Cadmium	mg/kg	0.44		0.37	0.092		0.51		0.51	0.13	U	3.3		3.3	1.6	U		
Calcium	mg/kg	14100		460	9.2		17700		630	13		14900		810	16	J		
Chromium	mg/kg	6.9		1.8	0.3		14.3		2.6	0.4		18.8		3.2	0.52			
Cobalt	mg/kg	5		4.6	0.077		7.6		6.3	0.11		8.1		8.1	0.13			
Copper	mg/kg	38.9		2.3	0.19		20.5		3.2	0.27		27.6		4.1	0.34			
Iron	mg/kg	15200		9.2	2.1		28400		13	2.9		50200		16	3.7	J		
Lead	mg/kg	12.2		9.2	0.18		6.7		13	0.25	B	9.8		16	0.32	B		
Magnesium	mg/kg	3130		460	9.2		1590		630	13		2110		810	16	J		
Manganese	mg/kg	217		1.4	0.046		280		1.9	0.063		253		2.4	0.081	J		
Mercury	mg/kg	0.066		0.14	0.02	B	0.079		0.22	0.032	B	0.071		0.24	0.034	B		
Nickel	mg/kg	13.1		3.7	0.21		17.9		5.1	0.29		20.7		6.5	0.37			
Potassium	mg/kg	634		920	9.2	B	948		1300	13	B	1280		1600	16	B		
Selenium	mg/kg	1.6		9.2	0.29	B	2.2		13	0.39	B	3.3		16	0.5	B		
Silver	mg/kg	0.92		0.92	0.11	U	1.3		1.3	0.15	U	1.6		1.6	0.19	U		
Sodium	mg/kg	168		920	46	B	470		1300	63	B	658		1600	81	B		
Thallium	mg/kg	0.92		0.92	0.31	U	1.3		1.3	0.43	U	1.6		1.6	0.55	U		
Vanadium	mg/kg	10.7		4.6	0.061		24.3		6.3	0.084		33.5		8.1	0.11			
Zinc	mg/kg	78.7		1.8	0.35		27.2		2.5	0.48		40.8		3.2	0.62			
Aroclor 1016	mg/kg	0.031		0.031	0.016	U	0.045		0.045	0.023	U	0.054		0.054	0.027	U		
Aroclor 1221	mg/kg	0.031		0.031	0.025	U	0.045		0.045	0.036	U	0.054		0.054	0.043	U		
Aroclor 1232	mg/kg	0.031		0.031	0.025	U	0.045		0.045	0.036	U	0.054		0.054	0.043	U		
Aroclor 1242	mg/kg	0.031		0.031	0.016	U	0.045		0.045	0.023	U	0.054		0.054	0.027	U		
Aroclor 1248	mg/kg	0.031		0.031	0.016	U	0.045		0.045	0.023	U	0.054		0.054	0.027	U		
Aroclor 1254	mg/kg	0.031		0.031	0.016	U	0.045		0.045	0.023	U	0.054		0.054	0.027	U		
Aroclor 1260	mg/kg	0.031		0.031	0.016	U	0.045		0.045	0.023	U	0.054		0.054	0.027	U		
3-Methylphenol and 4-Methylphenol	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Acenaphthene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Acenaphthylene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Anthracene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzo(a)anthracene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzo(a)pyrene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzo(b)fluoranthene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzo(ghi)perylene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzo(k)fluoranthene	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Benzoic acid	mg/kg	1.6		1.6	0.63	U	2.2		2.2	0.88	U	2.8		2.8	1.1	U		
Benzyl alcohol	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Bis(2-chloroethoxy)methane	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Bis(2-chloroethyl)ether	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Bis(2-chloroisopropyl)ether	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Bis(2-ethylhexyl)phthalate	mg/kg	0.63		0.63	0.31	U	0.88		0.88	0.44	U	1.1		1.1	0.55	U		
Bromophenyl phenyl ether, 4-	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Butyl benzyl phthalate	mg/kg	0.63		0.63	0.13	U	0.88		0.88	0.18	U	1.1		1.1	0.22	U		
Carbazole	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		
Chloro-3-methylphenol, 4-	mg/kg	0.31		0.31	0.063	U	0.44		0.44	0.088	U	0.55		0.55	0.11	U		

Table A-1

**Surface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB07 AP0051 21-Aug-09 REG 0-1						ASH PIT 3-SB08 AP0054 21-Aug-09 REG 0.3-1						ASH PIT 3-SB08 AP0056 21-Aug-09 FD 0.3-1					
	Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Chloroaniline, 4-	mg/kg	0.31	0.31	0.13	U	U	0.44	0.44	0.18	U	U	0.55	0.55	0.22	U	U		
Chloronaphthalene, 2-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Chlorophenol, 2-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Chlorophenyl phenyl ether, 4-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Chrysene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Dibenz(a,h)anthracene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Dibenzofuran	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Dichlorobenzene, 1,2-	mg/kg	0.31	0.31	0.081	U	U	0.44	0.44	0.11	U	U	0.55	0.55	0.14	U	U		
Dichlorobenzene, 1,3-	mg/kg	0.31	0.31	0.081	U	U	0.44	0.44	0.11	U	U	0.55	0.55	0.14	U	U		
Dichlorobenzene, 1,4-	mg/kg	0.31	0.31	0.075	U	U	0.44	0.44	0.11	U	U	0.55	0.55	0.13	U	U		
Dichlorobenzidine, 3,3'-	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Dichlorophenol, 2,4-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Diethyl phthalate	mg/kg	0.63	0.63	0.31	U	U	0.88	0.88	0.44	U	U	1.1	1.1	0.55	U	U		
Dimethyl phthalate	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Dimethylphenol, 2,4-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Di-n-butyl phthalate	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Dinitro-2-methylphenol, 4,6-	mg/kg	0.63	0.63	0.2	U	U	0.88	0.88	0.28	U	U	1.1	1.1	0.35	U	U		
Dinitrophenol, 2,4-	mg/kg	1.6	1.6	0.63	U	U	2.2	2.2	0.88	U	U	2.8	2.8	1.1	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Di-n-octyl phthalate	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Fluoranthene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Fluorene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Hexachlorobenzene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Hexachlorobutadiene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Hexachlorocyclopentadiene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Hexachloroethane	mg/kg	0.31	0.31	0.075	U	U	0.44	0.44	0.11	U	U	0.55	0.55	0.13	U	U		
Indeno(1,2,3-cd)pyrene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Isophorone	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Methylnaphthalene, 2-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Methylphenol, 2-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Naphthalene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Nitroaniline, 2-	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Nitroaniline, 3-	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Nitroaniline, 4-	mg/kg	0.63	0.63	0.13	U	U	0.88	0.88	0.18	U	U	1.1	1.1	0.22	U	U		
Nitrobenzene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Nitrophenol, 2-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Nitrophenol, 4-	mg/kg	1.6	1.6	0.63	U	U	2.2	2.2	0.88	U	U	2.8	2.8	1.1	U	U		
n-Nitroso-di-n-propylamine	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
n-Nitrosodiphenylamine	mg/kg	0.31	0.31	0.13	U	U	0.44	0.44	0.18	U	U	0.55	0.55	0.22	U	U		
Pentachlorophenol	mg/kg	1.6	1.6	0.63	U	U	2.2	2.2	0.88	U	U	2.8	2.8	1.1	U	U		
Phenanthrene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Phenol	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Pyrene	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Trichlorobenzene, 1,2,4-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Trichlorophenol, 2,4,5-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		
Trichlorophenol, 2,4,6-	mg/kg	0.31	0.31	0.063	U	U	0.44	0.44	0.088	U	U	0.55	0.55	0.11	U	U		

FD - Field duplicate.
LQ - Laboratory qualifier.
MDL - Method detection limit.
mg/kg - Milligram(s) per kilogram.
REG - Regular sample.
RL - Reporting limit.
VQ - Validation qualifier.

Validation Qualifiers:
U - Nondetect.
J - Estimated Concentration.
UJ - Nondetected; detection limit is estimated.
B - Analyte also found in blank.
R - Rejected.

Table A-2

**Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 6)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB01 AP0033 19-Aug-09 REG 3-5						ASH PIT 3-SB02 AP0037 19-Aug-09 REG 1.2-2.3						ASH PIT 3-SB04 AP0043 21-Aug-09 REG 0.8-1.5					
	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.15	0.15	0.038	U	U	0.18	0.18	0.044	U	U	0.16	0.16	0.039	U	U		
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.15	0.15	0.083	U	U	0.18	0.18	0.096	U	U	0.16	0.16	0.086	U	U		
Dinitrobenzene, 1,3-	mg/kg	0.15	0.15	0.04	U	U	0.18	0.18	0.047	U	U	0.16	0.16	0.042	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.15	0.15	0.04	U	U	0.18	0.18	0.053	U	U	0.16	0.16	0.047	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.15	0.15	0.04	U	U	0.0512	0.18	0.047	J	J	0.16	0.16	0.042	U	U		
HMX	mg/kg	0.15	0.15	0.066	U	U	0.18	0.18	0.076	U	U	0.16	0.16	0.068	U	U		
Nitrobenzene	mg/kg	0.15	0.15	0.05	U	U	0.18	0.18	0.058	U	U	0.16	0.16	0.052	U	U		
Nitrotoluene, 2-	mg/kg	0.15	0.15	0.038	U	U	0.18	0.18	0.044	U	U	0.16	0.16	0.039	U	U		
Nitrotoluene, 3-	mg/kg	0.15	0.15	0.064	U	U	0.18	0.18	0.074	U	U	0.16	0.16	0.066	U	U		
Nitrotoluene, 4-	mg/kg	0.15	0.15	0.047	U	U	0.18	0.18	0.055	U	U	0.16	0.16	0.049	U	U		
RDX	mg/kg	0.15	0.15	0.065	U	U	0.18	0.18	0.075	U	U	0.16	0.16	0.067	U	U		
Tetryl	mg/kg	0.15	0.15	0.039	U	U	0.18	0.18	0.045	U	U	0.16	0.16	0.04	U	U		
Trinitrobenzene, 1,3,5-	mg/kg	0.15	0.15	0.038	U	U	0.18	0.18	0.044	U	U	0.16	0.16	0.039	U	U		
Trinitrotoluene, 2,4,6-	mg/kg	0.15	0.15	0.038	U	U	0.18	0.18	0.044	U	U	0.16	0.16	0.039	U	U		
% Solids	Percent	80.6	0				36.4	0				81.5	0					
Aluminum	mg/kg	7010	12	0.68	J	J	10400	27	1.5	J	J	4870	12	0.64				
Antimony	mg/kg	7.4	7.4	0.56	U	UJ	8.1	8.1	0.61	U	UJ	7	7	0.53	U	U		
Arsenic	mg/kg	5	0.99	0.45			12.9	1.1	0.48			8.5	0.93	0.42				
Barium	mg/kg	41.2	12	0.31			85.7	27	0.67			41.5	12	0.29				
Beryllium	mg/kg	0.5	0.31	0.062			1.5	0.67	0.13			0.4	0.29	0.058				
Cadmium	mg/kg	0.18	0.25	0.062	B	J	0.54	0.54	0.13	U	U	0.2	0.23	0.058	B	J		
Calcium	mg/kg	49300	620	12	J	J	9690	670	13	J	J	47500	580	12				
Chromium	mg/kg	10.3	1.2	0.198	J	J	14.9	2.6	0.44	J	J	7.7	1.2	0.186				
Cobalt	mg/kg	6.7	3.1	0.051	J	J	6.3	6.7	0.11	B	J	5.9	2.9	0.048				
Copper	mg/kg	18.4	1.6	0.13			16.2	3.4	0.28			15	1.5	0.12				
Iron	mg/kg	14500	6.2	1.4	J	J	24700	13	3.1	J	J	11200	5.8	1.3				
Lead	mg/kg	9.3	12	0.25	B	J	3.4	13	0.27	B	J	8.1	12	0.23	B	J		
Magnesium	mg/kg	16600	310	6.2	J	J	891	670	13	J	J	14900	290	5.8				
Manganese	mg/kg	410	1.9	0.062	J	J	120	2	0.067	J	J	362	1.8	0.058				
Mercury	mg/kg	0.015	0.091	0.013	B	J	0.22	0.22	0.032	U	U	0.014	0.09	0.013	B	J		
Nickel	mg/kg	17.1	2.5	0.14			13.4	5.4	0.31			14.3	2.3	0.13				
Potassium	mg/kg	1970	620	6.2	J	J	1570	1300	13	J	J	1290	580	5.8				
Selenium	mg/kg	0.84	12	0.38	B	J	2.2	13	0.42	B	J	1.1	12	0.36	B	J		
Silver	mg/kg	0.62	0.62	0.074	U	UJ	1.3	1.3	0.16	U	UJ	0.58	0.58	0.07	U	U		
Sodium	mg/kg	293	620	31	B	J	640	1300	67	B	J	224	580	29	B	J		
Thallium	mg/kg	0.62	0.62	0.21	U	U	6.5	6.5	4.6	U	U	0.58	0.58	0.2	U	U		
Vanadium	mg/kg	16.3	3.1	0.041			24.3	6.7	0.089			13.3	2.9	0.039				
Zinc	mg/kg	44.7	1.2	0.24		J	30.5	2.7	0.51		J	37	1.2	0.22				
Aroclor 1016	mg/kg	0.021	0.021	0.01	U	U	0.047	0.047	0.023	U	U	0.021	0.021	0.01	U	U		
Aroclor 1221	mg/kg	0.021	0.021	0.017	U	U	0.047	0.047	0.037	U	U	0.021	0.021	0.017	U	U		
Aroclor 1232	mg/kg	0.021	0.021	0.017	U	U	0.047	0.047	0.037	U	U	0.021	0.021	0.017	U	U		
Aroclor 1242	mg/kg	0.021	0.021	0.01	U	U	0.047	0.047	0.023	U	U	0.021	0.021	0.01	U	U		
Aroclor 1248	mg/kg	0.021	0.021	0.01	U	U	0.047	0.047	0.023	U	U	0.021	0.021	0.01	U	U		
Aroclor 1254	mg/kg	0.021	0.021	0.01	U	U	0.047	0.047	0.023	U	U	0.021	0.021	0.01	U	U		
Aroclor 1260	mg/kg	0.021	0.021	0.01	U	U	0.047	0.047	0.023	U	U	0.021	0.021	0.01	U	U		
3-Methylphenol and 4-Methylphenol	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Acenaphthene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Acenaphthylene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Anthracene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzo(a)anthracene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzo(a)pyrene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzo(b)fluoranthene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzo(ghi)perylene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzo(k)fluoranthene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Benzoic acid	mg/kg	1	1	0.41	U	U	2.3	2.3	0.92	U	U	1	1	0.41	U	U		
Benzyl alcohol	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Bis(2-chloroethoxy)methane	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Bis(2-chloroethyl)ether	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Bis(2-chloroisopropyl)ether	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		
Bis(2-ethylhexyl)phthalate	mg/kg	0.41	0.41	0.2	U	U	0.92	0.92	0.46	U	U	0.41	0.41	0.2	U	U		
Bromophenyl phenyl ether, 4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U		

Table A-2

**Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 2 of 6)

Parameter	UNITS	ASH PIT 3-SB01						ASH PIT 3-SB02						ASH PIT 3-SB04					
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
Butyl benzyl phthalate	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Carbazole	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Chloro-3-methylphenol, 4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Chloroaniline, 4-	mg/kg	0.2	0.2	0.082	U	U	0.46	0.46	0.18	U	U	0.2	0.2	0.081	U	U			
Chloronaphthalene, 2-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Chlorophenol, 2-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Chlorophenyl phenyl ether, 4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Chrysene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Dibenz(a,h)anthracene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Dibenzofuran	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Dichlorobenzene, 1,2-	mg/kg	0.2	0.2	0.053	U	U	0.46	0.46	0.12	U	U	0.2	0.2	0.053	U	U			
Dichlorobenzene, 1,3-	mg/kg	0.2	0.2	0.053	U	U	0.46	0.46	0.12	U	U	0.2	0.2	0.053	U	U			
Dichlorobenzene, 1,4-	mg/kg	0.2	0.2	0.049	U	U	0.46	0.46	0.11	U	U	0.2	0.2	0.049	U	U			
Dichlorobenzidine, 3,3'-	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Dichlorophenol, 2,4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Diethyl phthalate	mg/kg	0.41	0.41	0.2	U	U	0.92	0.92	0.46	U	U	0.41	0.41	0.2	U	U			
Dimethyl phthalate	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Dimethylphenol, 2,4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Di-n-butyl phthalate	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Dinitro-2-methylphenol, 4,6-	mg/kg	0.41	0.41	0.13	U	U	0.92	0.92	0.29	U	U	0.41	0.41	0.13	U	U			
Dinitrophenol, 2,4-	mg/kg	1	1	0.41	U	U	2.3	2.3	0.92	U	U	1	1	0.41	U	U			
Dinitrotoluene, 2,4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Dinitrotoluene, 2,6-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Di-n-octyl phthalate	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Fluoranthene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Fluorene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Hexachlorobenzene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Hexachlorobutadiene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Hexachlorocyclopentadiene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Hexachloroethane	mg/kg	0.2	0.2	0.049	U	U	0.46	0.46	0.11	U	U	0.2	0.2	0.049	U	U			
Indeno(1,2,3-cd)pyrene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Isophorone	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Methylnaphthalene, 2-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Methylphenol, 2-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Naphthalene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Nitroaniline, 2-	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Nitroaniline, 3-	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Nitroaniline, 4-	mg/kg	0.41	0.41	0.082	U	U	0.92	0.92	0.18	U	U	0.41	0.41	0.081	U	U			
Nitrobenzene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Nitrophenol, 2-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Nitrophenol, 4-	mg/kg	1	1	0.41	U	U	2.3	2.3	0.92	U	U	1	1	0.41	U	U			
n-Nitroso-di-n-propylamine	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
n-Nitrosodiphenylamine	mg/kg	0.2	0.2	0.082	U	U	0.46	0.46	0.18	U	U	0.2	0.2	0.081	U	U			
Pentachlorophenol	mg/kg	1	1	0.41	U	U	2.3	2.3	0.92	U	U	1	1	0.41	U	U			
Phenanthrene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Phenol	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Pyrene	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Trichlorobenzene, 1,2,4-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Trichlorophenol, 2,4,5-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			
Trichlorophenol, 2,4,6-	mg/kg	0.2	0.2	0.041	U	U	0.46	0.46	0.092	U	U	0.2	0.2	0.041	U	U			

Table A-2

Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 3 of 6)

LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	SAMPLE_PURPOSE	DEPTH	ASH PIT 3-SB05 AP0046 21-Aug-09 REG 1-2						ASH PIT 3-SB06 AP0061A 20-Aug-09 REG 2-2.7						ASH PIT 3-SB07 AP0052 21-Aug-09 REG 1-2					
Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ						
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.18	0.18	0.044	U	U	0.16	0.16	0.04	U	U	0.17	0.17	0.043	U	U						
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.18	0.18	0.096	U	U	0.16	0.16	0.088	U	U	0.17	0.17	0.094	U	U						
Dinitrobenzene, 1,3-	mg/kg	0.18	0.18	0.046	U	U	0.16	0.16	0.043	U	U	0.17	0.17	0.046	U	U						
Dinitrotoluene, 2,4-	mg/kg	0.18	0.18	0.053	U	U	0.16	0.16	0.048	U	U	0.17	0.17	0.052	U	U						
Dinitrotoluene, 2,6-	mg/kg	0.18	0.18	0.046	U	U	0.16	0.16	0.043	U	U	0.17	0.17	0.046	U	U						
HMX	mg/kg	0.18	0.18	0.075	U	U	0.16	0.16	0.069	U	U	0.17	0.17	0.074	U	U						
Nitrobenzene	mg/kg	0.18	0.18	0.058	U	U	0.16	0.16	0.053	U	U	0.17	0.17	0.057	U	U						
Nitrotoluene, 2-	mg/kg	0.18	0.18	0.044	U	U	0.16	0.16	0.04	U	U	0.17	0.17	0.043	U	U						
Nitrotoluene, 3-	mg/kg	0.18	0.18	0.074	U	U	0.16	0.16	0.067	U	U	0.17	0.17	0.073	U	U						
Nitrotoluene, 4-	mg/kg	0.18	0.18	0.054	U	U	0.16	0.16	0.05	U	U	0.17	0.17	0.054	U	U						
RDX	mg/kg	0.18	0.18	0.075	U	U	0.16	0.16	0.068	U	U	0.17	0.17	0.074	U	U						
Tetryl	mg/kg	0.18	0.18	0.045	U	U	0.16	0.16	0.041	U	U	0.17	0.17	0.044	U	U						
Trinitrobenzene, 1,3,5-	mg/kg	0.18	0.18	0.044	U	U	0.16	0.16	0.04	U	U	0.17	0.17	0.043	U	U						
Trinitrotoluene, 2,4,6-	mg/kg	0.18	0.18	0.044	U	U	0.16	0.16	0.04	U	U	0.17	0.17	0.043	U	U						
% Solids	Percent	78.3	0				79.2	0				78.2	0									
Aluminum	mg/kg	6010	12	0.68			7840	12	0.67			9910	13	0.7								
Antimony	mg/kg	0.67	7.4	0.56	B	J	0.93	7.3	0.55	B	J	0.72	7.6	0.57	B	J						
Arsenic	mg/kg	20.3	0.99	0.45			30.6	0.97	0.44			9.3	1	0.46								
Barium	mg/kg	63.7	12	0.31			65.1	12	0.3			70.5	13	0.32								
Beryllium	mg/kg	0.47	0.31	0.062			0.56	0.3	0.061			0.67	0.32	0.063								
Cadmium	mg/kg	0.21	0.25	0.062	B	J	0.17	0.24	0.061	B	J	0.21	0.25	0.063	B	J						
Calcium	mg/kg	52800	620	12			46100	610	12			42400	630	13								
Chromium	mg/kg	9.6	1.2	0.198			11.8	1.2	0.194			15	1.3	0.2								
Cobalt	mg/kg	5.8	3.1	0.051			8.5	3	0.05			8.1	3.2	0.053								
Copper	mg/kg	17.5	1.5	0.13			17.6	1.5	0.13			21.7	1.6	0.13								
Iron	mg/kg	16700	6.2	1.4			20500	6.1	1.4			17700	6.3	1.5								
Lead	mg/kg	9.6	12	0.25	B	J	10	12	0.24	B	J	11.9	13	0.25	B	J						
Magnesium	mg/kg	17500	310	6.2			15700	300	6.1			15800	320	6.3								
Manganese	mg/kg	432	1.9	0.062			402	1.8	0.061			477	1.9	0.063								
Mercury	mg/kg	0.03	0.1	0.015	B	J	0.016	0.11	0.015	B	J	0.026	0.11	0.015	B	J						
Nickel	mg/kg	16.4	2.5	0.14			18.8	2.4	0.14			22.7	2.5	0.15								
Potassium	mg/kg	1490	620	6.2			1900	610	6.1			2110	630	6.3								
Selenium	mg/kg	1.1	12	0.38	B	J	2.1	12	0.38	B	J	0.9	13	0.39	B	J						
Silver	mg/kg	0.62	0.62	0.074	U	U	0.61	0.61	0.073	U	U	0.63	0.63	0.076	U	U						
Sodium	mg/kg	261	620	31	B	J	300	610	30	B	J	269	630	32	B	J						
Thallium	mg/kg	2.5	2.5	1.05	U	U	0.61	0.61	0.21	U	U	0.63	0.63	0.22	U	U						
Vanadium	mg/kg	15.5	3.1	0.041			17.7	3	0.04			21.9	3.2	0.042								
Zinc	mg/kg	41.6	1.2	0.24			49.3	1.2	0.23			53.2	1.3	0.24								
Aroclor 1016	mg/kg	0.022	0.022	0.011	U	U	0.021	0.021	0.011	U	U	0.021	0.021	0.011	U	U						
Aroclor 1221	mg/kg	0.022	0.022	0.017	U	U	0.021	0.021	0.017	U	U	0.021	0.021	0.017	U	U						
Aroclor 1232	mg/kg	0.022	0.022	0.017	U	U	0.021	0.021	0.017	U	U	0.021	0.021	0.017	U	U						
Aroclor 1242	mg/kg	0.022	0.022	0.011	U	U	0.021	0.021	0.011	U	U	0.021	0.021	0.011	U	U						
Aroclor 1248	mg/kg	0.022	0.022	0.011	U	U	0.021	0.021	0.011	U	U	0.021	0.021	0.011	U	U						
Aroclor 1254	mg/kg	0.022	0.022	0.011	U	U	0.021	0.021	0.011	U	U	0.021	0.021	0.011	U	U						
Aroclor 1260	mg/kg	0.022	0.022	0.011	U	U	0.021	0.021	0.011	U	U	0.021	0.021	0.011	U	U						
3-Methylphenol and 4-Methylphenol	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Acenaphthene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Acenaphthylene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Anthracene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzo(a)anthracene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzo(a)pyrene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzo(b)fluoranthene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzo(ghi)perylene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzo(k)fluoranthene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Benzoic acid	mg/kg	1	1	0.42	U	U	1	1	0.42	U	U	1.1	1.1	0.42	U	U						
Benzyl alcohol	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Bis(2-chloroethoxy)methane	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Bis(2-chloroethyl)ether	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Bis(2-chloroisopropyl)ether	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						
Bis(2-ethylhexyl)phthalate	mg/kg	0.42	0.42	0.21	U	U	0.42	0.42	0.21	U	U	0.42	0.42	0.21	U	U						
Bromophenyl phenyl ether, 4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U						

Table A-2

**Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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Parameter	Units	ASH PIT 3-SB05 AP0046 21-Aug-09 REG 1-2						ASH PIT 3-SB06 AP0061A 20-Aug-09 REG 2-2.7						ASH PIT 3-SB07 AP0052 21-Aug-09 REG 1-2					
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
Butyl benzyl phthalate	mg/kg	0.42	0.42	0.084	U	U	0.42	0.21	0.084	U	U	0.42	0.42	0.084	U	U			
Carbazole	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Chloro-3-methylphenol, 4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Chloroaniline, 4-	mg/kg	0.21	0.21	0.084	U	U	0.21	0.21	0.084	U	U	0.21	0.21	0.084	U	U			
Chloronaphthalene, 2-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Chlorophenol, 2-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Chlorophenyl phenyl ether, 4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Chrysene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Dibenz(a,h)anthracene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Dibenzofuran	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Dichlorobenzene, 1,2-	mg/kg	0.21	0.21	0.054	U	U	0.21	0.21	0.054	U	U	0.21	0.21	0.055	U	U			
Dichlorobenzene, 1,3-	mg/kg	0.21	0.21	0.054	U	U	0.21	0.21	0.054	U	U	0.21	0.21	0.055	U	U			
Dichlorobenzene, 1,4-	mg/kg	0.21	0.21	0.05	U	U	0.21	0.21	0.05	U	U	0.21	0.21	0.05	U	U			
Dichlorobenzidine, 3,3'-	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Dichlorophenol, 2,4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Diethyl phthalate	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Dimethyl phthalate	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Dimethylphenol, 2,4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Di-n-butyl phthalate	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Dinitro-2-methylphenol, 4,6-	mg/kg	0.42	0.42	0.13	U	U	0.42	0.42	0.13	U	U	0.42	0.42	0.13	U	U			
Dinitrophenol, 2,4-	mg/kg	1	1	0.42	U	U	1	1	0.42	U	U	1.1	1.1	0.42	U	U			
Dinitrotoluene, 2,4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Dinitrotoluene, 2,6-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Di-n-octyl phthalate	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Fluoranthene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Fluorene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Hexachlorobenzene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Hexachlorobutadiene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Hexachlorocyclopentadiene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Hexachloroethane	mg/kg	0.21	0.21	0.05	U	U	0.21	0.21	0.05	U	U	0.21	0.21	0.05	U	U			
Indeno(1,2,3-cd)pyrene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Isophorone	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Methylnaphthalene, 2-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Methylphenol, 2-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Naphthalene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Nitroaniline, 2-	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Nitroaniline, 3-	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Nitroaniline, 4-	mg/kg	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U	0.42	0.42	0.084	U	U			
Nitrobenzene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Nitrophenol, 2-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Nitrophenol, 4-	mg/kg	1	1	0.42	U	U	1	1	0.42	U	U	1.1	1.1	0.42	U	U			
n-Nitroso-di-n-propylamine	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
n-Nitrosodiphenylamine	mg/kg	0.21	0.21	0.084	U	U	0.21	0.21	0.084	U	U	0.21	0.21	0.084	U	U			
Pentachlorophenol	mg/kg	1	1	0.42	U	U	1	1	0.42	U	U	1.1	1.1	0.42	U	U			
Phenanthrene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Phenol	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Pyrene	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Trichlorobenzene, 1,2,4-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Trichlorophenol, 2,4,5-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			
Trichlorophenol, 2,4,6-	mg/kg	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U	0.21	0.21	0.042	U	U			

Table A-2

**Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

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Parameter	LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE DEPTH	ASH PIT 3-SB07 AP0059 21-Aug-09 FD 1-2						ASH PIT 3-SB08 AP0055 21-Aug-09 REG 1-2					
		Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Amino-2,6-dinitrotoluene, 4-		mg/kg	0.15	0.15	0.038	U	U	0.14	0.14	0.036	U	U	
Amino-4,6-dinitrotoluene, 2-		mg/kg	0.15	0.15	0.083	U	U	0.14	0.14	0.079	U	U	
Dinitrobenzene, 1,3-		mg/kg	0.15	0.15	0.04	U	U	0.14	0.14	0.038	U	U	
Dinitrotoluene, 2,4-		mg/kg	0.15	0.15	0.045	U	U	0.14	0.14	0.043	U	U	
Dinitrotoluene, 2,6-		mg/kg	0.15	0.15	0.04	U	U	0.14	0.14	0.038	U	U	
HMX		mg/kg	0.15	0.15	0.065	U	U	0.14	0.14	0.062	U	U	
Nitrobenzene		mg/kg	0.15	0.15	0.05	U	U	0.14	0.14	0.048	U	U	
Nitrotoluene, 2-		mg/kg	0.15	0.15	0.038	U	U	0.14	0.14	0.036	U	U	
Nitrotoluene, 3-		mg/kg	0.15	0.15	0.064	U	U	0.14	0.14	0.061	U	U	
Nitrotoluene, 4-		mg/kg	0.15	0.15	0.047	U	U	0.14	0.14	0.045	U	U	
RDX		mg/kg	0.15	0.15	0.064	U	U	0.14	0.14	0.061	U	U	
Tetryl		mg/kg	0.15	0.15	0.039	U	U	0.14	0.14	0.037	U	U	
Trinitrobenzene, 1,3,5-		mg/kg	0.15	0.15	0.038	U	U	0.14	0.14	0.036	U	U	
Trinitrotoluene, 2,4,6-		mg/kg	0.15	0.15	0.038	U	U	0.14	0.14	0.036	U	U	
% Solids		Percent	76.5	0				80.6	0				
Aluminum		mg/kg	9620	13	0.7			8970	12	0.65			
Antimony		mg/kg	7.6	7.6	0.57	U	U	0.5	3.5	0.27	B	J	
Arsenic		mg/kg	6.9	1	0.46			32.7	0.47	0.21		J	
Barium		mg/kg	70.1	13	0.32			98.4	12	0.3			
Beryllium		mg/kg	0.63	0.32	0.063			0.64	0.3	0.059		J	
Cadmium		mg/kg	0.19	0.25	0.063	B	J	0.14	0.24	0.059	B	J	
Calcium		mg/kg	47800	630	13			28000	300	5.9		J	
Chromium		mg/kg	14.5	1.3	0.2			12.6	1.2	0.19			
Cobalt		mg/kg	7.3	3.2	0.053			12.2	3	0.049			
Copper		mg/kg	19.4	1.6	0.13			20.9	1.5	0.12			
Iron		mg/kg	14900	6.3	1.5			28300	5.9	1.4		J	
Lead		mg/kg	11.2	13	0.25	B	J	8.6	5.9	0.12			
Magnesium		mg/kg	17500	320	6.3			12400	300	5.9		J	
Manganese		mg/kg	460	1.9	0.063			1270	8.9	0.3		J	
Mercury		mg/kg	0.022	0.1	0.015	B	J	0.029	0.1	0.014	B	J	
Nickel		mg/kg	21.4	2.5	0.15			31.9	2.4	0.14			
Potassium		mg/kg	2110	630	6.3			1400	590	5.9			
Selenium		mg/kg	0.98	13	0.39	B	J	0.66	5.9	0.18	B	J	
Silver		mg/kg	0.63	0.63	0.076	U	U	0.59	0.59	0.071	U	U	
Sodium		mg/kg	280	630	32	B	J	324	590	30	B	J	
Thallium		mg/kg	0.63	0.63	0.22	U	U	0.5	0.59	0.2	B	J	
Vanadium		mg/kg	21.1	3.2	0.042			20.3	3	0.039			
Zinc		mg/kg	49.5	1.3	0.24			50.6	1.2	0.22			
Aroclor 1016		mg/kg	0.021	0.021	0.011	U	U	0.021	0.021	0.01	U	U	
Aroclor 1221		mg/kg	0.021	0.021	0.017	U	U	0.021	0.021	0.017	U	U	
Aroclor 1232		mg/kg	0.021	0.021	0.017	U	U	0.021	0.021	0.017	U	U	
Aroclor 1242		mg/kg	0.021	0.021	0.011	U	U	0.021	0.021	0.01	U	U	
Aroclor 1248		mg/kg	0.021	0.021	0.011	U	U	0.021	0.021	0.01	U	U	
Aroclor 1254		mg/kg	0.021	0.021	0.011	U	U	0.021	0.021	0.01	U	U	
Aroclor 1260		mg/kg	0.021	0.021	0.011	U	U	0.021	0.021	0.01	U	U	
3-Methylphenol and 4-Methylphenol		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Acenaphthene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Acenaphthylene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Anthracene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzo(a)anthracene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzo(a)pyrene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzo(b)fluoranthene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzo(ghi)perylene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzo(k)fluoranthene		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Benzoic acid		mg/kg	1.1	1.1	0.43	U	U	1	1	0.42	U	U	
Benzyl alcohol		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Bis(2-chloroethoxy)methane		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Bis(2-chloroethyl)ether		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Bis(2-chloroisopropyl)ether		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	
Bis(2-ethylhexyl)phthalate		mg/kg	0.43	0.43	0.22	U	U	0.42	0.42	0.21	U	U	
Bromophenyl phenyl ether, 4-		mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U	

Table A-2

**Subsurface Soil Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 6 of 6)

Parameter	UNITS	ASH PIT 3-SB07 AP0059 21-Aug-09 FD 1-2						ASH PIT 3-SB08 AP0055 21-Aug-09 REG 1-2					
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Butyl benzyl phthalate	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Carbazole	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Chloro-3-methylphenol, 4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Chloroaniline, 4-	mg/kg	0.22	0.22	0.086	U	U	0.21	0.21	0.084	U	U		
Chloronaphthalene, 2-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Chlorophenol, 2-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Chlorophenyl phenyl ether, 4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Chrysene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Dibenz(a,h)anthracene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Dibenzofuran	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Dichlorobenzene, 1,2-	mg/kg	0.22	0.22	0.056	U	U	0.21	0.21	0.054	U	U		
Dichlorobenzene, 1,3-	mg/kg	0.22	0.22	0.056	U	U	0.21	0.21	0.054	U	U		
Dichlorobenzene, 1,4-	mg/kg	0.22	0.22	0.052	U	U	0.21	0.21	0.05	U	U		
Dichlorobenzidine, 3,3'-	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Dichlorophenol, 2,4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Diethyl phthalate	mg/kg	0.43	0.43	0.22	U	U	0.42	0.42	0.21	U	U		
Dimethyl phthalate	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Dimethylphenol, 2,4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Di-n-butyl phthalate	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Dinitro-2-methylphenol, 4,6-	mg/kg	0.43	0.43	0.14	U	U	0.42	0.42	0.13	U	U		
Dinitrophenol, 2,4-	mg/kg	1.1	1.1	0.43	U	U	1	1	0.42	U	U		
Dinitrotoluene, 2,4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Dinitrotoluene, 2,6-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Di-n-octyl phthalate	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Fluoranthene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Fluorene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Hexachlorobenzene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Hexachlorobutadiene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Hexachlorocyclopentadiene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Hexachloroethane	mg/kg	0.22	0.22	0.052	U	U	0.21	0.21	0.05	U	U		
Indeno(1,2,3-cd)pyrene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Isophorone	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Methylnaphthalene, 2-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Methylphenol, 2-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Naphthalene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Nitroaniline, 2-	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Nitroaniline, 3-	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Nitroaniline, 4-	mg/kg	0.43	0.43	0.086	U	U	0.42	0.42	0.084	U	U		
Nitrobenzene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Nitrophenol, 2-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Nitrophenol, 4-	mg/kg	1.1	1.1	0.43	U	U	1	1	0.42	U	U		
n-Nitroso-di-n-propylamine	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
n-Nitrosodiphenylamine	mg/kg	0.22	0.22	0.086	U	U	0.21	0.21	0.084	U	U		
Pentachlorophenol	mg/kg	1.1	1.1	0.43	U	U	1	1	0.42	U	U		
Phenanthrene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Phenol	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Pyrene	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Trichlorobenzene, 1,2,4-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Trichlorophenol, 2,4,5-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		
Trichlorophenol, 2,4,6-	mg/kg	0.22	0.22	0.043	U	U	0.21	0.21	0.042	U	U		

FD - Field duplicate.
LQ - Laboratory qualifier.
MDL - Method detection limit.
mg/kg - Milligram(s) per kilogram.
REG - Regular sample.
RL - Reporting limit.
VQ - Validation qualifier.

Validation Qualifiers:
U - Nondetect.
J - Estimated Concentration.
UJ - Nondetected; detection limit is estimated.
B - Analyte also found in blank.
R - Rejected.

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works
 Sandusky, Ohio

(Page 1 of 12)

LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE	AP3-MW01 AP3083 19-Dec-11 1_3 - 1_3 Ft REG					AP3-MW01 AP3084 19-Dec-11 1_3 - 1_3 Ft FD					AP3-MW01 AP3085 19-Dec-11 1_3 - 1_3 Ft FS				
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Amino-2,6-dinitrotoluene, 4-	N	µg/L	0.2	0.2	0.082	U	U	0.2	0.2	0.08	U	U	0.05	0.1	0.05	U	U		
Amino-4,6-dinitrotoluene, 2-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.1	0.2	0.1	U	U		
Dinitrobenzene, 1,3-	N	µg/L	0.2	0.2	0.097	U	U	0.2	0.2	0.095	U	U	0.05	0.1	0.05	U	U		
Dinitrotoluene, 2,4-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.05	U	U		
Dinitrotoluene, 2,6-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.05	U	U		
HMX	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.036	U	U		
Nitrobenzene	N	µg/L	0.2	0.2	0.084	U	U	0.2	0.2	0.082	U	U	0.05	0.1	0.05	U	U		
Nitrotoluene, 2-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.1	0.5	0.088	U	U		
Nitrotoluene, 3-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.1	0.5	0.057	U	U		
Nitrotoluene, 4-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.1	0.65	0.088	U	U		
RDX	N	µg/L	0.2	0.2	0.2	U	U	0.2	0.2	0.2	U	U	0.05	0.1	0.036	U	U		
Tetryl	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.05	U	U		
Trinitrobenzene, 1,3,5-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.03	U	U		
Trinitrotoluene, 2,4,6-	N	µg/L	0.2	0.2	0.08	U	U	0.2	0.2	0.078	U	U	0.05	0.1	0.05	U	U		
Ferrous Iron	N	mg/L	0	0.042															
Alkalinity	N	mg/L											300	5	2.7				
Alkalinity, Carbonate	N	mg/L	302		5	2.5	J	305		5	2.5	J							
Chloride	N	mg/L	1.8		2	1 B	J	1.7		2	1 B	J	1.7		1	0.1			
Cyanide, total	N	mg/L	0.01	0.01	0.005	U	U	0.01	0.01	0.005	U	U	0.01	0.01	0.005	U	U		
Hardness (as CaCO3)	N	mg/L	399	23	0.66			416	23	0.66			470	25	16				
Nitrate-Nitrite	N	mg/L	0.1	0.1	0.05	U	U	0.052	0.1	0.05	B	J	0.065	0.1	0.014	J B	B		
Sulfate	N	mg/L	162	4	2		J	167	4	2		J	180	2	0.24				
Total dissolved solids	N	mg/L	557	100	10		J	398	100	10		J	530	10	7.4				
Total suspended solids	N	mg/L	10	10	4	U	U	10	10	4	U	U	4	4	1.8	U	U		
Turbidity	N	NTU	1	1	1	U	U	1	1	1	U	U	1	0.5	0.11	H			
Aluminum	N	µg/L	200	200	24	U	U	200	200	24	U	U	300	300	75	U	U		
Aluminum	Y	µg/L	200	200	20	U	U	200	200	20	U	U	300	300	75	U	U		
Antimony	N	µg/L	6	6	1	U	U	6	6	1	U	U	25	25	7.4	U	U		
Antimony	Y	µg/L	6	6	1	U	U	6	6	1	U	U	25	25	7.4	U	U		
Arsenic	N	µg/L	10	10	1	U	U	10	10	1	U	U	10	10	3.3	U	U		
Arsenic	Y	µg/L	10	10	1	U	U	10	10	1	U	U	4.1	10	3.3	J	J		
Barium	N	µg/L	40.6	200	4	B	J	39.5	200	4	B	J	45	200	2.8	J	J		
Barium	Y	µg/L	40.5	200	1	B	J	43.5	200	1	B	J	46	200	2.8	J	J		
Beryllium	N	µg/L	4	4	0.1	U	U	4	4	0.1	U	U	5	5	0.56	U	U		
Beryllium	Y	µg/L	4	4	0.1	U	U	4	4	0.1	U	U	5	5	0.56	U	U		
Cadmium	N	µg/L	5	5	0.1	U	U	5	5	0.1	U	U	5	5	0.39	U	U		
Cadmium	Y	µg/L	5	5	0.1	U	U	5	5	0.1	U	U	5	5	0.39	U	U		
Calcium	N	µg/L	117000	1000	50		J	121000	1000	50		J	130000	5000	630				
Calcium	Y	µg/L	119000	1000	50		J	125000	1000	50		J	130000	5000	630				
Chromium	N	µg/L	10	10	1	U	U	10	10	1	U	U	7	7	1.4	U	U		
Chromium	Y	µg/L	10	10	1	U	U	10	10	1	U	U	7	7	1.4	U	U		
Cobalt	N	µg/L	1.2	50	1	B	J	1.1	50	1	B	J	1.7	7	1.5	J	J		
Cobalt	Y	µg/L	1.4	50	1	B	J	1.3	50	1	B	J	7	7	1.5	U	U		
Copper	N	µg/L	25	25	1	U	U	25	25	1	U	U	25	25	4.4	U	U		
Copper	Y	µg/L	7.3	25	1	B	J	25	25	1	U	U	25	25	4.4	U	U		
Iron	N	µg/L	57.5	300	23	B	J	70.6	300	23	B	J	130	250	64	J	J		
Iron	Y	µg/L	74.1	300	34	B	J	67.4	300	34	B	J	81	250	64	J	J		
Lead	N	µg/L	5	5	1	U	U	5	5	1	U	U	10	10	1.7	U	U		
Lead	Y	µg/L	5	5	1	U	U	5	5	1	U	U	10	10	1.7	U	U		
Magnesium	N	µg/L	25900	5000	50		J	27700	5000	50		J	29000	5000	120	B			
Magnesium	Y	µg/L	27400	5000	50		J	29000	5000	50		J	29000	5000	120	B			
Manganese	N	µg/L	1760	15	1			1780	15	1			1900	15	1.8				
Manganese	Y	µg/L	1720	15	1			1800	15	1			1900	15	1.8				
Mercury	N	µg/L	1	1	0.071	U	U	1	1	0.071	U	U	0.2	0.2	0.12	U	U		
Mercury	Y	µg/L	1	1	0.071	U	U	1	1	0.071	U	U	0.2	0.2	0.12	U	U		
Nickel	N	µg/L	40	40	1	U	U	40	40	1	U	U	40	40	2.2	U	U		
Nickel	Y	µg/L	40	40	1	U	U	40	40	1	U	U	40	40	2.2	U	U		
Potassium	N	µg/L	805	10000	50	B	J	716	10000	50	B	J	900	5000	300	J	J		
Potassium	Y	µg/L	744	10000	50	B	J	766	10000	50	B	J	860	5000	300	J	J		
Selenium	N	µg/L	10	10	2	U	U	10	10	2	U	U	15	15	4	U	U		

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works
Sandusky, Ohio

(Page 2 of 12)

Parameter	LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE	AP3-MW01 AP3083 19-Dec-11 1_3 - 1_3 Ft REG						AP3-MW01 AP3084 19-Dec-11 1_3 - 1_3 Ft FD						AP3-MW01 AP3085 19-Dec-11 1_3 - 1_3 Ft FS					
						Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Selenium	Y					10	10	10	2	U	U	U	15	15	15	4	U	U					
Silver	N					10	10	1	U	U	U	10	10	1	U	U	1.7	U					
Silver	Y					10	10	1	U	U	U	10	10	1	U	U	1.7	U					
Sodium	N					20100	10000	850				19500	10000	850			20000	5000					
Sodium	Y					19800	10000	750				21300	10000	750			19000	5000					
Thallium	N					10	10	1.5	U	U	U	10	10	1.5	U	U	30	30					
Thallium	Y					10	10	1	U	U	U	10	10	1	U	U	30	30					
Vanadium	N					50	50	1	U	U	U	50	50	1	U	U	7	7					
Vanadium	Y					50	50	1	U	U	U	50	50	1	U	U	7	7					
Zinc	N					20	20	1	U	U	U	20	20	1	U	U	50	50					
Zinc	Y					20	20	1	U	U	U	20	20	1	U	U	50	50					
3-Methylphenol and 4-Methylphenol	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	2	2					
Acenaphthene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Acenaphthylene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Acetophenone	N															0.98	0.98						
Anthracene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Atrazine	N															0.98	0.98						
Benzaldehyde	N															0.98	0.98						
Benzo(a)anthracene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Benzo(a)pyrene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Benzo(b)fluoranthene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Benzo(ghi)perylene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Benzo(k)fluoranthene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Benzoic acid	N					4.8	4.8	9.5	U	U	U	4.8	4.8	9.5	U	U							
Benzyl alcohol	N					4.8	4.8	0.95	U	U	U	4.8	4.8	0.95	U	U							
Bibenzene	N															0.98	0.98						
Bis(2-chloroethoxy)methane	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.98	0.98					
Bis(2-chloroethyl)ether	N					4.8	4.8	0.51	U	U	U	4.8	4.8	0.51	U	U	0.98	0.98					
Bis(2-chloroisopropyl)ether	N					4.8	4.8	0.51	U	U	U	4.8	4.8	0.51	U	U	0.98	0.98					
Bis(2-ethylhexyl)phthalate	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	0.8	2					
Bromophenyl phenyl ether, 4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	2	2					
Butyl benzyl phthalate	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	0.98	0.98					
Caprolactam	N															4.9	4.9						
Carbazole	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.98	0.98					
Chloro-3-methylphenol, 4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	2	2					
Chloroaniline, 4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	2	2					
Chloronaphthalene, 2-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.98	0.98					
Chlorophenol, 2-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.98	0.98					
Chlorophenyl phenyl ether, 4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	2	2					
Chrysene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Dibenz(a,h)anthracene	N					4.8	4.8	0.5	U	U	U	4.8	4.8	0.5	U	U	0.2	0.2					
Dibenzofuran	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.98	0.98					
Dichlorobenzene, 1,2-	N					4.8	4.8	0.95	U	U	U	4.8	4.8	0.95	U	U							
Dichlorobenzene, 1,3-	N					4.8	4.8	0.95	U	U	U	4.8	4.8	0.95	U	U							
Dichlorobenzene, 1,4-	N					4.8	4.8	0.95	U	U	U	4.8	4.8	0.95	U	U							
Dichlorobenzidine, 3,3'	N					9.5	9.5	0.95	U	U	U	9.5	9.5	0.95	U	U	4.9	4.9					
Dichlorophenol, 2,4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	2	2					
Diethyl phthalate	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	0.98	0.98					
Dimethyl phthalate	N					4.8	4.8	0.94	U	U	U	4.8	4.8	0.94	U	U	0.98	0.98					
Dimethylphenol, 2,4-	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	2	2					
Di-n-butyl phthalate	N					4.8	4.8	0.83	U	U	U	4.8	4.8	0.83	U	U	0.98	0.98					
Dinitro-2-methylphenol, 4,6-	N					9.5	9.5	1.9	U	U	U	9.5	9.5	1.9	U	U	4.9	4.9					
Dinitrophenol, 2,4-	N					24	24	9.5	U	U	U	24	24	9.5	U	U	4.9	4.9					
Dinitrotoluene, 2,4-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	4.9	4.9					
Dinitrotoluene, 2,6-	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	4.9	4.9					
Di-n-octyl phthalate	N					4.8	4.8	1	U	U	U	4.8	4.8	1	U	U	0.98	0.98					
Fluoranthene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Fluorene	N					4.8	4.8	0.48	U	U	U	4.8	4.8	0.48	U	U	0.2	0.2					
Hexachlorobenzene	N					4.8	4.8	0.53	U	U	U	4.8	4.8	0.53	U	U	0.2	0.2					
Hexachlorobutadiene	N					4.8	4.8	0.95	U	U	U	4.8	4.8	0.95	U	U	0.98	0.98					

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works
Sandusky, Ohio

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Parameter	LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE	AP3-MW01 AP3083 19-Dec-11 1_3 - 1_3 Ft REG						AP3-MW01 AP3084 19-Dec-11 1_3 - 1_3 Ft FD						AP3-MW01 AP3085 19-Dec-11 1_3 - 1_3 Ft FS					
						Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	
Hexachlorocyclopentadiene	N					9.5	9.5	1.8	U	U	9.5	9.5	1.8	U	U	9.8	9.8	0.78	U	U			
Hexachloroethane	N					4.8	4.8	0.95	U	U	4.8	4.8	0.95	U	U	0.98	0.98	0.78	U	U			
Indeno(1,2,3-cd)pyrene	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.2	0.2	0.098	U	U			
Isophorone	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.98	0.98	0.26	U	U			
Methylnaphthalene, 2-	N					4.8	4.8	0.54	U	U	4.8	4.8	0.54	U	U	0.2	0.2	0.098	U	U			
Methylphenol, 2-	N					4.8	4.8	0.51	U	U	4.8	4.8	0.51	U	U	0.98	0.98	0.78	U	U			
Naphthalene	N					4.8	4.8	0.76	U	U	4.8	4.8	0.76	U	U	0.2	0.2	0.098	U	U			
Nitroaniline, 2-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	2	2	0.78	U	U			
Nitroaniline, 3-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	2	2	0.27	U	U			
Nitroaniline, 4-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	2	2	0.78	U	U			
Nitrobenzene	N					4.8	4.8	0.56	U	U	4.8	4.8	0.56	U	U	0.98	0.98	0.039	U	U			
Nitrophenol, 2-	N					4.8	4.8	0.51	U	U	4.8	4.8	0.51	U	U	2	2	0.27	U	U			
Nitrophenol, 4-	N					24	24	4.8	U	U	24	24	4.8	U	U	4.9	4.9	2.4	U	U			
n-Nitroso-di-n-propylamine	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.98	0.98	0.78	U	U			
n-Nitrosodiphenylamine	N					4.8	4.8	0.95	U	U	4.8	4.8	0.95	U	U	0.98	0.98	0.3	U	U			
Pentachlorophenol	N					24	24	5.1	U	U	24	24	5.1	U	U	4.9	4.9	2.4	U	U			
Phenanthrene	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.2	0.2	0.098	U	U			
Phenol	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.98	0.98	0.59	U	U			
Pyrene	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	0.2	0.2	0.098	U	U			
Trichlorobenzene, 1,2,4-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U								
Trichlorophenol, 2,4,5-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	4.9	4.9	0.29	U	U			
Trichlorophenol, 2,4,6-	N					4.8	4.8	0.48	U	U	4.8	4.8	0.48	U	U	4.9	4.9	0.78	U	U			
Acetone	N					25	25	10	U	U	25	25	10	U	U	10	10	1.1	U	U			
Benzene	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.13	U	U			
Bromodichloromethane	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.15	U	U			
Bromoform	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.64	U	U			
Bromomethane	N					2	2	0.5	U	U	2	2	0.5	U	U	1	1	0.41	U	U			
Butanone, 2-	N					5	5	2	U	U	5	5	2	U	U	10	10	0.57	U	U			
Carbon disulfide	N					2	2	0.5	U	U	2	2	0.5	U	U	1	1	0.13	U	U			
Carbon tetrachloride	N					1	1	0.25	U	U	1	1	0.25	U	U	1	1	0.13	U	U			
Chlorobenzene	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.15	U	U			
Chloroethane	N					2	2	0.5	U	U	2	2	0.5	U	U	1	1	0.29	U	U			
Chloroform	N					1	1	0.22	U	U	1	1	0.22	U	U	1	1	0.16	U	U			
Chloromethane	N					2	2	0.5	U	U	2	2	0.5	U	U	1	1	0.3	U	U			
Cyclohexane	N															1	1	0.12	U	U			
Dibromo-3-chloropropane, 1,2-	N															2	2	0.67	U	U			
Dibromochloromethane	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.18	U	U			
Dibromoethane, 1,2-	N															1	1	0.24	U	U			
Dichlorobenzene, 1,2-	N															1	1	0.13	U	U			
Dichlorobenzene, 1,3-	N															1	1	0.14	U	U			
Dichlorobenzene, 1,4-	N															1	1	0.13	U	U			
Dichlorodifluoromethane	N															1	1	0.31	U	U			
Dichloroethane, 1,1-	N					1.7	1	0.25	U	U	1.6	1	0.25	U	U	1.3	1	0.15	U	U			
Dichloroethane, 1,2-	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.22	U	U			
Dichloroethene, 1,1-	N					1	1	0.23	U	U	1	1	0.23	U	U	1	1	0.19	U	U			
Dichloroethene, cis-1,2-	N					1	1	0.26	U	U	1	1	0.26	U	U	1	1	0.17	U	U			
Dichloroethene, trans-1,2-	N					1	1	0.35	U	U	1	1	0.35	U	U	1	1	0.19	U	U			
Dichloropropane, 1,2-	N					1	1	0.25	U	U	1	1	0.25	U	U	1	1	0.18	U	U			
Dichloropropene, cis-1,3-	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.14	U	U			
Dichloropropene, trans-1,3-	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.19	U	U			
Ethylbenzene	N					1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.17	U	U			
Hexanone, 2-	N					10	10	4	U	U	10	10	4	U	U	10	10	0.41	U	U			
Isopropylbenzene	N															1	1	0.13	U	U			
METHYL ACETATE	N															10	10	0.38	U	U			
Methyl Tertiary Butyl Ether	N															1	1	0.17	U	U			
Methyl-2-pentanone, 4-	N					5	5	2	U	U	5	5	2	U	U	10	10	0.32	U	U			
Methylcyclohexane	N															1	1	0.13	U	U			
Methylene chloride	N					5	5	2	U	U	5	5	2	U	U	1	1	0.33	U	U			

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works
 Sandusky, Ohio

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LOCATION_CODE			AP3-MW01					AP3-MW01					AP3-MW01				
SAMPLE_NO			AP3083					AP3084					AP3085				
SAMPLE_DATE			19-Dec-11					19-Dec-11					19-Dec-11				
DEPTH			1_3 - 1_3 Ft					1_3 - 1_3 Ft					1_3 - 1_3 Ft				
SAMPLE_PURPOSE			REG					FD					FS				
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Styrene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.11	U	U
Tetrachloroethane, 1,1,2,2-	N	µg/L	1	1	0.23	U	U	1	1	0.23	U	U	1	1	0.18	U	U
Tetrachloroethene	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U	1	1	0.29	U	U
Toluene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.13	U	U
Trichlorobenzene, 1,2,4-	N	µg/L											1	1	0.15	U	U
Trichloroethane, 1,1,1-	N	µg/L	0.27	1	0.2	J	J	0.26	1	0.2	J	J	1	1	0.22	U	U
Trichloroethane, 1,1,2-	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U	1	1	0.27	U	U
Trichloroethene	N	µg/L	1	1	0.26	U	U	1	1	0.26	U	U	1	1	0.17	U	U
Trichlorofluoromethane	N	µg/L											1	1	0.21	U	U
Trichlorotrifluoroethane	N	µg/L											1	1	0.28	U	U
Vinyl chloride	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U	1	1	0.22	U	U
Xylenes, total	N	µg/L	3	3	0.52	U	U	3	3	0.52	U	U	2	2	0.28	U	U

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works
 Sandusky, Ohio

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Parameter	LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE	AP3-MW01				AP3-MW02				AP3-MW02			
						AP3088	30-May-12	0 - 0 Ft	REG	AP3086	16-Dec-11	10_02 - 10_19 Ft	REG	AP3091	30-May-12	0 - 0 Ft	REG
	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Amino-2,6-dinitrotoluene, 4-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.08	U	U	0.4	0.4	0.16	U	U
Amino-4,6-dinitrotoluene, 2-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Dinitrobenzene, 1,3-	N	µg/L	0.4	0.4	0.19	U	U	0.19	0.19	0.094	U	U	0.4	0.4	0.19	U	U
Dinitrotoluene, 2,4-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Dinitrotoluene, 2,6-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
HMX	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Nitrobenzene	N	µg/L	0.4	0.4	0.17	U	U	0.19	0.19	0.082	U	U	0.4	0.4	0.17	U	U
Nitrotoluene, 2-	N	µg/L	0.18	0.4	0.16	J	J	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Nitrotoluene, 3-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Nitrotoluene, 4-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
RDX	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Tetryl	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Trinitrobenzene, 1,3,5-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Trinitrotoluene, 2,4,6-	N	µg/L	0.4	0.4	0.16	U	U	0.19	0.19	0.078	U	U	0.4	0.4	0.16	U	U
Ferrous Iron	N	mg/L						0	0.042								
Alkalinity	N	mg/L															
Alkalinity, Carbonate	N	mg/L						392	5	2.5							
Chloride	N	mg/L						11	10	5							
Cyanide, total	N	mg/L						0.01	0.01	0.005	U	U					
Hardness (as CaCO3)	N	mg/L	479	23	0.66			644	23	0.66			568	23	0.66		
Nitrate-Nitrite	N	mg/L						0.53	0.1	0.05							
Sulfate	N	mg/L						305	10	5							
Total dissolved solids	N	mg/L						788	100	10							
Total suspended solids	N	mg/L						10	10	4	U	U					
Turbidity	N	NTU						1.2	1	1							
Aluminum	N	µg/L	71	200	20	B	J	200	200	24	U	U	40.2	200	20	B	J
Aluminum	Y	µg/L															
Antimony	N	µg/L	6	6	1	U	U	6	6	1	U	U	6	6	1	U	U
Antimony	Y	µg/L															
Arsenic	N	µg/L	9.9	10	1	B	J	10	10	1	U	U	5.2	10	1	B	J
Arsenic	Y	µg/L															
Barium	N	µg/L	110	200	1	B	J	220	200	4	J	J	113	200	1	B	J
Barium	Y	µg/L															
Beryllium	N	µg/L	4	4	0.1	U	U	4	4	0.1	U	U	4	4	0.1	U	U
Beryllium	Y	µg/L															
Cadmium	N	µg/L	5	5	0.1	U	U	5	5	0.1	U	U	5	5	0.1	U	U
Cadmium	Y	µg/L															
Calcium	N	µg/L	116000	1000	50			140000	1000	50	J	J	118000	1000	50		
Calcium	Y	µg/L															
Chromium	N	µg/L	10	10	1	U	U	1.3	10	1	B	J	10	10	1	U	U
Chromium	Y	µg/L															
Cobalt	N	µg/L	50	50	1	U	U	50	50	1	U	U	2.1	50	1	B	J
Cobalt	Y	µg/L															
Copper	N	µg/L	25	25	1	U	U	25	25	1	U	U	25	25	1	U	U
Copper	Y	µg/L															
Iron	N	µg/L	2240	300	34			300	300	23	U	U	661	300	34		
Iron	Y	µg/L															
Lead	N	µg/L	5	5	1	U	U	5	5	1	U	U	5	5	1	U	U
Lead	Y	µg/L															
Magnesium	N	µg/L	45900	5000	50			71600	5000	50	J	J	66300	5000	50		
Magnesium	Y	µg/L															
Manganese	N	µg/L	779	15	1			52	15	1			270	15	1		
Manganese	Y	µg/L															
Mercury	N	µg/L	1	1	0.04	U	U	1	1	0.071	U	U	1	1	0.04	U	U
Mercury	Y	µg/L															
Nickel	N	µg/L	40	40	1	U	U	40	40	1	U	U	40	40	1	U	U
Nickel	Y	µg/L															
Potassium	N	µg/L	4050	10000	50	B	J	3840	10000	50	B	J	5030	10000	50	B	J
Potassium	Y	µg/L															
Selenium	N	µg/L	10	10	2	U	U	10	10	2	U	U	10	10	2	U	U

Table A-3

Groundwater Data Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
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LOCATION_CODE			AP3-MW01 AP3088 30-May-12 0 - 0 Ft REG						AP3-MW02 AP3086 16-Dec-11 10_02 - 10_19 Ft REG						AP3-MW02 AP3091 30-May-12 0 - 0 Ft REG					
SAMPLE_NO			Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
SAMPLE_DATE																				
DEPTH																				
SAMPLE_PURPOSE																				
Parameter	Filtered	Units																		
Styrene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.2	U	U			
Tetrachloroethane, 1,1,2,2-	N	µg/L	1	1	0.23	U	U	1	1	0.23	U	U	1	1	0.23	U	U			
Tetrachloroethene	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U	1	1	0.25	U	U			
Toluene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.2	U	U			
Trichlorobenzene, 1,2,4-	N	µg/L																		
Trichloroethane, 1,1,1-	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U	1	1	0.2	U	U			
Trichloroethane, 1,1,2-	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U	1	1	0.22	U	U			
Trichloroethene	N	µg/L	1	1	0.26	U	U	1	1	0.26	U	U	1	1	0.26	U	U			
Trichlorofluoromethane	N	µg/L																		
Trichlorotrifluoroethane	N	µg/L																		
Vinyl chloride	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U	1	1	0.22	U	U			
Xylenes, total	N	µg/L	3	3	0.52	U	U	3	3	0.52	U	U	3	3	0.52	U	U			

Table A-3

**Groundwater Data Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works
Sandusky, Ohio**

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LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE			AP3-MW03			AP3-MW03				
							AP3087			AP3092				
							20-Dec-11			30-May-12				
							7_22 - 7_41 Ft			0 - 0 Ft				
							REG			REG				
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Amino-2,6-dinitrotoluene, 4-	N	µg/L	0.2	0.2	0.082	U	U	0.4	0.4	0.16	U	U		
Amino-4,6-dinitrotoluene, 2-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Dinitrobenzene, 1,3-	N	µg/L	0.2	0.2	0.097	U	U	0.4	0.4	0.19	U	U		
Dinitrotoluene, 2,4-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Dinitrotoluene, 2,6-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
HMX	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Nitrobenzene	N	µg/L	0.2	0.2	0.084	U	U	0.4	0.4	0.17	U	U		
Nitrotoluene, 2-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Nitrotoluene, 3-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Nitrotoluene, 4-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
RDX	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Tetryl	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Trinitrobenzene, 1,3,5-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Trinitrotoluene, 2,4,6-	N	µg/L	0.2	0.2	0.08	U	U	0.4	0.4	0.16	U	U		
Ferrous Iron	N	mg/L	0	0.042										
Alkalinity	N	mg/L												
Alkalinity, Carbonate	N	mg/L	398	5	2.5		J							
Chloride	N	mg/L	27	10	5									
Cyanide, total	N	mg/L	0.01	0.01	0.005	U	U							
Hardness (as CaCO3)	N	mg/L	613	23	0.66			619	23	0.66				
Nitrate-Nitrite	N	mg/L	0.42	0.1	0.05									
Sulfate	N	mg/L	538	20	10									
Total dissolved solids	N	mg/L	1210	100	10									
Total suspended solids	N	mg/L	8	10	4 B		J							
Turbidity	N	NTU	1	1	1 U		U							
Aluminum	N	µg/L	88.9	200	24 B		J	34.9	200	20 B		J		
Aluminum	Y	µg/L	46.7	200	20 B		J							
Antimony	N	µg/L	6	6	1 U		U	6	6	1 U		U		
Antimony	Y	µg/L	6	6	1 U		U							
Arsenic	N	µg/L	10	10	1 U		U	10	10	1 U		U		
Arsenic	Y	µg/L	10	10	1 U		U							
Barium	N	µg/L	67	200	4 B		J	47.5	200	1 B		J		
Barium	Y	µg/L	75.3	200	1 B		J							
Beryllium	N	µg/L	4	4	0.1 U		U	4	4	0.1 U		U		
Beryllium	Y	µg/L	4	4	0.1 U		U							
Cadmium	N	µg/L	5	5	0.1 U		U	5	5	0.1 U		U		
Cadmium	Y	µg/L	5	5	0.1 U		U							
Calcium	N	µg/L	161000	1000	50		J	157000	1000	50				
Calcium	Y	µg/L	169000	1000	50									
Chromium	N	µg/L	10	10	1 U		U	10	10	1 U		U		
Chromium	Y	µg/L	10	10	1 U		U							
Cobalt	N	µg/L	50	50	1 U		U	1.6	50	1 B		J		
Cobalt	Y	µg/L	50	50	1 U		U							
Copper	N	µg/L	25	25	1 U		U	25	25	1 U		U		
Copper	Y	µg/L	25	25	1 U		U							
Iron	N	µg/L	300	300	23 U		U	631	300	34				
Iron	Y	µg/L	300	300	34 U		U							
Lead	N	µg/L	5	5	1 U		U	5	5	1 U		U		
Lead	Y	µg/L	5	5	1 U		U							
Magnesium	N	µg/L	51200	5000	50		J	55200	5000	50				
Magnesium	Y	µg/L	54100	5000	50									
Manganese	N	µg/L	151	15	1			472	15	1				
Manganese	Y	µg/L	151	15	1									
Mercury	N	µg/L	1	1	0.071 U		U	1	1	0.04 U		U		
Mercury	Y	µg/L	1	1	0.071 U		U							
Nickel	N	µg/L	2.4	40	1 B		J	40	40	1 U		U		
Nickel	Y	µg/L	40	40	1 U		U							
Potassium	N	µg/L	11100	10000	50			4680	10000	50 B		J		
Potassium	Y	µg/L	11300	10000	50									
Selenium	N	µg/L	10	10	2 U		U	10	10	2 U		U		

Table A-3

**Groundwater Data Used in the Baseline Human Health Risk Assessment
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Former Plum Brook Ordnance Works
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LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE			AP3-MW03				AP3-MW03						
							AP3087				AP3092						
							20-Dec-11				30-May-12						
							7_22 - 7_41 Ft				0 - 0 Ft						
							REG				REG						
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Selenium	Y	µg/L	2.9	10		2 B	J										
Silver	N	µg/L	10	10		1 U	U	10	10		1 U	U					
Silver	Y	µg/L	10	10		1 U	U										
Sodium	N	µg/L	154000	40000	3400			105000	40000	3400							
Sodium	Y	µg/L	153000	40000	3000												
Thallium	N	µg/L	10	10	1.5 U	U		10	10		1 U	U					
Thallium	Y	µg/L	10	10	1 U	U											
Vanadium	N	µg/L	2.1	50	1 B	J		50	50		1 U	U					
Vanadium	Y	µg/L	2.4	50	1 B	J											
Zinc	N	µg/L	7.7	20	1 B	J		18.4	20		1 B	J					
Zinc	Y	µg/L	20	20	1 U	U											
3-Methylphenol and 4-Methylphenol	N	µg/L	4.8	4.8		1 U	U										
Acenaphthene	N	µg/L	4.8	4.8	0.48 U	U											
Acenaphthylene	N	µg/L	4.8	4.8	0.48 U	U											
Acetophenone	N	µg/L															
Anthracene	N	µg/L	4.8	4.8	0.48 U	U											
Atrazine	N	µg/L															
Benzaldehyde	N	µg/L															
Benzo(a)anthracene	N	µg/L	4.8	4.8	0.48 U	U											
Benzo(a)pyrene	N	µg/L	4.8	4.8	0.48 U	U											
Benzo(b)fluoranthene	N	µg/L	4.8	4.8	0.48 U	U											
Benzo(ghi)perylene	N	µg/L	4.8	4.8	0.48 U	U											
Benzo(k)fluoranthene	N	µg/L	4.8	4.8	0.48 U	U											
Benzoic acid	N	µg/L	4.8	4.8	9.5 U	U											
Benzy alcohol	N	µg/L	4.8	4.8	0.95 U	U											
Bibenzene	N	µg/L															
Bis(2-chloroethoxy)methane	N	µg/L	4.8	4.8	0.48 U	U											
Bis(2-chloroethyl)ether	N	µg/L	4.8	4.8	0.51 U	U											
Bis(2-chloroisopropyl)ether	N	µg/L	4.8	4.8	0.51 U	U											
Bis(2-ethylhexyl)phthalate	N	µg/L	4.8	4.8	1 U	U											
Bromophenyl phenyl ether, 4-	N	µg/L	4.8	4.8	0.48 U	U											
Butyl benzyl phthalate	N	µg/L	4.8	4.8	1 U	U											
Caprolactam	N	µg/L															
Carbazole	N	µg/L	4.8	4.8	0.48 U	U											
Chloro-3-methylphenol, 4-	N	µg/L	4.8	4.8	0.48 U	U											
Chloroaniline, 4-	N	µg/L	4.8	4.8	0.48 U	U											
Chloronaphthalene, 2-	N	µg/L	4.8	4.8	0.48 U	U											
Chlorophenol, 2-	N	µg/L	4.8	4.8	0.48 U	U											
Chlorophenyl phenyl ether, 4-	N	µg/L	4.8	4.8	0.48 U	U											
Chrysene	N	µg/L	4.8	4.8	0.48 U	U											
Dibenz(a,h)anthracene	N	µg/L	4.8	4.8	0.5 U	U											
Dibenzofuran	N	µg/L	4.8	4.8	0.48 U	U											
Dichlorobenzene, 1,2-	N	µg/L	4.8	4.8	0.95 U	U											
Dichlorobenzene, 1,3-	N	µg/L	4.8	4.8	0.95 U	U											
Dichlorobenzene, 1,4-	N	µg/L	4.8	4.8	0.95 U	U											
Dichlorobenzidine, 3,3'-	N	µg/L	9.5	9.5	0.95 U	U											
Dichlorophenol, 2,4-	N	µg/L	4.8	4.8	0.48 U	U											
Diethyl phthalate	N	µg/L	4.8	4.8	1 U	U											
Dimethyl phthalate	N	µg/L	4.8	4.8	0.94 U	U											
Dimethylphenol, 2,4-	N	µg/L	4.8	4.8	1 U	U											
Di-n-butyl phthalate	N	µg/L	4.8	4.8	0.83 U	U											
Dinitro-2-methylphenol, 4,6-	N	µg/L	9.5	9.5	1.9 U	U											
Dinitrophenol, 2,4-	N	µg/L	24	24	9.5 U	U											
Dinitrotoluene, 2,4-	N	µg/L	4.8	4.8	0.48 U	U											
Dinitrotoluene, 2,6-	N	µg/L	4.8	4.8	0.48 U	U											
Di-n-octyl phthalate	N	µg/L	4.8	4.8	1 U	U											
Fluoranthene	N	µg/L	4.8	4.8	0.48 U	U											
Fluorene	N	µg/L	4.8	4.8	0.48 U	U											
Hexachlorobenzene	N	µg/L	4.8	4.8	0.53 U	U											
Hexachlorobutadiene	N	µg/L	4.8	4.8	0.95 U	U											

Table A-3

**Groundwater Data Used in the Baseline Human Health Risk Assessment
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Former Plum Brook Ordnance Works
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LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	DEPTH	SAMPLE_PURPOSE	AP3-MW03 AP3087 20-Dec-11 7_22 - 7_41 Ft REG					AP3-MW03 AP3092 30-May-12 0 - 0 Ft REG				
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Hexachlorocyclopentadiene	N	µg/L	9.5	9.5	1.8	U	U							
Hexachloroethane	N	µg/L	4.8	4.8	0.95	U	U							
Indeno[1,2,3-cd]pyrene	N	µg/L	4.8	4.8	0.48	U	U							
Isophorone	N	µg/L	4.8	4.8	0.48	U	U							
Methylnaphthalene, 2-	N	µg/L	4.8	4.8	0.54	U	U							
Methylphenol, 2-	N	µg/L	4.8	4.8	0.51	U	U							
Naphthalene	N	µg/L	4.8	4.8	0.76	U	U							
Nitroaniline, 2-	N	µg/L	4.8	4.8	0.48	U	U							
Nitroaniline, 3-	N	µg/L	4.8	4.8	0.48	U	U							
Nitroaniline, 4-	N	µg/L	4.8	4.8	0.48	U	U							
Nitrobenzene	N	µg/L	4.8	4.8	0.56	U	U							
Nitrophenol, 2-	N	µg/L	4.8	4.8	0.51	U	U							
Nitrophenol, 4-	N	µg/L	24	24	4.8	U	U							
n-Nitroso-di-n-propylamine	N	µg/L	4.8	4.8	0.48	U	U							
n-Nitrosodiphenylamine	N	µg/L	4.8	4.8	0.95	U	U							
Pentachlorophenol	N	µg/L	24	24	5.1	U	U							
Phenanthrene	N	µg/L	4.8	4.8	0.48	U	U							
Phenol	N	µg/L	4.8	4.8	0.48	U	U							
Pyrene	N	µg/L	4.8	4.8	0.48	U	U							
Trichlorobenzene, 1,2,4-	N	µg/L	4.8	4.8	0.48	U	U							
Trichlorophenol, 2,4,5-	N	µg/L	4.8	4.8	0.48	U	U							
Trichlorophenol, 2,4,6-	N	µg/L	4.8	4.8	0.48	U	U							
Acetone	N	µg/L	17.6	25	10	J	J	25	25	10	U	U		
Benzene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Bromodichloromethane	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Bromoform	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Bromomethane	N	µg/L	2	2	0.5	U	U	2	2	0.5	U	U		
Butanone, 2-	N	µg/L	5	5	2	U	U	5	5	2	U	U		
Carbon disulfide	N	µg/L	2	2	0.5	U	U	2	2	0.5	U	U		
Carbon tetrachloride	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U		
Chlorobenzene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Chloroethane	N	µg/L	2	2	0.5	U	U	2	2	0.5	U	U		
Chloroform	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U		
Chloromethane	N	µg/L	1.6	2	0.5	J	J	2	2	0.5	U	U		
Cyclohexane	N	µg/L												
Dibromo-3-chloropropane, 1,2-	N	µg/L												
Dibromochloromethane	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Dibromoethane, 1,2-	N	µg/L												
Dichlorobenzene, 1,2-	N	µg/L												
Dichlorobenzene, 1,3-	N	µg/L												
Dichlorobenzene, 1,4-	N	µg/L												
Dichlorodifluoromethane	N	µg/L												
Dichloroethane, 1,1-	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U		
Dichloroethane, 1,2-	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Dichloroethene, 1,1-	N	µg/L	1	1	0.23	U	U	1	1	0.23	U	U		
Dichloroethene, cis-1,2-	N	µg/L	1	1	0.26	U	U	1	1	0.26	U	U		
Dichloroethene, trans-1,2-	N	µg/L	1	1	0.35	U	U	1	1	0.35	U	U		
Dichloropropane, 1,2-	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U		
Dichloropropene, cis-1,3-	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Dichloropropene, trans-1,3-	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Ethylbenzene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U		
Hexanone, 2-	N	µg/L	10	10	4	U	U	10	10	4	U	U		
Isopropylbenzene	N	µg/L												
METHYL ACETATE	N	µg/L												
Methyl Tertiary Butyl Ether	N	µg/L												
Methyl-2-pentanone, 4-	N	µg/L	5	5	2	U	U	5	5	2	U	U		
Methylcyclohexane	N	µg/L												
Methylene chloride	N	µg/L	5	5	2	U	U	5	5	2	U	U		

Table A-3

**Groundwater Data Used in the Baseline Human Health Risk Assessment
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LOCATION_CODE	AP3-MW03							AP3-MW03				
SAMPLE_NO	AP3087							AP3092				
SAMPLE_DATE	20-Dec-11							30-May-12				
DEPTH	7_22 - 7_41 Ft							0 - 0 Ft				
SAMPLE_PURPOSE	REG							REG				
Parameter	Filtered	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Styrene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U
Tetrachloroethane, 1,1,2,2-	N	µg/L	1	1	0.23	U	U	1	1	0.23	U	U
Tetrachloroethene	N	µg/L	1	1	0.25	U	U	1	1	0.25	U	U
Toluene	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U
Trichlorobenzene, 1,2,4-	N	µg/L										
Trichloroethane, 1,1,1-	N	µg/L	1	1	0.2	U	U	1	1	0.2	U	U
Trichloroethane, 1,1,2-	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U
Trichloroethene	N	µg/L	1	1	0.26	U	U	1	1	0.26	U	U
Trichlorofluoromethane	N	µg/L										
Trichlorotrifluoroethane	N	µg/L										
Vinyl chloride	N	µg/L	1	1	0.22	U	U	1	1	0.22	U	U
Xylenes, total	N	µg/L	3	3	0.52	U	U	3	3	0.52	U	U

FD - Field duplicate.
LQ - Laboratory qualifier.
MDL - Method detection limit.
µg/L - micrograms per liter
REG - Regular sample.
RL - Reporting limit.
VQ - Validation qualifier.

Validation Qualifiers:
U - Nondetect.
J - Estimated Concentration.
UJ - Nondetected; detection limit is estimated.
B - Analyte also found in blank.
R - Rejected.

Table A-4

Sediment Samples Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 4)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE	Units	AP3-SD01 AP1009 23-May-09 REG						AP3-SD02 AP1010 23-May-09 REG						AP3-SD03 AP1011 23-May-09 REG					
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.16	0.16	0.041	U	U	0.18	0.18	0.045	U	U	0.18	0.18	0.045	U	U			
Amino-4,6-dinitrotoluene, 2-	mg/kg	0.16	0.16	0.089	U	U	0.18	0.18	0.099	U	U	0.18	0.18	0.099	U	U			
Dinitrobenzene, 1,3-	mg/kg	0.16	0.16	0.043	U	U	0.18	0.18	0.048	U	U	0.18	0.18	0.048	U	U			
Dinitrotoluene, 2,4-	mg/kg	0.16	0.16	0.049	U	U	0.18	0.18	0.055	U	U	0.18	0.18	0.055	U	U			
Dinitrotoluene, 2,6-	mg/kg	0.16	0.16	0.043	U	U	0.18	0.18	0.048	U	U	0.18	0.18	0.048	U	U			
HMX	mg/kg	0.16	0.16	0.07	U	U	0.18	0.18	0.078	U	U	0.18	0.18	0.078	U	U			
Nitrobenzene	mg/kg	0.16	0.16	0.054	U	U	0.18	0.18	0.06	U	U	0.18	0.18	0.06	U	U			
Nitrotoluene, 2-	mg/kg	0.16	0.16	0.041	U	U	0.18	0.18	0.045	U	U	0.18	0.18	0.045	U	U			
Nitrotoluene, 3-	mg/kg	0.16	0.16	0.069	U	U	0.18	0.18	0.076	U	U	0.18	0.18	0.076	U	U			
Nitrotoluene, 4-	mg/kg	0.16	0.16	0.051	U	U	0.18	0.18	0.056	U	U	0.18	0.18	0.056	U	U			
RDX	mg/kg	0.16	0.16	0.07	U	U	0.18	0.18	0.077	U	U	0.18	0.18	0.077	U	U			
Tetryl	mg/kg	0.16	0.16	0.042	U	U	0.18	0.18	0.046	U	U	0.18	0.18	0.046	U	U			
Trinitrobenzene, 1,3,5-	mg/kg	0.16	0.16	0.041	U	U	0.18	0.18	0.045	U	U	0.18	0.18	0.045	U	U			
Trinitrotoluene, 2,4,6-	mg/kg	0.16	0.16	0.041	U	U	0.18	0.18	0.045	U	U	0.18	0.18	0.045	U	U			
% Solids	Percent	54.3	0				55.4	0				68.4	0						
Total organic carbon	mg/kg	28000	0.4	0.4															
Aluminum	mg/kg	3610	18	0.99															
Antimony	mg/kg	0.54	5.4	0.41	B	J													
Arsenic	mg/kg	6.4	0.72	0.32															
Barium	mg/kg	26.6	18	0.45															
Beryllium	mg/kg	0.26	0.45	0.09	B	J													
Cadmium	mg/kg	0.3	0.36	0.09	B	J													
Calcium	mg/kg	9610	450	9		J													
Chromium	mg/kg	6.4	0.9	0.14															
Cobalt	mg/kg	4.1	4.5	0.075	B	J													
Copper	mg/kg	126	2.3	0.19															
Iron	mg/kg	13000	9	2.1		J													
Lead	mg/kg	8.7	9	0.18	B	J													
Magnesium	mg/kg	4370	450	9		J													
Manganese	mg/kg	130	1.4	0.045		J													
Mercury	mg/kg	0.027	0.14	0.021	B	J													
Nickel	mg/kg	10.5	3.6	0.21		J													
Potassium	mg/kg	337	1800	9	B	J													
Selenium	mg/kg	0.54	9	0.28	B	B													
Silver	mg/kg	0.9	0.9	0.11	U	U													
Sodium	mg/kg	900	900	45	U	U													
Thallium	mg/kg	9	9	3.1	U	U													
Vanadium	mg/kg	11.3	4.5	0.06															
Zinc	mg/kg	52.8	1.8	0.34		J													
Aroclor 1016	mg/kg	0.031	0.031	0.015	U	U	0.03	0.03	0.015	U	U	0.024	0.024	0.012	U	U			
Aroclor 1221	mg/kg	0.031	0.031	0.025	U	U	0.03	0.03	0.024	U	U	0.024	0.024	0.019	U	U			
Aroclor 1232	mg/kg	0.031	0.031	0.025	U	U	0.03	0.03	0.024	U	U	0.024	0.024	0.019	U	U			
Aroclor 1242	mg/kg	0.031	0.031	0.015	U	U	0.03	0.03	0.015	U	U	0.024	0.024	0.012	U	U			
Aroclor 1248	mg/kg	0.031	0.031	0.015	U	U	0.03	0.03	0.015	U	U	0.024	0.024	0.012	U	U			
Aroclor 1254	mg/kg	0.031	0.031	0.015	U	U	0.03	0.03	0.015	U	U	0.024	0.024	0.012	U	U			
Aroclor 1260	mg/kg	0.031	0.031	0.015	U	U	0.03	0.03	0.015	U	U	0.024	0.024	0.012	U	U			
3-Methylphenol and 4-Methylphenol	mg/kg	0.31	0.31	0.061	U	U													
Acenaphthene	mg/kg	0.31	0.31	0.061	U	U													
Acenaphthylene	mg/kg	0.31	0.31	0.061	U	U													
Anthracene	mg/kg	0.31	0.31	0.061	U	U													
Benzo(a)anthracene	mg/kg	0.143	0.31	0.061	J	J													
Benzo(a)pyrene	mg/kg	0.158	0.31	0.061	J	J													
Benzo(b)fluoranthene	mg/kg	0.24	0.31	0.061	J	J													
Benzo(ghi)perylene	mg/kg	0.101	0.31	0.061	J	J													
Benzo(k)fluoranthene	mg/kg	0.0805	0.31	0.061	J	J													
Benzoic acid	mg/kg	1.5	1.5	0.61	U	U													
Benzyl alcohol	mg/kg	0.31	0.31	0.061	U	U													
Bis(2-chloroethoxy)methane	mg/kg	0.31	0.31	0.061	U	U													
Bis(2-chloroethyl)ether	mg/kg	0.31	0.31	0.061	U	U													
Bis(2-chloroisopropyl)ether	mg/kg	0.31	0.31	0.061	U	U													
Bis(2-ethylhexyl)phthalate	mg/kg	0.61	0.61	0.31	U	U													
Bromophenyl phenyl ether, 4-	mg/kg	0.31	0.31	0.061	U	U													
Butyl benzyl phthalate	mg/kg	0.61	0.61	0.12	U	U													

Table A-4

Sediment Samples Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 2 of 4)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE	Units	AP3-SD01 AP1009 23-May-09 REG						AP3-SD02 AP1010 23-May-09 REG						AP3-SD03 AP1011 23-May-09 REG					
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
Amino-2,6-dinitrotoluene, 4-	mg/kg	0.16	0.16	0.041	U	U	0.18	0.18	0.045	U	U	0.18	0.18	0.045	U	U			
Carbazole	mg/kg	0.31	0.31	0.061	U	U													
Chloro-3-methylphenol, 4-	mg/kg	0.31	0.31	0.061	U	U													
Chloroaniline, 4-	mg/kg	0.31	0.31	0.12	U	U													
Chloronaphthalene, 2-	mg/kg	0.31	0.31	0.061	U	U													
Chlorophenol, 2-	mg/kg	0.31	0.31	0.061	U	U													
Chlorophenyl phenyl ether, 4-	mg/kg	0.31	0.31	0.061	U	U													
Chrysene	mg/kg	0.159	0.31	0.061	J	J													
Dibenzo(a,h)anthracene	mg/kg	0.31	0.31	0.061	U	U													
Dibenzofuran	mg/kg	0.31	0.31	0.061	U	U													
Dichlorobenzene, 1,2-	mg/kg	0.31	0.31	0.08	U	U													
Dichlorobenzene, 1,3-	mg/kg	0.31	0.31	0.08	U	U													
Dichlorobenzene, 1,4-	mg/kg	0.31	0.31	0.074	U	U													
Dichlorobenzidine, 3,3'-	mg/kg	0.61	0.61	0.12	U	U													
Dichlorophenol, 2,4-	mg/kg	0.31	0.31	0.061	U	U													
Diethyl phthalate	mg/kg	0.61	0.61	0.31	U	U													
Dimethyl phthalate	mg/kg	0.61	0.61	0.12	U	U													
Dimethylphenol, 2,4-	mg/kg	0.31	0.31	0.061	U	U													
Di-n-butyl phthalate	mg/kg	0.61	0.61	0.12	U	U													
Dinitro-2-methylphenol, 4,6-	mg/kg	0.61	0.61	0.2	U	U													
Dinitrophenol, 2,4-	mg/kg	1.5	1.5	0.61	U	U													
Dinitrotoluene, 2,4-	mg/kg	0.31	0.31	0.061	U	U													
Dinitrotoluene, 2,6-	mg/kg	0.31	0.31	0.061	U	U													
Di-n-octyl phthalate	mg/kg	0.61	0.61	0.12	U	U													
Fluoranthene	mg/kg	0.253	0.31	0.061	J	J													
Fluorene	mg/kg	0.31	0.31	0.061	U	U													
Hexachlorobenzene	mg/kg	0.31	0.31	0.061	U	U													
Hexachlorobutadiene	mg/kg	0.31	0.31	0.061	U	U													
Hexachlorocyclopentadiene	mg/kg	0.31	0.31	0.061	U	U													
Hexachloroethane	mg/kg	0.31	0.31	0.074	U	U													
Indeno(1,2,3-cd)pyrene	mg/kg	0.111	0.31	0.061	J	J													
Isophorone	mg/kg	0.31	0.31	0.061	U	U													
Methylnaphthalene, 2-	mg/kg	0.31	0.31	0.061	U	U													
Methylphenol, 2-	mg/kg	0.31	0.31	0.061	U	U													
Methylphenol, 4-	mg/kg																		
Naphthalene	mg/kg	0.31	0.31	0.061	U	U													
Nitroaniline, 2-	mg/kg	0.61	0.61	0.12	U	U													
Nitroaniline, 3-	mg/kg	0.61	0.61	0.12	U	U													
Nitroaniline, 4-	mg/kg	0.61	0.61	0.12	U	U													
Nitrobenzene	mg/kg	0.31	0.31	0.061	U	U													
Nitrophenol, 2-	mg/kg	0.31	0.31	0.061	U	U													
Nitrophenol, 4-	mg/kg	1.5	1.5	0.61	U	U													
n-Nitroso-di-n-propylamine	mg/kg	0.31	0.31	0.061	U	U													
n-Nitrosodiphenylamine	mg/kg	0.31	0.31	0.12	U	U													
Pentachlorophenol	mg/kg	1.5	1.5	0.61	U	U													
Phenanthrene	mg/kg	0.31	0.31	0.061	U	U													
Phenol	mg/kg	0.31	0.31	0.061	U	U													
Pyrene	mg/kg	0.206	0.31	0.061	J	J													
Trichlorobenzene, 1,2,4-	mg/kg	0.31	0.31	0.061	U	U													
Trichlorophenol, 2,4,5-	mg/kg	0.31	0.31	0.061	U	U													
Trichlorophenol, 2,4,6-	mg/kg	0.31	0.31	0.061	U	U													

Table A-4

Sediment Samples Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 3 of 4)

LOCATION_CODE	SAMPLE_NO	SAMPLE_DATE	SAMPLE_PURPOSE	PBOW99-SDA302 PBOW99SDA302 11-Jun-99 REG					PBOW99-SDA303 PBOW99SDA303 11-Jun-99 REG					PBOW99-SDA303 PBOW99SDA303DUP 11-Jun-99 FD				
Parameter	Units	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ		
Amino-2,6-dinitrotoluene, 4-	mg/kg																	
Amino-4,6-dinitrotoluene, 2-	mg/kg																	
Dinitrobenzene, 1,3-	mg/kg																	
Dinitrotoluene, 2,4-	mg/kg																	
Dinitrotoluene, 2,6-	mg/kg																	
HMX	mg/kg																	
Nitrobenzene	mg/kg																	
Nitrotoluene, 2-	mg/kg																	
Nitrotoluene, 3-	mg/kg																	
Nitrotoluene, 4-	mg/kg																	
RDX	mg/kg																	
Tetryl	mg/kg																	
Trinitrobenzene, 1,3,5-	mg/kg																	
Trinitrotoluene, 2,4,6-	mg/kg																	
% Solids	Percent																	
Total organic carbon	mg/kg																	
Aluminum	mg/kg	7750	33.1	33.1			1800	29.8	29.8		J	3150	30.9	30.9				
Antimony	mg/kg	1.7	1.7	1.7	U		1.5	1.5	1.5	U		1.5	1.5	1.5	U			
Arsenic	mg/kg	5.9	1.7	1.7			3.6	1.5	1.5			3.8	1.5	1.5				
Barium	mg/kg	47.8	33.1	33.1			29.8	29.8	29.8	U		30.9	30.9	30.9	U			
Beryllium	mg/kg	0.83	0.83	0.83	U		0.74	0.74	0.74	U		0.77	0.77	0.77	U			
Cadmium	mg/kg	0.33	0.33	0.33	U		0.3	0.3	0.3	U		0.31	0.31	0.31	U			
Calcium	mg/kg	18100	828	828			4560	744	744			5620	773	773				
Chromium	mg/kg	10.8	0.83	0.83			5.6	0.74	0.74			5.2	0.77	0.77				
Cobalt	mg/kg	8.3	8.3	8.3	U		7.4	7.4	7.4	U		7.7	7.7	7.7	U			
Copper	mg/kg	14.4	4.1	4.1			71.8	3.7	3.7			76.2	3.9	3.9				
Iron	mg/kg	12800	16.6	16.6			5880	14.9	14.9			7540	15.5	15.5				
Lead	mg/kg	11.9	0.5	0.5			5.1	0.45	0.45			8.3	0.46	0.46				
Magnesium	mg/kg	5670	828	828			2220	744	744			2920	773	773				
Manganese	mg/kg	318	2.5	2.5			122	2.2	2.2			159	2.3	2.3				
Mercury	mg/kg	0.17	0.17	0.17	U		0.15	0.15	0.15	U		0.15	0.15	0.15	U			
Nickel	mg/kg	12.5	6.6	6.6			6.3	6	6			6.8	6.2	6.2				
Potassium	mg/kg	1270	828	828			744	744	744	U		773	773	773	U			
Selenium	mg/kg	1.1	0.83	0.83			0.74	0.74	0.74	U		0.77	0.77	0.77	U			
Silver	mg/kg	0.83	0.83	0.83	U		0.74	0.74	0.74	U		0.77	0.77	0.77	U			
Sodium	mg/kg	828	828	828	U		744	744	744	U		773	773	773	U			
Thallium	mg/kg	1.7	1.7	1.7	U		1.5	1.5	1.5	U		1.5	1.5	1.5	U			
Vanadium	mg/kg	19.3	8.3	8.3			7.4	7.4	7.4	U		9.4	7.7	7.7				
Zinc	mg/kg	48.3	3.3	3.3	MBD		24.4	3	3	MBD		28.2	3.1	3.1	MBD			
Aroclor 1016	mg/kg																	
Aroclor 1221	mg/kg																	
Aroclor 1232	mg/kg																	
Aroclor 1242	mg/kg																	
Aroclor 1248	mg/kg																	
Aroclor 1254	mg/kg																	
Aroclor 1260	mg/kg																	
3-Methylphenol and 4-Methylphenol	mg/kg																	
Acenaphthene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Acenaphthylene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Anthracene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzo(a)anthracene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzo(a)pyrene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzo(b)fluoranthene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzo(ghi)perylene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzo(k)fluoranthene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Benzoic acid	mg/kg																	
Benzyl alcohol	mg/kg																	
Bis(2-chloroethoxy)methane	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Bis(2-chloroethyl)ether	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Bis(2-chloroisopropyl)ether	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Bis(2-ethylhexyl)phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Bromophenyl phenyl ether, 4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		
Butyl benzyl phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ		

Table A-4

Sediment Samples Used in the Baseline Human Health Risk Assessment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 4 of 4)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE	Units	PBOW99-SDA302 PBOW99SDA302 11-Jun-99 REG					PBOW99-SDA303 PBOW99SDA303 11-Jun-99 REG					PBOW99-SDA303 PBOW99SDA303DUP 11-Jun-99 FD				
		Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ
Amino-2,6-dinitrotoluene, 4-	mg/kg															
Carbazole	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chloro-3-methylphenol, 4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chloroaniline, 4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chloronaphthalene, 2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chlorophenol, 2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chlorophenyl phenyl ether, 4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Chrysene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dibenzo(a,h)anthracene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dibenzofuran	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dichlorobenzene, 1,2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dichlorobenzene, 1,3-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dichlorobenzene, 1,4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dichlorobenzidine, 3,3'-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dichlorophenol, 2,4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Diethyl phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dimethyl phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dimethylphenol, 2,4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Di-n-butyl phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dinitro-2-methylphenol, 4,6-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Dinitrophenol, 2,4-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Dinitrotoluene, 2,4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Dinitrotoluene, 2,6-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Di-n-octyl phthalate	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Fluoranthene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Fluorene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Hexachlorobenzene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Hexachlorobutadiene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Hexachlorocyclopentadiene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Hexachloroethane	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Indeno(1,2,3-cd)pyrene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Isophorone	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Methylnaphthalene, 2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Methylphenol, 2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Methylphenol, 4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Naphthalene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Nitroaniline, 2-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Nitroaniline, 3-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Nitroaniline, 4-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Nitrobenzene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Nitrophenol, 2-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Nitrophenol, 4-	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
n-Nitroso-di-n-propylamine	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
n-Nitrosodiphenylamine	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Pentachlorophenol	mg/kg	1.3	1.3	1.3	U	UJ	1.2	1.2	1.2	U	UJ	1.2	1.2	1.2	U	UJ
Phenanthrene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Phenol	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Pyrene	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Trichlorobenzene, 1,2,4-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Trichlorophenol, 2,4,5-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ
Trichlorophenol, 2,4,6-	mg/kg	0.55	0.55	0.55	U	UJ	0.49	0.49	0.49	U	UJ	0.51	0.51	0.51	U	UJ

FD - Field duplicate.
 LQ - Laboratory qualifier.
 MDL - Method detection limit.
 mg/kg - Milligram(s) per kilogram.
 REG - Regular sample.
 RL - Reporting limit.
 VQ - Validation qualifier.

Validation Qualifiers:
 U - Nondetect.
 J - Estimated Concentration.
 UJ - Nondetected; detection limit is estimated.
 B - Analyte also found in blank.
 R - Rejected.

Table A-5

**Surface Water Samples Used in the Baseline Human Health Risk Assessment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 2)

LOCATION_CODE SAMPLE_NO SAMPLE_DATE SAMPLE_PURPOSE	Filtered	Units	AP3-SW01 AP2009 23-May-09 REG				AP3-SW02 AP2010 23-May-09 REG				AP3-SW03 AP2011A 27-May-09 REG				PBOW99-SWA301 PBOW99SWA301 11-Jun-99 REG				PBOW99-SWA302 PBOW99SWA302 11-Jun-99 REG				PBOW99-SWA303 PBOW99SWA303 11-Jun-99 REG				PBOW99-SWA303 PBOW99SWA303DUP 11-Jun-99 FD								
			Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ	Result	RL	MDL	LQ	VQ			
Amino-2,6-dinitrotoluene, 4-	N	µg/L	0.19	0.19	0.048	U	0.19	0.19	0.048	U	0.19	0.19	0.049	U	U																				
Amino-4,6-dinitrotoluene, 2-	N	µg/L	0.19	0.19	0.073	U	0.19	0.19	0.073	U	0.19	0.19	0.075	U	U																				
Dinitrobenzene, 1,3-	N	µg/L	0.19	0.19	0.049	U	0.19	0.19	0.049	U	0.19	0.19	0.05	U	U																				
Dinitrotoluene, 2,4-	N	µg/L	0.19	0.19	0.062	U	0.19	0.19	0.062	U	0.19	0.19	0.063	U	U																				
Dinitrotoluene, 2,6-	N	µg/L	0.19	0.19	0.089	U	0.19	0.19	0.089	U	0.19	0.19	0.09	U	U																				
HMX	N	µg/L	0.19	0.19	0.1	U	0.19	0.19	0.1	U	0.19	0.19	0.11	U	U																				
Nitrobenzene	N	µg/L	0.19	0.19	0.048	U	0.19	0.19	0.048	U	0.19	0.19	0.049	U	U																				
Nitrotoluene, 2-	N	µg/L	0.19	0.19	0.061	U	0.19	0.19	0.061	U	0.19	0.19	0.062	U	U																				
Nitrotoluene, 3-	N	µg/L	0.19	0.19	0.092	U	0.19	0.19	0.092	U	0.19	0.19	0.094	U	U																				
Nitrotoluene, 4-	N	µg/L	0.19	0.19	0.072	U	0.19	0.19	0.072	U	0.19	0.19	0.074	U	U																				
RDX	N	µg/L	0.19	0.19	0.071	U	0.19	0.19	0.071	U	0.19	0.19	0.073	U	U																				
Tetryl	N	µg/L	0.19	0.19	0.074	U	0.19	0.19	0.074	U	0.19	0.19	0.076	U	U																				
Trinitrobenzene, 1,3,5-	N	µg/L	0.19	0.19	0.048	U	0.19	0.19	0.048	U	0.19	0.19	0.049	U	U																				
Trinitrotoluene, 2,4,6-	N	µg/L	0.19	0.19	0.066	U	0.19	0.19	0.066	U	0.19	0.19	0.067	U	U																				
Aluminum	N	µg/L														200	200	200	U		200	200	200	U		200	200	200	U						
Antimony	N	µg/L														10	10	10	U		10	10	10	U		10	10	10	U						
Arsenic	N	µg/L														10	10	10	U		10	10	10	U		10	10	10	U						
Barium	N	µg/L														200	200	200	U		200	200	200	U		200	200	200	U						
Beryllium	N	µg/L														5	5	5	U		5	5	5	U		5	5	5	U						
Cadmium	N	µg/L														2	2	2	U		2	2	2	U		2	2	2	U						
Calcium	N	µg/L														27900	5000	5000			27700	5000	5000			24400	5000	5000			24500	5000	5000		
Chromium	N	µg/L														5	5	5	U		5	5	5	U		5	5	5	U						
Cobalt	N	µg/L														50	50	50	U		50	50	50	U		50	50	50	U						
Copper	N	µg/L														25	25	25	U		25	25	25	U		25	25	25	U						
Iron	N	µg/L														687	100	100	MBD	U	429	100	100	MBD	U	431	100	100	MBD	U	251	100	100	MBD	U
Lead	N	µg/L														3	3	3	U		3	3	3	U		3	3	3	U						
Magnesium	N	µg/L														9410	5000	5000			9470	5000	5000			8940	5000	5000			9040	5000	5000		
Manganese	N	µg/L														195	15	15			153	15	15			48.4	15	15			34.9	15	15		
Mercury	N	µg/L														0.2	0.2	0.2	U		0.2	0.2	0.2	U		0.2	0.2	0.2	U		0.2	0.2	0.2	U	
Nickel	N	µg/L														40	40	40	U		40	40	40	U		40	40	40	U		40	40	40	U	
Potassium	N	µg/L														5000	5000	5000	U		5000	5000	5000	U		5000	5000	5000	U		5000	5000	5000	U	
Selenium	N	µg/L														5	5	5	U		5	5	5	U		5	5	5	U		5	5	5	U	
Silver	N	µg/L														5	5	5	U		5	5	5	U		5	5	5	U		5	5	5	U	
Sodium	N	µg/L														9740	5000	5000			9840	5000	5000			10200	5000	5000			10500	5000	5000		
Thallium	N	µg/L														10	10	10	U		10	10	10	U		10	10	10	U		10	10	10	U	
Vanadium	N	µg/L														50	50	50	U		50	50	50	U		50	50	50	U		50	50	50	U	
Zinc	N	µg/L														20	20	20	U		20	20	20	U		20	20	20	U		20	20	20	U	
Acenaphthene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Acenaphthylene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Anthracene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Benzo(a)anthracene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Benzo(a)pyrene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Benzo(b)fluoranthene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Benzo(ghi)perylene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Benzo(k)fluoranthene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Bis(2-chloroethoxy)methane	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Bis(2-chloroethyl)ether	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Bis(2-chloroisopropyl)ether	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Bis(2-ethylhexyl)phthalate	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Bromophenyl phenyl ether, 4-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Butyl benzyl phthalate	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Carbazole	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chloro-3-methylphenol, 4-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chloroaniline, 4-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chloronaphthalene, 2-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chlorophenol, 2-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chlorophenyl phenyl ether, 4-	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U	U	10	10	10	U	U
Chrysene	N	µg/L														10	10	10	U	U	10	10	10	U	U	10	10	10	U						

APPENDIX B

ProUCL OUTPUT FOR UPPER CONFIDENCE LIMITS ON THE MEAN

Surface Soil ProUCL input - Ash Pit 3, PBOW

Sample No.	Units	Arsenic	D_Arsenic	Thallium	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene
AP0032	mg/kg	7.7	1	1.45	0.156	0.168	0.268
AP0036	mg/kg	4.9	1	0.315	0.105	0.105	0.105
AP0039	mg/kg	12.4	1	3.25	0.215	0.215	0.215
AP0042	mg/kg	29	1	3	0.245	0.245	0.245
AP0045	mg/kg	13.7	1	2.6	0.215	0.215	0.215
AP0048	mg/kg	12.7	1	3	0.13	0.13	0.13
AP0051	mg/kg	7.7	1	0.46	0.155	0.155	0.155
AP0054/0056	mg/kg	44.05	1	0.725	0.2475	0.2475	0.2475

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

From File AP 3 ss UCL input.wst
 Full Precision OFF
 Confidence Coefficient 95%
 Number of Bootstrap Operations 2000

Arsenic - Surface Soil (AP 3)

General Statistics

Number of Valid Observations 8

Number of Distinct Observations 7

Raw Statistics

Minimum 4.9
 Maximum 44.05
 Mean 16.52
 Median 12.55
 SD 13.33
 Coefficient of Variation 0.807
 Skewness 1.586

Log-transformed Statistics

Minimum of Log Data 1.589
 Maximum of Log Data 3.785
 Mean of log Data 2.563
 SD of log Data 0.721

Warning: There are only 8 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

Shapiro Wilk Test Statistic 0.798
 Shapiro Wilk Critical Value 0.818

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.941
 Shapiro Wilk Critical Value 0.818

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 25.45

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 27.09
 95% Modified-t UCL (Johnson-1978) 25.89

Assuming Lognormal Distribution

95% H-UCL 35.87

95% Chebyshev (MVUE) UCL 34.49
 97.5% Chebyshev (MVUE) UCL 42.41
 99% Chebyshev (MVUE) UCL 57.96

Gamma Distribution Test

k star (bias corrected) 1.471
 Theta Star 11.23
 MLE of Mean 16.52
 MLE of Standard Deviation 13.62
 nu star 23.53
 Approximate Chi Square Value (.05) 13.49
 Adjusted Level of Significance 0.0195
 Adjusted Chi Square Value 11.61

Data appear Gamma Distributed at 5% Significance Level

Anderson-Darling Test Statistic 0.46
 Anderson-Darling 5% Critical Value 0.723
 Kolmogorov-Smirnov Test Statistic 0.267
 Kolmogorov-Smirnov 5% Critical Value 0.297

Assuming Gamma Distribution

95% Approximate Gamma UCL 28.81
 95% Adjusted Gamma UCL 33.46

Potential UCL to Use

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 24.27
 95% Jackknife UCL 25.45
 95% Standard Bootstrap UCL 23.76
 95% Bootstrap-t UCL 43.6
 95% Hall's Bootstrap UCL 76.16
 95% Percentile Bootstrap UCL 24.43
 95% BCA Bootstrap UCL 26.7
 95% Chebyshev(Mean, Sd) UCL 37.06
 97.5% Chebyshev(Mean, Sd) UCL 45.95
 99% Chebyshev(Mean, Sd) UCL 63.41

Use 95% Approximate Gamma UCL 28.81

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

General UCL Statistics for Full Data Sets

User Selected Options

From File AP 3 ss UCL input.wst
 Full Precision OFF
 Confidence Coefficient 95%
 Number of Bootstrap Operations 2000

Thallium - Surface Soil (AP 3)

General Statistics

Number of Valid Observations 8

Number of Distinct Observations 7

Raw Statistics

Minimum 0.315
 Maximum 3.25
 Mean 1.85
 Median 2.025
 SD 1.247
 Coefficient of Variation 0.674
 Skewness -0.156

Log-transformed Statistics

Minimum of Log Data -1.155
 Maximum of Log Data 1.179
 Mean of log Data 0.306
 SD of log Data 0.937

Warning: There are only 8 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

Shapiro Wilk Test Statistic 0.85
 Shapiro Wilk Critical Value 0.818

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.85
 Shapiro Wilk Critical Value 0.818

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 2.685

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 2.549
 95% Modified-t UCL (Johnson-1978) 2.681

Assuming Lognormal Distribution

95% H-UCL 6.712

95% Chebyshev (MVUE) UCL 4.86
 97.5% Chebyshev (MVUE) UCL 6.113
 99% Chebyshev (MVUE) UCL 8.574

Gamma Distribution Test

k star (bias corrected) 1.187
 Theta Star 1.558
 MLE of Mean 1.85
 MLE of Standard Deviation 1.698
 nu star 19

Approximate Chi Square Value (.05) 10.12
 Adjusted Level of Significance 0.0195
 Adjusted Chi Square Value 8.527

Anderson-Darling Test Statistic 0.592
 Anderson-Darling 5% Critical Value 0.726
 Kolmogorov-Smirnov Test Statistic 0.266
 Kolmogorov-Smirnov 5% Critical Value 0.298

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Normal at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 3.474
 95% Adjusted Gamma UCL 4.122

Nonparametric Statistics

95% CLT UCL 2.575
 95% Jackknife UCL 2.685
 95% Standard Bootstrap UCL 2.521
 95% Bootstrap-t UCL 2.64
 95% Hall's Bootstrap UCL 2.378
 95% Percentile Bootstrap UCL 2.533
 95% BCA Bootstrap UCL 2.502
 95% Chebyshev(Mean, Sd) UCL 3.771
 97.5% Chebyshev(Mean, Sd) UCL 4.603
 99% Chebyshev(Mean, Sd) UCL 6.236

Potential UCL to Use

Use 95% Student's-t UCL 2.685

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Benzo(a)anthracene - Surface Soil (AP 3)

General Statistics

Number of Valid Observations 8

Number of Distinct Observations 7

Raw Statistics

Minimum 0.105
 Maximum 0.248
 Mean 0.184
 Median 0.186
 SD 0.0541
 Coefficient of Variation 0.294
 Skewness -0.154

Log-transformed Statistics

Minimum of Log Data -2.254
 Maximum of Log Data -1.396
 Mean of log Data -1.737
 SD of log Data 0.315

Warning: There are only 8 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

Shapiro Wilk Test Statistic 0.909
 Shapiro Wilk Critical Value 0.818

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.908
 Shapiro Wilk Critical Value 0.818

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.22

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.214
 95% Modified-t UCL (Johnson-1978) 0.22

Assuming Lognormal Distribution

95% H-UCL 0.237

95% Chebyshev (MVUE) UCL 0.274
 97.5% Chebyshev (MVUE) UCL 0.312
 99% Chebyshev (MVUE) UCL 0.389

Gamma Distribution Test

k star (bias corrected) 7.722
 Theta Star 0.0238
 MLE of Mean 0.184
 MLE of Standard Deviation 0.0661
 nu star 123.6

Approximate Chi Square Value (.05) 98.89
 Adjusted Level of Significance 0.0195
 Adjusted Chi Square Value 93.31

Anderson-Darling Test Statistic 0.405
 Anderson-Darling 5% Critical Value 0.715
 Kolmogorov-Smirnov Test Statistic 0.246
 Kolmogorov-Smirnov 5% Critical Value 0.294

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Normal at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.229
 95% Adjusted Gamma UCL 0.243

Nonparametric Statistics

95% CLT UCL 0.215
 95% Jackknife UCL 0.22
 95% Standard Bootstrap UCL 0.212
 95% Bootstrap-t UCL 0.218
 95% Hall's Bootstrap UCL 0.208
 95% Percentile Bootstrap UCL 0.213
 95% BCA Bootstrap UCL 0.213
 95% Chebyshev(Mean, Sd) UCL 0.267
 97.5% Chebyshev(Mean, Sd) UCL 0.303
 99% Chebyshev(Mean, Sd) UCL 0.374

Potential UCL to Use

Use 95% Student's-t UCL 0.22

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Benzo(a)pyrene - Surface Soil (AP 3)

General Statistics

Number of Valid Observations 8

Number of Distinct Observations 7

Raw Statistics

Minimum 0.105
 Maximum 0.248
 Mean 0.185
 Median 0.192
 SD 0.0533
 Coefficient of Variation 0.288
 Skewness -0.251

Log-transformed Statistics

Minimum of Log Data -2.254
 Maximum of Log Data -1.396
 Mean of log Data -1.727
 SD of log Data 0.312

Warning: There are only 8 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

Shapiro Wilk Test Statistic 0.925
 Shapiro Wilk Critical Value 0.818

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.916
 Shapiro Wilk Critical Value 0.818

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.221

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.214
 95% Modified-t UCL (Johnson-1978) 0.221

Assuming Lognormal Distribution

95% H-UCL 0.238

95% Chebyshev (MVUE) UCL 0.275
 97.5% Chebyshev (MVUE) UCL 0.314
 99% Chebyshev (MVUE) UCL 0.39

Gamma Distribution Test

k star (bias corrected) 7.933
 Theta Star 0.0233
 MLE of Mean 0.185
 MLE of Standard Deviation 0.0657
 nu star 126.9

Approximate Chi Square Value (.05) 101.9
 Adjusted Level of Significance 0.0195
 Adjusted Chi Square Value 96.24

Anderson-Darling Test Statistic 0.346
 Anderson-Darling 5% Critical Value 0.715
 Kolmogorov-Smirnov Test Statistic 0.238

Kolmogorov-Smirnov 5% Critical Value 0.294

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 0.216
 95% Jackknife UCL 0.221
 95% Standard Bootstrap UCL 0.214
 95% Bootstrap-t UCL 0.219
 95% Hall's Bootstrap UCL 0.209
 95% Percentile Bootstrap UCL 0.213
 95% BCA Bootstrap UCL 0.213
 95% Chebyshev(Mean, Sd) UCL 0.267
 97.5% Chebyshev(Mean, Sd) UCL 0.303
 99% Chebyshev(Mean, Sd) UCL 0.373

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.231
 95% Adjusted Gamma UCL 0.244

Potential UCL to Use

Use 95% Student's-t UCL 0.221

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Benzo(b)fluoranthene - Surface Soil (AP 3)

General Statistics

Number of Valid Observations 8

Number of Distinct Observations 7

Raw Statistics

Minimum 0.105
 Maximum 0.268
 Mean 0.198
 Median 0.215
 SD 0.0601
 Coefficient of Variation 0.304
 Skewness -0.516

Log-transformed Statistics

Minimum of Log Data -2.254
 Maximum of Log Data -1.317
 Mean of log Data -1.669
 SD of log Data 0.342

Warning: There are only 8 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

Shapiro Wilk Test Statistic 0.909
 Shapiro Wilk Critical Value 0.818

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.882
 Shapiro Wilk Critical Value 0.818

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.238

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.228
 95% Modified-t UCL (Johnson-1978) 0.237

Assuming Lognormal Distribution

95% H-UCL 0.263

95% Chebyshev (MVUE) UCL 0.303
 97.5% Chebyshev (MVUE) UCL 0.349
 99% Chebyshev (MVUE) UCL 0.438

Gamma Distribution Test

k star (bias corrected) 6.789
 Theta Star 0.0291
 MLE of Mean 0.198
 MLE of Standard Deviation 0.0758
 nu star 108.6

Approximate Chi Square Value (.05) 85.57
 Adjusted Level of Significance 0.0195
 Adjusted Chi Square Value 80.41

Anderson-Darling Test Statistic 0.485
 Anderson-Darling 5% Critical Value 0.715
 Kolmogorov-Smirnov Test Statistic 0.274
 Kolmogorov-Smirnov 5% Critical Value 0.294

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 0.232
 95% Jackknife UCL 0.238
 95% Standard Bootstrap UCL 0.23
 95% Bootstrap-t UCL 0.234
 95% Hall's Bootstrap UCL 0.225
 95% Percentile Bootstrap UCL 0.23
 95% BCA Bootstrap UCL 0.227
 95% Chebyshev(Mean, Sd) UCL 0.29
 97.5% Chebyshev(Mean, Sd) UCL 0.33
 99% Chebyshev(Mean, Sd) UCL 0.409

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.251
 95% Adjusted Gamma UCL 0.267

Potential UCL to Use

Use 95% Student's-t UCL 0.238

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Total Soil ProUCL Input - Ash Pit 3, PBOW

Sample No.	Units	Arsenic	D_Arsenic	Thallium	D_Thallium	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene
AP0032	mg/kg	7.7	1	2.9	0	0.156	0.168	0.268
AP0033	mg/kg	5	1	0.62	0	0.1	0.1	0.1
AP0036	mg/kg	4.9	1	0.63	0	0.105	0.105	0.105
AP0037	mg/kg	12.9	1	6.5	0	0.23	0.23	0.23
AP0039	mg/kg	12.4	1	6.5	0	0.215	0.215	0.215
AP0042	mg/kg	29	1	6	0	0.245	0.245	0.245
AP0043	mg/kg	8.5	1	0.58	0	0.1	0.1	0.1
AP0045	mg/kg	13.7	1	5.2	0	0.215	0.215	0.215
AP0046	mg/kg	20.3	1	2.5	0	0.105	0.105	0.105
AP0048	mg/kg	12.7	1	3	1	0.13	0.13	0.13
AP0049	mg/kg	16.7	1	2.9	1	0.17	0.17	0.17
AP0051	mg/kg	7.7	1	0.92	0	0.155	0.155	0.155
AP0052/0059	mg/kg	8.1	1	0.63	0	0.1075	0.1075	0.1075
AP0061A	mg/kg	30.6	1	0.61	0	0.105	0.105	0.105
AP0054/0056	mg/kg	44.05	1	1.45	0	0.2475	0.2475	0.2475
AP0055	mg/kg	32.7	1	0.5	1	0.105	0.105	0.105

Thallium - Total Soil (AP 3)

General Statistics			
Number of Valid Data	16	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	13
		Percent Non-Detects	81.25%
Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.5	Minimum Detected	-0.693
Maximum Detected	3	Maximum Detected	1.099
Mean of Detected	2.133	Mean of Detected	0.49
SD of Detected	1.415	SD of Detected	1.025
Minimum Non-Detect	0.58	Minimum Non-Detect	-0.545
Maximum Non-Detect	6.5	Maximum Non-Detect	1.872
		Number treated as Non-Detect	16
		Number treated as Detected	0
		Single DL Non-Detect Percentage	100.00%

Note: Data have multiple DLs - Use of KM Method is recommended
 For all methods (except KM, DL/2, and ROS Methods),
 Observations < Largest ND are treated as NDs

Warning: There are only 3 Distinct Detected Values in this data set
The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods.
Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values for bootstrap methods.
However, results obtained using 4 to 9 distinct values may not be reliable.
It is recommended to have 10 to 15 or more observations for accurate and meaningful results and estimates.

Normal Distribution Test with Detected Values Only		UCL Statistics		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.78			Shapiro Wilk Test Statistic	0.764
5% Shapiro Wilk Critical Value	0.767			5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level				Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution				Assuming Lognormal Distribution	
DL/2 Substitution Method				DL/2 Substitution Method	
Mean	1.495			Mean	-0.033
SD	1.256			SD	1.019
95% DL/2 (t) UCL	2.045			95% H-Stat (DL/2) UCL	3.339
Maximum Likelihood Estimate(MLE) Method	N/A			Log ROS Method	
MLE method failed to converge properly				Mean in Log Scale	-0.384
				SD in Log Scale	0.621
				Mean in Original Scale	0.868
				SD in Original Scale	0.827
				95% t UCL	1.231
				95% Percentile Bootstrap UCL	1.233
				95% BCA Bootstrap UCL	1.327
Gamma Distribution Test with Detected Values Only				Data Distribution Test with Detected Values Only	
k star (bias corrected)	N/A			Data appear Normal at 5% Significance Level	
Theta Star	N/A				
nu star	N/A				
A-D Test Statistic	N/A			Nonparametric Statistics	
5% A-D Critical Value	N/A			Kaplan-Meier (KM) Method	
K-S Test Statistic	N/A			Mean	0.908
5% K-S Critical Value	N/A			SD	0.913
Data not Gamma Distributed at 5% Significance Level				SE of Mean	0.323
Assuming Gamma Distribution				95% KM (t) UCL	1.474
Gamma ROS Statistics using Extrapolated Data				95% KM (z) UCL	1.439
Minimum	N/A			95% KM (jackknife) UCL	2.395
Maximum	N/A			95% KM (bootstrap t) UCL	1.213
Mean	N/A			95% KM (BCA) UCL	3
Median	N/A			95% KM (Percentile Bootstrap) UCL	3
SD	N/A			95% KM (Chebyshev) UCL	2.316
k star	N/A			97.5% KM (Chebyshev) UCL	2.925
Theta star	N/A			99% KM (Chebyshev) UCL	4.121

General Statistics

Number of Valid Observations 16

Number of Distinct Observations 11

Raw Statistics

Minimum 0.1
 Maximum 0.248
 Mean 0.156
 Median 0.143
 SD 0.057
 Coefficient of Variation 0.365
 Skewness 0.499

Log-transformed Statistics

Minimum of Log Data -2.303
 Maximum of Log Data -1.396
 Mean of log Data -1.916
 SD of log Data 0.358

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic 0.834
 Shapiro Wilk Critical Value 0.887

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.84
 Shapiro Wilk Critical Value 0.887

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.181

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.182
 95% Modified-t UCL (Johnson-1978) 0.182

Gamma Distribution Test

k star (bias corrected) 6.833
 Theta Star 0.0229
 MLE of Mean 0.156
 MLE of Standard Deviation 0.0598
 nu star 218.7
 Approximate Chi Square Value (.05) 185.4
 Adjusted Level of Significance 0.0335
 Adjusted Chi Square Value 182

Anderson-Darling Test Statistic 1.072
 Anderson-Darling 5% Critical Value 0.74
 Kolmogorov-Smirnov Test Statistic 0.254
 Kolmogorov-Smirnov 5% Critical Value 0.215

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.184
 95% Adjusted Gamma UCL 0.188

Potential UCL to Use

Assuming Lognormal Distribution

95% H-UCL 0.187
 95% Chebyshev (MVUE) UCL 0.218
 97.5% Chebyshev (MVUE) UCL 0.245
 99% Chebyshev (MVUE) UCL 0.298

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL 0.18
 95% Jackknife UCL 0.181
 95% Standard Bootstrap UCL 0.179
 95% Bootstrap-t UCL 0.185
 95% Hall's Bootstrap UCL 0.179
 95% Percentile Bootstrap UCL 0.181
 95% BCA Bootstrap UCL 0.18
 95% Chebyshev(Mean, Sd) UCL 0.219
 97.5% Chebyshev(Mean, Sd) UCL 0.245
 99% Chebyshev(Mean, Sd) UCL 0.298

Use 95% Student's-t UCL 0.181
 or 95% Modified-t UCL 0.182

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Benzo(b)fluoranthene - Total Soil (AP 3)

General Statistics

Number of Valid Observations 16

Number of Distinct Observations 11

Raw Statistics

Minimum 0.1
 Maximum 0.268
 Mean 0.163
 Median 0.143
 SD 0.0635
 Coefficient of Variation 0.39
 Skewness 0.436

Log-transformed Statistics

Minimum of Log Data -2.303
 Maximum of Log Data -1.317
 Mean of log Data -1.887
 SD of log Data 0.388

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic 0.831
 Shapiro Wilk Critical Value 0.887

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.831
 Shapiro Wilk Critical Value 0.887

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.191

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.191
 95% Modified-t UCL (Johnson-1978) 0.191

Assuming Lognormal Distribution

95% H-UCL 0.198

95% Chebyshev (MVUE) UCL 0.233
 97.5% Chebyshev (MVUE) UCL 0.263
 99% Chebyshev (MVUE) UCL 0.322

Gamma Distribution Test

k star (bias corrected) 5.896
 Theta Star 0.0276
 MLE of Mean 0.163
 MLE of Standard Deviation 0.067
 nu star 188.7
 Approximate Chi Square Value (.05) 157.9
 Adjusted Level of Significance 0.0335
 Adjusted Chi Square Value 154.7

Anderson-Darling Test Statistic 1.171
 Anderson-Darling 5% Critical Value 0.74
 Kolmogorov-Smirnov Test Statistic 0.257
 Kolmogorov-Smirnov 5% Critical Value 0.216
 Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.194
 95% Adjusted Gamma UCL 0.198

Potential UCL to Use

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL 0.189
 95% Jackknife UCL 0.191
 95% Standard Bootstrap UCL 0.187
 95% Bootstrap-t UCL 0.193
 95% Hall's Bootstrap UCL 0.187
 95% Percentile Bootstrap UCL 0.189
 95% BCA Bootstrap UCL 0.19
 95% Chebyshev(Mean, Sd) UCL 0.232
 97.5% Chebyshev(Mean, Sd) UCL 0.262
 99% Chebyshev(Mean, Sd) UCL 0.321

Use 95% Student's-t UCL 0.191
 or 95% Modified-t UCL 0.191

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Groundwater ProUCL Input - Ash Pit 3, PBOW

Sample No.	Units	Arsenic	D_Arsenic	Cobalt	D_Cobalt	Iron	D_Iron	Manganese	D_Manganese
AP3083/084	ug/L	1	0	1.15	1	64.05	1	1770	1
AP3086	ug/L	1	0	1	0	23	0	52	1
AP3087	ug/L	1	0	1	0	23	0	151	1
AP3088	ug/L	9.9	1	1	0	2240	1	779	1
AP3091	ug/L	5.2	1	2.1	1	661	1	270	1
AP3092	ug/L	1	0	1.6	1	631	1	472	1

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

From File Sheet1.wst
 Full Precision OFF
 Confidence Coefficient 95%
 Number of Bootstrap Operations 2000

Arsenic - GW (AP3)

General Statistics			
Number of Valid Data	6	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	4
		Percent Non-Detects	66.67%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	5.2	Minimum Detected	1.649
Maximum Detected	9.9	Maximum Detected	2.293
Mean of Detected	7.55	Mean of Detected	1.971
SD of Detected	3.323	SD of Detected	0.455
Minimum Non-Detect	1	Minimum Non-Detect	0
Maximum Non-Detect	1	Maximum Non-Detect	0

Warning: Data set has only 2 Distinct Detected Values.

This may not be adequate enough to compute meaningful and reliable test statistics and estimates.

The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

Unless Data Quality Objectives (DQOs) have been met, it is suggested to collect additional observations.

The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods. Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values for bootstrap methods.

However, results obtained using 4 to 9 distinct values may not be reliable.

It is recommended to have 10 to 15 or more observations for accurate and meaningful results and estimates.

UCL Statistics		Lognormal Distribution Test with Detected Values Only	
Normal Distribution Test with Detected Values Only		Shapiro Wilk Test Statistic	N/A
Shapiro Wilk Test Statistic	N/A	5% Shapiro Wilk Critical Value	N/A
5% Shapiro Wilk Critical Value	N/A		
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2.85	Mean	0.195
SD	3.932	SD	1.391
95% DL/2 (t) UCL	6.085	95% H-Stat (DL/2) UCL	96.91
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	N/A
		SD in Log Scale	N/A
		Mean in Original Scale	N/A
		SD in Original Scale	N/A
		95% t UCL	N/A
		95% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap UCL	N/A
		95% H-UCL	N/A

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	N/A	Data do not follow a Discernable Distribution (0.05)	
Theta Star	N/A		
nu star	N/A		
A-D Test Statistic	N/A	Nonparametric Statistics	
5% A-D Critical Value	N/A	Kaplan-Meier (KM) Method	
K-S Test Statistic	N/A	Mean	5.983
5% K-S Critical Value	N/A	SD	1.752
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.011
		95% KM (t) UCL	8.021

Assuming Gamma Distribution		95% KM (z) UCL	7.647
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	N/A
Minimum	N/A	95% KM (bootstrap t) UCL	N/A
Maximum	N/A	95% KM (BCA) UCL	N/A
Mean	N/A	95% KM (Percentile Bootstrap) UCL	N/A
Median	N/A	95% KM (Chebyshev) UCL	10.39
SD	N/A	97.5% KM (Chebyshev) UCL	12.3
k star	N/A	99% KM (Chebyshev) UCL	16.05
Theta star	N/A		
Nu star	N/A	Potential UCLs to Use	
AppChi2	N/A	95% KM (t) UCL	8.021
95% Gamma Approximate UCL (Use when n >= 40)	N/A	95% KM (% Bootstrap) UCL	N/A
95% Adjusted Gamma UCL (Use when n < 40)	N/A		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006). For additional insight, the user may want to consult a statistician.

Cobalt - GW (AP3)

General Statistics			
Number of Valid Data	6	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	3
		Percent Non-Detects	50.00%
Raw Statistics		Log-transformed Statistics	
Minimum Detected	1.15	Minimum Detected	0.14
Maximum Detected	2.1	Maximum Detected	0.742
Mean of Detected	1.617	Mean of Detected	0.451
SD of Detected	0.475	SD of Detected	0.302
Minimum Non-Detect	1	Minimum Non-Detect	0
Maximum Non-Detect	1	Maximum Non-Detect	0

Warning: There are only 3 Distinct Detected Values in this data set. The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods. Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values for bootstrap methods. However, results obtained using 4 to 9 distinct values may not be reliable. It is recommended to have 10 to 15 or more observations for accurate and meaningful results and estimates.

UCL Statistics		UCL Statistics	
Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.999	Shapiro Wilk Test Statistic	0.997
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	
Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	1.058	Mean	-0.121
SD	0.681	SD	0.655
95% DL/2 (t) UCL	1.619	95% H-Stat (DL/2) UCL	2.675
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	1.027	Mean in Log Scale	-0.0608
SD	0.717	SD in Log Scale	0.621
95% MLE (t) UCL	1.617	Mean in Original Scale	1.099
95% MLE (Tiku) UCL	1.753	SD in Original Scale	0.651
		95% t UCL	1.634
		95% Percentile Bootstrap UCL	1.51
		95% BCA Bootstrap UCL	1.55
		95% H UCL	2.589
Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	N/A	Data appear Normal at 5% Significance Level	
Theta Star	N/A		
nu star	N/A		

A-D Test Statistic	N/A	Nonparametric Statistics	
5% A-D Critical Value	N/A	Kaplan-Meier (KM) Method	
K-S Test Statistic	N/A	Mean	1.383
5% K-S Critical Value	N/A	SD	0.36
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.18
Assuming Gamma Distribution		95% KM (t) UCL	1.746
Gamma ROS Statistics using Extrapolated Data		95% KM (z) UCL	1.68
Minimum	N/A	95% KM (jackknife) UCL	1.788
Maximum	N/A	95% KM (bootstrap t) UCL	1.538
Mean	N/A	95% KM (BCA) UCL	2.1
Median	N/A	95% KM (Percentile Bootstrap) UCL	2.1
SD	N/A	95% KM (Chebyshev) UCL	2.168
k star	N/A	97.5% KM (Chebyshev) UCL	2.508
Theta star	N/A	99% KM (Chebyshev) UCL	3.175
Nu star	N/A		
AppChi2	N/A	Potential UCLs to Use	
95% Gamma Approximate UCL (Use when n >= 40)	N/A	95% KM (t) UCL	1.746
95% Adjusted Gamma UCL (Use when n < 40)	N/A	95% KM (Percentile Bootstrap) UCL	2.1

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006). For additional insight, the user may want to consult a statistician.

Iron - GW (AP3)

General Statistics			
Number of Valid Data	6	Number of Detected Data	4
Number of Distinct Detected Data	4	Number of Non-Detect Data	2
		Percent Non-Detects	33.33%
Raw Statistics		Log-transformed Statistics	
Minimum Detected	64.05	Minimum Detected	4.16
Maximum Detected	2240	Maximum Detected	7.714
Mean of Detected	899	Mean of Detected	6.204
SD of Detected	935.2	SD of Detected	1.484
Minimum Non-Detect	23	Minimum Non-Detect	3.135
Maximum Non-Detect	23	Maximum Non-Detect	3.135

Warning: There are only 4 Distinct Detected Values in this data
 Note: It should be noted that even though bootstrap may be performed on this data set the resulting calculations may not be reliable enough to draw conclusions

It is recommended to have 10-15 or more distinct observations for accurate and meaningful results.

UCL Statistics		Lognormal Distribution Test with Detected Values Only	
Normal Distribution Test with Detected Values Only		Shapiro Wilk Test Statistic	0.909
Shapiro Wilk Test Statistic	0.858	5% Shapiro Wilk Critical Value	0.748
5% Shapiro Wilk Critical Value	0.748	Data appear Lognormal at 5% Significance Level	
Data appear Normal at 5% Significance Level		Assuming Lognormal Distribution	
Assuming Normal Distribution		DL/2 Substitution Method	
DL/2 Substitution Method		Mean	4.95
Mean	603.2	SD	2.257
SD	857.2	95% H-Stat (DL/2) UCL	11111369
95% DL/2 (t) UCL	1308		
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	362.1	Mean in Log Scale	4.904
SD	1061	SD in Log Scale	2.35
95% MLE (t) UCL	1235	Mean in Original Scale	603.3
95% MLE (Tiku) UCL	1300	SD in Original Scale	857.1
		95% t UCL	1308
		95% Percentile Bootstrap UCL	1177
		95% BCA Bootstrap UCL	1341
		95% H UCL	27167556

Gamma Distribution Test with Detected Values Only

k star (bias corrected) 0.409
 Theta Star 2198
 nu star 3.272

A-D Test Statistic 0.318
 5% A-D Critical Value 0.667
 K-S Test Statistic 0.667
 5% K-S Critical Value 0.403

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data
 Minimum 0.000001
 Maximum 2240
 Mean 599.3
 Median 347.5
 SD 860.4
 k star 0.169
 Theta star 3544
 Nu star 2.03
 AppChi2 0.155
 95% Gamma Approximate UCL (Use when n >= 40) 7863
 95% Adjusted Gamma UCL (Use when n < 40) N/A

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006). For additional insight, the user may want to consult a statistician.

Data Distribution Test with Detected Values Only

Data appear Normal at 5% Significance Level

Nonparametric Statistics

Kaplan-Meier (KM) Method
 Mean 620.7
 SD 769.6
 SE of Mean 362.8
 95% KM (t) UCL 1352
 95% KM (z) UCL 1217
 95% KM (jackknife) UCL 1324
 95% KM (bootstrap t) UCL 1638
 95% KM (BCA) UCL 1714
 95% KM (Percentile Bootstrap) UCL 1252
 95% KM (Chebyshev) UCL 2202
 97.5% KM (Chebyshev) UCL 2886
 99% KM (Chebyshev) UCL 4230

Potential UCLs to Use

95% KM (t) UCL 1352
 95% KM (Percentile Bootstrap) UCL 1252

Manganese - GW (AP3)

General Statistics

Number of Valid Observations 6

Number of Distinct Observations 6

Raw Statistics

Minimum 52
 Maximum 1770
 Mean 582.3
 Geometric Mean 333.7
 Median 371
 SD 636.6
 Std. Error of Mean 259.9
 Coefficient of Variation 1.093
 Skewness 1.653

Log-transformed Statistics

Minimum of Log Data 3.951
 Maximum of Log Data 7.479
 Mean of log Data 5.81
 SD of log Data 1.245

Warning: A sample size of 'n' = 6 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 6 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

Shapiro Wilk Test Statistic 0.832
 Shapiro Wilk Critical Value 0.788

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.995
 Shapiro Wilk Critical Value 0.788

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 1106
95% UCLs (Adjusted for Skewness)
 95% Adjusted-CLT UCL (Chen-1995) 1197
 95% Modified-t UCL (Johnson-1978) 1135

Assuming Lognormal Distribution

95% H-UCL 11557
 95% Chebyshev (MVUE) UCL 1901
 97.5% Chebyshev (MVUE) UCL 2462
 99% Chebyshev (MVUE) UCL 3564

Gamma Distribution Test

k star (bias corrected) 0.627
 Theta Star 928.2
 MLE of Mean 582.3
 MLE of Standard Deviation 735.2
 nu star 7.529
 Approximate Chi Square Value (.05) 2.465
 Adjusted Level of Significance 0.0122
 Adjusted Chi Square Value 1.55

 Anderson-Darling Test Statistic 0.16
 Anderson-Darling 5% Critical Value 0.715
 Kolmogorov-Smirnov Test Statistic 0.135
 Kolmogorov-Smirnov 5% Critical Value 0.341

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL (Use when n >= 40) 1778
 95% Adjusted Gamma UCL (Use when n < 40) 2828

Potential UCL to Use

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 1010
 95% Jackknife UCL 1106
 95% Standard Bootstrap UCL 966.3
 95% Bootstrap-t UCL 1736
 95% Hall's Bootstrap UCL 2948
 95% Percentile Bootstrap UCL 1007
 95% BCA Bootstrap UCL 1102
 95% Chebyshev(Mean, Sd) UCL 1715
 97.5% Chebyshev(Mean, Sd) UCL 2205
 99% Chebyshev(Mean, Sd) UCL 3168

Use 95% Student's-t UCL 1106

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

APPENDIX 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 U.S. Environmental Protection Agency (EPA) (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 ($\text{atm}\cdot\text{m}^3/\text{mole}$), 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

(cm²/s), diffusivity in water (D_w) in cm²/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 (ΔH_{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-m³/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):

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

where:

- H = Henry's law constant (atm-m³/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, 2013; NIST, 2013) 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 biomedica 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 (B_p) for SVOCs are estimated by a simple model by Travis and Arms (1988), which is based only on log K_{ow} :

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

where:

B_p = 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 (B_{pr}) and the vegetative parts of plants (B_{pv}) by the Travis and Arms model.

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

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

where:

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

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

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

where:

B_m = 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

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 listings in EPA (2004) 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/m³), for chronic or subchronic inhalation exposure. The inhalation RfC in units of mg/m³ 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 m³/day (EPA, 1989, 1991). In other words, the RfC expressed as mg/m³ is multiplied by the inhalation rate of 20 m³/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 BMD₁₀. The RfD or RfC is derived by dividing the benchmark level (e.g., NOAEL or BMD₁₀) 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 (LD₅₀) or the concentration in air associated with lethality of 50 percent of an exposed population (LC₅₀). 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, 2013). 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 ($\mu\text{g}/\text{m}^3$) 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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{mg}$.

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 (2013) 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, 2007; HSDB, 2013). 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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/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), 2013, 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), 2007, **Toxicological Profile for Arsenic**, U.S. Public Health Service, Atlanta, Georgia, August.

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, 2013). 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, 2007).

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, 2007). 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, 2007). 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, 2007). 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, 2007). 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, 2007). 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, 2013). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico. EPA (2013) 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 (2013) 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, 2013). 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 (2013) 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 (2007) 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 $\mu\text{g}/\text{m}^3$, 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, 2013).

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 microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.										

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
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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CHROMIUM (7440-47-3)

1.0 Introduction and Physical Properties

Chromium is a naturally occurring **metal** (ATSDR, 2012). 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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/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.

^aAgency for Toxic Substances and Disease Registry, (ATSDR), 2012, *Toxicological Profile for Chromium*, U.S. Public Health Service, Atlanta, Georgia, 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, 2012). 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, 2012). 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, 2012). 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, 2012; 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, 2012). 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, 2012). 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, 2012). 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, 2012). 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, 2012). 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, 2012). 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, 2012).

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, 2013).

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, 2012). EPA (2013) 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 (2013) 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 (20113) 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 (EPA, 1998a) revision to a previously verified derivation. EPA (2013) 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.

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, 2013). No adverse effects were observed in workers exposed to 1E-3 mg/m³ for 0.2 to 23.6 years (average 2.5 years). EPA (1998b, 2013) 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 (2013) 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/m³ for chromic acid mists and dissolved chromium (VI) aerosols is based on an adjusted LOAEL of 7.14E-4 mg/m³ 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 (2013) derived an RfC of 1E-4 mg/m³ for chronic inhalation exposure to chromium (VI) particulates from an adjusted benchmark concentration of 3.4E-2 mg/m³ 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/m³ yields a preliminary subchronic inhalation RfC of 2E-5 mg/m³ 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 $3.4E-2 \text{ mg/m}^3$ yields a preliminary subchronic inhalation RfC of $3E-4 \text{ mg/m}^3$ for chromium (VI) particulates. The preliminary subchronic inhalation RfC is equivalent to a subchronic inhalation RfD of $9.7E-5 \text{ mg/kg-day}$.

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 (2013) 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, 2013). 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 (2013) assigned chromium (VI) to Group D for oral exposure.

An inhalation URF of $1.2E-2$ per $\mu\text{g/m}^3$, 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, 2013). 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 (2013) presented the URF of $1.2E-2$ per $\mu\text{g/m}^3$ 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	ND	3E-4/ 9.7E-5	1E-4/ 2.9E-5	Lu	D	ND	A	1.2E-2	4.2E+1
Total Chromium ^c										
NA	2.1E-2	ND	NA	7E-4/ 2.0E-4	Lu	D	ND	A	1.2E-2	4.2E+1
<p>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.</p> <p>Target organ or critical effect abbreviations: Lu = lung.</p> <p>^asRfDo and cRfDo should be adjusted by the appropriate GAF values described above when used for dermal exposure.</p> <p>^bInhalation 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.</p> <p>^cFor 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 2013; EPA, 2004b).</p>										

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COBALT (7440-48-4)

1.0 Introduction and Physical Properties

Cobalt is a relatively scarce **metal**, constituting about 0.001 percent of the earth's crust (Elinder and Friberg, 1986). It often occurs in association with nickel, silver, lead, copper and iron ores. Cobalt is used in forming special metal alloys with unusual and useful properties (ATSDR, 2004; HSDB, 2013). It is also used in cutting materials, lacquers, varnishes, paint driers, enamels, inks, glazes, glass manufacture, as a catalyst in petroleum refining, and as a supplement in animal and human nutrition. Relevant physical properties are compiled below:

MW	log K _{ow}	H	K _d	D _a	D _w	VP	S
58.93 ^a	NA	NA	45 ^b	NA	NA	ND	Note 1 ^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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/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: Cobaltous chloride as a typical cobalt salt: soluble.

^aHazardous Substance Data Bank (HSDB), 2013, National Library of Medicine, on line.

^bBaes, C.F., R.D. Sharp, H. Sjoreen, and R.W. Shor, *A Review and Analysis for Assessing Transport of Environmentally Released Radionuclides through Agriculture, ORNL-5786, Oak Ridge National Laboratory*.

^cBudavari, S, Ed., 1989, *The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals*, Eleventh Edition, Merck & Co., Rahway, NJ.

2.0 Environmental Fate and Transport

Cobalt is released to the environment, particularly to the air, largely from industrial processes that involve grinding or polishing hard metal devices that contain cobalt (HSDB, 2013). Other anthropogenic sources include fossil fuel burning, vehicular and aircraft exhaust, production and use of cobalt-containing alloys and chemicals, copper and nickel smelting and refining, disposal of sewage sludge, and application of fertilizers derived from phosphate rock (ATSDR, 2004). Natural sources include soil and soil dust, seawater spray, and volcanic eruptions. Natural releases to the atmosphere slightly exceed anthropogenic releases.

Ionic cobalt compounds exist in the particulate phase in air and these compounds may be removed from the air by wet and dry deposition (HSDB, 2013).

HSDB (2013) reports that K_d values for cobalt range from 0.2 to 3,800 L/kg. The value of 45 L/kg presented above appears to be a reasonable intermediate point. Soils with higher pH and content of clay, natural organics, and hydrous manganese and iron oxides, bind cobalt to a greater degree; as these factors decrease, the mobility of cobalt increases. Microbial activity may increase the solubility of cobalt in soil. Chelating agents increase the solubility of cobalt and enhance its mobility in soil. Cobalt compounds do not volatilize from moist or dry soil surfaces due to their ionic character.

The transport and speciation of cobalt in natural waters and sediments is complicated by many factors (HSDB, 2013). The presence of sewage-derived organics appears to enhance the solubility of cobalt in freshwater by forming soluble complexes. Cobalt exists in the +2 or +3

oxidation state for the majority of its compounds and complexes. Volatilization from water surfaces is not expected, due to the ionic nature of dissolved cobalt compounds.

A few plant species are known to accumulate cobalt; however, translocation to aerial parts is generally insignificant (ATSDR, 2004; HSDB, 2013). Cobalt is bioaccumulated by aquatic organisms, particularly at the lower trophic levels. Biomagnification, however, does not occur and cobalt localizes in the viscera and on the skin, rather than in edible tissues. Therefore, cobalt is not expected to impact human health through the food-chain pathways and biotransfer factors are not provided.

3.0 Toxicokinetics

Inhaled cobalt particles deposit in the upper or lower respiratory tract, depending largely on particulate size (ATSDR, 2004). Uptake may occur by dissolution or phagocytosis; larger particles are removed by ciliary clearance. The extent of pulmonary uptake depends largely on particle size distribution.

GI absorption of ingested cobalt in humans ranges from 18 to 97 percent, depending on the form and dose, and on the nutritional status of the subjects (ATSDR, 2004). Cobalt is absorbed more efficiently in cases of iron deficiency. Animal studies show that soluble forms such as cobalt chloride are absorbed to a much greater extent (13 to 34 percent) than insoluble cobalt oxide (1 to 3 percent).

Experiments with humans indicate that cobalt in hard metal dust can be absorbed through the skin, although the rate or extent of uptake were not quantified (ATSDR, 2004). Experiments in animals indicate that cobalt is absorbed much more efficiently through abraded than intact skin.

Distribution of cobalt within body tissues depends in large measure on the route of exposure (ATSDR, 2004). Cobalt as a component of cyanocobalamin (vitamin B12) is generally distributed among the body tissues. Highest levels following inhalation exposure are found in the tissues and lymph nodes of the respiratory tract. Lesser but elevated levels are found in liver, spleen and kidney. Highest levels are found in the liver following ingestion exposure.

Elimination also depends on route of exposure (ATSDR, 2004). Inhaled particulates are cleared by mucociliary elevation, phagocytosis and dissolution. Elevated particles are swallowed and subjected to GI absorption or throughput in the feces. Absorbed cobalt is excreted primarily through the urine.

4.0 Dermal Exposure

The provisional chronic oral RfD of 3E-4 for cobalt was derived from a study in which humans were treated orally with cobalt (EPA, 2008). A GAF of 1, as recommended by EPA (2004) is selected for cobalt.

Data were not located regarding the extent of dermal uptake of cobalt 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 (2004) K_p for cobalt of 4E-4 cm/hour is selected for cobalt.

5.0 Noncancer Effects Evaluation

Human health effects following oral exposure to cobalt include decreased iodine uptake by the thyroid, dermatitis in sensitized individuals, increased erythrocyte production and hemoglobin levels, and cardiomyopathy. Related effects to each of these, except dermatitis, are supported by animal studies. Other effects observed in animal studies include neurobehavioral and testicular; these effects were observed only at relatively high doses. Thyroid toxicity was selected as the critical effect for the derivation of provisional oral reference doses.

Cobalt is nutritionally essential as a cofactor in cyanocobalamin (ATSDR, 2004). The element is ubiquitous and universally present in the diet. Data from a dietary survey performed in the 1980s indicate that daily U.S. dietary intakes of cobalt are in the range of $3\text{E-}3$ to $1.1\text{E-}2$ mg/day ($4\text{E-}5$ to $2\text{E-}4$ mg/kg-day, assuming an average body weight of 70 kg). Chronic ingestion from the consumption of beer containing high concentrations of cobalt is associated with "beer-cobalt cardiomyopathy," which includes polycythemia and goiter, as well as marked myocardial degeneration and mortality. However, confounding variables were identified and a dose-response could not be derived from the associated studies (EPA, 2008).

A consistently produced effect of exogenously administered cobalt in humans is the erythropoietic effect of stimulating erythrocyte production resulting in increased blood hemoglobin concentration (EPA, 2002). Cobalt chloride has been used therapeutically at dose rates of $1.6\text{E-}1$ to $3.2\text{E-}1$ mg cobalt/kg-day in anemic, anephric dialysis patients for 12 to 32 weeks to induce a significant, but reversible, rise in blood hemoglobin concentration. Clinical studies demonstrating this effect identified an effect level of $1.8\text{E-}1$ mg/kg-day, which EPA (2002) had previously designated a LOAEL. However, EPA (2008) has since identified confounding variables, and has judged the effects of cobalt in some of the patients in pertinent studies as beneficial rather than adverse. A LOAEL of 1 mg/kg-day for subchronic thyroid toxicity as the critical oral effect was identified by EPA (2008) from a subchronic study by Roche and Layrisse (1956). A provisional subchronic RfD of $3\text{E-}3$ mg/kg-day was derived assuming a composite UF of 300. This assumes a UF of 10 for LOAEL to NOAEL conversion, and a UF of 10 for inter-individual variability. An additional UF of 3 was added due to a lack of multigenerational studies, since testicular degeneration and sperm function were noted in some animal studies.

The provisional chronic oral RfD of $3\text{E-}4$ mg/kg-day was derived as described for the subchronic RfD, with an additional UF of 10 added to extrapolate from subchronic to chronic exposure for a composite UF of 3,000 (EPA, 2008). Confidence in the principal study is described as low-to-medium.

The human and animal database indicate that symptoms of inhaled cobalt include respiratory tract irritation and altered pulmonary function. These were identified as the critical effects for exposure to inhaled cobalt for the derivation of RfDs. A NOAEL adjusted for continuous exposure of $1.9 \mu\text{g}/\text{m}^3$ was identified by EPA (2008). A subchronic RfC of $2\text{E-}5$ mg/ m^3 was derived by EPA (2008) assuming a composite UF of 100, assuming a UF of 10 for database insufficiencies and a UF of 10 for inter-individual variability. The preliminary subchronic inhalation RfC is equivalent to a preliminary subchronic inhalation RfD of $5.7\text{E-}5$ mg/kg-day.

A provisional chronic RfC of 6E-6 mg/m³ was derived as described above for the subchronic RfC, but with an additional UF of 3 for the extrapolation from subchronic to chronic exposure (EPA, 2008). Thus, the composite UF is 300. This provisional RfC equals an inhalation RfD of 1.7E-6 mg/kg-day.

6.0 Carcinogenicity Evaluation

Human or animal studies examining the carcinogenicity of cobalt associated with oral exposure were not located. Therefore, cobalt is classified as having “inadequate information to assess carcinogenic potential” via the oral pathway, thus no cancer slope factor was derived (EPA, 2008).

Cobalt is described as “likely to be carcinogenic to humans by the inhalation route,” based on limited evidence of carcinogenicity in humans (occupational studies) and sufficient evidence of carcinogenicity in animals (EPA, 2008). Respiratory tract tumors have been suggested in occupational studies as the result of inhaled cobalt. Statistically increased incidence of respiratory tract tumors have been observed in animal studies. Limited evidence supports genotoxicity and cytotoxicity, followed by cellular regeneration as potential modes of cobalt tumorigenicity. A mutagenic effect may be plausible for inhaled cobalt, but has not been clearly established (EPA, 2008).

Human inhalation studies were not sufficiently detailed to use in the derivation of a URF. Rats and mice exposed via inhalation to cobalt sulfate heptahydrate for 2 years developed alveolar and bronchial lung tumors (neoplasms and adenomas). EPA (2008) identified a benchmark dose level of In the absence of an identified mode of action, EPA developed a provisional URF by linear extrapolation of benchmark dose level of 0.011 mg/m³. Using a benchmark response of 10 percent in extra risk, a provisional URF of 9E-3(μg/m³)⁻¹ was derived for cobalt sulfate. This equals an inhalation SF of 3.2E+1 (mg/kg-day)⁻¹.

7.0 Toxicity Summary

Toxicity values for cobalt are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	Woe	SFo	Woe	URFi	SFi
3E-3	3E-4	Thyroid	2E- 5/5.7E-5	6E-6/ 1.7E-6	RT	ND	ND	B	9E-3	3.2E+1
<p>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.</p> <p>Target organ or critical effect abbreviations: RT = respiratory tract.</p>										

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IRON (7439-89-6)

1.0 Introduction and Physical Properties

Iron is a very abundant, ubiquitous, naturally occurring **metal** that constitutes approximately 5 percent of the earth's crust (Spivey Fox and Rader, 1988). It is the second most prevalent metal in the earth's crust following aluminum (HSDB, 2013). Its primary use is in making steels and other metals that have a wide range of application. Less than one percent of iron is used to make iron compounds that have application in dyes and pigments, water treatment, organic chemical synthesis, and medicinal preparations. Relevant physical properties are compiled below:

MW	log K _{ow}	H	K _d	D _a	D _w	VP	S
55.85 ^a (Element)	NA	NA	25 ^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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/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: Ferrous sulfate as a typical iron salt: soluble.

^aHazardous Substance Data Bank (HSDB), 2013, National Library of Medicine, on line.

^bBaes, C.F., R.D. Sharp, H. Sjoreen, and R.W. Shor, *A Review and Analysis for Assessing Transport of Environmentally Released Radionuclides through Agriculture, ORNL-5786, Oak Ridge National Laboratory.*

2.0 Environmental Fate and Transport

There is little information available regarding the anthropogenic release of iron and its fate in the environmental, probably because of its prevalence in nature. Clearly, however, mining and handling of iron ores releases dust containing iron oxides (HSDB, 2013). Presumably the manufacture and use of iron compounds may result in release to the environment, but data were not located.

Insoluble iron compounds are most likely to occur in air as particulates, and are expected to deposit to soil and surface water (HSDB, 2013). Although data are unavailable, soluble compounds are expected to leach to groundwater, and to remain in solution in both groundwater and surface water. The extent of partitioning to sediment is expected to depend on water pH, organic matter and reduction-oxidation potential of the water system.

Biouptake of iron is known to occur, because the element is a nutritionally required trace element that is a structural component of heme proteins and some enzymes, which are common to mammals and several other orders of living organisms (Spivey Fox and Rader, 1988). However, there is no evidence that iron bioconcentrates in either aquatic or terrestrial food chain pathways. Therefore, biotransfer factors for iron are not presented.

3.0 Toxicokinetics

The GI absorption of iron is regulated by homeostatic mechanisms sited largely in the intestinal mucosa (Goyer, 1991; EPA, 2006). Approximately 2 to 15 percent of dietary iron is ordinarily absorbed; however, several dietary factors may increase or decrease absorption (HSDB, 2013). Dietary iron from animal sources is absorbed more readily (10 to 25 percent) than iron from vegetables and grains (1 to 10 percent) (Elinder, 1986). Absorption is greatly increased at times

of unusual need (rapid growth of childhood, pregnancy, blood loss). Divalent (ferrous) iron is absorbed into the intestinal mucosal cells and released to the blood plasma where it is oxidized to the trivalent (ferric) form, which binds with a specific protein, transferrin, for transport to storage sites.

Approximately 67 percent of iron in the body is associated with hemoglobin in the erythrocytes, 10 percent is associated with myoglobin and various iron-containing enzymes, and the remainder is bound to ferritin or hemosiderin, which are intracellular storage forms (Goyer, 1991). Ferritin and hemosiderin bind iron in a manner that renders it inactive for beneficial use (e.g., hemoglobin synthesis) and inactive as a toxicant. Some of the iron taken up by the cells of the reticuloendothelial system remains stored and some enters a labile pool that is available for erythropoiesis.

Excess iron is generally excreted, in part in shed intestinal epithelial cells, or in the bile or urine (Goyer, 1991). In cases of very high iron intake, excretion may be unable to remove iron as quickly as it is absorbed. Iron overload results, which stimulates increased synthesis of ferritin and increased iron storage, particularly in the liver, pancreas, spleen, various endocrine organs and the heart.

4.0 Dermal Exposure

As noted above, the GI absorption of iron appears to be under homeostatic control in normal humans (EPA, 2006), which suggests that GI absorption could vary widely between individuals depending on nutritional status. Given the uncertainty about the GI absorption of iron, a default GAF of 1 (EPA, 2004) is chosen for this evaluation.

Data were not located regarding the extent of dermal uptake of iron 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 iron from water were not located. The EPA (2004) default K_p for inorganic chemicals of $1E-3$ cm/hour is selected for iron.

5.0 Noncancer Effects Evaluation

Iron is a nutritionally required trace element that forms an integral part of hemoglobin, myoglobin and several enzymes (Spivey Fox and Rader, 1988). The NAS (1989) Recommended Daily Allowance (RDA) for iron is 10 mg/day (0.13 mg/kg-day) for adult males; 15 mg/day (0.24 to 0.33 mg/kg-day) for females aged 11 to 50 years; 30 mg/day (0.443 mg/kg-day) for pregnant females; and 10 mg/day (0.36 to 1.11 mg/kg-day) for children aged 6 months to 10 years.

Both acute and chronic toxicity syndromes are associated with ingestion exposure to iron. Acute toxicity generally involves children (Goyer, 1991). Acute ingestion of large quantities (e.g., children may ingest several ferrous sulfate tablets with candy-like coating) results in severe GI irritation and, in some cases, liver damage. Liver cirrhosis and blood coagulation defects (indicative of reduced circulating levels of blood clotting factors produced by the liver) are observed as sequelae to severe acute toxicity.

Three chronic toxicity syndromes that result in iron overload have been observed in humans (Goyer et al., 1991). The first chronic syndrome is idiopathic hemochromatosis, which arises from a genetic derangement of the homeostatic mechanisms that control iron absorption. This syndrome involves greatly increased GI absorption of iron, regardless of the level of iron in the diet, which results in massive iron overload.

The second chronic syndrome is iron overload resulting from high dietary intake (Goyer, 1991). Although high dietary intake seldom results in iron overload in humans with normal homeostatic control of iron absorption, rare exceptions occur (EPA, 2006). One such exception is the occurrence of iron overload in Bantus who regularly consumed Kaffir beer, an acid beer brewed in iron vessels. This syndrome is not entirely understood; however, the iron in Kaffir beer appears to have exceptionally high bioavailability – equivalent to that of nutritional iron supplements especially formulated for bioavailability – which may account for the increased absorption and iron overload. Other exceptions include persons who take large amounts of tonics or nutritional supplements containing iron.

The third chronic syndrome involves persons requiring multiple blood transfusions over a period of time (Goyer, 1991). This results in the parenteral administration of large quantities of iron that are released as the transfused erythrocytes expire and are removed from the circulation by the reticuloendothelial system. As noted above, excretion cannot keep up with unusually high circulating levels of iron, such as results from multiple blood transfusions. Clearly, the third syndrome has no relevance to environmental exposure.

Regardless of the chronic syndrome that produces it, the effects of iron overload involve the same pathogenesis (Goyer, 1991). Iron stores in the body increase from the normal level of 3 to 5 g, to levels of 20 to 40 g. The cells are induced to increase production of ferritin, which binds the iron cation inactivating its toxicity. Increasingly larger proportions of iron are located in hemosiderin than in ferritin. Hemosiderin is a larger storage protein, probably formed primarily from ferritin. The iron in hemosiderin is essentially completely unavailable and nontoxic. Hemosiderosis is the term given to large accumulations of hemosiderin that do not result in adverse effects because the iron is unavailable. Extremely large accumulations of hemosiderin that result in impaired cellular function is termed hemochromatosis. Hemochromatosis of the liver, endocrine glands and heart is associated with reduced liver function, eventually liver cirrhosis, pancreatic fibrosis, diabetes mellitus, other endocrine disruption and cardiovascular effects.

Goyer (1991) and EPA (2006) reviewed the available data regarding the toxicity of iron. Neither source located any data clearly associating *environmental* exposure with toxicity. EPA (1996) reviewed a study associating high circulating ferritin levels and high dietary iron intakes with myocardial infarction in Finnish men, but association with other factors (e.g., high levels of red meat consumption) confounds interpretation of this study. Furthermore, attempts to produce an animal model of chronic iron toxicity have failed, presumably because of the efficacy of the homeostatic mechanisms that regulate iron absorption from the GI tract.

EPA (1996) reasoned previously that a NOEL for chronic iron overload could be inferred from the estimates of dietary intake and iron status (circulating ferritin levels) established by the second National Health and Nutrition Examination Survey (NHANES II) data base. The NHANES II study determined that average dietary iron intakes, which ranged from 0.15 to 0.27

mg/kg-day, were associated with normal iron status. More recently, EPA (2006) has concluded that the assignment of a LOAEL based on iron overload for normal individuals cannot be determined based on existing data because of confounding factors in the studies.

EPA (2006) has determined that gastrointestinal toxicity, commonly associated with therapeutic use of iron supplements, is the critical effect. A provisional subchronic and chronic oral RfD of 0.7 mg/kg-day was derived, based on a LOAEL of 1 mg/kg-day and a UF of 1.5 (EPA, 2006). The same value was derived for both subchronic and chronic exposures because clinical experience with iron supplements indicates that the gastrointestinal irritation is associated with treatment and does not intensify with duration. Further, the effect is reversible once treatment is discontinued. A higher UF was not selected because of the reversibility and the effect is not regarded as serious (EPA, 2006).

Inhalation (occupational) exposure to iron oxide fumes may lead to radiographic densities in the lungs without demonstrable clinical effects (ACGIH, 1991). Inhalation of dusts or mists of ferric salts may irritate the respiratory tract. Inhalation data are insufficient for derivation of an inhalation RfC or RfD (EPA, 2005a).

6.0 Carcinogenicity Evaluation

Data regarding the potential carcinogenicity of iron were not located. Neither a cancer weight-of-evidence classification nor cancer potency factors are available (EPA, 2005b).

7.0 Toxicity Summary

Toxicity values for iron 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
7E-1	7E-1	GI	ND	ND	NA	ND	ND	ND	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; NA = not applicable. Target organ or critical effect abbreviations: GI = gastrointestinal. ^a sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.										

In most cases, it is inappropriate to apply the toxicity values summarized above to environmental exposure.

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INORGANIC MANGANESE (7439-96-5)

1.0 Introduction and Physical Properties

Manganese is a naturally occurring **metal** found in the earth's crust in the form of numerous minerals such as pyrolusite, romanechite, manganite and hausmannite (ATSDR, 2012; HSDB, 2013; Keen and Leach, 1988). It occurs ubiquitously in the environment, constituting approximately 0.085 percent of the earth's crust, commonly in valence states of +2, +4 and +7; valences of +1, +3, +5 and +6 are rare. Manganese is used in the manufacture of steel, and in other metallurgical processes, batteries, and various manganese-containing chemicals including matches, glass and porcelain, fireworks, varnishes, ceramics, diagnostic contrast media and fungicides. Relevant physical properties are compiled below:

MW	log K _{ow}	H	K _d	D _a	D _w	VP	S
54.938 ^a (Elemental)	NA	NA	65 ^b	NA	NA	NA	Note 1 ^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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.
Note 1: Metallic manganese decomposes in the presence of water. Manganous chloride as a typical manganese salt: 7.23E+5 mg/L (25° C).
^aHazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
^bBaes, 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.
^cAgency for Toxic Substances and Disease Registry, (ATSDR), 2000, **Update Toxicological Profile for Manganese**, U.S. Public Health Service, Atlanta, Georgia, September, on line.

2.0 Environmental Fate and Transport

Inorganic manganese compounds enter the atmosphere and other environmental compartments from the weathering of rocks and windblown soil (HSDB, 2013). Anthropogenic sources of atmospheric manganese include metal processing, disposal and use of manganese-containing materials (antiseptics, catalysts, dietary supplements, dry cells, feed additives, fertilizers, pesticides and pigments), resuspension of manganese-containing soil dust, and fly ash emissions from incinerators (ATSDR, 2012; HSDB, 2013). Another source of atmospheric manganese is the combustion of gasoline containing organic manganese anti-knock ingredients such as methylcyclopentadienyl manganese tricarbonyl (MMT). It is unclear whether manganese from this source remains as an organic form or is converted to an inorganic form. The use of MMT in gasoline has been discontinued because of deleterious effects on catalytic converters. Manganese may be released to water by discharge from industrial facilities or in leachate from landfills and soil. Landfill disposal of manganese-containing wastes is the predominant source of manganese release to soil.

Manganese exists in the air bound to particles; dry deposition is the primary removal mechanism, although wet deposition may also be significant (ATSDR, 2012). Manganese in fly ash exists as chlorides and oxides that are relatively soluble and mobile in the environment.

Manganese in water exists as any of several sparingly soluble salts that attach to suspended sediment (ATSDR, 2000). Sedimentation is the primary removal process. Soluble forms also exist, depending on pH of the water, and may be released from sediment. The extent of

absorption to constituents of soil is highly variable. Low concentrations may irreversibly bind to clay, but higher concentrations often manifest considerable mobility.

Manganese may accumulate in various kinds of plants such as legumes, nuts, heather, and tea, and in the lower aquatic trophic levels, but not significantly in the higher aquatic trophic levels or most terrestrial animals (ATSDR, 2012; HSDB, 2013). This indicates that food-chain pathways are unlikely to be significant for human health; therefore, biotransfer factors are not compiled or estimated.

3.0 Toxicokinetics

Manganese is nutritionally essential for humans (ATSDR, 2012). Stable tissue levels are maintained by homeostatic regulation of GI absorption and biliary excretion.

Manganese is absorbed by the respiratory tract (ATSDR, 2012), although data quantifying the extent of absorption were not located. Smaller particles are probably retained in the lung eventually to be absorbed; larger particles are probably removed by mucociliary elevation and swallowed so that they become available for GI absorption. The more soluble compounds are more readily absorbed by the respiratory tract. Studies in rats given radiolabeled manganese by intranasal administration suggest that olfactory uptake and direct transmission to the brain may occur.

GI absorption of manganese in humans averages 3 to 5 percent, but homeostatic mechanisms reduce the extent of absorption at higher doses (ATSDR, 2012). The more soluble compounds are more readily absorbed. Iron deficiency enhances manganese uptake. Absorption appears to be more efficient and resulting tissue levels appear to be higher in infants than adults.

Quantitative data regarding dermal uptake of manganese were not located.

Normal tissue levels of manganese in humans range from 0.1 to 1 mg/kg wet weight with highest levels in liver, pancreas and kidney, and lowest levels in bone and fat (ATSDR, 2012). Animal studies (inhalation, intranasal, intratracheal and oral administration) confirm that many tissues including the brain, and in some instances specific regions of the brain, show significant increases in manganese levels after treatment. Tissue levels recede toward normal after treatment ceases.

Metabolism of manganese appears to be limited to oxidation-reduction changes in valence state (ATSDR, 2000). The evidence suggests that manganese (+2) is oxidized to manganese (+3), which alters its tissue distribution and slows excretion. Manganese is removed from the blood in the liver and is excreted principally through the bile. Urine, sweat and milk represent minor excretory routes. Animal studies show that the young are less efficient than adults in excreting manganese, although the young do not appear to be more sensitive to its effects.

4.0 Dermal Exposure

Data were not located regarding the extent of dermal uptake of manganese 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 manganese from water were not located. ATSDR (2012), however, notes that the uptake of manganese across intact skin is insignificant with respect to exposure, suggesting that uptake of toxicologically significant amounts is unlikely. EPA (2004) provides no chemical-specific estimate of K_p for manganese; therefore, the default K_p value of $1E-3$ cm/hr is used to estimate dermal uptake of manganese from water.

5.0 Noncancer Effects Evaluation

Manganese is nutritionally required by humans for normal growth and health (EPA, 2010). Humans exposed to approximately 0.8 mg manganese/kg-day in drinking water (28 mg/L) exhibited lethargy, increased muscle tonus, tremor and mental disturbances. The elderly appeared to be more sensitive than children. Oral treatment of laboratory rodents induces biochemical changes in the brain, but rodents do not exhibit the neurological signs exhibited by humans.

EPA (2013) verified a chronic oral RfD for manganese of $1.4E-1$ mg/kg-day from a NOAEL of $1.4E-1$ mg/kg-day for neurologic effects from human dietary studies and an uncertainty factor of 1. Confidence in the RfD is medium. The oral RfD of $1.4E-1$ mg/kg-day will be used for dietary items other than drinking water. EPA (2013) recommends that the contribution of background diet should be subtracted and that a modifying factor of 3 should be used to adjust for use when oral exposure involves drinking water and non-dietary ingestion. The oral RfD resulting from these manipulations is $2.4E-2$ mg/kg-day, which will be used for drinking water and incidental ingestion of non-dietary items. The CNS is the target organ for chronic oral exposure to manganese. EPA (1997) effectually adopts the chronic oral RfD as sufficiently protective for subchronic oral exposure.

The NOAEL described above is associated with a dose rate of approximately 10 mg/day for an adult. However, EPA (2002) notes that normal intake levels may well exceed 10 mg/day, especially with diets containing substantial amounts of whole-grain cereals, nuts, green leafy vegetables, and tea. Adverse effects have not been reported at dietary levels of 10 mg/day. It is likely that larger dose rates are innocuous as well.

Occupational exposure to high concentrations in air induces a generally typical spectrum of neurological effects, and increased incidence of pneumonia (ACGIH, 1991). EPA (2013) presents a verified chronic inhalation RfC of $5E-5$ mg/m³ (equivalent to an inhalation RfD of $1.4E-5$ mg/kg-day) based on a LOAEL for neurological effects in occupationally exposed humans and an uncertainty factor of 1000. The uncertainty factor consists of three factors of 10 each to provide additional protection for unusually sensitive individuals, to extrapolate a NOAEL from a LOAEL, and to address database limitations including the less-than-chronic periods of exposure, the lack of developmental toxicity data, and uncertainty regarding potential but unquantified differences in the toxicity of different forms of manganese. Confidence in the RfC is medium. The CNS is the target organ for inhalation exposure to manganese. The chronic inhalation RfC is adopted as sufficiently protective for subchronic inhalation exposure.

6.0 Carcinogenicity Evaluation

EPA (2013) classified manganese in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans). Quantitative cancer risk estimates are not derived for Group D chemicals.

7.0 Toxicity Summary

Noncancer Effects						Carcinogenicity				
Oral Exposure			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1.4E-1 ^a / 2.4E-2 ^b	1.4E-1 ^a / 2.4E-2 ^b	CNS	5E-5/ 1.4E-4	5E-5/ 1.4E-5	CNS	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 or critical effect abbreviations: CNS = central nervous system. ^a Dietary items other than drinking water. ^b Ingestion of environmental media and drinking water.										

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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, 2013). 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, 2013). 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·m³/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm²/second); D_w = diffusivity in water (cm²/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).
^aAgency for Toxic Substances and Disease Registry, (ATSDR), 1992, *Toxicological Profile for Thallium*, U.S. Public Health Service, Atlanta, Georgia, July, on line.
^bU.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.
^cpH = 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, 2013). 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, 2013). 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, 2013). 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 mucociliary 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 (2013) 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, 2013). 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 treated 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, 2000).

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, 2013); 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, 2013). EPA (2013) had previously 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; these RfDs were removed from IRIS in September 2009. Currently, no RfD exists for thallium, thallium salts, or thallium compounds because of insufficient toxicity

data (EPA, 2013). Toxicity data were also insufficient for the development of a peer-reviewed provisional toxicity value (PPRTV) RfD (EPA, 2012).

The previous oral RfD that had been listed on IRIS 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) is considered the target organ for oral exposure to thallium, based on other data reviewed by EPA (2013). 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 is 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, 2013). 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,	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 =

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
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: S = skin (alopecia). ^a sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.										

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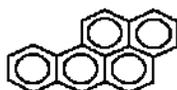
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Semivolatile Organic Compounds

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, 1995; HSDB, 2013). 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, 2005). Relevant physical properties are compiled below:

MW	log K _{ow}	H	log K _{oc}	D _a	D _w	VP	S	T _b	T _c	ΔH _{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 ^a (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	4.57E-7 ^f (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	6.57E-7 ^f (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 ^a (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	ΔH _{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	4.40E-4 ^k (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
<p>MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m³/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 (cm²/second); D_w = diffusivity in water (cm²/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.</p> <p>^aHazardous Substance Data Bank (HSDB), 2013, National Library of Medicine, on line.</p> <p>^bU.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.</p> <p>^cU.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.</p> <p>^dCalculated as described in Introduction to Toxicological profiles.</p> <p>^eAssumed.</p> <p>^fEstimated 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.</p> <p>^gEstimated 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.</p> <p>^hU.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.</p> <p>ⁱU.S. Environmental Protection Agency (EPA), 2012, <i>EPISuite electronic database</i></p> <p>^jNational Institute of Standards and Technology (NIST), 2004, Standard Reference Data Program, Online Data Bases.</p>										

2.0 Environmental Fate and Transport

The PAHs are ubiquitous products of incomplete combustion; natural sources include volcanoes and forest fires (ATSDR, 1995, 2005; HSDB, 2013). 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

Source ^a	Individual PAHs
Diesel total emissions	Acenaphthene, fluorene, phenanthrene
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, Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) , U.S. Public Health Service, Atlanta, Georgia, August.	

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, 1995). 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, 1995; 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 (1995) 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, 1995; HSDB, 2013). 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, 1995; 2005). 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-m³/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, 1995; 2005). 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, 1995). 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, 1995). 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, 1995). 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, 1995). 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 (1995) 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, 1995). 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, 2005).

Distribution of absorbed PAHs is generally widespread, with highest levels located initially in lipid-rich tissues (ATSDR, 1995). 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, 1995). 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 (1995) 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, 1995). 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, 1995). 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, 1995). 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, 1995). 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, 2005). 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).

^aEstimated as described in Introduction to Toxicological profiles.

^bProvided 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, 1995). 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. (2008) 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 LD₅₀ 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, 2005). 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 $3\text{E-}3 \text{ mg/m}^3$, which is equivalent to a chronic inhalation RfD of $8.6\text{E-}4 \text{ mg/kg-day}$. 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 $3\text{E-}3 \text{ mg/m}^3$, equivalent to an inhalation RfD of $8.6\text{E-}4 \text{ mg/kg-day}$, 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 $3\text{E-}2 \text{ mg/kg-day}$ 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 $3\text{E-}1 \text{ mg/kg-day}$ 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, 2013). 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, 2013). 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, 1995). 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, 1995).

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, 2013). 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 (2013) 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				
PAH	Relative Potency	Oral Slope Factor (per mg/kg-day)	Inhalation	
			Unit Risk Factor (per $\mu\text{g}/\text{m}^3$)	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 (2013) 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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APPENDIX D

RISK CHARACTERIZATION SPREADSHEETS

Table D-1

Groundskeeper, Current Scenario, Exposure to Surface Soil
 Ash Pit No. 3
 Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

Chemicals of Potential Concern	Incidental Ingestion					Dermal Contact				
	EPC mg/kg	Cancer	Noncancer	ILCR	HQ	Dose	Cancer	Noncancer	ILCR	HQ
		Dose	Dose			Absorbed	Dose	Dose		
Inorganics										
Arsenic	2.88E+01	1.01E-05	2.82E-05	1.51E-05	9.40E-02	1.73E-07	1.99E-06	5.58E-06	2.99E-06	1.86E-02
Thallium	2.69E+00	9.38E-07	2.63E-06	NA	4.04E-02	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	5.45E-08	1.53E-07	3.98E-08	NA	4.06E-09	4.68E-08	1.31E-07	3.41E-08	NA
Benzo(a)pyrene	1.68E-01	5.87E-08	1.64E-07	4.29E-07	NA	4.37E-09	5.04E-08	1.41E-07	3.68E-07	NA
Benzo(b)fluoranthene	2.38E-01	8.32E-08	2.33E-07	6.07E-08	NA	6.19E-09	7.14E-08	2.00E-07	5.21E-08	NA
Total ILCR or HI				1.56E-05	1.34E-01				3.44E-06	1.86E-02

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/m³ - Milligrams per cubic meter.

Table D-1

Groundskeeper, Current Scenario, Exposure to Surface Soil
 Ash Pit No. 3
 Plum Brook Ordnance Works, Sandusky, Ohio

(Page 2 of 2)

Chemicals of Potential Concern	Concentration in Air C _a mg/m ³	Inhalation				All Pathways	
		Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Total ILCR	Total HI
		Inorganics					
Arsenic	2.88E-06	2.01E-07	5.64E-07	3.02E-06	NA	2.11E-05	1.13E-01
Thallium	2.69E-07	1.88E-08	5.25E-08	NA	NA	NA	4.04E-02
Semivolatile Organic Compounds							
Benzo(a)anthracene	1.56E-08	1.09E-09	3.05E-09	3.38E-10	NA	7.43E-08	NA
Benzo(a)pyrene	1.68E-08	1.17E-09	3.29E-09	3.64E-09	NA	8.00E-07	NA
Benzo(b)fluoranthene	2.38E-08	1.66E-09	4.66E-09	5.16E-10	NA	1.13E-07	NA
Total ILCR or HI				3.02E-06	NA	2.21E-05	1.53E-01

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/m³ - Milligrams per cubic meter.

Table D-2

Groundskeeper, Future Scenario, Exposure to Total Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

Page 1 of 2

Chemicals of Potential Concern	Incidental Ingestion					Dermal Contact				
	EPC mg/kg	Cancer	Noncancer	ILCR	HQ	Dose	Cancer	Noncancer	ILCR	HQ
		Dose	Dose			Absorbed	Dose	Dose		
Inorganics										
Arsenic	2.28E+01	7.95E-06	2.23E-05	1.19E-05	7.42E-02	1.37E-07	1.57E-06	4.41E-06	2.36E-06	1.47E-02
Thallium	1.47E+00	5.15E-07	1.44E-06	NA	2.22E-02	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	5.45E-08	1.53E-07	3.98E-08	NA	4.06E-09	4.68E-08	1.31E-07	3.41E-08	NA
Benzo(a)pyrene	1.68E-01	5.87E-08	1.64E-07	4.29E-07	NA	4.37E-09	5.04E-08	1.41E-07	3.68E-07	NA
Benzo(b)fluoranthene	1.91E-01	6.67E-08	1.87E-07	4.87E-08	NA	4.97E-09	5.73E-08	1.60E-07	4.18E-08	NA
Total ILCR or HI				1.24E-05	9.64E-02				2.81E-06	1.47E-02

EPC - Exposure Point Concentration.
ILCR - Incremental Lifetime Cancer Risk.
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - Milligrams per kilogram.
mg/m³ - Milligrams per cubic meter.

Table D-2

Groundskeeper, Future Scenario, Exposure to Total Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

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Chemicals of Potential Concern	Concentration in Air C _a mg/m ³	Inhalation				All Pathways	
		Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Total	Total
						ILCR	HI
Inorganics							
Arsenic	2.28E-06	1.59E-07	4.45E-07	2.39E-06	NA	1.67E-05	8.89E-02
Thallium	1.47E-07	1.03E-08	2.88E-08	NA	NA	NA	2.22E-02
Semivolatile Organic Compounds							
Benzo(a)anthracene	1.56E-08	1.09E-09	3.05E-09	3.38E-10	NA	7.43E-08	NA
Benzo(a)pyrene	1.68E-08	1.17E-09	3.29E-09	3.64E-09	NA	8.00E-07	NA
Benzo(b)fluoranthene	1.91E-08	1.33E-09	3.74E-09	4.14E-10	NA	9.09E-08	NA
Total ILCR or HI				2.39E-06	NA	1.76E-05	1.11E-01

EPC - Exposure Point Concentration.
ILCR - Incremental Lifetime Cancer Risk.
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - Milligrams per kilogram.
mg/m³ - Milligrams per cubic meter.

Table D-3

**Groundskeeper, Future Scenario, Exposure to Overburden Monitoring Well Groundwater
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/L	Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer Dose	Noncancer Dose	ILCR	HQ	Dose Absorbed	Cancer Dose	Noncancer Dose	ILCR	HQ	Total	Total
		mg/kg-day	mg/kg-day			mg/cm ² -day	mg/kg-day	mg/kg-day			ILCR	HI
Inorganics												
Arsenic	8.02E-03	2.80E-05	7.85E-05	4.20E-05	2.62E-01	8.02E-09	9.25E-08	2.59E-07	1.39E-07	8.63E-04	4.22E-05	2.62E-01
Chromium, Total	1.30E-03	4.54E-06	1.27E-05	NA	6.06E-04	1.30E-09	1.50E-08	4.20E-08	NA	1.55E-04	NA	7.61E-04
Cobalt	1.75E-03	6.10E-06	1.71E-05	NA	5.69E-02	6.98E-10	8.05E-09	2.26E-08	NA	7.52E-05	NA	5.70E-02
Iron	1.35E+00	4.72E-03	1.32E-02	NA	1.89E-02	1.35E-06	1.56E-05	4.37E-05	NA	NA	NA	1.89E-02
Manganese	1.11E+00	3.86E-03	1.08E-02	NA	2.30E-01	1.11E-06	1.28E-05	3.57E-05	NA	1.88E-02	NA	2.49E-01
General Chemistry												
Sulfate	5.38E+02	1.88E+00	5.26E+00	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total ILCR or HI				4.20E-05	5.68E-01				1.39E-07	1.99E-02	4.22E-05	5.88E-01

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/L - Milligrams per liter.
 mg/cm² - Milligrams per square meter.
 mg/kg - Milligrams per kilogram.

Table D-4

**Indoor Worker, Future Scenario, Exposure to Surface Soil
Ash Pit No. 3
Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion				Total ILCR	Total HI
		Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ		
Inorganics							
Arsenic	2.88E+01	5.03E-06	1.41E-05	7.55E-06	4.70E-02	7.55E-06	4.70E-02
Thallium	2.69E+00	4.69E-07	1.31E-06	NA	2.02E-02	NA	2.02E-02
Semivolatile Organic Compounds							
Benzo(a)anthracene	1.56E-01	2.73E-08	7.63E-08	1.99E-08	NA	1.99E-08	NA
Benzo(a)pyrene	1.68E-01	2.94E-08	8.22E-08	2.14E-07	NA	2.14E-07	NA
Benzo(b)fluoranthene	2.38E-01	4.16E-08	1.16E-07	3.04E-08	NA	3.04E-08	NA
Total ILCR or HI						7.82E-06	6.72E-02

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.

Table D-5

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

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Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion				Dermal Contact				
		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										
Arsenic	2.28E+01	5.25E-07	7.33E-05	7.87E-07	2.44E-01	2.05E-07	4.72E-08	6.60E-06	7.09E-08	2.20E-02
Thallium	1.47E+00	3.40E-08	4.75E-06	NA	7.30E-02	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	3.60E-09	5.02E-07	2.63E-09	NA	6.08E-09	1.40E-09	1.96E-07	1.02E-09	NA
Benzo(a)pyrene	1.68E-01	3.87E-09	5.41E-07	2.83E-08	NA	6.55E-09	1.51E-09	2.11E-07	1.10E-08	NA
Benzo(b)fluoranthene	1.91E-01	4.41E-09	6.15E-07	3.22E-09	NA	7.45E-09	1.72E-09	2.40E-07	1.25E-09	NA
Total ILCR or HI				8.22E-07	3.17E-01				8.42E-08	2.20E-02

EPC - Exposure Point Concentration
 ILCR - Incremental Lifetime Cancer Risk
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - milligram per kilogram
 mg/m³ - milligram per cubic meter

Table D-5

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

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Chemicals of Potential Concern	Concentration in Air C _a mg/m ³	Inhalation				All Pathways	
		Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Total	Total
						ILCR	HI
Inorganics							
Arsenic	7.97E-06	1.11E-08	1.55E-06	1.67E-07	NA	1.03E-06	2.66E-01
Thallium	5.16E-07	7.21E-10	1.01E-07	NA	NA	NA	7.30E-02
Semivolatile Organic Compounds							
Benzo(a)anthracene	5.46E-08	7.63E-11	1.07E-08	2.37E-11	NA	3.67E-09	NA
Benzo(a)pyrene	5.88E-08	8.22E-11	1.15E-08	2.55E-10	NA	3.96E-08	NA
Benzo(b)fluoranthene	6.69E-08	9.34E-11	1.30E-08	2.90E-11	NA	4.50E-09	NA
Total ILCR or HI				1.67E-07	NA	1.07E-06	3.39E-01

EPC - Exposure Point Concentration
 ILCR - Incremental Lifetime Cancer Risk
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - milligram per kilogram
 mg/m³ - milligram per cubic meter

Table D-6

**Indoor Worker, Future Scenario, Exposure to Overburden Monitoring Well Groundwater
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/L	Incidental Ingestion				Dermal Contact					All Pathways	
		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	Total ILCR	Total HI
Inorganics												
Arsenic	8.02E-03	2.80E-05	7.85E-05	4.20E-05	2.62E-01	8.02E-09	9.25E-08	2.59E-07	1.39E-07	8.63E-04	4.22E-05	2.62E-01
Chromium, Total	1.30E-03	4.54E-06	1.27E-05	NA	6.06E-04	1.30E-09	1.50E-08	4.20E-08	NA	1.55E-04	NA	7.61E-04
Cobalt	1.75E-03	6.10E-06	1.71E-05	NA	5.69E-02	6.98E-10	8.05E-09	2.26E-08	NA	7.52E-05	NA	5.70E-02
Iron	1.35E+00	4.72E-03	1.32E-02	NA	1.89E-02	1.35E-06	1.56E-05	4.37E-05	NA	NA	NA	1.89E-02
Manganese	1.11E+00	3.86E-03	1.08E-02	NA	2.30E-01	1.11E-06	1.28E-05	3.57E-05	NA	1.88E-02	NA	2.49E-01
General Chemistry												
Sulfate	5.38E+02	1.88E+00	5.26E+00	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total ILCR or HI				4.20E-05	5.68E-01				1.39E-07	1.99E-02	4.22E-05	5.88E-01

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/L - Milligrams per liter.
 mg/cm² - Milligrams per square meter.
 mg/kg - Milligrams per kilogram.

Table D-7

**Construction Worker, Current and Future Scenarios, Exposure to Sediment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion				Dermal Contact					All Pathways	
		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	Total ILCR	Total HI
Inorganics												
Arsenic	6.40E+00	1.48E-07	2.06E-05	2.21E-07	6.87E-02	5.76E-08	1.33E-08	1.85E-06	1.99E-08	6.18E-03	2.41E-07	7.49E-02
Chromium, total	1.08E+01	2.49E-07	3.48E-05	NA	1.66E-03	NA	NA	NA	NA	NA	NA	1.66E-03
Semivolatiles Organic Compounds												
Benzo(a)pyrene	1.58E-01	3.64E-09	5.09E-07	2.66E-08	NA	6.16E-09	1.42E-09	1.98E-07	1.04E-08	NA	3.70E-08	NA
Benzo(b)fluoranthene	2.40E-01	5.54E-09	7.73E-07	4.04E-09	NA	9.36E-09	2.16E-09	3.01E-07	1.58E-09	NA	5.62E-09	NA
Total ILCR or HI				2.52E-07	7.04E-02				3.19E-08	6.18E-03	2.84E-07	7.65E-02

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per centimeter squared.

Table D-8

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion			Dermal Contact				
		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									
Arsenic	2.28E+01	9.62E-06	2.24E-05	4.81E-05	4.78E-08	1.28E-06	1.37E-07	2.10E-06	5.06E-06
Thallium	1.47E+00	6.23E-07	1.45E-06	NA	NA	NA	NA	NA	NA
Semivolatile Organic Compounds									
Benzo(a)anthracene	1.56E-01	6.59E-08	1.54E-07	1.60E-07	1.42E-09	3.80E-08	4.06E-09	6.22E-08	7.32E-08
Benzo(a)pyrene	1.68E-01	7.10E-08	1.66E-07	1.73E-06	1.53E-09	4.09E-08	4.37E-09	6.70E-08	7.88E-07
Benzo(b)fluoranthene	1.91E-01	8.07E-08	1.88E-07	1.96E-07	1.74E-09	4.65E-08	4.97E-09	7.62E-08	8.96E-08
Total ILCR				5.02E-05					6.01E-06

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per square meter.
 mg/m³ - Milligrams per cubic meter.

Table D-8

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 2 of 2)

Chemicals of Potential Concern	Concentration in Air C _a mg/m ³	Inhalation		Resident ILCR	All Pathways Total ILCR
		Adult Dose mg/kg-day	Child Dose mg/kg-day		
Inorganics					
Arsenic	4.48E-08	4.21E-09	2.46E-09	1.00E-07	5.33E-05
Thallium	2.90E-09	2.73E-10	1.59E-10	NA	NA
Semivolatile Organic Compounds					
Benzo(a)anthracene	3.07E-10	2.89E-11	1.68E-11	1.42E-11	2.34E-07
Benzo(a)pyrene	3.31E-10	3.11E-11	1.81E-11	1.53E-10	2.52E-06
Benzo(b)fluoranthene	3.76E-10	3.53E-11	2.06E-11	1.73E-11	2.86E-07
Total ILCR				1.00E-07	5.63E-05

EPC - Exposure Point Concentration.
ILCR - Incremental Lifetime Cancer Risk.
mg/kg - Milligrams per kilogram.
mg/cm² - Milligrams per square meter.
mg/m³ - Milligrams per cubic meter.

Table D-9

**On-Site Child Resident, Noncancer Risk, Future Scenario, Exposure to Total Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion		Dermal Contact			Concentration in Air C _a mg/m ³	Inhalation		All Pathways Total HI
		Child Dose mg/kg-day	Child Resident HQ	Dose Absorbed mg/cm ² -day	Child Dose mg/kg-day	Child Resident HQ		Child Dose mg/kg-day	Child Resident HQ	
Inorganics										
Arsenic	2.28E+01	2.62E-04	8.73E-01	1.37E-07	2.44E-05	8.15E-02	4.48E-08	2.87E-08	NA	9.54E-01
Thallium	1.47E+00	1.70E-05	2.61E-01	NA	NA	NA	2.90E-09	1.86E-09	NA	2.61E-01
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	1.80E-06	NA	4.06E-09	7.26E-07	NA	3.07E-10	1.96E-10	NA	NA
Benzo(a)pyrene	1.68E-01	1.93E-06	NA	4.37E-09	7.82E-07	NA	3.31E-10	2.12E-10	NA	NA
Benzo(b)fluoranthene	1.91E-01	2.20E-06	NA	4.97E-09	8.89E-07	NA	3.76E-10	2.41E-10	NA	NA
Total HI			1.13E+00			8.15E-02			NA	1.22E+00

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per square meter.
 mg/m³ - Milligrams per cubic meter.

Table D-10

**On-Site Adult Resident, Noncancer Risk, Future Scenario, Exposure to Total Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion		Dermal Contact			Concentration in Air C _a mg/m ³	Inhalation		All Pathways Total HI
		Adult Dose mg/kg-day	Adult Resident HQ	Dose Absorbed mg/cm ² -day	Adult Dose mg/kg-day	Adult Resident HQ		Adult Dose mg/kg-day	Adult Resident HQ	
Inorganics										
Arsenic	2.28E+01	2.81E-05	9.35E-02	4.78E-08	3.73E-06	1.24E-02	4.48E-08	1.23E-08	NA	1.06E-01
Thallium	1.47E+00	1.82E-06	2.80E-02	NA	NA	NA	2.90E-09	7.95E-10	NA	2.80E-02
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	1.92E-07	NA	1.42E-09	1.11E-07	NA	3.07E-10	8.42E-11	NA	NA
Benzo(a)pyrene	1.68E-01	2.07E-07	NA	1.53E-09	1.19E-07	NA	3.31E-10	9.07E-11	NA	NA
Benzo(b)fluoranthene	1.91E-01	2.35E-07	NA	1.74E-09	1.36E-07	NA	3.76E-10	1.03E-10	NA	NA
Total HI			1.21E-01			1.24E-02			NA	1.34E-01

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per square meter.
 mg/m³ - Milligrams per cubic meter.

Table D-11

**On-Site Resident, Cancer Risk, Future Scenario, Exposure to Sediment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion			Dermal Contact					All Pathways Total ILCR
		Adult Dose mg/kg-day	Child Dose mg/kg-day	Resident ILCR	Dose Absorbed mg/cm ² -day	Adult Dose mg/kg-day	Dose Absorbed mg/cm ² -day	Child Dose mg/kg-day	Resident ILCR	
Inorganics										
Arsenic	6.40E+00	3.01E-07	7.01E-07	1.50E-06	1.34E-09	5.35E-09	3.84E-09	8.75E-09	2.11E-08	1.52E-06
Chromium, total	1.08E+01	5.07E-07	1.18E-06	NA	NA	NA	NA	NA	NA	NA
Semivolatiles Organic Compounds										
Benzo(a)pyrene	1.58E-01	7.42E-09	1.73E-08	1.81E-07	1.44E-10	5.72E-10	4.11E-10	9.36E-10	1.10E-08	1.92E-07
Benzo(b)fluoranthene	2.40E-01	1.13E-08	2.63E-08	2.74E-08	2.18E-10	8.69E-10	6.24E-10	1.42E-09	1.67E-09	2.91E-08
Total ILCR				1.71E-06					3.38E-08	1.74E-06

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per centimeter squared.

Table D-12

**On-Site Child Resident Noncancer Risk, Future Scenario, Exposure to Sediment
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion		Dermal Contact			All Pathways Total HI
		Child Dose mg/kg-day	Child HQ	Dose Absorbed mg/cm ² -day	Child Dose mg/kg-day	Child HQ	
Inorganics							
Arsenic	6.40E+00	8.18E-06	2.73E-02	3.84E-09	1.02E-07	3.40E-04	2.76E-02
Chromium, total	1.08E+01	1.38E-05	6.58E-04	NA	NA	NA	6.58E-04
Semivolatiles Organic Compounds							
Benzo(a)pyrene	1.58E-01	2.02E-07	NA	4.11E-10	1.09E-08	NA	NA
Benzo(b)fluoranthene	2.40E-01	3.07E-07	NA	6.24E-10	1.66E-08	NA	NA
Total HI			2.79E-02			3.40E-04	2.83E-02

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per centimeter squared.

Table D-13

On-Site Adult Resident, Noncancer Risk, Future Scenario, Exposure to Sediment
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion		Dermal Contact			All Pathways
		Adult Dose mg/kg-day	Adult HQ	Dose Absorbed mg/cm ² -day	Adult Dose mg/kg-day	Adult HQ	Total HI
Inorganics							
Arsenic	6.40E+00	8.77E-07	2.92E-03	1.34E-09	1.56E-08	5.20E-05	2.97E-03
Chromium, total	1.08E+01	1.48E-06	7.05E-05	NA	NA	NA	7.05E-05
Semivolatiles Organic Compounds							
Benzo(a)pyrene	1.58E-01	2.16E-08	NA	1.44E-10	1.67E-09	NA	NA
Benzo(b)fluoranthene	2.40E-01	3.29E-08	NA	2.18E-10	2.53E-09	NA	NA
Total HI			2.99E-03			5.20E-05	3.04E-03

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per centimeter squared.

Table D-14

**On-Site Resident, Future Scenario, Cancer Risk from Exposure to Overburden Monitoring Well Groundwater
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/L	Incidental Ingestion			Dermal Contact					Inhalation of VOCs				All Pathways Total ILCR
		Adult Dose mg/kg-day	Child Dose mg/kg-day	ILCR	Dose Absorbed mg/cm ² -day	Adult Dose mg/kg-day	Dose Absorbed mg/cm ² -day	Child Dose mg/kg-day	ILCR	Concentration in Air mg/m ³	Adult Dose mg/kg-day	Child Dose mg/kg-day	ILCR	
Inorganics														
Arsenic	8.02E-03	7.53E-05	4.40E-05	1.79E-04	1.60E-09	1.51E-07	2.67E-09	9.66E-08	3.71E-07	NA	NA	NA	NA	1.79E-04
Chromium, Total	1.30E-03	1.22E-05	7.12E-06	NA	2.60E-10	2.44E-08	4.33E-10	1.57E-08	NA	NA	NA	NA	NA	NA
Cobalt	1.75E-03	1.64E-05	9.57E-06	NA	1.40E-10	1.31E-08	2.33E-10	8.41E-09	NA	NA	NA	NA	NA	NA
Iron	1.35E+00	1.27E-02	7.41E-03	NA	2.70E-07	2.54E-05	4.50E-07	1.63E-05	NA	NA	NA	NA	NA	NA
Manganese	1.11E+00	1.04E-02	6.06E-03	NA	2.21E-07	2.08E-05	3.68E-07	1.33E-05	NA	NA	NA	NA	NA	NA
General Chemistry														
Sulfate	5.38E+02	5.05E+00	2.95E+00	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total ILCR				1.79E-04					3.71E-07				NA	1.79E-04

EPC - Exposure Point Concentration.
 ILCR - Incremental Lifetime Cancer Risk.
 mg/L - Milligrams per liter.
 mg/cm² - Milligrams per square meter.
 mg/kg - Milligrams per kilogram.
 mg/m³ - Milligrams per cubic meter.

Table D-15

Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Monitoring Well Groundwater
 Ash Pit No. 3
 Former Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	EPC mg/L	Incidental Ingestion		Dermal Contact			Inhalation of VOCs			All Pathways Total HI
		Child Dose mg/kg-day	HQ	Dose Absorbed mg/cm ² -day	Child Dose mg/kg-day	HQ	Concentration in Air mg/m ³	Child Dose mg/kg-day	HQ	
Inorganics										
Arsenic	8.02E-03	5.13E-04	1.71E+00	2.67E-09	1.13E-06	3.76E-03	NA	NA	NA	1.71E+00
Chromium, Total	1.30E-03	8.31E-05	3.96E-03	4.33E-10	1.83E-07	6.76E-04	NA	NA	NA	4.63E-03
Cobalt	1.75E-03	1.12E-04	3.72E-01	2.33E-10	9.81E-08	3.27E-04	NA	NA	NA	3.72E-01
Iron	1.35E+00	8.64E-02	1.23E-01	4.50E-07	1.90E-04	NA	NA	NA	NA	1.23E-01
Manganese	1.11E+00	7.07E-02	1.50E+00	3.68E-07	1.55E-04	8.18E-02	NA	NA	NA	1.59E+00
General Chemistry										
Sulfate	5.38E+02	3.44E+01	NA	NA	NA	NA	NA	NA	NA	NA
Total HI			3.71E+00			8.65E-02			NA	3.80E+00

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/L - Milligrams per liter.
 mg/cm² - Milligrams per square meter.
 mg/kg - Milligrams per kilogram.
 mg/m³ - Milligrams per cubic meter.

Table D-16

**Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Monitoring Well Groundwater
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

Chemicals of Potential Concern	EPC mg/L	Incidental Ingestion		Dermal Contact			Inhalation of VOCs			All Pathways Total HI
		Noncancer Dose mg/kg-day	HQ	Dose Absorbed mg/cm ² -day	Adult Dose mg/kg-day	HQ	Concentration in Air mg/m ³	Adult Dose mg/kg-day	HQ	
Inorganics										
Arsenic	8.02E-03	2.20E-04	7.33E-01	1.60E-09	4.40E-07	1.47E-03	NA	NA	NA	7.34E-01
Chromium, Total	1.30E-03	3.56E-05	1.70E-03	2.60E-10	7.12E-08	2.64E-04	NA	NA	NA	1.96E-03
Cobalt	1.75E-03	4.78E-05	1.59E-01	1.40E-10	3.83E-08	1.28E-04	NA	NA	NA	1.60E-01
Iron	1.35E+00	3.70E-02	5.29E-02	2.70E-07	7.41E-05	NA	NA	NA	NA	5.29E-02
Manganese	1.11E+00	3.03E-02	6.45E-01	2.21E-07	6.06E-05	3.19E-02	NA	NA	NA	6.77E-01
General Chemistry										
Sulfate	5.38E+02	1.47E+01	NA	NA	NA	NA	NA	NA	NA	NA
Total HI			1.59E+00			3.38E-02			NA	1.63E+00

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 mg/L - Milligrams per liter.
 mg/cm² - Milligrams per square meter.
 mg/kg - Milligrams per kilogram.
 mg/m³ - Milligrams per cubic meter.

Table D-17

**Adult Hunter, Future Scenario, Exposure to Surface Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio**

(Page 1 of 2)

Chemicals of Potential Concern	EPC mg/kg	Incidental Ingestion				Dermal Contact				
		Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	Adult Hunter ILCR	Adult Hunter HQ	Dose Absorbed mg/cm ² -day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	Adult Hunter ILCR	Adult Hunter HQ
Inorganics										
Arsenic	2.88E+01	6.77E-07	1.58E-06	1.01E-06	5.26E-03	1.73E-07	1.34E-07	3.13E-07	2.01E-07	1.04E-03
Thallium	2.69E+00	6.31E-08	1.47E-07	NA	2.26E-03	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.56E-01	3.66E-09	8.55E-09	2.67E-09	NA	4.06E-09	3.14E-09	7.33E-09	2.29E-09	NA
Benzo(a)pyrene	1.68E-01	3.95E-09	9.21E-09	2.88E-08	NA	4.37E-09	3.38E-09	7.90E-09	2.47E-08	NA
Benzo(b)fluoranthene	2.38E-01	5.59E-09	1.30E-08	4.08E-09	NA	6.19E-09	4.80E-09	1.12E-08	3.50E-09	NA
Total ILCR and HI				1.05E-06	7.53E-03				2.31E-07	1.04E-03

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 ILCR - Incremental Lifetime Cancer Risk.
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per square meter.

Table D-17

Adult Hunter, Future Scenario, Exposure to Surface Soil
Ash Pit No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio

(Page 2 of 2)

Chemicals of Potential Concern	Concentration in Forage C _p mg/kg	Concentration in Venison C _v mg/kg	Consumption of Venison				All Pathways	
			Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Total	Total
							ILCR	HI
Inorganics								
Arsenic	NA	NA	NA	NA	NA	NA	1.22E-06	6.30E-03
Thallium	1.34E-03	2.80E-07	2.14E-11	4.99E-11	NA	7.68E-07	NA	2.26E-03
Semivolatile Organic Compounds								
Benzo(a)anthracene	NA	NA	NA	NA	NA	NA	4.97E-09	NA
Benzo(a)pyrene	NA	NA	NA	NA	NA	NA	5.35E-08	NA
Benzo(b)fluoranthene	NA	NA	NA	NA	NA	NA	7.58E-09	NA
Total ILCR and HI					NA	7.68E-07	1.28E-06	8.57E-03

EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 ILCR - Incremental Lifetime Cancer Risk.
 mg/kg - Milligrams per kilogram.
 mg/cm² - Milligrams per square meter.

Table D-18

Hunter's Child, Future Scenario, Exposure to Surface Soil
 Ash Pit No. 3
 Former 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				Total ILCR	Total HI
				Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ		
Inorganics									
Arsenic	2.88E+01	NA	NA	NA	NA	NA	NA	NA	NA
Thallium	2.69E+00	1.34E-03	2.80E-07	7.68E-12	8.96E-11	NA	1.38E-06	NA	1.38E-06
Semivolatile Organic Compounds									
Benzo(a)anthracene	1.56E-01	NA	NA	NA	NA	NA	NA	NA	NA
Benzo(a)pyrene	1.68E-01	NA	NA	NA	NA	NA	NA	NA	NA
Benzo(b)fluoranthene	2.38E-01	NA	NA	NA	NA	NA	NA	NA	NA
Total ILCR and HI						NA	1.38E-06	NA	1.38E-06

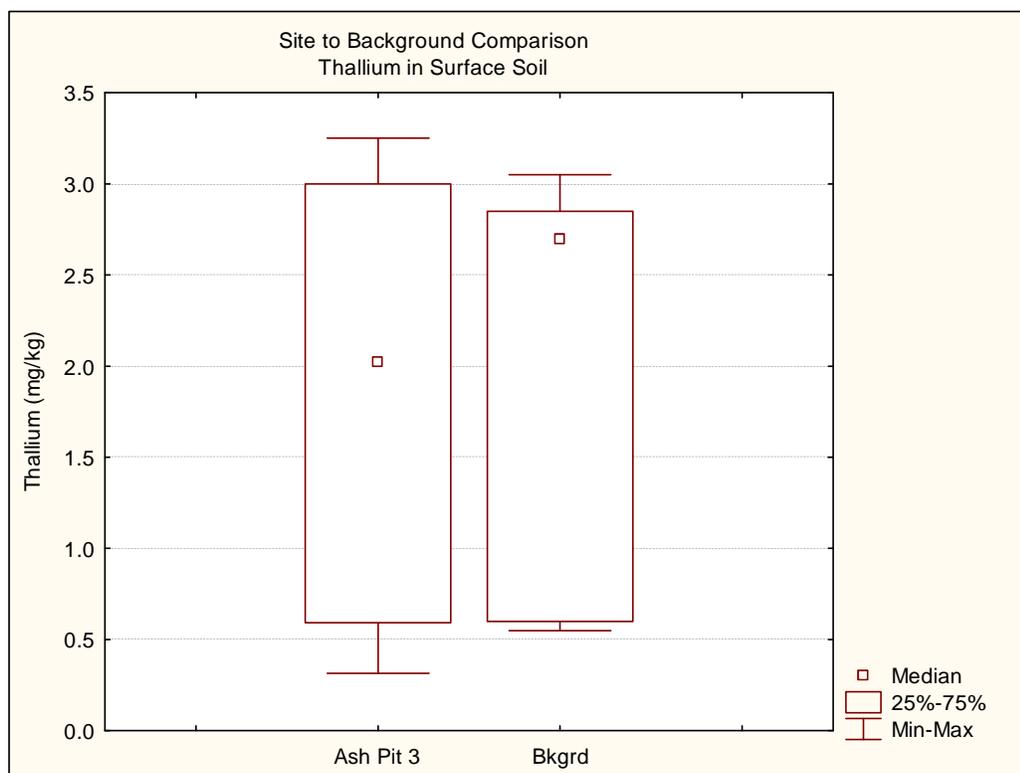
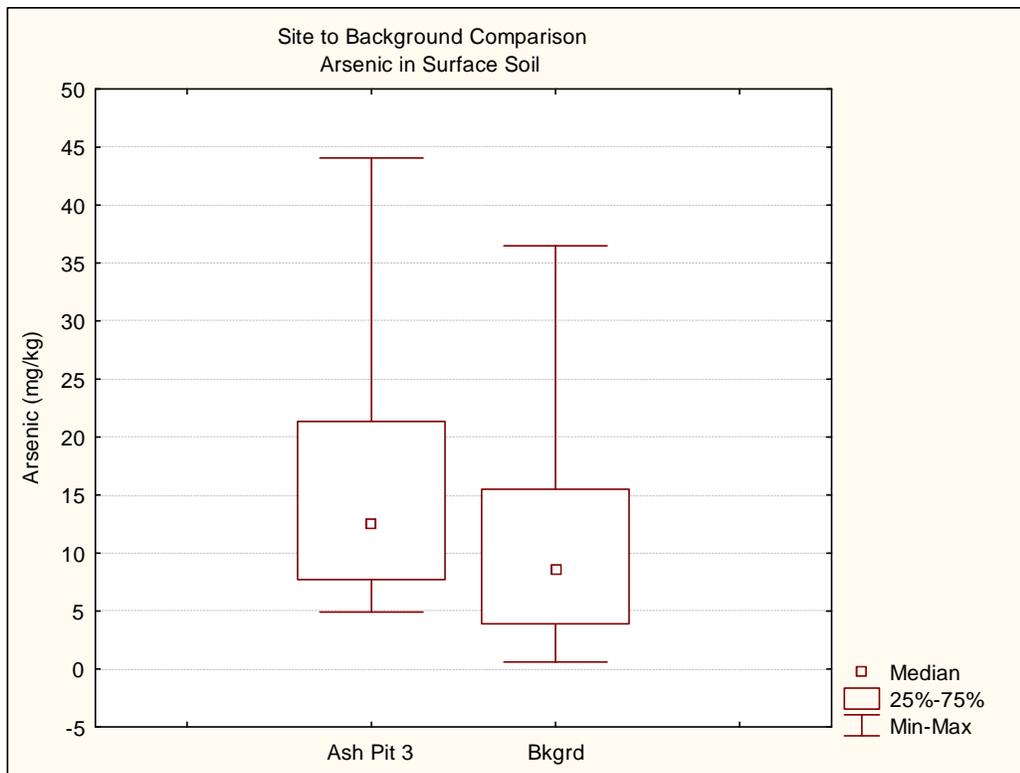
EPC - Exposure Point Concentration.
 HQ - Hazard Quotient; HI - Hazard Index
 ILCR - Incremental Lifetime Cancer Risk.
 mg/kg - Milligrams per kilogram.

APPENDIX E

WILCOXON RANK SUM STATISTICAL TEST OUTPUT

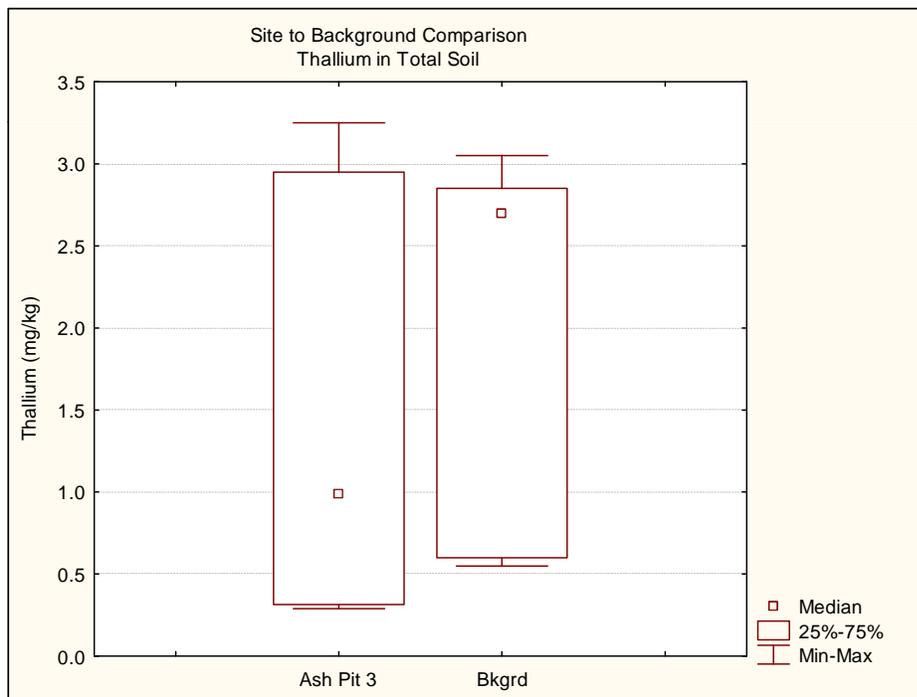
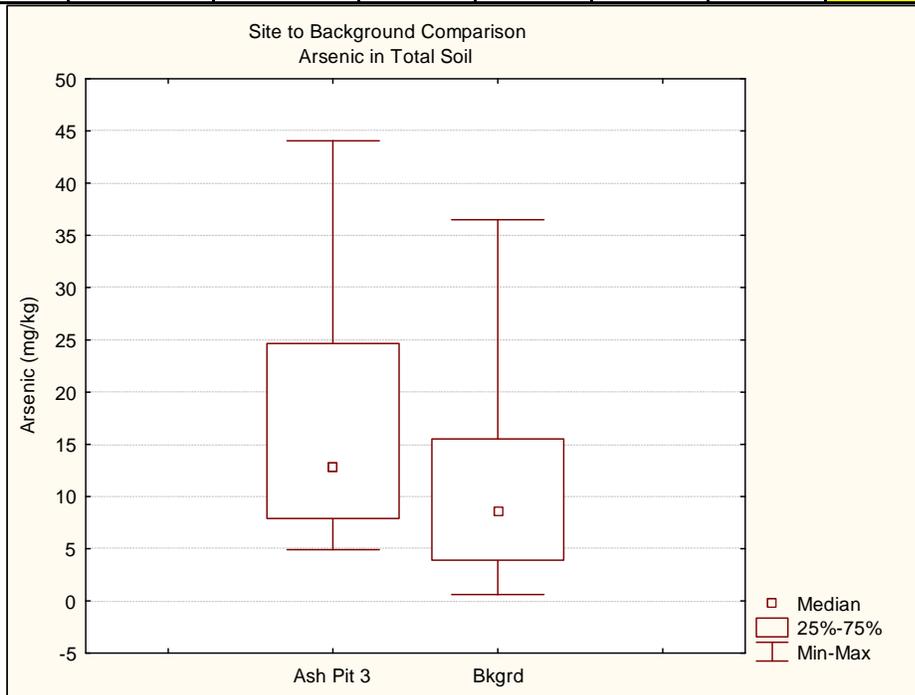
Site to Background Evaluation for Metals in Surface Soil - AP 3

	Rank Sum	Rank Sum	U	Z	p-level	Z	p-level	Valid N	Valid N	2*1sided
Arsenic	167.0000	428.0000	77.00000	1.096197	0.272993	1.096449	0.272883	8	26	0.288221
Thallium	139.0000	422.0000	97.00000	0.126025	0.899712	0.126608	0.899250	8	25	0.918057



Site to Background Evaluation for Metals in Total Soil - AP 3

	Rank Sum	Rank Sum	U	Z	p-level	Z	p-level	Valid N	Valid N	2*1sided
Arsenic	408.0000	495.0000	144.0000	1.65764	0.097392	1.65784	0.097351	16	26	0.100440
Thallium	295.5000	565.5000	159.5000	-1.08241	0.279072	-1.08515	0.277855	16	25	0.282616



RESPONSE TO COMMENTS

**Responses to Ohio Environmental Protection Agency Comments on the
Draft Baseline Human Health Risk Assessment (BHHRA) for Ash Pit No. 3 and the
BHHRA and Screening-Level Ecological Risk Assessment Addendum for Coal Yard No. 3
Former Plum Brook Ordnance Works, Sandusky, Ohio,
Dated June 12, 2013**

The BHHRA and SLERA documents were reviewed by Ohio EPA, who had no comments as indicated by correspondence received from Paul Jayko on August 6, 2013.