Baseline Human Health Risk Assessment

Garage Maintenance Area
(Locomotive Building Area)

Plum Brook Ordnance Works
Sandusky, Ohio

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List of Acronyms

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

List of Acronyms *(Continued)*

kg	kilogram
kg/day	kilograms per day
LMS	linearized multistage
K_p	permeability coefficient
LBA	Locomotive Building Area
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
µg/L	micrograms per liter
mg/L	milligrams per liter
ml/min	milliliter per minute
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
PEF	particulate emission factor
ppm	parts per million
PRG	preliminary remediation goal
QC	quality control
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, Inc.
TNT	trinitrotoluene
TNTA	TNT Area A
TNTB	TNT Area B
TNTC	TNT Area C
UCL	95th percent upper confidence limit on the arithmetic mean
UF	uncertainty factor
USACE	U.S. Army Corps of Engineers
VOC	volatile organic compound
WRS	Wilcoxon Rank Sum

Executive Summary

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

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

Accountability and custody were transferred to the National Aeronautics and Space Administration (NASA) on March 15, 1963 for the entire portion of the former PBOW property that had been under the accountability and custody of the Department of the Army. 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. NASA currently controls approximately 6,400 acres.

Locomotive Building Area Description and History. The LBA is part of the Garage Maintenance Area. It is located in the central portion of the PBOW facility, and the study area comprises approximately 3 acres and includes the Locomotive Shop and the Rail Car Washing Area. The Locomotive Shop is located north of Maintenance Road. The former Rail Car Washing Area is located east of the Locomotive Shop.

The Locomotive Shop was used for the maintenance of equipment, vehicles, and rail cars. A work pit built to allow maintenance personnel to work below the locomotives and railway cars during the operation of PBOW is the primary area of concern in this building. The pit measures 60 feet long, 3 feet, 8 inches wide, and 5 feet deep. NASA has backfilled this pit with loose stone and currently uses this building as a storage garage. When the maintenance pit was operational, the waste effluent drained to a sump in the bottom of the maintenance pit and was sent into the sewer system via a pump and small sump collection basin. This sump basin is located just inside the south side of the building. A dark petroleum color and strong odor were noted in the 2000 limited site inspection report concerning a sample collected from the sump within the maintenance pit.

The Rail Car Washing Area is located approximately 260 feet east of the Locomotive Shop. During the operation of PBOW, the Rail Car Washing Area consisted of tracks which carried locomotives and railway cars above a pit. This area was used as a locomotive and railway car cleaning operation that involved an acid. The Rail Car Washing Area surrounded the terminus of the southern track in the pair of railroad tracks that exited the Locomotive Shop from the eastern end of the building. During 2008 and 2009 site visits by USACE and Shaw personnel, it was observed that the Locomotive Shop is used for equipment storage (e.g., backhoe tractor) and possibly maintenance.

Approach. The BHHRA evaluated exposure to chemicals in surface soil, subsurface soil, maintenance pit material, bedrock groundwater, overburden groundwater, surface water, and sediment for cancer risks and noncancer hazards. Only validated analytical data were used in the BHHRA. Validated analytical data are from samples collected during 2009, as reported in the 2010 site characterization report, and those collected in 1999 and reported in the limited site inspection report. A screening for chemicals of potential concern (COPC) was used to focus the evaluation on those chemicals most likely to present a risk to potentially exposed individuals. Each COPC in each medium was evaluated for exposure via the relevant exposure pathways and the resultant risk and hazards were estimated. The receptors listed below were evaluated for exposure and risk. The media evaluated for each receptor are shown in parentheses.

- Current groundskeeper (surface soil)
- Future groundskeeper (combined surface and subsurface soil [referred to as “total soil], bedrock groundwater, overburden groundwater)

- Future indoor worker (surface soil, subsurface soil [inhalation pathway only], bedrock groundwater, overburden groundwater)
- Future/current construction worker (total soil, surface water, sediment, maintenance pit material)
- Future on-site resident (total soil, surface water, sediment, bedrock groundwater, overburden groundwater)
- Future hunter (surface soil)
- Future hunter's child (surface soil [venison pathway only]).

The current groundskeeper and construction worker are regarded as current receptors. No construction is planned for the LBA, but a construction worker is assumed to be potentially exposed under current or future land use. Hunting is currently allowed by permit on parts of PBOW, but not at the LBA. Hunting may continue at the PBOW and under future land use may include the LBA. 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 neither the overburden nor bedrock groundwater units produced a sufficient volume of water for potable use, these future receptors were also evaluated assuming no groundwater use.

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

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

- Current groundskeeper: ILCR = 9E-6 (3E-6 excluding background-related chromium); HI = 0.007
- Future groundskeeper – bedrock groundwater use: ***ILCR = 1E-3; HI = 11***

- Future groundskeeper – overburden groundwater use: **ILCR = 6E-5**; HI = 0.9
- Future groundskeeper – excluding groundwater use: ILCR = 8E-6 (3E-6 excluding background-related chromium); HI = 0.2
- Future indoor worker – bedrock groundwater use: ***ILCR = 1E-3; HI = 11***
- Future indoor worker – overburden groundwater use: **ILCR = 5E-5**; HI = 0.7
- Future indoor worker – excluding groundwater use: ILCR = 8E-7; HI = 0.002
- Future/current construction worker: ILCR = 1E-6; HI = 0.9
- Current/future construction worker – maintenance pit material: ILCR= 2E-7 ; HI= (no noncarcinogenic COPCs identified)
- Future resident – bedrock groundwater use: ***ILCR = 8E-3***; ***child HI = 167***; ***adult HI = 72***
- Future resident – overburden groundwater use: ***ILCR = 2E-4***; ***child HI = 6***; ***adult HI = 2***
- Future resident – excluding groundwater use: ILCR = 1E-5; child HI = 0.7; adult HI = 0.1
- Future hunter: ILCR = 2E-7; HI = 0.0003
- Future hunter's child: ILCR = 3E-12; HI = 0.0000003.

The ILCR and HI values for the two current exposure scenarios (current groundskeeper and construction worker) each meet the NCP risk management range (1E-6 to 1E-4), the PBOW cancer risk criterion (1E-5), and the noncancer hazard criterion (HI≤1). Note that no construction is currently planned at the LBA and the current worker scenario is likely to greatly overestimate the exposure levels of any current on-site worker or other receptor.

Each of the potential future exposure scenarios that does not assume potable use of groundwater also meets the PBOW risk and the target hazard criteria. This includes the future residential scenario. However, the cancer risks associated with the following future receptors assumed to use groundwater as a hypothetical source of tap water exceed the NCP risk management range and PBOW cancer risk criterion: future groundskeeper (bedrock), future indoor worker (bedrock), and resident (both overburden and bedrock). Each of these also exceeds the target HI criterion of 1. The groundskeeper and indoor worker assumed to use overburden groundwater also exceed the PBOW criterion of 1E-5, but not the NCP risk management range.

Use of either the overburden or bedrock groundwater is regarded as implausible because of insufficient yield. Additionally, the naturally poor quality of the bedrock groundwater and associate hydrogen sulfide off-gassing make it unsuitable as a potable source. The readily available municipal water supply obviates the need for use of groundwater as a potential source of tap water in the general vicinity of PBOW.

Recommendations. None of the receptors for which groundwater use was excluded had cancer risks or noncancer hazards that exceed the PBOW target criteria. This indicates that the associated environmental media at the LBA are unlikely to pose unacceptable risks to any receptor. Because groundwater use is not regarded as plausible based on insufficient yield in both the overburden and bedrock units, the risks associated with LBA groundwater are accordingly implausible. The naturally occurring poor quality in the LBA bedrock groundwater further renders the use of bedrock groundwater as implausible. Therefore, no further investigation of LBA environmental media is recommended.

No unacceptable risks were found associated with the maintenance pit material and exposure to this material is assumed to be negligible to minimal. However, evidence of petroleum was found in a sample collected from the eastern portion of this pit in 2009. This sampling location is near the maintenance pit sump that was sampled during the limited site inspection conducted in 1999. Both petroleum odor and visual signs of petroleum were also observed in the 1999 maintenance pit sample. Although no risk-based reason has been identified to perform a cleanup of this pit, it is suggested that removal of the gravel backfill and associated materials be considered by the PBOW Project Delivery Team based on the presence of petroleum contamination.

1.0 Introduction

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

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

1.1 Facility Location and Description

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

1.2 Facility History and Background

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

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

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

NASA has operated and maintained the former PBOW property since 1963, and the facility is currently the NASA Glenn Research Center, Plum Brook Station. NASA operates the property as a space research facility in support of their John Glenn Research Center at Lewis Field, Cleveland, Ohio. Most of the aerospace testing facilities built in the 1960s at the site are currently on standby or inactive status. On April 18, 1978, NASA declared approximately 2,152 acres of PBOW as excess. This excess included former buffer areas that had not been used by the Army and were thus not subject to decontamination efforts. The Perkins Township Board of Education acquired 46 acres of the excess acreage and uses this area as a bus transportation area. The GSA retains ownership of the remaining excess acreage and currently has a use agreement with the Ohio National Guard for 604 acres of this land. NASA currently controls approximately

6,400 acres. The details of land transactions are listed in the site management plan (USACE, 1995).

1.3 Locomotive Building Area Description and History

The LBA is part of the Garage Maintenance Area. As noted above, PBOW was built in early 1941 and manufactured 2,4,6-TNT, DNT, and pentolite until 1945. The Garage Maintenance Area included three buildings used for the maintenance of various vehicles and equipment used on site during facility operation. The other two buildings, including the main garage and vehicle maintenance shop have been actively used by NASA and are thus, not part of this investigation. The easternmost of these is Building 718, referred to herein as the Locomotive Shop, which was used specifically for locomotive maintenance. The LBA includes the Locomotive Shop, the nearby Rail Car Washing Area, and the surrounding area.

The LBA is located in the central portion of the PBOW facility and the study area comprises approximately 3 acres (Figure 1-2). The Locomotive Shop is located north of Maintenance Road. The former Rail Car Washing Area is located east of the Locomotive Shop (Figure 1-2).

The Locomotive Shop was used for the maintenance of equipment, vehicles, and rail cars. A work pit built to allow maintenance personnel to work below the locomotives and railway cars during the operation of PBOW is the primary area of concern in this building. The pit measures 60 feet long, 3 feet, 8 inches wide, and 5 feet deep (USACE, 2000). The pit is located beneath the northern set of railroad tracks that run through the Locomotive Shop. This work pit is labeled "Maintenance Pit" in the depiction on Figure 1-2. NASA has backfilled this pit with loose stone and currently uses this building as a storage garage. When the maintenance pit was operational, the waste effluent drained to a sump in the bottom of the maintenance pit and was sent into the sewer system via a pump into a small sump collection basin. This sump measures 9 feet, 2 inches deep and 2 feet, 6 inches in diameter and is located just inside the south side of the building, approximately 40 feet south of the maintenance pit.

The Rail Car Washing Area is located approximately 260 feet east of the Locomotive Shop, across Maintenance Road from Ash Pit No. 1. During the operation of PBOW, the Rail Car Washing Area consisted of tracks which carried locomotives and railway cars above a pit. It was used as a locomotive and railway car cleaning operation that involved an acid; thus, it also referred to as the acid area in the limited site inspection report (USACE, 2000). The Rail Car Washing Area surrounded the terminus of the southern track in the pair of railroad tracks that exited the Locomotive Shop from the eastern end of the building. A third track, known as the

"Z" track and oriented east-to-west just north of the Locomotive Shop, also ran to the Rail Car Washing Area (USACE, 2000).

During 2008 and 2009 site visits by USACE and Shaw personnel, it was observed that the Locomotive Shop is used for equipment storage (e.g., backhoe tractor) and possibly maintenance. It is also noted that an approximately 30-foot-long addition was built onto the eastern end of the Locomotive Shop at some point after the PBOW operational period. It was apparent that this addition was post-operational based on a comparison of current site observations versus historical photographs and historical use (including the fact that the presence of this addition would not have allowed access to the Rail Car Washing Area through the Locomotive Shop).

The location of the former Rail Car Washing Area pit is not apparent from visual survey. As shown on Figure 1-2, there are no buildings at the Rail Car Washing Area, and none are known to have been present historically. The area is not located near any easily recognizable man-made structure other than the locomotive building. Three rubble piles were observed within the Rail Car Washing Area; these piles contain railroad ties and railroad ballast rock.

1.4 Groundwater Use and Site Use

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

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

- Industrial use (NASA activities and programs) may continue.
- Portions of the site may be used for recreation by hunters and fishermen.

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

In summary, future site use of the LBA is considered to be industrial or residential for the purpose of developing receptor and exposure scenarios. Hunting is currently not allowed in the LBA, but hunting is allowed by permit in other PBOW areas; therefore, hunting is evaluated in this BHHRA. It is assumed that groundwater may be developed as a source of potable water in the future. Refer to Section 3.1.3 for a discussion of receptors and exposure scenarios.

1.5 Protocol for the Baseline Human Health Risk Assessment

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

- Ohio Environmental Protection Agency (OEPA), 2009a***, Use of U.S. EPA's Regional Screening Levels as Screening Values in Human Health Risk Assessments***, Technical Decision Compendium, Division of Emergency and Remedial Response, August.
- Ohio Environmental Protection Agency (OEPA), 2009b***, Human Health Cumulative Carcinogenic Risk and Non-carcinogenic Hazard Goals for the DERR Remedial Response Program***, Technical Decision Compendium, Division of Emergency and Remedial Response, August.
- U.S. Army Corps of Engineers (USACE), 1999, ***Risk Assessment Handbook, Volume I: Human Health Evaluation***, Engineer Manual EM 200-1-4.
- U.S. Environmental Protection Agency (EPA), 1989a, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A)***, Interim Final, Office of Emergency and Remedial Response, Washington, D.C., EPA/540/1-89/002.
- U.S. Environmental Protection Agency (EPA), 1991a, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance, Standard Default Exposure Factors***, Interim Final, Office of Solid Waste and Emergency Response, OSWER Directive: 9285.6-03.
- U.S. Environmental Protection Agency (EPA), 1992, ***Guidance on Risk Characterization for Risk Managers and Risk Assessors***, Memorandum from F.

Henry Habicht II, Deputy Administrator, to Assistant Administrators, Regional Administrators, February.

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

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

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

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

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

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

1.6 Report Organization

The remainder of this document is organized as follows:

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

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

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

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

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

- ***Chapter 7.0, Summary, Conclusions, and Recommendations.*** Provides a brief summary of the BHHRA, including quantitative results, uncertainties, and pertinent site information. Summary and discussion is focused on those results and issues that are most likely to directly affect site management decisions.

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

2.0 Data Evaluation

Data evaluation consists of a description of the appropriate data sources for each environmental medium sampled for each site, a discussion of data quality, a description of the methodology used for identification of the COPCs, and a summary of the COPCs for each LBA 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 include surface soil, subsurface soil, maintenance pit material, surface water, sediment, and monitoring well samples. The associated analytical suite for each environmental medium is also shown. This sample list includes those collected as part of the limited site inspection for the LBA (USACE, 2000) and the remedial investigation (RI) samples (Shaw, 2010b). All sampling locations are shown on Figure 2-1.

The three samples collected from the bottom of the concrete maintenance pit in the Locomotive Shop. As described in Section 1.3, this pit, formerly used to service locomotives and railcars, has been backfilled with loose stone. The pit samples were presumably composed of residual materials that were present in the pit prior to backfilling as well as fines and small stones from the gravel backfill. It is noted that samples from overburden well RCA-MW01 were conservatively excluded from the BHHRA exposure concentration calculations because this well was upgradient of the LBA, and the analytical results (Shaw, 2010b) indicated that no chemical was detected above its risk-based screening concentration (RBSC) and background screening concentration (BSC).

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, 2010a) and the limited site inspection report (USACE, 2000). The surface soil and subsurface soil are considered separate media. Surface and subsurface soil data are combined to assess exposures under the construction worker, future groundskeeper, and residential site use scenarios, which would involve excavation and mixing of surface and subsurface soil. Combined surface and subsurface soil data are termed “total soil” in the BHHRA. 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 generated from the combined data sets.

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, 2009). At one sampling location (LBA-SB04), the upper 0.9 foot was covered with roots, gravel fill, and scraps of wood; therefore, the surface soil sample collected at this location was taken from the 0.9 to 1.9 foot interval. The limited site inspection (USACE, 2000) historical surface soil samples were collected from a depth of either 0 to 0.5 or 0 to 2 feet, and the subsurface soil samples were collected at depth intervals that begin at a depth of 1 foot or greater below the surface; these intervals are consistent with surface soil and subsurface soil, respectively, as described in the RI report (Shaw, 2010b). As possible, the RI subsurface soil samples were generally collected at depths of 3 to 5 and 8 to 10 feet bgs. Note that where refusal or the water table was encountered before a depth of 10 feet bgs, samples from the deepest 2-foot interval were collected.

The samples collected from the bottom of the concrete-lined Locomotive Shop maintenance pit were evaluated separately from the other subsurface soil samples, because this material was from a very limited area and is composed mostly of stones and fines from backfill; thus, exposure to this material and any contaminants present in it would be negligible to minimal.

2.3 Evaluation of Data Quality

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

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

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

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

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

UJ - The analyte was not detected above the reporting limit. However, the reporting limit is approximate and may or may not represent the actual limit

of quantitation necessary to accurately and precisely measure the analyte in the sample.

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

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

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

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

2.4 Identification of Chemicals of Potential Concern

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

2.4.1 Risk-Based Screening

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

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 regional screening levels (RSL) table "tap water" values and RBSCs for soil are derived from "residential soil" RSL values (EPA, 2010a). This is a change in the source of the RBSCs based on discussion between USACE and OEPA (2009c), and this change is consistent with recent OEPA (OEPA, 2009a) guidelines. Previously, the groundwater and soil RBSCs were derived from the corresponding EPA (2004b) Region 9 preliminary remediation goals (PRG). The soil RBSCs are applied to both surface and subsurface soil.

RSL values are based on a concentration equal to either an incremental lifetime cancer risk (ILCR) of 1E-6 or a noncancer hazard quotient (HQ) of 1, the threshold at (or below) which adverse noncancer effects are regarded as unlikely to occur. For the BHHRA, the noncancer values listed in the RSL tables are multiplied by a factor of 0.1 to provide additional protection for simultaneous exposure to multiple chemicals (OEPA, 2009a; EPA, 2010a). This results in RBSC values associated with an HQ of 0.1. For cancer risk, the RSL values based on an ILCR of 1E-6 were used directly as RBSCs in the BHHRA. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) identifies acceptable exposure levels that are generally associated with concentration levels that represent an excess upper bound lifetime cancer risk to an individual of 1E-6 to 1E-4 (EPA, 1990). This range is hereinafter referred to as the "NCP risk management range." Cancer risks associated with RSL values represent the lower end of this range. The OEPA recognizes an overall cancer risk of 1E-5, which represents the logarithmic midpoint of the EPA risk management range, as a remedial goal (OEPA, 2009b). The RBSC for a chemical that elicits both cancer and noncancer health effects is selected based on either a cancer risk of 1E-6 or an HQ of 0.1, whichever associated concentration is lower.

Risks associated with exposure to sediment and surface water along the tributary to Plum Brook in the vicinity of the LBA are also evaluated in the BHHRA. Although RSLs have not been developed specifically for sediment and surface water, RBSCs can be derived from the RSLs based on site conditions at PBOW and the types of exposure to these media that may reasonably be anticipated. The routes by which receptors may be exposed to sediment (i.e., incidental ingestion, dermal contact) are similar to those by which receptors may be exposed to soil. However, sediment contact is expected to be appreciably less intense than soil contact, due to the lower duration and frequency of contact with sediment as compared with soil. Similarly, surface water exposure is expected to be much less intense than exposure to groundwater as surface water from Plum Brook is not regarded as a plausible source of drinking water, partly because the Erie County Health Department does not permit using surface water as private drinking water. Consequently, the exposure frequency is expected to be much lower for surface water, and the incidental ingestion of surface water would be much lower than the assumed intentional ingestion and use of groundwater from the tap. For these reasons, OEPA (1999) stated that unadjusted tap water PRG values (i.e., HQ = 1; ILCR = 1E-6) should be used for screening PBOW surface water. This screening protocol was adopted specifically because it was agreed that the magnitude of exposure associated with PBOW surface water exposure would be far less than that associated with household tap water. In other words, it was agreed that analytes with a maximum concentration at the PRG level would not contribute appreciably to overall risks and hazards for PBOW sites based on the exposure pathways of the surface water exposure scenarios for PBOW. The same agreement was reached for screening sediment against unadjusted residential soil PRGs. This protocol has been updated to base RBSCs on RSLs rather than PRGs. Even though the sediment and surface water RBSCs are an order of magnitude higher for noncarcinogens than the respective soil and groundwater RBSCs, these sediment and surface water RBSCs are regarded as protective of sediment and surface water receptors for screening because of the lower exposure rate to these media. Note that this previously made PBOW Team agreement for screening surface water and sediment (OEPA, 1999), which considered site-specific conditions, takes precedence over current OEPA (2009b) guidance, which simply states that adjusted RSLs (i.e., RBSCs) for soil may be used to screen contaminants in sediment, and those for groundwater may be used to screen surface water. The surface water RBSCs also meet the outside-of-the-mixing-zone average non-drinking water concentrations for the Lake Erie Basin.

The screening of lead in soil and groundwater is a special case. The EPA (2009d) Office of Water treatment technique action level of 15 µg/L for lead is listed in the RSL table, and the RSL User's Guide recommends this level for use as an RSL. Lead exposure and risk is evaluated separately from other chemicals using the EPA (2004c) Integrated Exposure Uptake Biokinetic

(IEUBK) model. The selection of the action level as the drinking water RSL is based partly on IEUBK model. Section 5.2 of the RSL User's Guide states that if the average tap water concentration exceeds 15 µg/L and the average soil concentration exceeds a value of 250 milligrams per kilogram (mg/kg), then more than the IEUBK target (EPA, 2004c) of 5 percent of the population of exposed children would exceed 10 micrograms per deciliter of lead in blood. Because an RSL of 15 µg/L sufficiently screens for a potential average concentration of 15 µg/L, the RSL of 15 µg/L is used as the RBSC. However, it is possible that the residential soil RSL of 400 mg/kg, which is selected as the soil RBSC, may not screen for an average soil concentration of 250 mg/kg within a given data set. Therefore, the following conditions were placed on the screening of lead: 1) If either the soil RBSC or groundwater RBSC is exceeded, then the IEUBK blood-lead model is run using both average soil and groundwater concentrations, and 2) if the average soil concentration exceeds 250 mg/kg, then the IEUBK model is run, even if neither RBSC is exceeded, using average concentrations of lead in both soil and groundwater. Note that for LBA soil, the MDC for lead (222 mg/kg) is less than both the RBSC (400 mg/kg) and the criterion for average concentration (250 mg/kg); the average lead concentration in LBA total soil was 20.6 mg/kg. Likewise, the lead MDCs in overburden (2.3 µg/L) and bedrock (2.6 µg/L) groundwater are both less than the RBSC (15 µg/L). Therefore, the IEUBK lead model was not run for the LBA soil and groundwater.

There are no RSLs for sulfate in tap water, but the drinking water outside-of-the-mixing-zone average value of 250 milligrams per liter (mg/L) for the Lake Erie Basin is used as the RBSC. This value is the same as the secondary drinking water regulation of 250 mg/L (EPA, 2006). Secondary drinking water regulations are non-promulgated values, based on aesthetic characteristics, which are used as guidelines for public water systems. A health-based advisory level of 500 mg/L also exists for sulfate (EPA, 2006).

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

2.4.2 Frequency of Detection

When confidence 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 present in the vicinity of PBOW (see Section 3.1.1).

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

Inorganics and organics are treated similarly from a quantitative perspective. However, all organics not eliminated on the basis of RBSC exceedance (Section 2.4.1) or infrequent detection (Section 2.4.2) are carried through the risk calculation process (exposure assessment, toxicity assessment, and risk characterization). As discussed 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 BSC. The background data set and derivation of soil BSCs for all PBOW soil investigations are described in IT (1998), and the background data set and derivation of BSCs for PBOW bedrock groundwater are described in the 2004 groundwater report (Shaw, 2005). It is noted that the method agreed upon for the development of BSCs, as recorded in the September 11, 2002 PBOW Team Meeting minutes, differs from that shown in current OEPA (2009d) guidance. This PBOW Team agreement, which has been used for all PBOW risk assessments to date, takes precedence over the subsequent OEPA guidance. Summary tables of the background data sets for soil and groundwater are provided as Tables 2-2 and 2-3, respectively. The background soil samples were collected from near the property boundary, away from any potential source areas, and the background groundwater wells were installed in off-site areas upgradient of PBOW sources. Briefly, BSCs were calculated for use at PBOW based on concentrations found in these background soil and bedrock monitoring well samples. Each BSC is either the MDC or the calculated 95th percent upper tolerance limit of the background data set, whichever value is lower (IT, 1998; Shaw, 2005). The background monitoring well samples were collected using low-flow samples and were unfiltered.

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

2.4.3.2 Statistical Data Set Testing of Inorganics

Statistical testing is performed to compare data sets of site inorganics data against the appropriate PBOW background data sets. As described in Section 2.4.3.1, the background data set for groundwater is found in the 2004 groundwater report (Shaw, 2005), and the background data set for soil is found in the site investigation for the acid areas (IT, 1998). As mentioned previously, background data sets do not exist for overburden groundwater, surface water, or sediment; therefore, a statistical background evaluation for COPCs in these media cannot be performed. The method for statistical comparison of the site data sets to the background data sets, described

in Appendix M of Shaw (2005), is the Wilcoxon Rank Sum (WRS) statistical test (also known as the Mann-Whitney U test). WRS testing is performed for inorganics having MDCs that exceed the respective BSCs and are identified as COPCs based on RBSC comparison (Section 2.4.1) and frequency of detection (Section 2.4.2). All COPCs are carried through the risk characterization process; thus, statistical testing results are not used to screen out any chemicals.

Site data sets are interpreted as being significantly different from PBOW background if the associated p-level is less than 0.05. WRS statistical output and box-and-whisker plots of the various inorganic COPC data sets are appended to the BHHRA for each inorganic data set evaluated against the appropriate site background data set; the WRS results are discussed as part of the uncertainty analysis (Section 6.2.10). 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 test 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). However, for those background-related analytes which are risk drivers, the risk characterization results without the contribution of these analytes are also presented as appropriate (Section 5.3).

2.4.3.3 Treatment of Organics

As mentioned in Section 2.4.3, certain organic compounds (e.g., BTEX and PAHs) in site media may be attributable to background conditions. However, no organic compounds are summarily screened out. Instead, all detected organic compounds are carried through the risk assessment process (i.e., exposure assessment, toxicity assessment, risk characterization) unless screened out on the basis of comparison to RBSCs (Section 2.4.1) or characterized as infrequently detected (Section 2.4.2). Background contributions of organics are discussed in the uncertainties analysis, as applicable (Section 6.2.10).

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), maintenance pit material (Table 2-7), bedrock groundwater (Table 2-8) and overburden groundwater (wells) (Table 2-9), sediment (Table 2-10), and surface water (Table 2-11). These tables provide the following information for each detected chemical in each environmental medium:

- Chemical name
- Frequency of detection
- Percent detection
- Range of detected concentrations
- Range of detection 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)
- EPC (for COPCs only).

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

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

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

- ***Surface Soil*** – Chromium, Aroclor 1221, benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, 2,4-DNT

- ***Subsurface Soil*** –Aluminum, 2,4-DNT, 2,6-DNT, 2-nitrotoluene , 3-nitrotoluene

- ***Total Soil*** **–** Aluminum, chromium, Aroclor 1221, benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, 2,4-DNT, 2,6-DNT, 2-nitrotoluene, 3-nitrotoluene

- ***Maintenance Pit Material*** – Benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene

- ***Bedrock Groundwater*** – Arsenic, iron, manganese, thallium, 2-methylnapthalene, naphthalene, benzene, chloroform, chloromethane, ethylbenzene, methylene chloride, 1,2,2,2-tetrachloroethane, toluene, xylenes, cyanide

- ***Overburden Groundwater*** – Chromium, cobalt, iron, manganese, sulfate, 2,4-DNT, 2,6-DNT, 2-nitrotoluene , 3-nitrotoluene , 4-nitrotoluene, carbon tetrachloride, dibromochloromethane, 1,1-dichloroethane, trichloroethene.

- ***Surface Water*** – Arsenic, chromium, cobalt, iron, lead, manganese

- ***Sediment*** – Arsenic, chromium, cobalt, manganese, thallium.

As discussed in Section 2.2, the total soil COPCs include all surface soil and subsurface soil COPCs. Note that the MDC for lead in surface water (18.0 µg/L) marginally exceeded the RBSC (15 µg/L); thus, lead was identified as a COPC in surface water. As described in Section 2.4.1, combined exposure to lead in drinking water and soil is evaluated using the IEUBK model. Exposure to surface water is assumed to occur only via dermal contact, and surface water in the vicinity of PBOW cannot be used as drinking water. Therefore, it is not appropriate to use the IEUBK model to evaluate this medium. It is also noted that, as stated in Section 2.4.1, arithmetic mean drinking water and soil concentrations are used as input values in the IEUBK model. The mean lead concentration of LBA surface water is 6.15 µg/L and the average lead concentration in LBA total soil is 20.6 mg/kg. Note that if a hypothetical average drinking water lead concentration of 15 µg/L is combined with a hypothetical average soil lead concentration of up to 250 mg/kg, the IEUBK model will result in an acceptable blood lead level (see Section 2.4.1). Therefore, even if surface water were used as drinking water, the IEUBK model would show an acceptable result for the LBA. Therefore, lead is not further evaluated in this BHHRA.

3.0 Exposure Assessment

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

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

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

3.1 Conceptual Site Exposure Model

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

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

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

The receptors and pathways on Figure 3-1 reflect scenarios developed from information regarding site background and history, topography, climate, and demographics as presented by D&M (1997) and the sitewide groundwater investigation (IT, 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 are evaluated. Most of the off-site residents are serviced by municipal water (from large surface water sources). Although there are numerous private groundwater wells in the vicinity, including eight within 1 mile of the facility boundary, none of these is used as a potable source. Based on the investigations of other PBOW sites, natural hydrocarbons and hydrogen sulfide are known to be present within the bedrock limestone, and shale formation groundwater generally provides low yields and is of low quality (e.g., Shaw [2008]). This was also found to be the case for LBA groundwater, where four of the six bedrock wells had petroleum on the cores at depth during drilling and a petroleum sheen was observed on a fifth (see Sections 3.1.1 and 6.2.5). Additionally, the groundwater yields in both the overburden and bedrock groundwater units were observed to be much too low to be used as a source of potable water, regardless of quality. Even though the development of groundwater for on-site residential (or on-site worker) use as tap water is regarded as implausible, potable groundwater use was evaluated for purposes of this BHHRA.

As stated in Section 2.2, material from the bottom of the maintenance pit was evaluated separately from the soil samples. Exposure to this material would be negligible for several reasons: 1) Because the material in the maintenance pit is primarily backfill, it would not be used as surface soil, where exposure is generally highest; 2) The larger particle sizes associated with this material will not likely adhere to the skin as readily as does soil, thus minimizing dermal absorption and ingestion via hand-to-mouth contact; 3) The volume of the maintenance pit is approximately only 40 cubic yards, most of which is fill gravel; thus, the fraction of "soil-like" material to which an individual may be exposed over a chronic or subchronic time period that would be composed of the maintenance pit material is considered minimal or negligible, as compared to all other sources of soil-like material to which he would be exposed. Although exposure to the maintenance pit material is thought to be negligible to minimal, COPCs identified in this material were evaluated assuming a normal construction worker scenario. This was performed for purposes of comparison to other soil media, but is not regarded as a reasonable scenario because it is overly conservative for this material.

3.1.1 Physical Setting

Climate/Meteorology. The climate in the Sandusky area is continental and strongly affected by Lake Erie. July is generally the warmest month (average high and low temperatures of 82 and 65 degrees Fahrenheit [°F], respectively), and January is generally the coldest (average high and

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

Geology. Three formations, all of Devonian Age, outcrop across PBOW, each of which was encountered in the upper 100 feet of bedrock at PBOW (Shaw, 2005). The Delaware Limestone is the lowermost formation screened by site wells. It is characterized as a hard, dense, finely crystalline limestone and dolomite. The unit is typically buff colored and usually is described as fossiliferous. In the vicinity of PBOW, quarries mine limestone from the Delaware. Traces of natural petroleum-derived hydrocarbons and hydrogen sulfide are common in area quarries (Shaw, 2005). Hydrogen sulfide was detected in the groundwater during sample collection of LBA monitoring wells IT-MNTA-BEDGW-001 (more than 200 parts per million [ppm]), which is one of the highest hydrogen sulfide producers on PBOW, and RCA-BEDGW-002 (102 ppm). A strong hydrogen sulfide odor was also noted during the development of LBA-BEDGW-002. Hydrocarbon seepage was noted on the rock cores at depth in the boring logs of LBA bedrock wells LBA-BEDGW-002, RCA-BEDGW-001, RCA-BEDGW-002, and RCA-BEDGW-003. Petroleum sheens have also been noted on the water surface of these wells and that of IT-MNTA-BEDGW-001.

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- to 50-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 is present in the PBOW area, the Huron Shale. 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). In the vicinity of the LBA, the shale was found to be between 11 and18 feet thick (Shaw, 2010b).

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

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

Hydrology. The two main water-bearing zones at PBOW are located in the overburden/shale unit and the limestone bedrock and are thus called the overburden/shale and bedrock water-bearing zones. The overburden and shale groundwater units show similar water levels in these two units, suggesting substantial vertical communication. Therefore, these two geologic units are combined for purposes of PBOW groundwater evaluation. Data collected during the more recent investigations (Shaw, 2005; IT, 1997, 1999, 2001b) indicate that groundwater in the overburden at PBOW is in discontinuous pockets during dry time periods. The shallow overburden generally has low yields over most of the site due to the high percentage of silt and clay. In contrast, the limestone bedrock water-bearing zone is saturated year round. During periods of low precipitation, only limited migration of contaminants would occur in the overburden due to less

infiltration. During the LBA RI, four of the six overburden wells had to sampled via bailer during wet season and dry season sampling events because the flow rate in each case was less than 50 milliliters per minute (ml/min) (i.e., 3 liters per hour). Only wells LBA-MW-02 and LBA-MW03 could be sampled using the low-flow method. Well LBA-MW-02 could be sampled using flow-low method (i.e., >50 ml/min) during both sampling events, but the flow rate was only minimally greater than 50 ml/min, at 70 and 120 ml/min (Shaw, 2010b) in May and November 2009, respectively. Well LBA-MW03 was sampled using low-flow methodology, even though this well did not meet the 50 ml/min flow criterion in May 2009 (42 ml/min) and marginally met the criterion in November 2009.

The general flow direction in the overburden water-bearing zone in the LBA is topographically controlled, toward the unnamed tributary that divides the Locomotive Shop area and the Rail Car Washing Area. Thus, flow in the vicinity of the Locomotive Shop is toward the east-northeast, and flow within the Rail Car Washing Area is toward the west-northwest. A hydrogeological study by the U.S. Geological Survey (1992) conducted in the glacial deposits of Sandusky in 1990 reported a horizontal hydraulic conductivity of 0.046 feet per day and a vertical hydraulic conductivity of 1.2 feet per day.

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

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). The LBA appears to be in Zone 1.

Surface Water. A small, unnamed tributary which flows southwest to northeast bisects the area between the Locomotive Shop and the Rail Car Washing Area. Flow has been observed during some site visits, but not during others. However, water has always been observed in this unnamed tributary. The average width of this tributary is approximately 6 feet and the average depth is roughly 1 foot. A clay pipe has been observed that surfaces along the eastern bank of the unnamed tributary, just west of the Rail Car Washing Area, several feet up from the level of the water. Water was observed flowing from this pipe during a sampling event in March 2009. A ditch flowing southeast to northwest, from a culvert under Maintenance Road (from Ash Pit No. 1), empties into the unnamed tributary (Figure 1-2). Water flows in this ditch during precipitation events, but it is dry at other times.

3.1.2 Contaminant Sources, Release Mechanisms, and Migration Pathways

Contaminant sources, release mechanisms, and migration pathways are summarized on Figure 3-1. Briefly, the Locomotive Shop area may have received hydrocarbons, solvents, and metals associated with locomotive repair. Materials may have been carried to the surface soil of the adjacent area via windborne deposition, tracking by mechanics and other personnel, washing, and possibly via leaking or spilling. The maintenance pit and sump could potentially provide a preferential pathway for contamination of the subsurface soil and groundwater. It is possible that contamination from the Locomotive Shop area could have adversely affected the surface water and sediment in the unnamed stream via overland surface flow associated with rain events. Any contamination present in groundwater associated with the Locomotive Shop area may also potentially impact surface water and sediment in the unnamed tributary to Plum Brook, as this tributary likely receives groundwater recharge from the overburden groundwater unit.

The same types of contaminants would potentially have been generated during the Rail Car Washing Area operation. The adjacent surface and subsurface soils may have been impacted via washing of contaminants into the pit. The waste water may have impacted the groundwater, as well as surface water and sediment at the adjacent unnamed tributary to Plum Brook. Any contamination present in groundwater associated with the Rail Car Washing Area may also potentially impact surface water and sediment in the unnamed tributary to Plum Brook, as this tributary likely receives groundwater recharge from the overburden groundwater unit.

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 the LBA 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. 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. Although no construction is currently planned, the construction worker scenario 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.

- Current groundskeeper
- Future groundskeeper
- Future indoor worker
- Current/future construction worker
- Future on-site resident
- Future hunter
- Future hunter's child.

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

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

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

Receptors and the associated exposure pathways are presented in the following subsections. 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. Total soil is typically evaluated under the future groundskeeper use scenario because hypothetical future construction may include considerable excavation of subsurface soil. This soil may be spread on the surface and regraded such that some of the soil currently in the subsurface (i.e., 1 to 10 feet bgs) will be spread as surface soil (0 to 1 foot bgs). Samples collected from within the upper foot to a depth of 2 feet are regarded as surface soil in the BHHRA (see Section 2.2). Groundwater use is also evaluated for the groundskeeper in the future site use scenario, as discussed in Section 3.1, which could theoretically be developed as a source of drinking water. It is assumed that any contact with surface water or sediment associated with the tributary to Plum Brook by this receptor would be infrequent and sporadic, because such contact would not be a part of the groundskeeper's regular duties or activities. Therefore, exposure to these media is not quantified.

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

Shaw's experience has been that surface soil that is contaminated with VOCs and that has been in place for extended periods is not a significant source of airborne VOCs, because infiltration

and dissipation over time reduces residues at the surface (i.e., first few centimeters) from which volatilization would occur. However, as noted above, the data set for surface soil may include samples taken from up to 1 foot bgs, which would include the soil zone deeper than the top few centimeters, where dissipation has not reduced VOC concentrations. In other words, the surface soil data set might indicate the presence of VOCs, although volatilization to the air is unlikely to be significant. Therefore, a surface soil-to-air volatilization model is not used in addition to the activity-based dust emissions model to estimate airborne concentrations of VOCs. Instead, the airborne concentrations estimated by the dust emissions model are assumed to sufficiently estimate levels of VOCs that may arise from volatilization, because the dust emissions model treats the VOCs as if they were located at the surface. It is assumed that VOC emissions from subsurface soil (i.e., at depths greater than 1 foot bgs) would be attenuated by the overlying soil so that concentrations in ambient air would not be toxicologically significant.

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

3.1.3.2 Future Groundskeeper

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

In addition, a hypothetical future groundskeeper is assumed to be exposed to groundwater, which could theoretically be developed as a source of drinking water in the future. His drinking water ingestion rate is assumed to be 1 liter per day (L/day) (EPA, 1991a). He may also experience dermal contact with groundwater used to clean equipment and to rinse dust or perspiration from his body. For this evaluation, it is assumed that the head, forearms, and hands, approximately 3,300 cm^2 of his body (EPA, 2004a), would be exposed intermittently for up to 1 hour/day. Because exposure is assumed to be intermittent rather than continuous, organic

chemical uptake across the dermis would not reach steady state, which guides the selection of the EPA (2004a) model used to quantify this pathway (see Section 3.3).

3.1.3.3 Indoor Worker

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

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

A future indoor worker is assumed to be exposed to groundwater, which could theoretically be developed as a source of drinking water (see Section 3.1). His drinking water ingestion rate is assumed to be 1 L/day (EPA, 1991a). Some indoor worker positions may require relatively frequent dermal contact with groundwater as well, e.g., a food preparer/cafeteria worker who would wash his hands, produce, equipment, etc. For this evaluation, it is assumed that the head, forearms, and hands, approximately 3,300 cm^2 of his body (EPA, 2004a), would be exposed intermittently for up to 1 hour per day. Because exposure is assumed to be intermittent rather

than continuous, organic chemical uptake across the dermis would not reach steady state, which guides the selection of the EPA (2004a) model used to quantify this pathway (see Section 3.3.4).

3.1.3.4 Construction Worker

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

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

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

The construction worker may be exposed to surface water and sediment during the 6-month construction period. The construction worker dermal exposure parameters for sediment are assumed to be exactly the same as those for soil. Dermal exposure to surface water is assumed to occur for up to 4 hours per day or one-half the normal work day. It is assumed the exposure to surface water is intermittent during this period. An exposed body surface area of 3,300 cm^2, the same as for sediment and soil, is assumed for exposure of the construction worker to surface water. It is expected that the construction worker would wear appropriate footgear and leg protection to minimize surface water and sediment exposure to the legs.

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

The construction worker scenario was also selected to evaluate exposure to the maintenance pit material separately from the evaluation to total soil. As discussed in Section 3.1, the rate of exposure to this material is expected to be negligible to minimal, and much lower than the rate of exposure to total soil. Nonetheless, the same exposure parameters were used for evaluating this material as were used to evaluate total soil for the sake of consistency.

3.1.3.5 On-Site Resident

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

The on-site residential scenario is evaluated assuming a 30-year residential exposure scenario, considering exposure to a resident as a young child (6-year duration, ages 1 through 6 years) through the adult portion of life spent at this residence (24-year duration) (EPA, 1991a). Noncancer hazard estimates are derived separately for the child and adult life stages. Cancer risk is estimated as the sum of the risks calculated for the adult (24 years) and the child (6 years) (EPA, 2002; EPA, 2010a).

The resident is assumed to be exposed directly to total soil, because residential development would involve excavation and regrading, which would mix surface and subsurface soil. Relevant pathways for total soil exposure include incidental ingestion, dermal contact, and inhalation of dust and VOCs. Evaluation of VOCs from total soil is addressed during evaluation of airborne dust, as described for the groundskeeper. For evaluating inhalation of airborne dust, it is assumed that 80 percent of the soil surface is covered with pavement or vegetation. Inhalation of VOCs released from subsurface soil entrapped in indoor air is also evaluated. The resident is also assumed to be exposed to VOCs that have been released from subsurface soil through cracks in the building foundation to indoor air. It is noted that because some of the subsurface soil is expected to be brought to the surface in the future, using only subsurface soil data will conservatively result in some double counting of exposure to any VOC COPCs that may be present in the subsurface soil. This may be addressed in the uncertainty analysis should the subsurface soil-to-indoor air pathway significantly affects risk and hazard estimates. This pathway did not contribute significantly to risk at the LBA, as 2-nitrotoluene is the only volatile chemical identified as a COPC in total soil; this marginally volatile compound (Henry's constant of 1.25E-5 atmospheres/cubic meter-mole) was detected in only 7 percent of total soil samples and has no toxicity value for the inhalation pathway.

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

inhalation of VOCs released to the air during household use of tap water associated with multiple household uses.

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

The adult resident is assumed to be a 70-kg person with an incidental soil ingestion rate of 100 mg/day and an inhalation rate of 20 cubic meters per day (m^3/day) or 0.83m^3/hr (EPA, 1991a). A body surface area of 5,700 cm^2, representing the hands, forearms, head, and lower legs, is assumed to be available for dermal exposure to soil (EPA, 2004a). A soil AF of 0.07 mg/cm^2 is used as the default RME value for the adult resident (EPA, 2004a). The adult resident is assumed to be exposed for 350 days/year for 24 years (EPA, 1991a; 2002).

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

It is assumed that the resident would visit the unnamed tributary to Plum Brook for 8 hours/day, 2 days/week during the warmer half of the year. This resident is assumed to wade for 3 hours/day on 52 days of the year. Mechanisms of exposure to soil and sediment are likely to be similar. Therefore, the incidental soil ingestion rate of 100 mg/day, the surface area of 5,700 cm^2, and the AF of 0.07 mg/cm^2 are also applied to sediment exposure in the adult. Similarly, the resident child soil ingestion rate of 200 mg/day, skin surface area of 2,800 cm^2, and soil AF of 0.2 mg/cm^2 are applied to sediment exposure for this receptor. The tributary in the vicinity of the LBA generally contains less than 2 feet of water, limiting the surface area of the body that would

typically be exposed. It is assumed that an adult body surface area of 7,000 cm^2 is available for exposure to surface water. This represents the same body parts to which soil and sediment would be exposed (i.e., hands, forearms, head, and lower legs) plus the feet (EPA, 1997a; 2004a). The body surface area of 2,800 cm^2, representing the hands, forearms, head, lower legs, and feet, used for soil and sediment exposure in the young child are also used for surface water exposure for this receptor.

EPA (1989a) permits the development of an FI to reflect the proportion of total daily exposure that a receptor obtains from potentially contaminated medium (refer to Section 3.1.3). For this receptor, the FI is used to apportion the resident's time of exposure between site soil and sediment. It is assumed that the resident spends 16 hours/day awake and potentially exposed to soil or sediment. As previously noted, 350 days/year are available for contact with soil; 52 of those days are also available for contact with sediment. It is assumed that contact with soil and sediment does not occur simultaneously; i.e., on those days when the resident spends time at the streams, 8 hours would be spent in contact with soil and 8 hours would be spent in contact with sediment. Therefore, the fraction of exposure to soil is 16 hours/16 hours = 1 on the 298 days without time spent at the streams, and the fraction of exposure to soil is estimated as 8 hours/16 hours = 0.5 on the 52 days with some time spent at the tributary. A weighted fraction of 0.93 (rounded to 0.9) is estimated for exposure to soil over the entire 350 days/year exposure frequency. A weighted fraction of 0.07 (rounded to 0.1) is estimated for exposure to sediment over the entire 350 days/year exposure frequency.

An adolescent resident may be the individual most likely to have regular exposure with sediment and surface water associated with the tributary to Plum Brook. It is not expected that adults would regularly visit this tributary, as it does not support game fish and would seemingly not provide any attraction. It is unlikely that a young child (i.e., ages 1 through 6) would frequent this unnamed tributary 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 as a young child and 24 years as an adult. For cancer effects, the 30-year exposure to surface water and sediment represented by both the young child and adult are combined. This approach is more conservative than evaluating an adolescent and is also consistent with BHHRAs performed for PBOW sites in the past.

With respect to groundwater exposure, it is assumed that an adult resident ingests 2 L/day of tap water (EPA, 1991a), and that the young child drinks 1 L/day (EPA, 2010b). The total body surface areas of the adult and of the young child resident are assumed to be exposed to tap water

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

3.1.3.6 Hunter

This scenario is created to evaluate the potential for contaminants in soil to affect food chain pathways. The LBA provides habitat for deer and other wildlife, especially in the eastern portion near the Rail Car Washing Area. Although hunting is not permitted on the LBA, deer hunting is permitted on other portions of the PBOW facility. Therefore, a hunter who consumes his game is evaluated in the LBA BHHRA, as hunting is a potential future land use.

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

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

The deer hunter is assumed to be a 70-kg adult who harvests deer and consumes venison over a 30-year period. It is assumed that he spends 14 days per year hunting on PBOW. His incidental soil ingestion rate is assumed to be 100 mg/day (EPA, 1991a). Hunting at PBOW occurs in the

fall and winter. Given the temperate climate of northern Ohio during hunting season, a hunter would dress appropriately, with typically only the hands and head exposed at most. The default industrial RME exposed skin surface area of 3,300 cm^2, which represents the hands, forearms, and head (EPA, 2004a), is conservatively assumed for the hunter. The default industrial RME soil AF of 0.2 mg/cm^2 (EPA, 2004a) is also assumed.

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

3.1.3.7 Hunter's Child

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

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

3.1.3.8 Other Receptors Not Considered

Another plausible receptor group is delivery personnel. These receptors, however, would be less intensively exposed to soil than the groundskeeper; therefore, their exposures are not evaluated.

The LBA could potentially become part of the area used for National Guard activities. National Guard trainees would be unlikely to spend a total of more than 2 months at the site if they were to receive all their training in this area. A 2-month exposure period would represent a subchronic duration, as opposed to the 25-year duration assumed for the groundskeeper. A National Guard Reservist may be required to frequent the site for 1 weekend per month throughout the year and serve an additional 2 weeks per year (total of 38 days per year). A 20-year Reservist career is unlikely to include more than two 2-month calls to training (120 days total) for active duty throughout this career. Conservatively assuming that all of this training is spent in the LBA at PBOW and that the 20-year Reserve soldier career is spent at Plum Brook, the exposure frequency for this receptor would be 44 days per year. This is considerably less than the groundskeeper scenario, where a 25-year, 250 days/year exposure assumption is made. Because the Nation Guard Reservist scenario 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. Therefore, fish ingestion as an indirect pathway for exposure to surface water and sediment is not evaluated. Also, as discussed in Section 3.1, off-site use of groundwater is not evaluated because nearby residents use municipal water (from surface water sources) as a potable source, and potential on-site users would be exposed to higher concentrations of contaminants in groundwater.

3.2 Quantification of Exposure-Point Concentrations

The EPC is an estimate of the concentration of a COPC in a given medium to which a receptor may be exposed over the duration of the exposure. An EPC may be based on chemical concentrations in media that have been directly measured using laboratory analysis, or it may be derived based on environmental medium-to-medium transport modeling. The EPCs of COPCs in soil, groundwater, surface water, and sediment are derived based on measured analytical data. Note that the EPC for dermal exposure to VOCs in groundwater is based on one-half the EPC concentration derived from the measured concentrations in groundwater (Sections 3.2.1 and 3.2.2.3). This value is used because it is assumed that 50 percent of the groundwater VOC concentration is volatilized during normal household use (Section 3.2.2.2). Concentrations of COPCs in air and venison are not measured (and in some cases cannot reasonably be measured) but are based on models that use the EPCs of COPCs in the appropriate directly measured media (i.e., soil and groundwater) as input values.

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

3.2.1 Soil, Groundwater, Surface Water, and Sediment Concentrations

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

The EPA (2009a,b,c) ProUCL (Version 4.00.04) software was used to estimate UCLs for the data sets of all environmental media represented by at least five samples. If the data set consists of fewer than five data points, the MDC was selected as the EPC. Analytical data from field duplicates are averaged with originals to yield one result for use in the statistical manipulations (see Section 2.5). One-half the reporting limit is used as the ProUCL input concentration for nondetects. Nondetect sample results with aberrantly high detection limits, due to matrix interferences or other sample-specific causes, are included in the initial ProUCL calculations. This is a conservative approach, as EPA (1989a) recommends that nondetected results with aberrantly high detection limits be removed from the data set so that calculation of the UCL is not unduly skewed by a nondetect. Because the latest version of ProUCL (EPA, 2009a) includes mathematical manipulations 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 LBA BHHRA.

ProUCL generates a variety of UCL estimates for each data set. The ProUCL output for each COPC is included in Appendix B. Generally, the results of one or two (sometimes more) of the UCL estimates are recommended. This recommendation is based on a variety of factors, including the distribution (i.e., normal, lognormal, gamma, or not discernable) that provides the best fit, number of nondetects, size of the data set, and skewness. If the recommended value(s) under the default mode equals or exceeds the MDC, ProUCL is re-run 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 subsections for estimating COPC concentrations in air include only those relevant and used specifically for LBA 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):

Eq. 3.1

$$C_a = (D)(C_s)(CF_1)$$

where:

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

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

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

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

Eq. 3.2

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

where:

PEF	=	particulate emission factor (cubic meters per kilogram [m^3/kg], calculated)
Q/C	=	inverse of the mean concentration at center of square source (55.99 grams per square meter-second per kg/m^3, site-specific value from Table 3 in EPA [1996] [Zone 7, Cleveland, 5-acre site])
3600	=	seconds/hour

V	=	fraction of surface covered with vegetation (0.8, unitless, assumed)
U_m	=	mean annual wind speed (default, 4.60 meters per second [m/second] equals mean annual wind speed of 10.3 miles per hour [see Section 3.1.1])
U_t	=	equivalent threshold value of wind speed at 7 meters (default, 11.32 m/second)
F(x)	=	function dependent on U_m/U_t (default, 0.194).

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

Eq. 3.3

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

where:

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

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

3.2.2.2 Concentrations in Household Air from Groundwater Use

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

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

Eq. 3.4

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

where:

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

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

3.2.2.3 Concentrations of VOCs in Groundwater: Resident Dermal Uptake

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

Eq. 3.5

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

where:

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

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

3.2.2.4 Exposure-Point Concentrations of COPCs in Venison

The hunter is assumed to harvest and consume game and share it with family members, including small children. The game is assumed to be venison, because the white-tailed deer is the species hunted most widely and most likely to provide a regular contribution to the diet. Data to reliably estimate contaminant concentrations in venison do not exist, 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 the LBA therefore, the fraction of total browse consumed from the LBA 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 chemicals, physical properties, behavior in environmental media, biotransfer capability, and toxicity values.

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

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

Eq. 3.7

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

where:

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

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

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

Eq. 3.8

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

where:

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

3.3 Quantification of Chemical Intake

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

3.3.1 Inhalation of COPCs in Air

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

Eq. 3.9

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

where:

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

3.3.2 Incidental Ingestion of COPCs in Soil or Sediment

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

Eq. 3.10

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

where:

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

3.3.3 Incidental Ingestion of COPCs in Water

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

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

where:

I_w	=	I_{gw} for groundwater, I_{sw} for surface water, = ingested dose of COPC in water (mg/kg-day, calculated)
C_w	=	C_{gw} for groundwater, C_{sw} for surface water, = concentration of COPC in water (mg/L)
IR_w	=	C_{gw} for groundwater, C_{sw} for surface water, = water ingestion rate (L/day)
FI_w	=	FI_{gw} for groundwater, FI_{sw} for surface water, = fraction of exposure attributed to site water (unitless)
EF_w	=	EF_{gw} for groundwater, EF_{sw} for surface water, = fraction of exposure attributed to site water exposure frequency (days/year)
ED_w	=	ED_{gw} for groundwater, ED_{sw} for surface water, = exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days).

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

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

Eq. 3.12

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

where:

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

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

Eq. 3.13

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

where:

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

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

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

Eq. 3.14

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

where:

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

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

Eq. 3.15

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

where:

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

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

Eq. 3.16

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

where:

DA	=	dose absorbed per unit body surface area per day (mg/cm^2-day, calculated)
C_w	=	concentration of COPC in water (mg/L)
FA	=	fraction absorbed from the water (unitless)
K_p	=	permeability coefficient (cm/hour)

CF = conversion factor (1E-3 L/cm^3)
τ = lag time for contaminant to cross stratum corneum per event (hours)
ET_w = time of contact (hour(s)/day)
B = Ratio of the permeability coefficient of a compound through the stratum corneum relative to its permeability coefficient across the viable epidermis (unitless).

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

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

Eq. 3.17

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

where:

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

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

Eq. 3.18

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

where:

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

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

3.3.5 Consumption of Venison

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

Eq. 3.19

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

where:

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

4.0 Toxicity Evaluation

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

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

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

4.1 Evaluation of Carcinogenicity

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

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

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

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

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

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

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

Eq. 4.1

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

where:

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

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

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

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

4.2 Evaluation of Noncarcinogenic Effects

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

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

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

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

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

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

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

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

4.3 Dermal Toxicity Values

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

4.4 Target Organ Toxicity

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

Mechanisms of toxicity data sufficient for predicting additivity with a high level of confidence are available for very few chemicals. In the absence of such data, EPA (1989a) assumes that chemicals that act on the same target organ may do so by the same mechanism of toxicity; that is, the target organ serves as a surrogate for mechanism of toxicity. When total HI for all media for a receptor exceeds 1 due to the contributions of several chemicals, it may be 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, 2010c) database.
- **Tier 2** values: These are EPA's provisional peer-reviewed toxicity values. The provisional peer-reviewed toxicity values are developed by the Office of Research and Development, the National Center for Environmental Assessment, and the Superfund Health Risk Technical Support Center on a chemical-specific basis when requested by the Superfund program.
- **Tier 3** values: These are other toxicity values from additional EPA and non-EPA sources of toxicity information. As stated in the EPA Office of Solid Waste and Emergency Response directive, "priority should be given to those sources of information that are the most current, the basis for which is transparent and publicly available, and which have been peer reviewed." Two common examples of Tier 3 values are the EPA's Health Effects Assessment Summary Tables (EPA, 1997b) and

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

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

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

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

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

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

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

5.0 Risk Characterization

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

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

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

5.1 Cancer Risk

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

Eq. 5.1

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

where:

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

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

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

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

Eq. 5.2

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

where:

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

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

Eq. 5.3

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

where:

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

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

Eq. 5.4

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

where:

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

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

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

5.2 Noncancer Effects of Chemicals

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

Eq. 5.5

$$HQ = I \ / \ RfD$$

where:

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

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

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

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

Eq. 5.6

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

where:

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

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

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

Eq. 5.7

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

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. Segregation by target organ was not performed for the LBA BHHRA because either the total HI for the receptor met the target criterion of 1, or in the case of hypothetical overburden (child resident only) and bedrock groundwater use, a single chemical exhibited an HQ exceeding a value of 1.

5.3 Risk Characterization Results

Cancer and noncancer risk characterization results were evaluated for each receptor and each environmental medium, using the methods described in Sections 5.1 and 5.2. The following subsections describe the risk characterization results for each receptor, which are summarized in Tables 5-1 and 5-2. The detailed quantitative evaluation tables that include the exposure

equations presented in Chapter 3.0 and the risk characterization equations presented in Chapter 5.0 are provided in Appendix D.

5.3.1 Current Groundkeeper

The ILCR for the current groundskeeper (Table 5-1) exposed to surface soil both through direct contact and via inhalation of suspended particulates is estimated as 9E-6. This is within the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5. It is noted that approximately 70 percent of this value is associated with the inhalation of chromium (6.6E-6). As presented in Section 6.2.10, chromium appears to be present in LBA surface soil at background concentrations. Excluding chromium, the ILCR for the current groundskeeper is 3E-6.

The total HI for the current groundskeeper (Table 5-1) is 0.007. This is far less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

5.3.2 Future Groundskeeper

The exposure assumptions for the future groundskeeper differ from those of the current groundskeeper (Section 5.3.1) in that the future groundskeeper is assumed to be exposed to total soil rather than surface soil, and the future groundkeeper is also assumed to be exposed to groundwater via direct contact (ingestion and dermal exposure). The risk characterization was run separately for bedrock groundwater exposure and overburden (well) groundwater exposure.

Bedrock Groundwater Use. The total ILCR for the future groundskeeper, assuming hypothetical bedrock groundwater use, is 1E-3 (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk criterion of 1E-5. Over 99 percent of this ILCR is associated with benzene (3.5E-4) and arsenic (6.9E-4) in groundwater.

Benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and petroleum was reported seeping at depth in four of the six LBA monitoring wells: LBA-BEDGW-002 (49.6 to 56.4 feet; petroleum sheen noted on water surface), RCA-BEDGW-001 (53.8 to 70.2 feet), RCA-BEDGW-002 (57.0 to 58.2 feet), RCA-BEDGW-003 (62.4 to 80.2 feet). Also, a petroleum sheen was seen on the surface of well IT-MNTA-BEDGW-001, which is also one of the highest hydrogen sulfide producers at PBOW.

Arsenic was detected in two bedrock monitoring wells, LBA-BEDMW-002 (131 µg/L [May 2009]) and RCA-BEDMW-002 (28.0 µg/L [May 2009]; 19.2 µg/L [November 2009]), and at relatively high concentrations. It is noted that three of the six LBA wells were dry and could not

be sampled in May or November 2009; LBA-BEDMW-002 could only be sampled in May, as it was dry in November 2009. It is also noted that the 5 samples that could be collected from the 12 bedrock well samples all had to be collected via bailer because of insufficient water volume for the low-flow sampling method. The low yield for each of the bedrock monitoring wells indicates that potable use by a site worker is not a plausible exposure pathway. In addition, the arsenic present in the LBA bedrock groundwater appears to be unrelated to PBOW activities based on the following observations:

- Arsenic was not detected in any of the overlying overburden well samples, including those nested with the wells LBA-BEGW-002 and RCA-BEDGW-002.
- Arsenic was found to be present at background concentrations in LBA soil samples. The maximum detected (17.7 mg/kg) and mean concentrations (8.1 mg/kg) of arsenic in total soil are both less than those of PBOW background data set (36.5 mg/kg and 10.1 mg/kg, respectively).

The ILCR for the future groundskeeper without the contributions of groundwater is 8E-6, which is within the NCP cancer risk management range and less than the 1E-5 PBOW cancer risk criterion. Also, it is noted that 65 percent of this value is associated with chromium in total soil. As discussed in Section 6.2.10, chromium in total soil is shown to be present at background concentrations. The total soil ILCR for this receptor without the contribution of background chromium is 3E-6.

The total HI for the future groundskeeper (Table 5-1) is 11. This exceeds the target HI value of 1, which would seem to indicate that noncancer health effects cannot be regarded as unlikely to occur. Virtually all of this HI is associated with the hypothetical use of bedrock groundwater. As discussed previously for cancer risk to the future groundskeeper, LBA groundwater is not present in sufficient quantity. Also, note that approximately 95 percent of this HI value is associated with benzene (HQ=4.4), 2-methylnapthalene (HQ=1.0), arsenic (HQ=4.3), and thallium (HQ=1.0) in groundwater. As stated above, benzene is not associated with former PBOW operations but is associated with naturally occurring petroleum. The same is true of 2-methylnapthalene, which likewise was not detected in overburden groundwater and was detected in only 2 of 37 total soil samples and at concentrations less than the RBSC.

As discussed previously for cancer effects, arsenic was not detected in LBA overburden groundwater and was found to be present in soil at background concentrations. Similarly, the thallium found in bedrock groundwater is apparently not associated with former LBA activities. This is evident from a lack of detections of thallium in overburden groundwater and the

observation that the MDC for thallium (0.678 mg/kg) in total soil is less than the total soil BSC (1.3 mg/kg). The HI for this receptor without the contributions from bedrock groundwater would be 0.2.

Overburden Groundwater Use. 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 it exceeds the PBOW target cancer risk criterion of 1E-5. Approximately 85 percent of this ILCR is associated with 2,4-DNT (1.1E-5), 2,6-DNT (9.7E-6), and 2-nitrotoluene (3.1E-5) in overburden groundwater. These nitroaromatic COPCs clearly appear to be related to former TNT manufacturing operations. However, overburden groundwater underlying the LBA would not provide adequate volume for use assumed under the future groundskeeper scenario. This is apparent from the following observations:

- Of the six LBA monitoring wells, only LBA-MW02 and LBA-MW03 had adequate volume to sample using low-flow sampling methodology.
- One well (RCA-MW01) was too dry in November 2009 to be sampled even by bailer.
- LBA-MW02 could be sampled using low-flow at a rate of 120 ml/min in May 2009 and 70 ml/min in November 2009. A well would have to produce many times this amount of water to be considered a viable potable source with respect to yield (e.g., 3 gpm [i.e., >10,000 ml/min]).

If overburden groundwater exposure is appropriately excluded, the ILCR is 8E-6. This value is within the NCP risk management range and is less than the PBOW criterion of 1E-5.

The total HI for the future groundskeeper, assuming hypothetical use of overburden groundwater, is 0.9 (Table 5-1). This is less than the target HI criterion of 1, indicating that noncancer health effects would be unlikely to occur under this scenario. As noted previously, the overburden groundwater underlying the LBA does not provide sufficient yield. The total HI for the future groundskeeper excluding use of overburden water is 0.2, which also meets the target HI criterion.

5.3.3 Indoor Worker

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

Bedrock Groundwater Use. The total ILCR for the future indoor worker, assuming hypothetical bedrock groundwater use, is 1E-3 (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk criterion of 1E-5. Over 99 percent of this ILCR is associated with benzene and arsenic in groundwater. As described in Section 5.3.2, benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and petroleum was reported seeping at depth in four of the six LBA bedrock wells and a petroleum sheen was observed on a fifth well (Shaw, 2010b). Also, as discussed in Section 5.3.2, the presence of elevated arsenic in LBA bedrock groundwater does not appear to be related to former site activities. The ILCR for the future indoor worker is 8E-7 if the contribution of bedrock groundwater is appropriately excluded. This value is less than both the NCP risk management range and the PBOW 1E-5 cancer risk criterion.

The total HI for the future indoor worker (Table 5-1) is 11. This exceeds the target HI value of 1, which would seem to indicate that noncancer health effects cannot be regarded as unlikely to occur. Virtually all of this HI is associated with the hypothetical use of bedrock groundwater. As discussed in Section 5.3.2 for the future groundskeeper, LBA groundwater is not present in quantities sufficient for use. Also, note that approximately 95 percent of this HI value is associated with benzene (HQ=4.4), 2-methylnapthalene (HQ=1.0), arsenic (HQ=4.3), and thallium (HQ=1.0) in bedrock groundwater. As stated above, benzene is not associated with former PBOW operations but is associated with naturally occurring petroleum. The same is true of 2-methylnapthalene, which likewise was not detected in overburden groundwater and was detected in only 2 of 37 total soil samples and at concentrations less than the RBSC. Neither do the presence of elevated arsenic concentrations and thallium appear to be associated with site-related activities, as discussed in Section 5.3.2.

If the contributions of hypothetical bedrock groundwater are excluded, the resulting HI for the indoor worker is 0.002. This is far less than the target criterion of 1 and suggests that adverse noncancer health effects are unlikely.

Overburden Groundwater Use. The ILCR for the future indoor worker assuming hypothetical use of overburden groundwater is 5E-5 (Table 5-1). This value is within the NCP cancer risk management range but exceeds the 1E-5 PBOW cancer risk criterion. Approximately 99 percent of this ILCR is resultant from the contributions of nitroaromatics, which are apparently resultant from former TNT manufacturing activities. However, as discussed in Section 5.3.2 for the groundskeeper, the overburden groundwater unit underlying the LBA provides insufficient groundwater yield. If the contributions of overburden groundwater use are

excluded, the ILCR for the indoor worker is 8E-7. This value is less than the NCP risk management range and the PBOW cancer risk criterion.

The total HI for the indoor worker is 0.7, assuming hypothetical use of overburden groundwater (Table 5-1). This is less than the target HI criterion of 1, indicating that noncancer health effects would be unlikely to occur under this scenario. As noted in Section 5.3.2, the overburden groundwater underlying the LBA does not provide sufficient yield. Therefore, the total HI for this receptor assuming overburden groundwater use is exaggerated. If the contributions of overburden groundwater are excluded, the total HI for the indoor worker is 0.002 (Table 5-2).

5.3.4 Construction Worker

Total Soil. The ILCR for the construction worker (Table 5-1) exposed to total soil both through direct contact and via inhalation of suspended particulates is estimated as 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 criterion of 1E-5.

The total HI for the construction worker (Table 5-1) is 0.9. This is less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

Maintenance Pit Material. As discussed in Section 3.1, the material sampled from the bottom of the concrete-lined maintenance pit is composed of gravel and associated fines that have been stained with petroleum-containing materials. Thus, it is not soil and could not be used as topsoil or growth medium. It is limited to a 3.8-foot by 60-foot pit that is 5 feet deep. Even though exposure to this material is regarded as negligible to minimal, it was evaluated for comparison purposes under a construction worker scenario, essentially assuming that the material is soil and that the construction worker would be exposed solely to this material during a 6-month, on-site work duration.

The ILCR for the construction worker (Table 5-1) exposed to this maintenance pit material both through direct contact and via inhalation of suspended particulates is estimated as 2E-7. This is less than the 1E-6 to 1E-4 NCP risk management range and the PBOW cancer risk criterion of 1E-5. No noncancer COPCs were identified for the maintenance pit material. Therefore, noncancer hazards associated with exposure to this material are regarded as negligible.

5.3.5 Resident

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

Bedrock Groundwater Use. The total ILCR for the resident, assuming bedrock groundwater exposure, is 8E-3 (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk criterion. Over 99 percent of this ILCR is associated with arsenic (2.9E-3), benzene (4.8E-3), and naphthalene (4.2E-4) in groundwater. As discussed in Section 5.3.2, the presence of elevated arsenic in LBA bedrock groundwater does not appear to be related to former site activities. As described in Section 5.3.2, benzene is a petroleum constituent that is known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and during well installation, petroleum was observed seeping at depth from four of the six LBA monitoring wells, petroleum was reported seeping at depth in four of the six LBA bedrock wells, and a petroleum sheen was observed on a fifth well (Shaw, 2010b). Naphthalene is also a naturally occurring constituent in petroleum. Evidence that the naphthalene found in LBA bedrock groundwater is not related to PBOW activities includes the following observations: 1) Naphthalene and other petroleum-related compounds were not detected in the overlying overburden groundwater, and 2) naphthalene was not identified as a soil COPC, 3) naphthalene was detected in only 2 of 34 soil samples in which it was analyzed. Further, as discussed in Section 5.3.2, bedrock groundwater underlying the LBA could not be sampled in three of the six LBA bedrock wells and could not be sampled during the dry season in a fourth. All bedrock groundwater samples had to be sampled via bailer because sufficient groundwater volume was not available to use the low-flow sampling method. Therefore, if the bedrock groundwater use is appropriately excluded, the ILCR for the resident is 1E-5. This value is within the NCP risk management range and meets the PBOW 1E-5 cancer risk criterion.

The total HI for the child resident, assuming bedrock groundwater exposure, is 167 (Table 5-1). This exceeds the target HI value of 1, indicating that noncancer health effects cannot be regarded as unlikely to occur. Note that virtually all of this HI is associated with exposure to bedrock groundwater, and nearly 90 percent of the groundwater HI value is associated with benzene (HQ=90), arsenic (HQ=28), naphthalene (HQ=18) and xylenes (HQ=13). The non-site-

relatedness of arsenic, benzene, and naphthalene are discussed in the preceding paragraph. Similar to benzene and naphthalene, xylenes are a component of naturally occurring petroleum. Xylenes appear to be unrelated to former PBOW activities based on the observations that xylenes were not detected in the overlying overburden unit and were not detected in LBA soil.

As discussed above, bedrock groundwater underlying the LBA does not provide adequate yield for potable use. Therefore, if the contribution of bedrock groundwater is excluded, the resultant HI for the child resident is 0.7. This meets the target HI, indicating that adverse noncancer health effects are unlikely to result from exposure by the child resident receptor to LBA total soil, sediment, and surface water (Table 5-2).

The total HI for the adult resident, assuming bedrock groundwater exposure, is 72 (Table 5-1). This exceeds the target HI value of 1, indicating that noncancer health effects cannot be regarded as unlikely to occur. Note that virtually all of this HI is associated with exposure to bedrock groundwater, and approximately 90 percent of the groundwater HI value is associated with benzene (HQ=39), arsenic (HQ=12), naphthalene (HQ=8), and xylenes (HQ=6). The non-site-relatedness of these four compounds is discussed for the child resident in the preceding paragraphs. Also, as discussed above, bedrock groundwater underlying the LBA provides insufficient yield to consider as a source of residential groundwater. Therefore, the contribution of bedrock groundwater may be appropriately excluded; the resultant HI for the adult resident is 0.1. This meets the target HI, indicating that adverse noncancer health effects are unlikely to result from exposure by the adult resident receptor to LBA total soil, sediment, and surface water (Table 5-2).

Overburden Groundwater Use. The total ILCR for the future resident, assuming overburden groundwater exposure, is 2E-4 (2.43E-5, prior to rounding) (Table 5-1). This value exceeds both the NCP risk management range and the PBOW target cancer risk criterion. Approximately 95 percent of this ILCR value (2.30E-4) is resultant from hypothetical exposure to overburden groundwater (Table 5-1), and most of this groundwater ILCR is associated with apparent site-related nitroaromatics. However, as discussed in Section 5.3.2, the overburden yields inadequate groundwater volume. If the groundwater pathway is appropriately excluded, then the ILCR for the resident is 1E-5. This value is within the NCP risk management range and meets the PBOW 1E-5 cancer risk criterion.

The total HI for the child resident, assuming overburden groundwater exposure, is 6 (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. However, as discussed in Section 5.3.2, the overburden

yields insufficient groundwater volume for potable use. If the groundwater pathway is appropriately excluded, then the total HI for the child resident exposed to LBA soil, sediment, and surface water is 0.7. This value is meets the target HI criterion of 1.

The total HI for the adult resident, assuming overburden groundwater exposure, is 2 (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. However, as discussed in Section 5.3.2, the overburden yields inadequate groundwater volume. If the groundwater pathway is appropriately excluded, then the total HI for the adult resident exposed to LBA soil, sediment, and surface water is 0.1, which meets the target HI criterion of 1.

5.3.6 Adult Hunter

The ILCR for the adult hunter (Table 5-1) exposed to surface soil both through direct contact and via the ingestion of venison is estimated as 2E-7. This is less than the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5.

The total HI for the adult hunter is (Table 5-1) is 0.0003. 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 is estimated as 3E-12. This is less than the 1E-6 to 1E-4 NCP risk management range and is less than the PBOW cancer risk criterion of 1E-5.

The total HI for the hunter's child is (Table 5-1) is 3E-7. This is far less than the target HI value of 1, indicating that noncancer health effects are unlikely to occur.

6.0 Uncertainty Analysis

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

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

6.1 Types of Uncertainty

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

6.2 Sources of Uncertainty

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

6.2.1 Sample Selection

Soil samples were collected from within the former LBA, near the Locomotive Shop and within the Rail Car Washing Area pit. The well locations were selected adjacent to the Locomotive Shop and within the Rail Car Washing Area pit as well as downgradient areas. Surface water and sediment samples were collected along areas of unnamed tributary to Plum Brook that had potentially been impacted by LBA materials. The sample locations appear to be representative of the site, and no information suggests that they have introduced an identifiable bias. Because the material sampled from the maintenance pit consisted predominantly of gravel, the bottom-most materials could not be retained in the sampler. Therefore, this BHHRA may not have adequately characterized the maintenance pit material, as it may be expected the materials in the bottom of the pit would be most highly contaminated. This adds a potentially nonconservative bias to the evaluation of the maintenance pit material.

6.2.2 Laboratory Analysis

State-of-the-practice laboratory methods were used for analysis of the RI samples (Shaw, 2010b). Site investigation samples, also used in this LBA BHHRA, were collected in 1999 (USACE, 2000). No laboratory analytical data quality issues were identified that affected the BHHRA results.

6.2.3 COPC Identification

COPCs were identified using a screening protocol described in the work plan (Shaw, 2009) and employed for more than a decade at PBOW sites. The purpose of screening is to focus the risk assessment effort only on those compounds that may potentially represent an unacceptable risk or hazard and eliminate those that clearly represent no appreciable risk. This protocol was developed through coordination of the PBOW Project Delivery Team, including members from the USACE Nashville and Huntington Districts and their contractors, NASA, and the OEPA. This includes a risk-based screening for all chemicals detected in all media, as well as a background screening for inorganics in soil and bedrock groundwater.

Subsequent to development of the PBOW background screening protocol, the OEPA (2009d) issued background screening guidance that recommends a nonparametric method that is somewhat different from the PBOW protocol. The OEPA (2009a) also issued subsequent risk-

based screening guidance that recommends the screening of contaminants in surface water and sediment to the same respective human health-based criteria as potable tap water and residential soil. PBOW surface water cannot legally or reasonably be used as tap water, and PBOW sediment is very limited in extent as compared to soil. Therefore, the PBOW Project Delivery Team agreed that for noncancer effects, the surface water and sediment screening criteria could be based on the tap water and residential soil criteria multiplied by a factor of 10 (OEPA, 1999). Note that for cancer effects, the surface water and sediment are screened against the respective tap water and residential soil criteria without the multiplier, effectively following the current OEPA guidance for carcinogens.

If the current OEPA background screening protocol were followed, the list of COPCs would differ slightly, possibly having fewer or a greater number inorganic COPCs in soil and bedrock groundwater. However, based on the background data set, knowledge of historic site operations, and a review of the detected inorganics in LBA soil and bedrock groundwater, the use of a different screening method would not have any impact on site remediation decisions with respect to analytes associated with former Department of Defense activities. If the OEPA generic method for screening sediment and surface water were used rather than the PBOW protocol that considers site-specific surface water and sediment conditions, additional COPCs would likely be added, but their contribution to the noncancer HI values for these media would be negligible at most, and their inclusion would have no effect on remediation decisions. In summary, the current PBOW screening protocol is judged to appropriately screen out chemicals that do not represent or contribute appreciably to a significant risk or hazard.

6.2.4 Exposure-Point Concentration Estimates

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

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

Because three of the six bedrock wells were dry and only one sample could be collected from one of the remaining wells (LBA-BEDMW-002), the bedrock groundwater data set was reduced to five samples. As a result, the MDC was typically used as the EPC. This tends to bias the exposure and resultant risk estimates high. However, the concentrations of the risk-driving compounds (especially arsenic and benzene) were detected in multiple samples at relatively high concentrations. Therefore, even if all 12 well samples could have been collected, the EPC would likely result in residential risks for hypothetical use of groundwater that would still exceed the NCP risk management range.

6.2.5 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 minimal NASA activity at the Locomotive Shop and no identified activity at the Rail Car Washing Area, the use of this receptor likely overestimates risks and hazards to a current site worker. The use of the hunter and the hunter's child are reasonable, as this area may be legally hunted by permit under future land use. Unrestricted land use, including residential use, is a reasonable assumption for the future, given the rural residential use of property adjacent to the PBOW facility. However, the assumption that either the overburden or bedrock groundwater will be used as a potable water source in the future is regarded as implausible for reasons described below.

The use of bedrock groundwater is regarded as unlikely because it contains natural petroleum and hydrogen sulfide, which can be commonly observed in area quarries (Shaw, 2005). Hydrogen sulfide was detected in the groundwater during sample collection of LBA monitoring wells RCA-BEDGW-002 (102 ppm) and IT-MNTA-BEDGW-001 (more than 200 ppm), which is one of the highest hydrogen sulfide producers on PBOW. A strong hydrogen sulfide odor was also noted during the development of LBA-BEDGW-002. Hydrocarbon seepage was noted on the rock cores at depth in the boring logs of four of the six LBA bedrock wells. These include LBA-BEDGW-002, RCA-BEDGW-001, RCA-BEDGW-002, and RCA-BEDGW-003. Petroleum sheens have also been noted on the water surface of these wells and that of IT-MNTA-BEDGW-001. Additionally, three of the six LBA wells could not be sampled because they were dry, and LBA-BEDGW-002 could only be sampled during one of the two sampling events. All samples had to be collected via bailer, because there was inadequate yield to use the low-flow sampling method.

The overburden groundwater has also been found to have low yield and to be regionally and seasonally discontinuous throughout much of PBOW (Shaw, 2006). This also appears to be true of the LBA. Of the six overburden groundwater monitoring wells, four had to be sampled using a bailer because of inadequate yield. Only LBA-MW02 and LBA-MW03 could be sampled using low-flow sampling. The flow rate of LBA-MW03 was actually less than the criterion for low-flow sampling of 50 ml/min in May 2009 (42 ml/min) and only marginally above the criterion in November 2009 (58 ml/min). LBA-MW02 could only be sampled at a low rate in November (70 ml/min) and was sampled at a relatively low rate of 120 ml/min in November. Note that potable wells need to provide a flow rate that is orders of magnitude greater.

In summary, use of the bedrock groundwater and overburden groundwater as a potable source at the LBA is regarded as implausible for the following reasons: 1) Both bedrock and overburden groundwater yield water volumes that are clearly inadequate, 2) bedrock groundwater has naturally occurring petroleum and hydrodgen sulfide constituents that make the water unsuitable regardless of yield, 3) this poor bedrock quality is associated with the presence of hydrogen sulfide off-gassing, a concern with respect to nuisance odors and potential health issues, 4) no residential wells in the area have been identified as potable, and 5) municipal water is available in the area.

6.2.6 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 the PBOW sites.

6.2.7 Groundwater-to-Air Household Model

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

of the default values from EPA (1991b), the resultant estimated household air concentrations would be less than those estimated in the BHHRA by a factor of nearly 5. In summary, the selection of the groundwater-to-air model parameter values used by EPA (1991b) may contribute a conservative bias to the resultant risk estimates.

6.2.8 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 RfC, using professional judgment. This modifying factor is intended to address gaps in the database and steepness of the dose-response curve. In practice, the overall UF, derived by multiplying the individual uncertainty factors by the modifying factor, associated with RfD and RfC values may span up to four orders of magnitude.

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

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

6.2.9 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.

6.2.10 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 (Section 2.4.3.2). The WRS test was performed for the following inorganic chemical concentrations in the media specified, with the resultant p level shown:

- Surface soil (chromium, p=1.0)
- Total soil (aluminum, p=0.83; chromium, p=0.16; vanadium, p=0.76)
- Overburden groundwater (iron, p=0.26).

None of the of inorganics failed the WRS at a p value of $p<0.05$. These results indicate that the concentrations of none of the LBA analytes tested differed significantly from those of the PBOW background data sets. The WRS result and box-and-whisker plot for each WRS test are included in Appendix E.

A background evaluation was also performed for arsenic in groundwater. WRS testing was not performed because the arsenic concentration found in the three LBA detections (19.9 to 131 µg/L) appreciably exceed the MDC of the PBOW background data set (7.4 µg/L). Also, the small set (n=5) for LBA groundwater is generally insufficient for WRS testing. Therefore, a

qualitative evaluation was performed assuming that if the arsenic found in the bedrock groundwater truly originated from former LBA operations, then it would be expected that elevated arsenic concentrations would be found in the overlying soil and/or overburden groundwater. This was clearly not the case. The mean and maximum arsenic concentrations found in LBA soil (8.1 and 17.7 mg/kg) were less than those of the PBOW background data set (10.8 and 36.5 mg/kg, respectively). Arsenic was not detected in any of the 12 overburden groundwater samples. Therefore, the arsenic concentrations found in the LBA bedrock groundwater exceed background concentrations, and this elevated arsenic does not appear to be related to former PBOW operations. The source of the elevated arsenic in the LBA bedrock groundwater remains uncertain.

A qualitative background evaluation was also performed for petroleum-related organic compounds observed in LBA bedrock groundwater, most notably BTEX compounds as well as naphthalene. These compounds are petroleum constituents that are known to be naturally occurring in the limestone groundwater at PBOW (Shaw, 2006), and petroleum was reported seeping at depth in four of the six LBA monitoring wells. These include LBA-BEDGW-002 (49.6 to 56.4 feet; petroleum sheen noted on water surface), RCA-BEDGW-001 (53.8 to 70.2 feet), RCA-BEDGW-002 (57.0 to 58.2 feet; petroleum sheen noted on water surface), and RCA-BEDGW-003 (62.4 to 80.2 feet). Also, a petroleum sheen was seen on the surface of well IT-MNTA-BEDGW-001, which is also one of the highest hydrogen sulfide producers at PBOW. Based on the observance of petroleum in the rock cores during drilling and the known regional presence of petroleum associated with the limestone bedrock, the observed elevated concentrations of the BTEX compounds and naphthalene that drive risks/hazards to a hypothetical bedrock groundwater user are not regarded as related to former PBOW activities.

7.0 Summary, Conclusions, and Recommendations

7.1 Summary

Cancer risks and noncancer hazards were evaluated for soil, sediment, surface water, overburden groundwater, and bedrock groundwater at the LBA. Risk and hazards were also evaluated for materials collected from the bottom of the maintenance pit within the Locomotive Shop. A risk-based screening approach was used to identify COPCs and focus the risk assessment on those chemicals detected at the LBA that may contribute appreciably to risk or hazard.

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

- Current groundskeeper (surface soil)
- Future groundskeeper (total soil, groundwater)
- Future indoor worker (surface soil, subsurface [air pathway only], groundwater)
- Current/future construction worker (total soil, sediment)
- Future resident (total soil, groundwater, surface water, sediment)
- Future adult hunter (surface soil, venison)
- Future hunter's child (venison).

Each receptor scenario that includes groundwater was run separately for overburden groundwater and bedrock groundwater. Because groundwater yield was found to be insufficient in the overburden and bedrock units underlying the LBA for potable use, and the LBA bedrock groundwater was observed to be of naturally poor quality due to the presence of petroleum and hydrogen sulfide, the receptors evaluated for potable groundwater use were also evaluated assuming no groundwater use. The resident was evaluated for noncancer hazards separately for the young child (ages 1 through 6 years) and adult life stages.

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

- Current groundskeeper: ILCR = 9E-6 (3E-6 excluding background-related chromium); HI = 0.007
- Future groundskeeper – bedrock groundwater use: ***ILCR = 1E-3; HI = 11***

- Future groundskeeper – overburden groundwater use: **ILCR = 6E-5**; HI = 0.9
- Future groundskeeper – excluding groundwater use: ILCR = 8E-6 (3E-6 excluding background-related chromium); HI = 0.2

- Future indoor worker – bedrock groundwater use: ***ILCR = 1E-3; HI = 11***
- Future indoor worker – overburden groundwater use: **ILCR = 5E-5**; HI = 0.7
- Future indoor worker – excluding groundwater use: ILCR = 8E-7; HI = 0.002

- Future/current construction worker: ILCR = 1E-6; HI = 0.9
- Future/current construction worker – maintenance pit material: ILCR= 2E-7 ; HI= (no noncarcinogenic COPCs identified)

- Future resident – bedrock groundwater use: ***ILCR = 8E-3***; ***child HI = 167***; ***adult HI = 72***
- Future resident – overburden groundwater use: ***ILCR = 2E-4***; ***child HI = 6***; ***adult HI = 2***
- Future resident – excluding groundwater use: ILCR = 1E-5; child HI = 0.7; adult HI = 0.1

- Future hunter: ILCR = 2E-7; HI = 0.0003
- Future hunter's child: ILCR = 3E-12; HI = 0.0000003.

7.2 Conclusions

The ILCR and HI values for the two current exposure scenarios (current groundskeeper and construction worker) each meet the PBOW cancer risk criterion (1E-5) and the noncancer hazard criterion (HI$\leq$1). Note that no construction is currently planned at the LBA. For the current groundskeeper, this assumes a full-time, 25-year employee who works exclusively at the LBA for 250 days per year. The current worker scenario is likely to greatly overestimate the exposure levels of any current on-site worker or other receptor.

Each of the potential future exposure scenarios that does not assume potable use of groundwater also meets the PBOW risk and hazard criteria. This includes even the future resident. However, the cancer risks associated with the following future receptors assumed to use groundwater as a hypothetical source of tap water exceed the NCP risk management range and PBOW cancer risk criterion: future groundskeeper (bedrock), future indoor worker (bedrock), and resident (both overburden and bedrock). Each of these also exceeds the target HI criterion of 1. Additionally, the groundskeeper and indoor worker assumed to use overburden groundwater also exceed the PBOW criterion of 1E-5. The risks associated with overburden and bedrock groundwater differ with respect to one another in that the overburden risks/hazards are mostly associated with apparent PBOW-related nitroaromatics compounds, whereas the bedrock groundwater risks/hazards are associated almost entirely with naturally occurring arsenic and petroleum-related compounds. Use of either the overburden or bedrock groundwater is regarded as implausible because of insufficient yield. Additionally, the naturally poor quality of the bedrock groundwater and associate hydrogen sulfide off-gassing make it unsuitable as a potable source.

The readily available municipal water supply obviates the need for use of groundwater as a potential source of tap water in the general vicinity of PBOW.

7.3 Recommendations

None of the receptors for which groundwater use was excluded had cancer risks or noncancer hazards that exceed the PBOW target criteria. This indicates that the associated environmental media at the LBA are unlikely to pose risk to any receptor. Because groundwater use is not regarded as plausible based on insufficient yield in both the overburden and bedrock units, the risks associated with LBA groundwater are accordingly implausible. The naturally poor quality in the LBA bedrock groundwater further supports the implausibility of bedrock groundwater use. Therefore, no further investigation of LBA environmental media is recommended.

No unacceptable risks were found associated with the maintenance pit material and exposure to this material is assumed to negligible to minimal. However, evidence of petroleum was found in a sample collected from the eastern portion of this pit in 2009. This sampling location is near the maintenance pit sump that was sampled during the limited site inspection conducted in 1999. Both petroleum odor and visual signs of petroleum were also observed in the 1999 maintenance pit sample. Although no risk-based reason has been identified to perform a cleanup of this pit, it is suggested that the backfill gravel be removed so that the material closest to the bottom of the pit may be adequately sampled and characterized for the presence of petroleum and other contaminants. Also, once the material is removed, the integrity of the pit can be assessed to ensure that it does not represent a pathway for leaching, especially if the Locomotive Shop building were to be razed.

It is noted that the Screening Level Ecological Risk Assessment for the LBA (Shaw, 2010c) recommends no further investigation or other actions based only on ecological concerns.

8.0 References

Burmaster, D.E., and R.H. Harris, 1993, "The Magnitude of Compounding Conservatisms in Superfund Risk Assessment," ***Risk Analysis, 13(2): 131-134.***

California Environmental Protection Agency, 2005, ***Toxicity Criteria Database***, Office of Environmental Health Hazard Assessment, Sacramento, California, http://www.oehha.co.gov/risk/ChemicalDB/index.asp.

City-Data.com, 2004, on-line data search for Sandusky, Ohio, performed November 11, 2004, http://www.city-data.com/city/Sandusky-South-Ohio.html.

Cogliano, V.J., 1997, "Plausible Upper Bounds: Are Their Sums Plausible?" ***Risk Analysis***, 17(1): 77-84.

Dames and Moore, Inc. (D&M), 1997, ***TNT Areas Site Investigation Final Report for Plum Brook Ordnance Works, Plum Brook Station/NASA, Sandusky, Ohio***, prepared for U.S. Army Corps of Engineers, Nashville District/Huntington District, April.

Environmental Council of States-U.S. Department of Defense, 2007, ***Identification and Selection of Toxicity Values/Criteria for CERCLA and Hazardous Waste Site Risk Assessments in the Absence of IRIS Values,*** ECO-DOD Sustainability Work Group, Emerging Contaminants Task Group, Risk Assessment Provisional Values Subgroup Issue Paper, 23 April.

IT Corporation (IT), 2001a, ***TNT Areas A and C Remedial Investigation, Volume II – Human Health Risk Assessment, Final, Former Plum Book Ordnance Works, Sandusky, Ohio,*** November.

IT Corporation (IT), 2001b, ***TNT Areas A and C Remedial Investigation, Volume I – Report of Findings, Final, Former Plum Book Ordnance Works, Sandusky, Ohio,*** November.

IT Corporation (IT), 2000, ***Draft Baseline Human Health Risk Assessment Work Plan for the Former TNT Manufacturing Area, West Virginia Ordnance Works, Mason Count, West Virginia***, January.

IT Corporation (IT), 1999, ***Final - Summary Report, Site-Wide Groundwater Monitoring (1997-1998), Former Plum Brook Ordnance Works, Sandusky, Ohio,*** June.

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

IT Corporation (IT), 1997, ***Site-Wide Groundwater Investigation, Former Plum Brook Ordnance Works, Sandusky, Ohio***, September.

Mautz, W.W., H. Silver, and J.B. Holter, 1976, "Digestibility and Related Nutritional Data for Seven Northern Deer Browse Species," ***Journal of Wildlife Management***, 40(4): 630-638.

National Council on Radiation Protection and Measurements, 1984, ***Radiological Assessment: Predicting the Transport, Bioaccumulation, and Uptake by Man of Radionuclides Released to the Environment***, NCRP Report No. 76.

National Oceanic and Atmospheric Administration, 1990, ***Comparative Climatic Data for the United States through 1989***, National Climatic Data Center, Asheville, North Carolina.

Nutrient Database, 1997, Data compiled from U.S. Department of Agriculture releases, and published nutrient information from 71 chain references, on line, http://www.nutribase.com.

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.

Ohio Environmental Protection Agency (OEPA), 2009c, Email Correspondence from B. Buthker, OEPA Risk Assessor, to L. Long, USACE Risk Assessor, March 18.

Ohio Environmental Protection Agency (OEPA), 2009d***, Use of Use of Background for Remedial Response Sites***, Technical Decision Compendium, Division of Emergency and Remedial Response, August.

Ohio Environmental Protection Agency (OEPA), 1999, email correspondence between Lawrence Sirinek (OEPA Risk Assessor) and Paul Goetchius (Shaw Risk Assessor), March 5.

Sample, B.E. and Suter II, G.W., 1994, ***Estimating Exposure of Terrestrial Wildlife Contaminants***, Environmental Sciences Division, Oak Ridge National Laboratory, ES/ER/TM-125.

Sample, B.E., D.M. Opresko, and G.W. Suter II, 1996, ***Toxicological Benchmarks for Wildlife: 1996 Revision***, prepared for the U.S. Department of Energy by Health Sciences Research Division, Oak Ridge National Laboratory.

Science Applications International Corporation, 1991, ***Plum Brook Station Preliminary Assessment,*** June.

Sharp, G., 1995, Personal Communication: Telephone conversation between P. Goetchius, IT Corp. and Gary Sharp, Wildlife Biologist, West Virginia Division of Natural Resources, Point Pleasant, West Virginia, 17 February.

Shaw Environmental, Inc. (Shaw), 2010a, ***Baseline Human Health Risk Assessment Work Plan, Garage Maintenance Area (Locomotive Building Area), Plum Brook Ordnance Works, Sandusky, Ohio***, May.

Shaw Environmental, Inc. (Shaw), 2010b, ***Site Characterization Report, Garage Maintenance Area (Locomotive Building Area), Final, Plum Brook Ordnance Works, Sandusky, Ohio***, September.

Shaw Environmental, Inc. (Shaw), 2010c, ***Screening Level Ecological Risk Assessment, Garage Maintenance Area (Locomotive Building Area), Draft, Plum Brook Ordnance Works, Sandusky, Ohio***, September.

Shaw Environmental, Inc. (Shaw), 2009, ***Site-Specific Sampling and Analysis Plan, Remedial Investigation, Locomotive Building Area***, Plum Brook Ordnance Works, Sandusky, Ohio, January.

Shaw Environmental, Inc. (Shaw), 2008, ***Feasibility Study for Groundwater, TNT and Red Water Pond Areas, Final,*** *Former Plum Brook Ordnance Works, Sandusky, Ohio*, December.

Shaw Environmental, Inc. (Shaw), 2006, ***Baseline Human Health Risk Assessment of Groundwater, Former Plum Brook Ordnance Works, Sandusky, Ohio***, September.

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

The Weather Channel, 2004, on-line data search for weather averages and records for Sandusky, Ohio, performed November 11, 2004, http://www.weather.com.

U.S. Army Corps of Engineers (USACE), 2000, ***Limited Site Inspection, Final Report, Garage Maintenance Area (Locomotive Building Area), Plum Brook Ordnance Works, Sandusky, Ohio***, July.

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

U.S. Army Corps of Engineers (USACE), 1995, ***Site Management Plan, Plum Brook Ordnance Works, Sandusky, Ohio, Part B, Areas of Concern***, September.

U.S. Department of Energy (DOE), 1989, ***A Manual for Implementing Residual Radioactive Material Guidelines***, Argonne National Laboratory, Argonne, Illinois, ANL/ES-160, DOE/CH/8901.

U.S. Department of Energy (DOE), 1983, ***Pathway Analysis and Radiation Dose Estimates for Radioactive Residues at Formerly Utilized MED/AEC Sites***, U.S. Dept. of Energy, Oak Ridge Operations Office, Oak Ridge, Tennessee, DOE ORO-832.

U.S. Environmental Protection Agency (EPA), 2010a, ***Regional Screening Level Table***, May, on line at www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/Generic_Tables/index.htm.

U.S. Environmental Protection Agency (EPA), 2010b, ***Regional Screening Level Table, User's Guide (May 2010)***, on line at www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/usersguide.htm.

U.S. Environmental Protection Agency (EPA), 2010c, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, Ohio, on line.

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

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

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

U.S. Environmental Protection Agency (EPA), 2009d, ***2009 Edition of the Drinking Water Standards and Health Advisories***, Office of Water, EPA 822-R-09-011, October.

U.S. Environmental Protection Agency (EPA), 2006, ***2006 Edition of the Drinking Water Standards and Health Advisories***, Office of Water, August, EPA 822-R-06-013.

U.S. Environmental Protection Agency (EPA), 2005, ***Guidelines for Carcinogen Risk Assessment***, Risk Assessment Forum, Washington, D.C., March, EPA/630/P-03/001B.

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), 2004b, ***Region 9 Preliminary Remediation Goals (PRG) Table***, San Francisco, California, October.

U.S. Environmental Protection Agency (EPA), 2004c, ***User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows 32-bit Version,*** Lead and IEUBK Technical Review Workgroup, Washington, D.C., May http://www.epa.gov/superfund/programs/lead/products.htm.

U.S. Environmental Protection Agency (EPA), 2004d, ***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>.

U.S. Environmental Protection Agency (EPA), 2003, ***Human Health Toxicity Values in Superfund Risk Assessments***, Office of Solid Waste and Emergency Response, Washington, D.C., OSWER Directive 9285.7-53, December.

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), 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), 1997b, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

U.S. Environmental Protection Agency (EPA), 1996, ***Soil Screening Guidance: Technical Background Document***, Office of Solid Waste and Emergency Response, EPA/540/R-95/128, NTIS No. PB96-963502.

U.S. Environmental Protection Agency (EPA), 1994, ***Exposure Assessment Guidance for RCRA Hazardous Waste Combustion Facilities,*** Office of Solid Waste and Emergency Response, Draft, April, EPA530-R-94-021, including Errata, "Guidance for Performing Screening Level Risk Analyses at Combustion Facilities Burning Hazardous Wastes," October, and "Modification of Screening Guidance Fate and Transport Equations, November.

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), 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), 1991b, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals***, Interim, Office of Emergency and Remedial Response, December, EPA/540/R-92/003.

U.S. Environmental Protection Agency (EPA), 1990, "National Oil and Hazardous Substances Pollution Contingency Plan," ***Federal Register*** 55(46): 8666-8865.

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), 1989b, ***General Quantitative Risk Assessment Guidelines for Noncancer Health Effects***, Prepared by the Office of Health and Environmental Assessment, Cincinnati, OH for the Risk Assessment Forum, ECAO-CIN-538.

U.S. Environmental Protection Agency (EPA), 1986, "Guidelines for Carcinogen Risk Assessment," ***Federal Register***, 51(185): 33992-34003.

U.S. Geological Survey, 1992, ***Hydraulic Properties of Three Types of Glacial Deposits in Ohio***, Water-Resources Investigation Report 92-4135.

TABLES

Table 2-1

Summary of Samples Used for Baseline Human Health Risk Assessment
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample No	Sample Purpose	Date	Depth (ft bgs)	Analyses
SOIL					
Surface Soil					
LBA-SB01	LBA0001	REG	20-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB02	LBA0004	REG	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB03	LBA0007	REG	20-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB03	LBA0008	FD	20-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB05	LBA0015	REG	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB06	LBA0018	REG	20-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB10	LBA0026	REG	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB10	LBA0027	FD	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB11	LBA0031	REG	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB12	LBA0034	REG	19-Jan-09	0 - 1	Explosives, Metals, PCB, SVOC, VOC
LBA-SB04	LBA0012	REG	19-Jan-09	0.9 - 1.9	Explosives, Metals, PCB, SVOC, VOC
PBOW99-SB001	PBOW99-SB001-001	REG	9-Jun-99	0 - 0.5	Metals, PCB, SVOC
PBOW99-DP002	PBOW99-DP002-0002	REG	11-Jun-99	0 - 2	Metals, PCB, SVOC
Subsurface Soil					
LBA-SB03	LBA0010	REG	20-Jan-09	2.5 - 3.2	Explosives, Metals, PCB, SVOC, VOC
LBA-SB01	LBA0002	REG	20-Jan-09	3 - 5	Explosives, Metals, PCB, SVOC, VOC
LBA-SB02	LBA0005	REG	19-Jan-09	3 - 5	Explosives, Metals, PCB, SVOC, VOC
LBA-SB05	LBA0016	REG	20-Jan-09	3 - 5	Explosives, Metals, PCB, SVOC, VOC
LBA-SB06	LBA0019	REG	20-Jan-09	3 - 5	Explosives, Metals, PCB, SVOC, VOC
LBA-SB10	LBA0029	REG	19-Jan-09	3 - 4.4	Explosives, Metals, PCB, SVOC, VOC
LBA-SB12	LBA0035	REG	19-Jan-09	3 - 3.9	Explosives, Metals, PCB, SVOC, VOC
LBA-SB04	LBA0013	REG	19-Jan-09	3.9 - 5.9	Explosives, Metals, PCB, SVOC, VOC
LBA-SB11	LBA0032	REG	19-Jan-09	3.9 - 5.9	Explosives, Metals, PCB, SVOC, VOC
LBA-SB01	LBA0003	REG	20-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB02	LBA0006	REG	19-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB03	LBA0011	REG	20-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB04	LBA0014	REG	19-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB05	LBA0017	REG	20-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB06	LBA0020	REG	20-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB06	LBA0021	FD	20-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB10	LBA0030	REG	19-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB11	LBA0033	REG	19-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
LBA-SB12	LBA0036	REG	19-Jan-09	8 - 10	Explosives, Metals, SVOC, VOC
PBOW99-DP001	PBOW99-DP001-0004	REG	11-Jun-99	2 - 4	Metals, PCB, SVOC
PBOW99-DP001	PBOW99-DP001-0008AA	REG	11-Jun-99	4 - 6	Metals

Table 2-1

Summary of Samples Used for Baseline Human Health Risk Assessment
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample No	Sample Purpose	Date	Depth (ft bgs)	Analyses
PBOW99-DP002	PBOW99-DP002-0004	REG	11-Jun-99	2 - 4	Metals, PCB, SVOC
PBOW99-DP002	PBOW99-DP002-0006	REG	11-Jun-99	4 - 6	Metals
PBOW99-DP003	PBOW99-DP003-0004	REG	11-Jun-99	2 - 4	Metals, PCB, SVOC
PBOW99-DP003	PBOW99-DP003-0006	REG	11-Jun-99	4 - 6	Metals
PBOW99-DP004	PBOW99-DP004-0006	REG	11-Jun-99	4 - 6	Metals
PBOW99-SB001	PBOW99-SB001-0005	REG	9-Jun-99	3 - 5	Metals, PCB, SVOC
PBOW99-SB001	PBOW99-SB001-003	REG	9-Jun-99	1 - 3	Metals, PCB, SVOC
Maintenance Pit Material					
LBA-SB07	LBA0023	REG	20-Jan-09	3 - 4.1	Explosives, Metals, SVOC
LBA-SB08	LBA0024	REG	20-Jan-09	3 - 4.9	Explosives, Metals, SVOC
LBA-SB09	LBA0025	REG	20-Jan-09	3 - 4.2	Explosives, Metals, SVOC
SEDIMENT					
LBA-SD01	LBA1000	REG	20-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD02	LBA1001	REG	21-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD03	LBA1002	REG	21-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD04	LBA1003	REG	21-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD05	LBA1004	REG	22-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD06	LBA1005	REG	22-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
LBA-SD06	LBA1006	FD	22-May-09	0 - 0.5	Explosives, Metals, PCB, SVOC, VOC
SURFACE WATER					
LBA-SW01	LBA2000	REG	20-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW02	LBA2001	REG	21-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW03	LBA2002	REG	21-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW03	LBA2003	FD	21-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW04	LBA2005	REG	21-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW05	LBA2006	REG	22-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW06	LBA2007	REG	22-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
LBA-SW07	LBA2008	REG	22-May-09	NA	Explosives, Metals, PCB, SVOC, VOC
GROUNDWATER					
Overburden-Monitoring Well					
LBA-MW01	LB3067	REG	12-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW02	LB3068	REG	19-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW02	LB3069	FD	19-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW03	LB3071	REG	10-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW01	LB3075	REG	4-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW02	LB3076	REG	5-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC

Table 2-1

Summary of Samples Used for Baseline Human Health Risk Assessment
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Location	Sample No	Sample Purpose	Date	Depth (ft bgs)	Analyses
LBA-MW02	LB3077	FD	5-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-MW03	LB3080	REG	3-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-MW02	RC3075	REG	11-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-MW03	RC3076	REG	11-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-MW02	RC3081	REG	3-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-MW03	RC3082	REG	4-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
Bedrock					
IT-MNTA-BEDGW-001	BEDGW-001-1109	REG	6-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
IT-MNTA-BEDGW-001	LB3074	REG	19-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-BEDGW-002	LB3073	REG	12-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-BEDGW-002	RC3078	REG	11-May-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
RCA-BEDGW-002	RC3084	REG	5-Nov-09	NA	Explosives, Gen Chem, Metals (f & uf), SVOC, VOC
Overburden Piezometer [a]					
LBA-PZ01	LBA3000	REG	31-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ05	LBA3001	FD	28-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ02	LBA3003	REG	29-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ03	LBA3004	REG	29-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ04	LBA3005	REG	30-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ05	LBA3006	REG	28-Jan-09	NA	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC

[a] These samples are summarized and screened (Appendix A), but no exposure calculations are presented.

f & uf - Metals analysis performed on filtered and unfiltered samples, respectively.
FD - Field Duplicate sample; REG - regular sample.
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.
ft bgs - Feet below ground surface.
NA - Not applicable.

Table 2-2

Background Screening Concentrations of Inorganics in Soil
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

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

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

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

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

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

Table 2-3

Background Screening Concentrations for Inorganics in Groundwater Locomotive Building Area Former Plum Brook Ordnance Works, Sandusky, Ohio

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

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

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

µg/L - Micrograms per liter.

BTEX - Benzene, toluene, ethylbenzene, and xylenes.

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

Table 2-4

Statistical Summary and COPC Selection of Chemicals Detected in Surface Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Detected Concentrations Maximum	VQ	Reporting Limits Minimum	Reporting Limits Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]	95% UCL [e] mg/kg	EPC [f] mg/kg
Inorganics														
Aluminum	11 / 11	100	4.40E+03		8.26E+03		1.81E+00	2.35E+01	6.58E+03	1.55E+04	7700	N (b)		---
Antimony	6 / 11	55	2.50E-01	J	8.57E-01		4.51E-01	1.20E+00	4.03E-01	9.30E+00	3.1	N (b)		---
Arsenic	11 / 11	100	4.50E+00	J	1.13E+01	J	9.02E-01	1.20E+00	7.45E+00	3.65E+01	0.39	N (b)		---
Barium	11 / 11	100	1.63E+01		2.18E+02		2.00E-01	2.35E+01	7.11E+01	8.26E+02	1500	N (b)		---
Beryllium	9 / 11	82	5.22E+00	J	9.26E+00	J	1.81E-01	5.90E-01	5.29E+00	1.00E+00	16	N (a)		---
Cadmium	7 / 11	64	2.53E-01	J	6.99E-01		2.10E-01	5.02E-01	3.82E-01		7	N (a)		---
Calcium	11 / 11	100	1.18E+03		1.38E+04		4.51E+00	5.88E+02	4.90E+03	5.23E+04	Nutrient	N (c)		---
Chromium	11 / 11	100	1.04E+01		5.10E+01		4.51E-01	5.90E-01	1.53E+01	2.90E+01	0.29	Y	2.24E+01	2.24E+01
Cobalt	9 / 11	82	3.52E+00		2.13E+01	J	3.61E-01	5.90E+00	8.67E+00	1.16E+02	2.3	N (b)		---
Copper	11 / 11	100	6.54E+00		1.87E+01		4.51E-01	2.90E+00	1.37E+01	5.62E+01	310	N (b)		---
Iron	11 / 11	100	1.30E+04		3.14E+04	J	3.64E+00	1.18E+01	1.84E+04	2.34E+05	5500	N (b)		---
Lead	11 / 11	100	5.29E+00		2.22E+02		3.20E-01	5.02E-01	3.68E+01	4.86E+01	400	N (a)		---
Magnesium	11 / 11	100	7.96E+02	J	2.87E+03		1.81E+00	5.88E+02	1.57E+03	1.04E+04	Nutrient	N (c)		---
Manganese	11 / 11	100	1.15E+02		1.56E+03	J	1.81E-01	1.80E+00	5.22E+02	3.51E+03	180	N (b)		---
Mercury	9 / 11	82	1.88E-02	J	4.56E-02		2.13E-02	1.20E-01	3.59E-02	8.50E-02	2.3	N (b)		---
Nickel	11 / 11	100	9.01E+00	J	2.26E+01	J	2.71E-01	4.70E+00	1.64E+01	5.51E+01	150	N (b)		---
Potassium	11 / 11	100	1.88E+02		1.07E+03		2.26E+01	5.88E+02	4.74E+02	3.39E+03	Nutrient	N (c)		---
Sodium	9 / 11	82	1.88E+01		5.04E+01		9.02E+00	5.88E+02	7.89E+01		Nutrient	N (c)		---
Thallium	3 / 11	27	2.94E-01	J	5.58E-01		4.45E-01	1.20E+00	3.40E-01	1.30E+00	0.51 g	N (b)		---
Vanadium	11 / 11	100	2.03E+01		2.75E+01		1.81E-01	5.90E+00	2.36E+01	4.09E+01	39	N (b)		---
Zinc	11 / 11	100	2.60E+01		2.73E+02		2.10E+00	2.51E+00	7.06E+01	3.22E+02	2300	N (b)		---
Polychlorinated biphenyls (PCB)														
Aroclor 1221	1 / 11	9	1.90E-01		1.90E-01		3.50E-02	4.29E-02	3.54E-02		0.14	Y	1.03E-01	1.03E-01
Aroclor 1260	3 / 11	27	6.29E-02	J	1.90E-01		3.50E-02	4.29E-02	4.61E-02		0.22	N (a)		---
Nitroaromatics														
Amino-2,6-dinitrotoluene, 4-	2 / 9	22	3.24E-01		5.27E-01		2.88E-01	3.00E-01	2.10E-01		15	N (a)		---
Amino-4,6-dinitrotoluene, 2-	1 / 9	11	2.94E-01		2.94E-01		2.88E-01	3.00E-01	1.64E-01		15	N (a)		---
Semivolatile Organic Compounds														
Acenaphthylene	2 / 11	18	7.06E-02	J	2.24E-01	J	3.50E-01	2.06E+00	2.92E-01		340 h	N (a)		---
Anthracene	2 / 11	18	8.11E-02	J	3.81E-01	J	3.50E-01	2.06E+00	3.07E-01		1700	N (a)		---
Benzo(a)anthracene	3 / 11	27	4.26E-02	J	6.52E-01	J	3.50E-01	2.06E+00	3.25E-01		0.15	Y	2.92E-01	2.92E-01
Benzo(a)pyrene	2 / 11	18	1.21E-01	JJ	3.95E-01	J	3.50E-01	2.06E+00	3.12E-01		0.015	Y	3.41E-01	3.41E-01
Benzo(b)fluoranthene	2 / 11	18	1.92E-01	JJ	8.78E-01	J	3.50E-01	2.06E+00	3.62E-01		0.15	Y	7.42E-01	7.42E-01
Benzo(ghi)perylene	1 / 11	9	1.06E-01	J	1.06E-01	J	3.50E-01	2.06E+00	3.68E-01		170 i	N (a)		---
Chrysene	3 / 11	27	3.95E-02	J	7.84E-01	J	3.50E-01	2.06E+00	3.37E-01		15	N (a)		---
Dibenzofuran	2 / 11	18	4.61E-01	J	1.40E+00	J	3.50E-01	2.06E+00	4.09E-01		7.8	N (a)		---
Di-n-butyl phthalate	1 / 11	9	5.90E-02	J	5.90E-02	J	3.50E-01	2.06E+00	3.64E-01		610	N (a)		---
Dinitrotoluene, 2,4-	3 / 11	27	2.68E-01	J	4.00E+00		3.50E-01	2.06E+00	7.26E-01		0.71	Y	1.44E+00	1.44E+00
Dinitrotoluene, 2,6-	1 / 11	9	2.04E-01	J	2.04E-01	J	3.50E-01	2.06E+00	3.77E-01		0.71	N (a)		---
Fluoranthene	5 / 11	45	4.01E-02	J	1.26E+00	J	3.50E-01	2.06E+00	3.65E-01		230	N (a)		---
Indeno(1,2,3-cd)pyrene	1 / 11	9	1.30E-01	J	1.30E-01	J	3.50E-01	2.06E+00	3.71E-01		0.15	N (a)		---
Methylnaphthalene, 2-	2 / 11	18	1.66E+00	J	5.60E+00		3.50E-01	2.06E+00	9.00E-01		31	N (a)		---
Naphthalene	2 / 11	18	1.02E+00	J	3.40E+00		3.50E-01	2.06E+00	6.41E-01		3.6	N (a)		---
Phenanthrene	3 / 11	27	1.17E-01	JJ	1.50E+00		3.50E-01	2.06E+00	4.45E-01		170 i	N (a)		---

Table 2-4

Statistical Summary and COPC Selection of Chemicals Detected in Surface Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]	95% UCL [e] mg/kg	EPC [f] mg/kg
Pyrene	3 / 11	27	6.27E-02	J	1.13E+00	J	3.50E-01	2.06E+00	3.77E-01		170	N (a)		---
Volatile Organic Compounds														
Acetone	9 / 9	100	3.03E-02	J	2.65E-01	J	4.62E-02	5.92E-02	1.27E-01		6100	N (a)		---
Butanone, 2-	7 / 7	100	8.27E-03	J	1.36E-02	J	5.28E-02	5.92E-02	1.08E-02		2800	N (a)		---
Carbon disulfide	1 / 9	11	4.95E-03	JJ	4.95E-03	JJ	9.23E-03	1.18E-02	5.35E-03		82	N (a)		---
Toluene	1 / 9	11	1.04E-03	J	1.04E-03	J	9.23E-03	1.18E-02	4.92E-03		500	N (a)		---
Trichloroethane, 1,1,1-	3 / 9	33	1.39E-03	J	1.56E-02	J	9.23E-03	1.18E-02	6.08E-03		870	N (a)		---
Trichloroethene	1 / 9	11	2.63E-03	J	2.63E-03	J	9.23E-03	1.18E-02	5.06E-03		2.8	N (a)		---

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

[a] IT Corporation (IT), 1998, *Site Investigation of Acid Areas* , Plum Brook Ordnance Works, Sandusky, Ohio, August.
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) 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 Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC
[f] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.
[g] RBSC based on thallium from EPA, 2010, Integrated Risk Information System.
[h] RBSC based on anthracene.
[i] RBSC based on pyrene.

Table 2-5

Statistical Summary and COPC Selection of Chemicals Detected in Subsurface Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg		COPC? [c,d]
Inorganics													
Aluminum	26 / 26	100	5.39E+03		1.72E+04		1.81E+00	3.00E+01	9.24E+03	1.55E+04	7700		Y
Antimony	17 / 26	65	2.70E-01	J	1.30E+00		4.54E-01	1.50E+00	5.23E-01	9.30E+00	3.1		N (b)
Arsenic	25 / 26	96	4.40E+00		1.77E+01		9.07E-01	1.50E+00	8.32E+00	3.65E+01	0.39		N (b)
Barium	26 / 26	100	2.07E+01		4.25E+02		1.85E-01	3.00E+01	8.59E+01	8.26E+02	1500		N (b)
Beryllium	19 / 26	73	6.60E-01		9.03E+00	J	1.81E-01	7.50E-01	4.77E+00	1.00E+00	16		N (a)
Cadmium	15 / 26	58	2.54E-01	J	6.71E-01		2.30E-01	5.26E-01	2.98E-01		7		N (a)
Calcium	26 / 26	100	1.32E+03	J	7.40E+04		4.54E+00	7.50E+02	2.13E+04	5.23E+04	Nutrient		N (c)
Chromium	26 / 26	100	1.10E+01		2.86E+01		4.54E-01	7.50E-01	1.57E+01	2.90E+01	0.29		N (b)
Cobalt	24 / 26	92	4.35E+00		5.38E+01	J	3.63E-01	7.50E+00	1.35E+01	1.16E+02	2.3		N (b)
Copper	26 / 26	100	7.80E+00		3.16E+01		4.54E-01	3.70E+00	2.11E+01	5.62E+01	310		N (b)
Iron	26 / 26	100	1.19E+04		4.64E+04		3.63E+00	1.50E+01	2.33E+04	2.34E+05	5500		N (b)
Lead	26 / 26	100	7.39E+00		2.30E+01		3.40E-01	5.26E-01	1.37E+01	4.86E+01	400		N (b)
Magnesium	26 / 26	100	1.05E+03	J	1.94E+04	J	1.81E+00	7.50E+02	8.33E+03	1.04E+04	Nutrient		N (c)
Manganese	26 / 26	100	1.46E+02		3.00E+03		1.81E-01	2.20E+00	6.70E+02	3.51E+03	180		N (b)
Mercury	15 / 26	58	1.18E-02	J	1.58E-01	J	2.11E-02	1.50E-01	4.37E-02	8.50E-02	2.3		N (a)
Nickel	26 / 26	100	1.07E+01		5.03E+01		2.72E-01	6.00E+00	2.90E+01	5.51E+01	150		N (b)
Potassium	26 / 26	100	2.05E+02		2.26E+03		2.27E+01	7.50E+02	9.59E+02	3.39E+03	Nutrient		N (c)
Selenium	1 / 26	4	5.16E-01	J	5.16E-01	J	5.70E-01	1.05E+00	4.36E-01	1.11E+01	39		N (b)
Sodium	18 / 26	69	2.67E+01		1.30E+02		9.07E+00	7.50E+02	1.48E+02		Nutrient		N (c)
Thallium	14 / 26	54	2.69E-01	J	6.78E-01		4.54E-01	1.50E+00	4.37E-01	1.30E+00	0.51	g	N (b)
Vanadium	26 / 26	100	1.83E+01		4.99E+01		1.81E-01	7.50E+00	2.57E+01	4.09E+01	39		Y
Zinc	26 / 26	100	3.00E+01		1.13E+02	J	2.27E+00	3.00E+00	7.33E+01	3.22E+02	2300		N (b)
Polychlorinated biphenyls (PCB)													
Aroclor 1260	1 / 14	7	6.10E-02	J	6.10E-02	J	3.70E-02	4.90E-02	2.32E-02		0.22		N (a)
Nitroaromatics													
Amino-2,6-dinitrotoluene, 4-	1 / 18	6	5.30E-01		5.30E-01		2.91E-01	3.00E-01	1.69E-01		15		N (a)
Amino-4,6-dinitrotoluene, 2-	2 / 18	11	1.63E-01	J	4.38E-01		2.91E-01	3.00E-01	1.65E-01		15		N (a)
Dinitrotoluene, 2,4-	6 / 18	33	3.21E-01		5.68E+00		2.91E-01	1.46E+00	6.04E-01		0.71		Y
Dinitrotoluene, 2,6-	2 / 18	11	8.42E-01	J	4.12E+00		2.91E-01	1.46E+00	4.07E-01		0.71		Y
Nitrotoluene, 2-	2 / 18	11	2.34E+00		2.27E+01		2.91E-01	2.91E+00	1.52E+00		2.9		Y
Nitrotoluene, 3-	2 / 18	11	3.08E-01		1.52E+00		2.91E-01	3.00E-01	2.33E-01		0.61		Y
Nitrotoluene, 4-	2 / 18	11	7.07E-01		1.18E+01		2.91E-01	1.46E+00	8.26E-01		24		N (a)
Semivolatile Organic Compounds													
Dinitrotoluene, 2,4-	8 / 23	35	2.30E-01	J	9.88E+00		3.70E-01	2.08E+00	9.91E-01		0.71		Y
Dinitrotoluene, 2,6-	5 / 23	22	7.71E-02	J	3.82E+00		3.70E-01	4.90E-01	4.59E-01		0.71		Y
Volatile Organic Compounds													
Acetone	12 / 12	100	2.10E-02	J	5.25E-02	J	1.46E-02	5.54E-02	3.34E-02		6100		N (a)

Table 2-5

Statistical Summary and COPC Selection of Chemicals Detected in Subsurface Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]
Benzene	6 / 18	33	1.21E-03	J	3.81E-03	J	8.89E-03	2.15E-02	4.27E-03		1.1	N (a)
Butanone, 2-	3 / 3	100	5.55E-03	J	7.91E-03	J	4.62E-02	4.74E-02	6.40E-03		2800	N (a)
Carbon disulfide	2 / 18	11	9.88E-04	J	3.07E-03	J	8.89E-03	2.15E-02	5.38E-03		82	N (a)
Dichloroethane, 1,1-	6 / 18	33	2.63E-03	J	3.45E-02	J	8.89E-03	2.15E-02	8.16E-03		3.3	N (a)
Dichloroethene, 1,1-	3 / 18	17	2.54E-03	J	5.63E-03	J	8.89E-03	2.15E-02	5.03E-03		24	N (a)
Dichloroethene, cis-1,2-	2 / 18	11	3.20E-03	J	9.86E-03	J	8.89E-03	2.15E-02	5.55E-03		78	N (a)
Naphthalene	2 / 18	11	1.51E-03	J	3.46E-03	J	8.89E-03	2.15E-02	4.99E-03		3.6	N (a)
Toluene	7 / 18	39	1.86E-03	J	5.99E-03	J	8.89E-03	2.15E-02	4.57E-03		500	N (a)
Trichloroethane, 1,1,1-	7 / 18	39	1.32E-02		1.21E+00	J	8.89E-03	5.49E-01	1.68E-01		870	N (a)
Trichloroethene	4 / 18	22	1.22E-02		1.21E-01		8.89E-03	2.15E-02	1.77E-02		2.8	N (a)
Trimethylbenzene, 1,2,4-	3 / 18	17	9.88E-04	J	1.78E-03	J	8.89E-03	2.15E-02	4.89E-03		6.2	N (a)

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

[a] IT Corporation (IT), 1998, *Site Investigation of Acid Areas* , Plum Brook Ordnance Works, Sandusky, Ohio, August.
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) 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 Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC.
[f] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.
[g] RBSC based on thallium from EPA, 2010, Integrated Risk Information System.

Table 2-6

Statistical Summary and COPC Selection of Chemicals Detected in Total Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg		COPC? [c,d]	95% UCL [e] mg/kg	EPC [f] mg/kg
Inorganics															
Aluminum	37 / 37	100	4.40E+03		1.72E+04		1.81E+00	3.00E+01	8.45E+03	1.55E+04	7700		Y	9.11E+03	9.11E+03
Antimony	23 / 37	62	2.50E-01	J	1.30E+00		4.51E-01	1.50E+00	4.88E-01	9.30E+00	3.1		N (b)		---
Arsenic	36 / 37	97	4.40E+00		1.77E+01		9.02E-01	1.50E+00	8.06E+00	3.65E+01	0.39		N (b)		---
Barium	37 / 37	100	1.63E+01		4.25E+02		1.85E-01	3.00E+01	8.15E+01	8.26E+02	1500		N (b)		---
Beryllium	28 / 37	76	6.60E-01		9.26E+00	J	1.81E-01	7.50E-01	4.93E+00	1.00E+00	16		N (a)		---
Cadmium	22 / 37	59	2.53E-01	J	6.99E-01		2.10E-01	5.26E-01	3.23E-01		7		N (a)		---
Calcium	37 / 37	100	1.18E+03		7.40E+04		4.51E+00	7.50E+02	1.64E+04	5.23E+04	Nutrient		N (c)		---
Chromium	37 / 37	100	1.04E+01		5.10E+01		4.51E-01	7.50E-01	1.56E+01	2.90E+01	0.29		Y	1.77E+01	1.77E+01
Cobalt	33 / 37	89	3.52E+00		5.38E+01	J	3.61E-01	7.50E+00	1.20E+01	1.16E+02	2.3		N (b)		---
Copper	37 / 37	100	6.54E+00		3.16E+01		4.51E-01	3.70E+00	1.89E+01	5.62E+01	310		N (b)		---
Iron	37 / 37	100	1.19E+04		4.64E+04		3.63E+00	1.50E+01	2.18E+04	2.34E+05	5500		N (b)		---
Lead	37 / 37	100	5.29E+00		2.22E+02		3.20E-01	5.26E-01	2.06E+01	4.86E+01	400		N (a)		---
Magnesium	37 / 37	100	7.96E+02	J	1.94E+04	J	1.81E+00	7.50E+02	6.32E+03	1.04E+04	Nutrient		N (c)		---
Manganese	37 / 37	100	1.15E+02		3.00E+03		1.81E-01	2.20E+00	6.26E+02	3.51E+03	180		N (b)		---
Mercury	24 / 37	65	1.18E-02	J	1.58E-01	J	2.11E-02	1.50E-01	4.14E-02	8.50E-02	2.3		N (a)		---
Nickel	37 / 37	100	9.01E+00	J	5.03E+01		2.71E-01	6.00E+00	2.52E+01	5.51E+01	150		N (b)		---
Potassium	37 / 37	100	1.88E+02		2.26E+03		2.26E+01	7.50E+02	8.15E+02	3.39E+03	Nutrient		N (c)		---
Selenium	1 37	3	5.16E-01	J	5.16E-01	J	5.30E-01	1.05E+00	4.38E-01	2.00E+00	39		N (b)		
Sodium	27 / 37	73	1.88E+01		1.30E+02		9.02E+00	7.50E+02	1.28E+02		Nutrient		N (c)		---
Thallium	17 / 37	46	2.69E-01	J	6.78E-01		4.45E-01	1.50E+00	4.08E-01	1.30E+00	0.51	g	N (b)		---
Vanadium	37 / 37	100	1.83E+01		4.99E+01		1.81E-01	7.50E+00	2.51E+01	4.09E+01	39		Y	2.65E+01	2.65E+01
Zinc	37 / 37	100	2.60E+01		2.73E+02		2.10E+00	3.00E+00	7.25E+01	3.22E+02	2300		N (b)		---
Polychlorinated biphenyls (PCB)															
Aroclor 1221	1 / 25	4	1.90E-01		1.90E-01		3.50E-02	4.90E-02	2.69E-02		0.14		Y	3.96E-02	3.96E-02
Aroclor 1260	4 / 25	16	6.10E-02	J	1.90E-01		3.50E-02	4.90E-02	3.33E-02		0.22		N (a)		---
Nitroaromatics															
Amino-2,6-dinitrotoluene, 4-	3 / 27	11	3.24E-01		5.30E-01		2.88E-01	3.00E-01	1.82E-01		15		N (a)		---
Amino-4,6-dinitrotoluene, 2-	3 / 27	11	1.63E-01	J	4.38E-01		2.88E-01	3.00E-01	1.64E-01		15		N (a)		---
Dinitrotoluene, 2,6-	2 / 27	7	8.42E-01	J	4.12E+00		2.88E-01	1.46E+00	3.20E-01		0.71		Y	9.75E-01	9.75E-01
Nitrotoluene, 2-	2 / 27	7	2.34E+00		2.27E+01		2.88E-01	2.91E+00	1.06E+00		2.9		Y	7.66E+00	7.66E+00
Nitrotoluene, 3-	2 / 27	7	3.08E-01		1.52E+00		2.88E-01	3.00E-01	2.04E-01		0.61		Y	1.52E+00	1.52E+00
Nitrotoluene, 4-	2 / 27	7	7.07E-01		1.18E+01		2.88E-01	1.46E+00	6.00E-01		24		N (a)		---
Semivolatile Organic Compounds															
Acenaphthylene	2 / 34	6	7.06E-02	J	2.24E-01	J	3.50E-01	2.06E+00	2.33E-01		340	h	N (a)		---
Anthracene	2 / 34	6	8.11E-02	J	3.81E-01	J	3.50E-01	2.06E+00	2.38E-01		1700		N (a)		---
Benzo(a)anthracene	3 / 34	9	4.26E-02	J	6.52E-01	J	3.50E-01	2.06E+00	2.43E-01		0.15		Y	1.98E-01	1.98E-01
Benzo(a)pyrene	2 / 34	6	1.21E-01	JJ	3.95E-01	J	3.50E-01	2.06E+00	2.39E-01		0.015		Y	2.02E-01	2.02E-01
Benzo(b)fluoranthene	2 / 34	6	1.92E-01	JJ	8.78E-01	J	3.50E-01	2.06E+00	2.55E-01		0.15		Y	3.12E-01	3.12E-01
Benzo(ghi)perylene	1 / 34	3	1.06E-01	J	1.06E-01	J	3.50E-01	2.06E+00	2.57E-01		170	i	N (a)		---
Chrysene	3 / 34	9	3.95E-02	J	7.84E-01	J	3.50E-01	2.06E+00	2.47E-01		15		N (a)		---
Dibenzofuran	2 / 34	6	4.61E-01	J	1.40E+00	J	3.50E-01	2.06E+00	2.70E-01		7.8		N (a)		---
Di-n-butyl phthalate	1 / 34	3	5.90E-02	J	5.90E-02	J	3.50E-01	2.06E+00	2.56E-01		610		N (a)		---
Dinitrotoluene, 2,4-	11 / 34	32	2.30E-01	J	9.88E+00		3.50E-01	2.08E+00	9.05E-01		0.71		Y	1.52E+00	1.52E+00
Fluoranthene	5 / 34	15	4.01E-02	J	1.26E+00	J	3.50E-01	2.06E+00	2.56E-01		230		N (a)		---
Indeno(1,2,3-cd)pyrene	1 / 34	3	1.30E-01	J	1.30E-01	J	3.50E-01	2.06E+00	2.58E-01		0.15		N (a)		---

Table 2-6

Statistical Summary and COPC Selection of Chemicals Detected in Total Soil
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg	COPC? [c,d]	95% UCL [e] mg/kg	EPC [f] mg/kg
Methylnaphthalene, 2-	2 / 34	6	1.66E+00	J	5.60E+00		3.50E-01	2.06E+00	4.29E-01		31	N (a)		---
Naphthalene	2 / 34	6	1.02E+00	J	3.40E+00		3.50E-01	2.06E+00	3.46E-01		3.6	N (a)		---
Phenanthrene	3 / 34	9	1.17E-01	JJ	1.50E+00		3.50E-01	2.06E+00	2.82E-01		170 [i]	N (a)		---
Pyrene	3 / 34	9	6.27E-02	J	1.13E+00	J	3.50E-01	2.06E+00	2.60E-01		170	N (a)		---
Volatile Organic Compounds														
Acetone	21 / 21	100	2.10E-02	J	2.65E-01	J	1.46E-02	5.92E-02	7.36E-02		6100	N (a)		---
Benzene	6 27	22	1.21E-03	J	3.81E-03	J	8.89E-03	2.15E-02	4.64E-03		1.1	N (a)		
Butanone, 2-	10 / 10	100	5.55E-03	J	1.36E-02	J	4.62E-02	5.92E-02	9.50E-03		2800	N (a)		---
Carbon disulfide	3 / 27	11	9.88E-04	J	4.95E-03	JJ	8.89E-03	2.15E-02	5.37E-03		82	N (a)		---
Dichloroethane, 1,1-	6 27	22	2.63E-03	J	3.45E-02	J	8.89E-03	2.15E-02	7.24E-03		3.3	N (a)		
Dichloroethene, 1,1-	3 27	11	2.54E-03	J	5.63E-03	J	8.89E-03	2.15E-02	5.15E-03		24	N (a)		
Dichloroethene, cis-1,2-	2 27	7	3.20E-03	J	9.86E-03	J	8.89E-03	2.15E-02	5.49E-03		78	N (a)		
Naphthalene	2 27	7	1.51E-03	J	3.46E-03	J	8.89E-03	2.15E-02	5.12E-03		3.6	N (a)		
Toluene	8 / 27	30	1.04E-03	J	5.99E-03	J	8.89E-03	2.15E-02	4.69E-03		500	N (a)		---
Trichloroethane, 1,1,1-	10 / 27	37	1.39E-03	J	1.21E+00	J	8.89E-03	5.49E-01	1.14E-01		870	N (a)		---
Trichloroethene	5 / 27	19	2.63E-03	J	1.21E-01		8.89E-03	2.15E-02	1.35E-02		2.8	N (a)		---
Trimethylbenzene, 1,2,4-	3 27	11	9.88E-04	J	1.78E-03	J	8.89E-03	2.15E-02	5.05E-03		6.2	N (a)		

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

[a] IT Corporation (IT), 1998, *Site Investigation of Acid Areas* , Plum Brook Ordnance Works, Sandusky, Ohio, August.
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) 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 Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC.
[f] Concentration used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.
[g] RBSC based on thallium from EPA, 2010, Integrated Risk Information System.
[h] RBSC based on anthracene.
[i] RBSC based on pyrene.

Table 2-7

Statistical Summary and COPC Selection of Chemicals Detected in Maintenance Pit Material
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	BSC [a] mg/kg	RBSC [b] mg/kg		COPC? [c,d]	EPC [e] mg/kg
Inorganics														
Aluminum	3 / 3	100	2.20E+03		9.16E+03		1.72E+00	1.76E+00	6.37E+03	1.55E+04	7700		N (b)	---
Antimony	3 / 3	100	2.88E-01	J	1.46E+00	J	4.30E-01	4.39E-01	9.96E-01	9.30E+00	3.1		N (b)	---
Arsenic	3 / 3	100	1.38E+00	J	3.41E+00	J	8.60E-01	8.78E-01	2.08E+00	3.65E+01	0.39		N (b)	---
Barium	3 / 3	100	2.67E+01		1.20E+02		1.72E-01	1.76E-01	8.03E+01	8.26E+02	1500		N (b)	---
Beryllium	3 / 3	100	1.14E+00	J	1.20E+00	J	1.72E-01	1.76E-01	1.16E+00	1.00E+00	16		N (a)	---
Cadmium	2 / 3	67	2.21E-01	J	2.64E-01	J	4.30E-01	4.39E-01	2.35E-01		7		N (a)	---
Calcium	3 / 3	100	2.40E+05	J	2.88E+05	J	8.60E+01	8.78E+01	2.72E+05	5.23E+04	Nutrient		N (c)	---
Chromium	3 / 3	100	4.63E+00		9.62E+00		4.30E-01	4.39E-01	6.85E+00	2.90E+01	0.29		N (b)	---
Cobalt	3 / 3	100	1.65E+00	J	2.13E+00	J	3.44E-01	3.51E-01	1.84E+00	1.16E+02	2.3		N (b)	---
Copper	3 / 3	100	9.11E+00	J	1.14E+01	J	4.30E-01	4.39E-01	1.03E+01	5.62E+01	310		N (b)	---
Iron	3 / 3	100	4.92E+03		6.98E+03		3.44E+00	3.51E+00	5.76E+03	2.34E+05	5500		N (b)	---
Lead	3 / 3	100	7.71E+00		1.29E+01		4.30E-01	4.39E-01	1.05E+01	4.86E+01	400		N (b)	---
Magnesium	3 / 3	100	2.35E+04	J	2.96E+04	J	1.72E+00	1.76E+00	2.72E+04	1.04E+04	Nutrient		N (c)	---
Manganese	3 / 3	100	3.69E+02	J	1.05E+03	J	1.72E-01	1.76E-01	7.95E+02	3.51E+03	180		N (b)	---
Mercury	3 / 3	100	9.31E-03	J	1.70E-02	J	1.86E-02	1.99E-02	1.41E-02	8.50E-02	2.3		N (b)	---
Nickel	3 / 3	100	5.06E+00	J	7.33E+00	J	2.58E-01	2.63E-01	5.99E+00	5.51E+01	150		N (b)	---
Potassium	3 / 3	100	3.05E+02		4.62E+02		2.15E+01	2.19E+01	3.93E+02	3.39E+03	Nutrient		N (c)	---
Silver	3 3	100	4.80E-01		2.25E+00		4.30E-01	4.39E-01	1.25E+00	1.11E+01	39		N (b)	---
Sodium	3 / 3	100	1.42E+02		2.62E+02		8.60E+00	8.78E+00	2.09E+02		Nutrient		N (c)	---
Vanadium	3 / 3	100	6.31E+00		7.96E+00		1.72E-01	1.76E-01	6.96E+00	4.09E+01	39		N (b)	---
Zinc	3 / 3	100	1.63E+01		4.47E+01		2.15E+00	2.19E+00	3.16E+01	3.22E+02	2300		N (b)	---
Semivolatile Organic Compounds														
Acenaphthylene	1 / 3	33	1.95E-01	J	1.95E-01	J	1.78E+00	1.82E+00	6.63E-01		340	f	N (a)	---
Anthracene	1 / 3	33	2.57E-01	J	2.57E-01	J	1.78E+00	1.82E+00	6.84E-01		1700		N (a)	---
Benzo(a)anthracene	3 / 3	100	1.63E-01	J	6.51E-01	J	1.78E+00	1.82E+00	3.31E-01		0.15		Y	6.51E-01
Benzo(a)pyrene	1 / 3	33	4.98E-01	J	4.98E-01	J	1.78E+00	1.82E+00	7.64E-01		0.015		Y	4.98E-01
Benzo(b)fluoranthene	2 / 3	67	3.27E-01	J	1.01E+00	J	1.78E+00	1.82E+00	7.42E-01		0.15		Y	1.01E+00
Bis(2-ethylhexyl)phthalate	2 3	67	2.41E-01	J	7.40E-01	J	1.78E+00	1.82E+00	6.29E-01		35		N (a)	---
Chrysene	3 / 3	100	2.51E-01	J	7.19E-01	J	1.78E+00	1.82E+00	4.14E-01		15		N (a)	---
Fluoranthene	3 / 3	100	1.71E-01	J	1.02E+00	J	1.78E+00	1.82E+00	4.74E-01		230		N (a)	---
Phenanthrene	1 / 3	33	2.02E-01	J	2.02E-01	J	1.78E+00	1.82E+00	6.66E-01		170	g	N (a)	---
Pyrene	3 / 3	100	2.13E-01	J	9.10E-01	J	1.78E+00	1.82E+00	4.48E-01		170		N (a)	---

Table 2-7

Statistical Summary and COPC Selection of Chemicals Detected in Maintenance Pit Material
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

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

[a] IT Corporation (IT), 1998, *Site Investigation of Acid Areas* , Plum Brook Ordnance Works, Sandusky, Ohio, August.
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) 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] Concentration used in risk assessment is equal to the maximum detected concentration.
[f] RBSC based on anthracene.
[g] RBSC based on pyrene.

Table 2-8

Statistical Summary and COPC Selection of Chemicals Detected in Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	BSC [a] µg/L	RBSC [b] µg/L	COPC? [c,d]	95% UCL [e] µg/L	EPC [f] µg/L
Inorganics														
Aluminum	3 / 3	100	7.96E+01	J	1.55E+02	J	2.00E+02	2.00E+02	1.24E+02	3.09E+02	3700	N (a)		---
Arsenic	3 / 5	60	1.99E+01		1.31E+02		1.00E+01	1.00E+01	3.78E+01	7.40E+00	0.045	Y	1.31E+02	1.31E+02
Barium	5 / 5	100	3.05E+02		7.62E+02		2.00E+02	2.00E+02	4.91E+02	1.18E+04	730	N (b)		---
Cadmium	1 / 5	20	1.40E+00	J	1.40E+00	J	5.00E+00	5.00E+00	2.28E+00		1.8	N (a)		---
Calcium	5 / 5	100	2.80E+05		4.62E+05		1.00E+03	1.00E+03	3.52E+05	3.16E+05	Nutrient	N (c)		---
Iron	4 / 5	80	1.80E+02	J	2.94E+03		3.00E+02	3.00E+02	8.49E+02	1.55E+03	2600	Y	2.02E+03	2.02E+03
Lead	1 / 5	20	2.60E+00	J	2.60E+00	J	5.00E+00	1.00E+01	3.02E+00		15 [g]	N (a)		---
Magnesium	5 / 5	100	2.12E+05		3.52E+05		5.00E+03	5.00E+03	2.63E+05	2.17E+05	Nutrient	N (c)		---
Manganese	5 / 5	100	8.58E+01		1.07E+03		1.50E+01	1.50E+01	3.27E+02	6.36E+02	88	Y	1.21E+03	1.07E+03
Nickel	1 / 5	20	7.20E+00	J	7.20E+00	J	4.00E+01	4.00E+01	1.74E+01	8.60E+00	73	N (a)		---
Potassium	5 / 5	100	5.76E+04		1.20E+05		1.00E+04	2.00E+04	8.97E+04	1.16E+05	Nutrient	N (c)		---
Selenium	2 / 4	50	8.50E+00	J	1.17E+01	J	1.00E+01	2.00E+01	8.80E+00		18	N (a)		---
Sodium	5 / 5	100	6.38E+05		1.23E+06		1.00E+05	4.00E+05	9.82E+05	1.39E+06	Nutrient	N (c)		---
Thallium	2 / 5	40	3.80E+00	J	6.40E+00	J	1.00E+01	1.00E+01	5.04E+00		0.24	Y	7.87E+00	6.40E+00
Vanadium	4 / 5	80	1.40E+00	J	2.80E+00	J	5.00E+01	5.00E+01	6.48E+00		18	N (a)		---
Zinc	1 / 2	50	1.16E+01	J	1.16E+01	J	2.00E+01	4.00E+01	1.58E+01	5.01E+02	1100	N (a)		---
Semivolatile Organic Compounds														
3-Methylphenol and 4-Methylphenol	1 / 5	20	5.20E+00		5.20E+00		4.80E+00	1.00E+02	1.40E+01		93	N (a)		---
Dimethylphenol, 2,4-	3 / 5	60	1.50E+00	J	1.23E+01		4.80E+00	1.00E+02	1.51E+01		73	N (a)		---
Fluorene	1 / 5	20	5.40E+00	J	5.40E+00	J	4.80E+00	1.00E+02	1.26E+01		150	N (a)		---
Methylnaphthalene, 2-	5 / 5	100	6.30E+00		2.56E+02	J	4.80E+00	1.00E+02	7.25E+01		15	Y	4.06E+02	2.56E+02
Methylphenol, 2-	1 / 5	20	5.60E+00		5.60E+00		4.80E+00	1.00E+02	1.41E+01		180	N (a)		---
Naphthalene	5 / 5	100	4.50E+00	J	6.49E+01	J	4.80E+00	1.00E+02	2.40E+01		0.14	Y	4.72E+01	4.72E+01
Phenanthrene	2 / 5	40	1.27E+01	J	3.73E+01	J	4.80E+00	1.00E+02	1.15E+01		110	N (a)		---
Phenol	1 / 5	20	8.00E+00		8.00E+00		4.80E+00	1.00E+02	1.46E+01		1100	N (a)		---
Volatile Organic Compounds														
Acetone	2 / 3	67	5.07E+01	J	3.45E+02	J	2.50E+01	5.00E+02	1.36E+02		2200	N (a)		---
Benzene	4 / 5	80	3.70E+00		1.68E+03	J	1.00E+00	2.00E+01	3.68E+02		0.41	Y	1.85E+03	1.68E+03
Butanone, 2-	3 / 5	60	1.32E+01		8.13E+01	J	5.00E+00	1.00E+02	3.12E+01		710	N (a)		---
Carbon disulfide	2 / 5	40	3.80E+00		1.16E+01	J	2.00E+00	4.00E+01	7.48E+00		100	N (a)		---
Chloroform	2 / 5	40	2.68E+01		6.67E+01		1.00E+00	2.00E+01	1.90E+01		0.19	Y	5.63E+01	5.63E+01
Chloromethane	2 / 5	40	1.28E+01		3.56E+01	J	2.00E+00	4.00E+01	1.41E+01		19	Y	3.34E+01	3.34E+01
Ethylbenzene	5 / 5	100	9.90E+00		1.69E+02		1.00E+00	2.00E+01	5.12E+01		1.5	Y	2.06E+02	1.69E+02
Methyl-2-pentanone, 4-	1 / 5	20	3.30E+00	J	3.30E+00	J	5.00E+00	1.00E+02	2.17E+01		200	N (a)		---
Methylene chloride	1 / 5	20	7.76E+01	J	7.76E+01	J	5.00E+00	1.00E+02	2.70E+01		4.8	Y	6.04E+01	6.04E+01
Tetrachloroethane, 1,1,2,2-	2 / 5	40	1.20E+00		3.70E+00		1.00E+00	2.00E+01	5.08E+00		0.067	Y	4.09E+00	3.70E+00
Toluene	5 / 5	100	1.00E+01		6.92E+02		1.00E+00	2.00E+01	1.68E+02		230	Y	1.15E+03	6.92E+02
Xylenes, total	5 / 5	100	9.72E+01		1.13E+03		3.00E+00	6.00E+01	3.66E+02		20	Y	1.22E+03	1.13E+03
General Chemistry														
Chloride	5 / 5	100	1.32E+05		3.26E+06		5.00E+04	2.00E+05	1.99E+06		Nutrient	N (c)		---
Cyanide, total	3 / 5	60	1.40E+01	J	1.10E+02	J	1.00E+01	1.00E+01	4.10E+01		73	Y	1.10E-01	1.10E-01
Sulfate	4 / 5	80	9.60E+03		2.18E+05		2.00E+03	5.00E+04	6.14E+04		250000 [h]	N (a)		---

Table 2-8

Statistical Summary and COPC Selection of Chemicals Detected in Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

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

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

[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) tap water 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% Upper confidence limit (UCL) determined using ProUCL Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC

[f] Exposure point concentration (EPC) used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.

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

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

Table 2-9

Statistical Summary and COPC Selection of Chemicals Detected in Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	RBSC [b] µg/L		COPC? [c,d]	95% UCL [e] µg/L	EPC [f] µg/L
Inorganics														
Aluminum	7 / 8	88	2.04E+01	J	1.34E+03		2.00E+02	2.00E+02	4.24E+02	3700		N (a)		---
Barium	10 / 10	100	5.58E+01	J	2.60E+02		2.00E+02	2.00E+02	1.01E+02	730		N (a)		---
Calcium	10 / 10	100	2.54E+04		1.89E+05		1.00E+03	1.00E+03	9.74E+04	Nutrient		N (b)		---
Chromium	2 / 10	20	2.80E+00	J	3.30E+00	J	1.00E+01	1.00E+01	4.61E+00	0.043		Y	3.51E+00	3.30E+00
Cobalt	3 / 10	30	2.30E+00	J	6.10E+00	J	5.00E+01	5.00E+01	1.87E+01	1.1		Y	6.07E+00	6.07E+00
Iron	5 / 10	50	2.04E+02	JJ	2.83E+03		3.00E+02	3.00E+02	7.22E+02	2600		Y	1.48E+03	1.48E+03
Lead	2 / 7	29	2.10E+00	J	2.30E+00	J	5.00E+00	1.00E+01	3.49E+00	15	g	N (a)		---
Magnesium	10 / 10	100	1.50E+04		4.79E+04		5.00E+03	5.00E+03	2.98E+04	Nutrient		N (b)		---
Manganese	10 / 10	100	5.30E+01		4.58E+02		1.50E+01	1.50E+01	2.33E+02	88		Y	3.29E+02	3.29E+02
Nickel	7 / 10	70	2.40E+00	J	2.10E+01	J	4.00E+01	4.00E+01	1.14E+01	73		N (a)		---
Potassium	10 / 10	100	1.90E+03	JJ	1.90E+04	J	1.00E+04	1.00E+04	9.26E+03	Nutrient		N (b)		---
Selenium	3 / 10	30	4.50E+00	J	6.30E+00	J	1.00E+01	2.00E+01	6.07E+00	18		N (a)		---
Sodium	10 / 10	100	5.78E+03	JJ	9.71E+04		1.00E+04	2.00E+04	4.37E+04	Nutrient		N (b)		---
Vanadium	2 / 10	20	3.00E+00	J	3.40E+00	J	5.00E+01	5.00E+01	2.06E+01	18		N (a)		---
Zinc	4 / 8	50	9.20E+00	J	1.83E+01	J	2.00E+01	4.00E+01	1.33E+01	1100		N (a)		---
Nitroaromatics														
Amino-2,6-dinitrotoluene, 4-	2 / 10	20	2.80E-01		4.80E-01	J	1.90E-01	2.00E-01	1.54E-01	7.3		N (a)		---
Amino-4,6-dinitrotoluene, 2-	2 / 10	20	2.00E-01		4.15E-01	JJ	1.90E-01	2.00E-01	1.39E-01	7.3		N (a)		---
Dinitrotoluene, 2,4-	3 / 10	30	2.30E-01		1.06E+01	JJ	1.90E-01	2.00E-01	1.96E+00	0.099		Y	4.67E+00	4.67E+00
Dinitrotoluene, 2,6-	3 / 10	30	2.90E-01		9.15E+00	JJ	1.90E-01	2.00E-01	1.67E+00	0.099		Y	3.99E+00	3.99E+00
HMX	1 / 10	10	3.20E-01		3.20E-01		1.90E-01	2.00E-01	1.19E-01	180		N (a)		---
Nitrotoluene, 2-	4 / 10	40	1.80E+00		4.14E+01	JJ	1.90E-01	8.95E-01	8.27E+00	0.31		Y	3.77E+01	3.77E+01
Nitrotoluene, 3-	2 / 10	20	4.30E+00	JJ	5.15E+00	JJ	1.90E-01	2.00E-01	1.02E+00	0.37		Y	4.59E+00	4.59E+00
Nitrotoluene, 4-	4 / 10	40	6.10E-01		4.58E+00	JJ	1.90E-01	2.00E-01	1.13E+00	4.2		Y	4.40E+00	4.40E+00
Volatile Organic Compounds														
Acetone	1 / 8	13	1.00E+01	J	1.00E+01	J	2.50E+01	2.50E+01	1.22E+01	2200		N (a)		---
Bromoform	1 / 10	10	3.60E-01	J	3.60E-01	J	1.00E+00	1.00E+00	4.86E-01	8.5		N (a)		---
Carbon disulfide	1 / 10	10	5.40E-01	J	5.40E-01	J	2.00E+00	2.00E+00	9.54E-01	100		N (a)		---
Carbon tetrachloride	1 / 10	10	4.00E+00		4.00E+00		1.00E+00	1.00E+00	8.50E-01	0.2		Y	2.38E+00	2.38E+00
Dibromochloromethane	1 / 10	10	2.00E-01	J	2.00E-01	J	1.00E+00	1.00E+00	4.70E-01	0.15		Y	5.25E-01	2.00E-01
Dichloroethane, 1,1-	6 / 10	60	1.55E+00		8.30E+00		1.00E+00	1.00E+00	2.57E+00	2.4		Y	4.44E+00	4.44E+00
Dichloroethene, 1,1-	4 / 10	40	6.80E-01	J	1.40E+00		1.00E+00	1.00E+00	7.38E-01	34		N (a)		---
Dichloroethene, cis-1,2-	2 / 10	20	8.05E-01	JJ	8.40E-01	JJ	1.00E+00	1.00E+00	5.65E-01	37		N (a)		---
Trichloroethane, 1,1,1-	5 / 10	50	1.30E+00		3.84E+01		1.00E+00	1.00E+00	1.31E+01	910		N (a)		---
Trichloroethene	2 / 10	20	4.55E+00		5.40E+00		1.00E+00	1.00E+00	1.40E+00	2		Y	4.84E+00	4.84E+00
General Chemistry														
Chloride	10 / 10	100	6800		195000	J	2.00E+03	2.00E+05	5.40E+04	Nutrient		N (b)		---
Cyanide, total	4 / 10	40	7.5	J	13		1.00E+01	1.00E+01	7.20E+00	73		N (a)		---
Nitrate-Nitrite	5 / 10	50	32	J	730	JJ	1.00E+02	2.00E+03	5.48E+02	1000	f	N (a)		---
Sulfate	10 / 10	100	4300		282000		2.00E+03	1.00E+04	1.02E+05	250000	g	Y		2.82E+05

Table 2-9

Statistical Summary and COPC Selection of Chemicals Detected in Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	RBSC [b] µg/L	COPC? [c,d]	95% UCL [e] µg/L	EPC [f] µg/L

RBSC - Risk based screening concentration.
COPC - Chemical of Potential Concern.
µg/L - micrograms per liter.
mg/L - milligrams 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 analyzec

[a] Shaw Environmental, Inc. (Shaw), 2005b, 2004 Groundwater Data Summary and Evaluation Report, Final, Former Plum Brook Ordnance Works, Sandusky, Ohio, Apri
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) tap water 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) = essential nutrient.
[d] Y = Chemical is chosen as COPC.
[e] Screening criteria for lead based on the action level of 15 µg/L (EPA, 1996, *Drinking Water Regulations and Health Advisories*, Office of Water, Washington, DC, October).
[f] Because no Regional Screening Level exists for nitrate-nitrite, 0.1 X the nitrate-nitrite MCL (10 mg/L) is used for screening.
[g] US Environmental Protection Agency (EPA), 2009, ***2009 Edition of the Drinking Water Standards and Health Advisories***, Office of Water, October, EPA 822-R-09-011.

Table 2-10

Statistical Summary and COPC Selection of Chemicals Detected in Sediment
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent Detection	Range of Values, mg/kg: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Mean mg/kg	RBSC [a] mg/kg	COPC? [b,c]	95% UCL [d] mg/kg	EPC [e] mg/kg
Inorganics													
Aluminum	6 / 6	100	5.71E+03	J	1.03E+04		1.30E+01	1.40E+01	7.78E+03	7.70E+04	N (a)		---
Arsenic	6 / 6	100	8.30E+00	J	1.78E+01	J	5.10E-01	5.60E-01	1.20E+01	3.90E-01	Y	1.52E+01	1.52E+01
Barium	6 / 6	100	5.88E+01	J	9.21E+01	J	1.30E+01	1.40E+01	7.61E+01	1.50E+04	N (a)		---
Beryllium	6 / 6	100	6.10E-01		9.60E-01		3.20E-01	3.50E-01	7.46E-01	1.60E+02	N (a)		---
Cadmium	6 / 6	100	4.60E-01	J	1.90E+00		2.50E-01	2.80E-01	8.32E-01	7.00E+01	N (a)		---
Calcium	6 / 6	100	3.26E+03		1.12E+04	J	3.20E+02	3.50E+02	6.54E+03	Nutrient	N (b)		---
Chromium	6 / 6	100	1.04E+01		1.82E+01		6.30E-01	6.90E-01	1.37E+01	2.90E-01	Y	1.62E+01	1.62E+01
Cobalt	6 / 6	100	1.08E+01		2.60E+01	J	3.20E+00	3.50E+00	1.71E+01	2.30E+01	Y	2.28E+01	2.28E+01
Copper	6 / 6	100	1.05E+01	J	2.28E+01		1.60E+00	1.70E+00	1.75E+01	3.10E+03	N (a)		---
Iron	6 / 6	100	1.87E+04	J	3.02E+04	J	6.30E+00	6.90E+00	2.28E+04	5.50E+04	N (a)		---
Lead	6 / 6	100	9.00E+00	J	1.79E+01	J	6.30E+00	6.90E+00	1.15E+01	4.00E+02	N (a)		---
Magnesium	6 / 6	100	1.59E+03	J	4.94E+03		3.20E+02	3.50E+02	3.00E+03	Nutrient	N (b)		---
Manganese	6 / 6	100	7.37E+02		5.36E+03		5.00E+00	2.10E+01	1.84E+03	1.80E+03	Y	3.98E+03	3.98E+03
Mercury	5 / 6	83	2.20E-02	J	3.10E-02	J	9.90E-02	1.20E-01	2.88E-02	2.30E+01	N (a)		---
Nickel	6 / 6	100	2.61E+01	J	5.22E+01		2.50E+00	2.80E+00	3.42E+01	1.50E+03	N (a)		---
Potassium	6 / 6	100	6.14E+02	J	1.40E+03		1.30E+03	1.40E+03	9.43E+02	Nutrient	N (b)		---
Selenium	6 / 6	100	1.70E+00	J	3.00E+00	J	6.30E+00	6.90E+00	2.09E+00	3.90E+02	N (a)		---
Sodium	6 / 6	100	1.10E+02	J	1.92E+02	J	1.30E+03	1.40E+03	1.57E+02	Nutrient	N (b)		---
Thallium	2 / 6	33	2.60E-01	J	1.90E+00		6.30E-01	1.40E+00	5.78E-01	5.10E-01 [f]	Y	1.90E+00	1.90E+00
Vanadium	6 / 6	100	1.94E+01	J	2.65E+01	J	3.20E+00	3.50E+00	2.35E+01	3.90E+02	N (a)		---
Zinc	6 / 6	100	5.24E+01	J	8.03E+01		1.30E+00	1.40E+00	6.28E+01	2.30E+04	N (a)		---
Semivolatiles Organic Compounds													
Benzo(b)fluoranthene	1 / 6	17	4.70E-02	JJ	4.70E-02	JJ	2.10E-01	2.40E-01	1.03E-01	1.50E-01	N (a)		---
Fluoranthene	1 / 6	17	8.36E-02	JJ	8.36E-02	JJ	2.10E-01	2.40E-01	1.09E-01	2.30E+03	N (a)		---
Phenanthrene	1 / 6	17	5.46E-02	J	5.46E-02	J	2.10E-01	2.40E-01	1.04E-01	1.70E+02 [g]	N (a)		---
Pyrene	1 / 6	17	6.28E-02	JJ	6.28E-02	JJ	2.10E-01	2.40E-01	1.05E-01	1.70E+02	N (a)		---
Volatiles Organic Compounds													
Acetone	1 / 6	17	9.21E-02	J	9.21E-02	J	5.80E-02	7.10E-02	4.31E-02	6.10E+03	N (a)		---
Carbon disulfide	1 / 6	17	1.90E-03	J	1.90E-03	J	5.80E-03	7.10E-03	3.11E-03	8.20E+01	N (a)		---
Methylene chloride	1 / 5	20	5.82E-02		5.82E-02		1.20E-02	1.40E-02	1.69E-02	1.10E+01	N (a)		---

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

[a] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) residential soil and are based on a risk level of 1.0E-06 and a hazard index of 1.

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

[c] Y = Chemical is chosen as COPC.

Table 2-10

Statistical Summary and COPC Selection of Chemicals Detected in Sediment
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

[d] 95% Upper confidence limit (UCL) determined using ProUCL Version 4.0 (EPA, 2007, ProUCL Version 4.0, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, April,on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC.

[e] Expsoure point concentration (EPC) used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower.

[g] RBSC based on thallium from EPA, 2010, Integrated Risk Information System.

[g] RBSC based on pyrene.

Table 2-11

Statistical Summary and COPC Selection of Chemicals Detected in Surface Water
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentration Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	RBSC [a] µg/L	COPC? [b,c]	95% UCL [d] µg/L	EPC [e] µg/L
Inorganics													
Aluminum	6 / 6	100	1.66E+03		2.38E+04		2.00E+02	2.00E+02	5.67E+03	3.70E+04	N (a)		---
Arsenic	1 / 7	14	8.80E+00	J	8.80E+00	J	1.00E+01	1.00E+01	5.54E+00	4.50E-02	Y	6.69E+00	6.69E+00
Barium	7 / 7	100	4.16E+01	J	1.55E+02	J	2.00E+02	2.00E+02	7.62E+01	7.30E+03	N (a)		---
Beryllium	1 / 7	14	1.10E+00	J	1.10E+00	J	4.00E+00	4.00E+00	1.87E+00	7.30E+00	N (a)		---
Calcium	7 / 7	100	1.10E+05		1.51E+05		1.00E+03	1.00E+03	1.35E+05	Nutrient	N (b)		---
Chromium	6 / 7	86	2.60E+00	J	3.98E+01		1.00E+01	1.00E+01	8.85E+00	4.30E-02	Y	3.16E+01	3.16E+01
Cobalt	5 / 7	71	2.35E+00	JJ	1.63E+01	J	5.00E+01	5.00E+01	1.12E+01	1.10E+01	Y	1.74E+01	1.63E+01
Copper	1 / 7	14	3.85E+01		3.85E+01		2.50E+01	2.50E+01	1.62E+01	1.50E+02	N (a)		---
Iron	7 / 7	100	1.09E+03		4.39E+04		3.00E+02	3.00E+02	9.50E+03	2.60E+04	Y	2.28E+04	2.28E+04
Lead	6 / 7	86	2.60E+00	J	1.80E+01		5.00E+00	1.00E+01	6.15E+00	1.50E+01 [d]	Y	NA	6.15E+00
Magnesium	7 / 7	100	1.74E+04		2.93E+04		5.00E+03	5.00E+03	2.66E+04	Nutrient	N (b)		---
Manganese	7 / 7	100	3.81E+02		2.22E+03		1.50E+01	1.50E+01	1.19E+03	8.80E+02	Y	1.71E+03	1.71E+03
Nickel	6 / 7	86	4.80E+00	J	7.16E+01		4.00E+01	4.00E+01	1.85E+01	7.30E+02	N (a)		---
Potassium	7 / 7	100	1.28E+03	J	6.54E+03	J	1.00E+04	2.00E+04	3.03E+03	Nutrient	N (b)		---
Selenium	3 / 7	43	3.50E+00	J	4.40E+00	J	1.00E+01	1.00E+01	4.50E+00	1.80E+01	N (a)		---
Sodium	7 / 7	100	1.82E+04		3.45E+04		1.00E+04	1.00E+04	2.81E+04	Nutrient	N (b)		---
Vanadium	6 / 7	86	4.50E+00	J	4.61E+01	J	5.00E+01	5.00E+01	1.40E+01	1.80E+02	N (a)		---
Zinc	6 / 7	86	1.97E+01	J	1.77E+02		2.00E+01	2.00E+01	4.80E+01	1.10E+04	N (a)		---
Volatile Organics													
Dichloroethane, 1,1-	4 / 7	57	2.50E-01	J	3.90E-01	J	1.00E+00	1.00E+00	4.12E-01	2.4	N (a)		---
Dichloroethene, cis-1,2-	1 / 7	14	2.30E-01	JJ	2.30E-01	JJ	1.00E+00	1.00E+00	4.61E-01	37	N (a)		---
Trichloroethane, 1,1,1-	2 / 7	29	4.70E-01	J	5.60E-01	JJ	1.00E+00	1.00E+00	5.04E-01	910	N (a)		---

RBSC - Risk based screening concentration.
COPC - Chemical of Potential Concern.
NA - Not applicable.
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 USEPA Regional Screening Level Table (May 2010) tap water values are based on a risk level of 1.0E-06 and a hazard index of 1.
[b] N = Chemical is not chosen as a COPC:
(a) = maximum detected concentration is less than the RBSC.
(b) = essential nutrient.
[c] Y = Chemical is chosen as COPC.
[d] Screening criteria for lead based on the action level of 15 µg/L (EPA, 1996, *Drinking Water Regulations and Health Advisories*, Office of Water, Washington, DC, October).
[d] 95% Upper confidnece limit (UCL) determined using ProUCL Version 4.00.04 (U.S. Environmental Protection Agency (EPA), 2009, ProUCL Version 4.00.04, Office of Research and Development, Technology Support Center Characterization and Monitoring Branch, Las Vegas, Nevada, February, on line at http://www.epa.gov/esd/tsc/form.htm). Calculated only for COPC
[e] Exposure point concentration (EPC) used in risk assessment equal to 95% UCL or maximum detected concentration, whichever is lower. The EPC for lead is the mean.

Table 3-1

Receptor/Exposure Scenarios
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

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

Table 3-1

Receptor/Exposure Scenarios
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

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

Table 3-1

Receptor/Exposure Scenarios
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

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

[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] 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).

[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] Maintenance pit material was conservatively evaluated using the construction worker and these same exposure pathways.

[f] There is no plausible pathway for exposure.

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
General Variables Used in All Intake Models					
Body weight (BW), kg	70[a]	70[a]	Child: 15[a] Adult: 70[a]	70[a]	Child: 15[a] Adult: 70[a]
Averaging time, noncancer (AT), days[b]	9125	183	Child: 2190 Adult: 8760	9125	Child: 2190 Adult: 10950
Averaging time, cancer (AT), days[b]	25550	25550	25550	25550	25550
Inhalation of VOCs and Resuspended Dust from Surface Soil, Total Soil or Subsurface Soil					
Fraction exposed to contaminated medium (FI_a), unitless	1[c]	1[c]	1[c]	NA	NA
Inhalation rate (IR_a), m^3/day	20[d]	20[d]	Child: 10[e] Adult: 20[d]	NA	NA
Exposure frequency (EF), days/year	250[d]	250[c]	350[a]	NA	NA
Exposure duration (ED), years	25[a]	0.5[c]	Child: 6[a] Adult: 24[a]	NA	NA
Inhalation of VOCs in Indoor Air from Subsurface Soil					
Fraction exposed to contaminated medium (FI_a), unitless	NA	NA	1[c]	1[c]	NA
Inhalation rate (IR_a), m^3/day	NA	NA	Child: 10[e] Adult: 20[d]	20[d]	NA
Exposure frequency (EF), days/year	NA	NA	350[a]	250[a]	NA
Exposure duration (ED), years	NA	NA	Child: 6[a] Adult: 24[a]	25[a]	NA
Incidental Ingestion of Soil					
Fraction exposed to contaminated medium (FI_{so}), unitless	1[c]	1[c]	0.9[f]	1[c]	1[c]

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

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
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

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 (Fl_{gw}), unitless	1[c]	NA	1[c]	1[c]	NA
Drinking water ingestion rate (IR_{gw}), L/day	1[d]	NA	Child: 1[e] Adult: 2[d]	1[d]	NA
Exposure frequency (EF), days/year	250[d]	NA	350[a]	250[a]	NA
Dermal Contact with Groundwater					
Fraction exposed to contaminated medium (Fl_{gw}), unitless	1[c]	NA	1[c]	1[c]	NA
Body surface area exposed to water (SA_{gw}), cm^2	3,300[c]	NA	Child: 6,600[i] Adult: 20,000[i]	3,300[i]	NA
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 (Fl_{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
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Pathway Variable	Grounds-keeper	Construction Worker	On-Site Resident	Indoor Worker	Hunter and Hunter's Child
Dermal Contact with 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]
Exposure duration (ED), years	NA	NA	NA	NA	Child: 6[a] Adult: 30[c]

Table 3-2

Variables Used to Estimate Potential Chemical Intakes and Contact Rates for Receptors
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

[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, DC, 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 Site-Related Chemicals[a]
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical of Potential Concern	Soil-to-forage Biotransfer Factor B_p (unitless)	Biotransfer Factor for Beef B_b (days/kg)	Biotransfer Factor for Venison B_v (unitless)	Diffusivity In Air (D_i) (cm^2/second)	Soil Organic Carbon-Water Partition Coefficient K_{oc} (cm^3/g)	log K_{ow}	Absorption Fraction ABS (unitless)	Permeability Coefficient K_p (cm/hour)	*tau* (hour/event)	*t** (hour)	Fraction Absorbed from Water FA (unitless)	B (unitless)	Molecular Weight (g/mole)	Henry's Law Constant (atm-m^3/mole)
Metals														
Aluminum	NA	NA	NA	NA	1500	NA	NA	NA	NA	NA	NA	NA	26.98	NA
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
Vanadium	NA	NA	NA	NA	1.55E+02	NA	0	1.00E-03	NA	NA	NA	NA	50.94	NA
Pesticides and PCBs														
Aroclor 1221	7.44E-02	1.26E-03	7.55E-06	5.54E-02	2.75E+02	4.53E+00	0.14	4.30E-01	11.29	47.9	0.5	3.2	200.7	7.36E-08
Nitroaromatics														
Dinitrotoluene, 2,4-	NA	NA	NA	2.03E-01	9.55E+01	1.98E+00	0.1	3.10E-03	1.12	2.69	1	0	182.1	5.40E-08
Dinitrotoluene, 2,6-	NA	NA	NA	5.91E-02	4.57E+01	1.72E+00	0.1	2.10E-03	1.12	2.69	1	0	182.1	7.47E-07
Nitrotoluene, 2-	NA	NA	NA	7.14E-02	2.75E+02	2.30E+00	NA	8.91E-03	0.62	1.48	1	0	137.14	1.25E-05
Nitrotoluene, 3-	NA	NA	NA	7.14E-02	1.45E+02	2.45E+00	NA	1.12E-02	0.62	1.48	1	0.1	137.15	9.30E-06
Nitrotoluene, 4-	NA	NA	NA	7.14E-02	3.09E+02	2.37E+00	NA	9.91E-03	0.62	1.48	1	0	137.14	5.63E-06
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
Methylnaphthalene, 2-	NA	NA	NA	6.97E-02	3.98E+03	3.86E+00	0.13	8.94E-02	0.657	1.58	1	0.41	142.2	5.18E-04
Naphthalene	NA	NA	NA	5.90E-02	2.00E+03	3.30E+00	0.13	4.70E-02	0.56	1.34	1	0.2	128.2	4.40E-04
Volatile Organic Compounds														
Benzene	NA	NA	NA	8.80E-02	5.89E+01	2.13E+00	NA	1.50E-02	0.29	0.7	1	0.1	78.1	5.55E-03
Carbon tetrachloride	NA	NA	NA	7.80E-02	1.74E+02	2.83E+00	NA	1.60E-02	0.78	1.86	1	0.1	153.8	2.76E-02
Chloroform	NA	NA	NA	1.04E-01	3.98E+01	1.97E+00	NA	6.80E-03	0.5	1.19	1	0	119.4	3.67E-03
Chloromethane (Methyl Chloride)	NA	NA	NA	1.39E-01	1.41E+01	9.10E-01	NA	3.30E-03	0.2	0.49	1	0	50.5	8.82E-03
Dibromochloromethane	NA	NA	NA	1.96E-02	6.31E+01	2.23E+00	NA	3.20E-03	1.57	3.77	1	0	208.3	7.83E-04
Dichloroethane, 1,1-	NA	NA	NA	7.42E-02	3.16E+01	1.79E+00	NA	6.70E-03	0.38	0.92	1	0	99	5.62E-03
Ethylbenzene	NA	NA	NA	7.50E-02	3.63E+02	3.15E+00	NA	4.90E-02	0.42	1.01	1	0.2	106.2	7.88E-03
Methylene chloride	NA	NA	NA	1.01E-01	1.17E+01	1.25E+00	NA	3.50E-03	0.32	0.76	1	0	84.9	3.25E-03
Tetrachloroethane, 1,1,2,2-	NA	NA	NA	7.10E-02	9.33E+01	2.39E+00	NA	6.90E-03	0.93	2.24	1	0	167.9	3.67E-04
Toluene	NA	NA	NA	8.70E-02	1.82E+02	2.73E+00	NA	3.10E-02	0.35	0.84	1	0.1	92.1	6.64E-03
Trichloroethene	NA	NA	NA	7.90E-02	1.66E+02	2.42E+00	NA	1.20E-02	0.58	1.39	1	0.1	131.4	9.85E-03
Xylenes	NA	NA	NA	7.00E-02	4.07E+02	3.20E+00	NA	5.30E-02	0.42	1.01	1	0.2	106.2	5.18E-03
General Chemistry														
Cyanide, total	NA	NA	NA	NA	2.84E+00	NA	NA	1.00E-03	NA	NA	NA	NA	26.02	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-m^3 - atmospheres per cubic meter

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

Table 4-1

Summary of Toxicity Assessment [a]
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

Chemical of Potential Concern	GAF	Weight of Evidence	Oral Slope Factor (mg/kg-day) $^{-1}$	Dermal Slope Factor (mg/kg-day) $^{-1}$	Weight of Evidence	Inhalation Unit Risk (µg/m^3) $^{-1}$	Inhalation Slope Factor (mg/kg-day) $^{-1}$
Metals							
Aluminum	NA	D	NA	NA	NA	NA	NA
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-03	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
Vanadium	0.026	ND	NA	NA	ND	NA	NA
Pesticides and PCBs							
Aroclor 1221	1	B2	2.00E+00	2.00E+00	B2	5.70E-04	2.00E+00
Nitroaromatics							
Nitrotoluene, 2-	1	B2	2.20E-01	2.20E-01	ND	NA	NA
Nitrotoluene, 3-	1	D	NA	NA	ND	NA	NA
Nitrotoluene, 4-	1	C	1.60E-02	1.60E-02	ND	NA	NA
Dinitrotoluene, 2,4-	1	B2	6.80E-01	6.80E-01	D	NA	NA
Dinitrotoluene, 2,6-	1	B2	6.80E-01	6.80E-01	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
Methylnaphthalene, 2-	1	D	NA	NA	D	NA	NA
Naphthalene	1	C	ND	NA	C	3.40E-05	1.20E-01
Volatile Organic Compounds							
Benzene	1	A	5.50E-02	5.50E-02	A	7.80E-06	2.70E-02
Carbon tetrachloride	1	B	7.00E-02	7.00E-02	B	6.00E-06	2.10E-02
Chloroform	1	B2	NC	NA	B2	ND	NC
Chloromethane (Methyl Chloride)	1	D	NA	NA	D	NA	NA
Dibromochloromethane	1	C	8.40E-02	8.40E-02	ND	NA	NA
Dichloroethane, 1,1-	1	C	ND	NA	C	ND	NA
Ethylbenzene	1	D	NA	NA	D	NA	NA
Methylene chloride	1	B2	7.50E-03	7.50E-03	B2	4.70E-07	1.60E-03
Tetrachloroethane, 1,1,2,2-	1	C	2.00E-01	2.00E-01	C	5.80E-05	2.00E-01
Toluene	1	D	NA	NA	D	NA	NA
trans-1,3-Dichloropropene	1	B2	1.00E-01	1.00E-01	B2	4.00E-06	1.40E-02
Trichloroethene	1	B	5.90E-03	5.90E-03	B	2.00E-06	7.00E-03
Xylenes	1	D	NA	NA	D	NA	NA
General Chemistry							
Cyanide, total	1	D	NA	NA	D	NA	NA

mg/kg-day = Milligram per kilogram - day.
µg/m^3 - Milligram per cubic meter.
GAF - Gastrointestinal absorption factor.
NA - Not available or not applicable.
ND - No data or not determined.
NC - Noncancer value is regarded as protective of cancer effects in the Integrated Risk Information System (IRIS) evaluation (EPA, 2010).

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

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 4-1

Summary of Toxicity Assessment [a]
Locomotive Building Area, Plum Brook Ordnance Works
Sandusky, Ohio

		Oral		Dermal	Reference	Inhalation	
Chemical of Potential Concern	GAF	Reference Dose mg/kg-day	Target Organs	Reference Dose mg/kg-day	Concentration mg/m^3	Reference Dose mg/kg-day	Target Organs
Metals							
Aluminum	NA	1	CNS	NA	5.00E-03	0.0014	CNS
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
Vanadium	0.026	5.00E-03	Hair	1.30E-04	ND	NA	NA
Pesticides and PCBs							
Aroclor 1221	1	7.00E-05	F	7.00E-05	ND	NA	NA
Nitroaromatics							
Nitrotoluene, 2-	1	9.00E-04	E	9.00E-04	ND	NA	NA
Nitrotoluene, 3-	1	1.00E-03	E	1.00E-03	ND	NA	NA
Nitrotoluene, 4-	1	4.00E-03	E,K	4.00E-03	ND	NA	NA
Dinitrotoluene, 2,4-	1	2.00E-03	CNS,E,L	2.00E-03	ND	NA	NA
Dinitrotoluene, 2,6-	1	1.00E-03	CNS,E,L	1.00E-03	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
Methylnaphthalene, 2-	1	4.00E-03	RT	4.00E-03	ND	NA	NA
Naphthalene	1	2.00E-02	RB/OW	2.00E-02	3.00E-03	8.60E-04	RT
Volatile Organic Compounds							
Benzene	1	4.00E-03	IS	4.00E-03	3.00E-03	8.60E-03	BC
Carbon tetrachloride	1	4.00E-03	L	4.00E-03	1.00E-01	2.90E-02	L
Chloroform	1	1.00E-02	L	1.00E-02	5.00E-02	1.40E-02	L,K
Chloromethane (Methyl Chloride)	1	ND	NA	NA	9.00E-02	2.60E-02	CNS
Dibromochloromethane	1	2.00E-02	L	2.00E-02	ND	NA	NA
Dichloroethane, 1,1-	1	2.00E-01	K	2.00E-01	ND	NA	NA
Ethylbenzene	1	1.00E-01	L,K	1.00E-01	1.00E+00	2.90E-01	F
Methylene chloride	1	6.00E-02	L	6.00E-02	9.00E-01	3.00E-01	NA
Tetrachloroethane, 1,1,2,2-	1	4.00E-03	L	4.00E-03	ND	NA	NA
Toluene	1	8.00E-02	K	8.00E-02	5.00E+00	1.40E+00	NA
trans-1,3-Dichloropropene	1	3.00E-02	Stomach Irritation	3.00E-02	2.00E-02	5.70E-03	NE
Trichloroethene	1	ND	NA	ND	ND	NA	NA
Xylenes	1	2.00E-01	RB/OW, Su	2.00E-01	1.00E-01	2.90E-02	CNS
General Chemistry							
Cyanide, total	1	2.00E-02	BW/Thyroid/NS	2.00E-02	3.00E-03	8.60E-04	CNS,Thyroid

mg/kg-day = milligram per kilogram - day
mg/m^3 - milligram per cubic meter
GAF - gastrointestinal absorption factor
NA - not available or not applicable
ND - no data
NC - Noncancer value is regarded as protective of cancer effects in the Integrated Risk Information System (IRIS) evaluation (EPA, 2010).

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

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

Table 5-1

Summary of Risk for All Receptors
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

	Groundskeeper [a]		Construction Worker		Indoor Worker		Resident			Hunter		Hunter's Child	
	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total HI	Total ILCR	Total Adult - HI	Total Child - HI	Total ILCR	Total HI	Total ILCR	Total HI
Bedrock Groundwater Use													
Exposure Media													
Surface Soil	9.40E-06	0.007	NE	NE	7.74E-07	0.002	NE	NE	NE	1.89E-07	0.0003	3.45E-12	0.0000003
Total Soil	8.01E-06	0.2	5.15E-07	0.5	NE	NE	9.77E-06	0.04	0.3	NE	NE	NE	NE
Maintenance Pit[b]	NE	NE	1.56E-07	NA	NE	NE	NE	NE	NE	NE	NE	NE	NE
Groundwater	1.04E-03	11	NE	NE	1.04E-03	11	8.24E-03	71	166	NE	NE	NE	NE
Sediment	NE	NE	5.75E-07	0.2	NE	NE	3.63E-06	0.03	0.3	NE	NE	NE	NE
Surface Water	NE	NE	9.26E-09	0.1	NE	NE	2.16E-07	0.05	0.08	NE	NE	NE	NE
Total ILCR or HI	**1.E-03**	**11**	**1.E-06**	**0.9**	**1.E-03**	**11**	**8.E-03**	**72**	**167**	**2.E-07**	**0.0003**	**3.E-12**	**0.0000003**
Overburden Groundwater Use													
Exposure Media													
Surface Soil	9.40E-06	0.007	NE	NE	7.74E-07	0.002	NE	NE	NE	1.89E-07	0.0003	3.45E-12	0.0000003
Total Soil	8.01E-06	0.2	5.15E-07	0.5	NE	NE	9.77E-06	0.04	0.3	NE	NE	NE	NE
Maintenance Pit[b]	NE	NE	1.56E-07	NA	NE	NE	NE	NE	NE	NE	NE	NE	NE
Groundwater	5.31E-05	0.7	NE	NE	5.31E-05	0.7	2.30E-04	2	5	NE	NE	NE	NE
Sediment	NE	NE	5.75E-07	0.2	NE	NE	3.63E-06	0.03	0.3	NE	NE	NE	NE
Surface Water	NE	NE	9.26E-09	0.1	NE	NE	2.16E-07	0.05	0.08	NE	NE	NE	NE
Total ILCR or HI	**6.E-05**	**0.9**	**1.E-06**	**0.9**	**5.E-05**	**0.7**	**2.E-04**	**2**	**6**	**2.E-07**	**0.0003**	**3.E-12**	**0.0000003**

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

[a] Total ILCR and total HI values for the groundskeeper reflect the respective totals for the future groundskeeper assumed to use either bedrock or overburden groundwater. The total ILCR and HI values for the current groundskeeper are simply those shown for surface soil. The rounded current groundskeeper ILCR is 9E-6 and the rounded HI is 0.01. The ILCR for the current groundskeeper without the contributior of background-related chromium is 3E-6.

[b] The Maintenance Pit material is composed of backfill gravel and associated fines. This stony material is unsuitable for use as topsoil or growth medium, thus exposure to this material would be negligible to minimal. Because the construction worker scenario assumes exposure to subsurface soil, this pit material was conservatively evaluated under this scenario for comparative purposes, even though the construction worker scenario makes the unrealistic assumption (in this case) of constant daily exposure.

Table 5-2

Summary of Risk for Receptors[a] Excluding Groundwater Exposure[b]
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

	Future Groundskeeper		Indoor Worker		Resident		
	Total ILCR[c]	Total HI	Total ILCR	Total HI	Total ILCR	Total Adult - HI	Total Child - HI
Exposure Media							
Surface Soil	NE	NE	7.74E-07	0.002	NE	NE	NE
Total Soil	8.01E-06	0.2	NE	NE	9.77E-06	0.04	0.3
Sediment	NE	NE	NE	NE	3.63E-06	0.03	0.3
Surface Water	NE	NE	NE	NE	2.16E-07	0.05	0.08
Total ILCR or HI	**8.E-06**	**0.2**	**8.E-07**	**0.002**	**1.E-05**	**0.1**	**0.7**

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 above receptors are those which were evaluated for groundwater exposure as shown in Table 5-1.
[b] Groundwater use is not regarded as plausible because of low yield in the overburden and bedrock units and naturally poor quality in the bedrock unit.
[c] The total ILCR for the future groundskeeper excluding the contribution of background-related chromium is 3E-6.

FIGURES

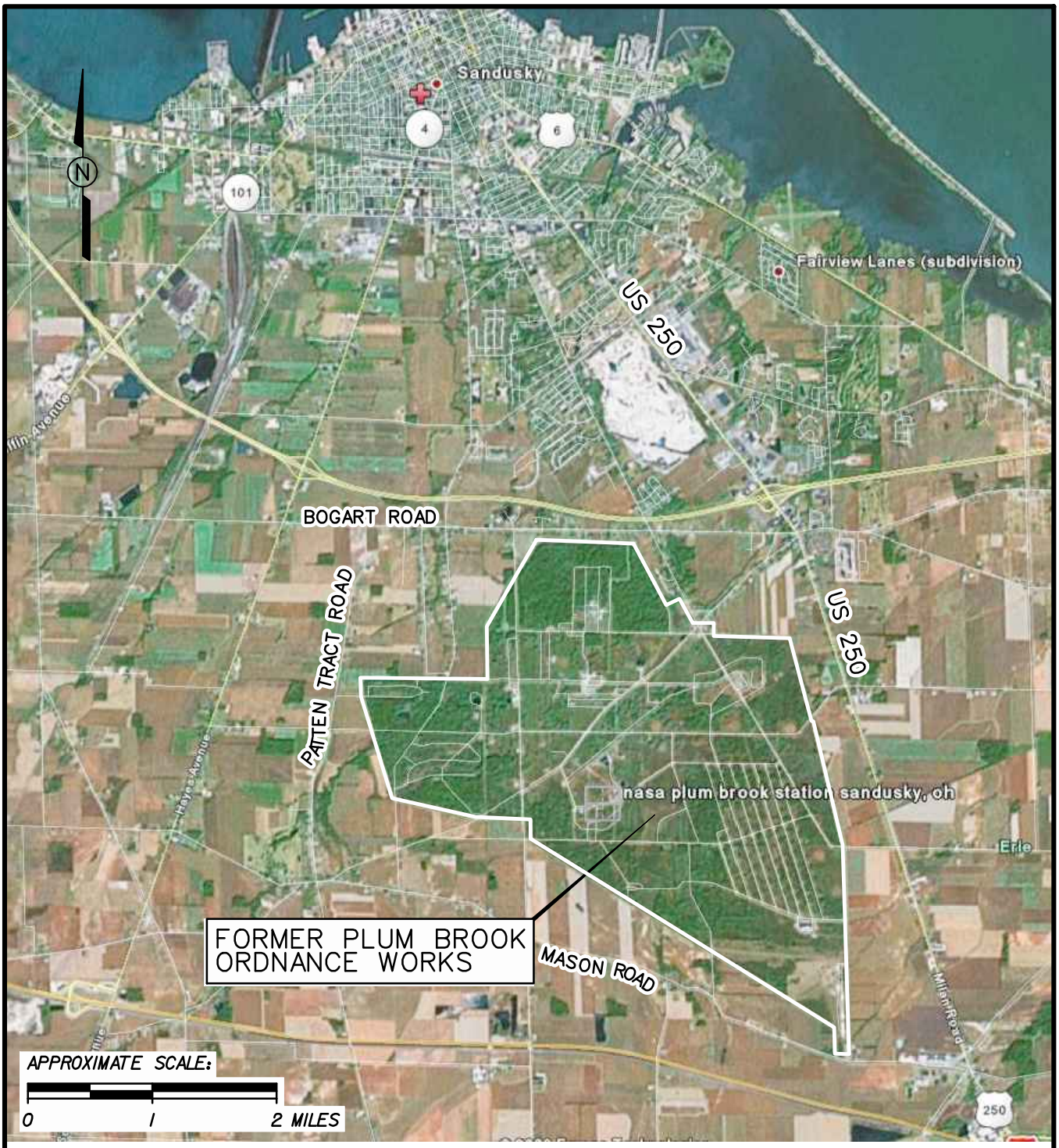

FIGURE 1-1
PBOW VICINITY MAP

LOCOMOTIVE BUILDING AREA BASELINE HUMAN HEALTH RISK ASSESSMENT FORMER PLUM BROOK ORDNANCE WORKS NASA PLUM BROOK STATION SANDUSKY, OHIO

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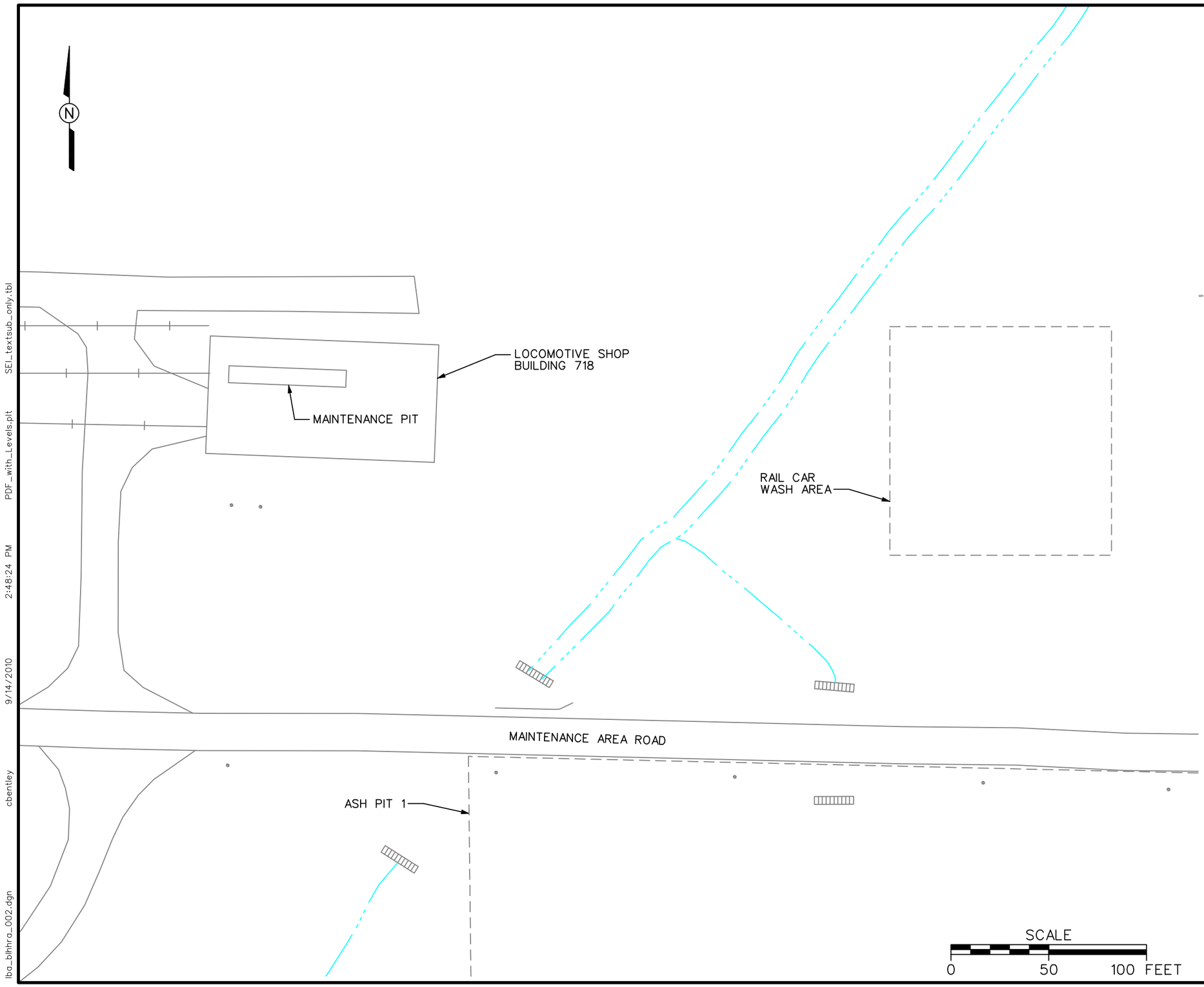
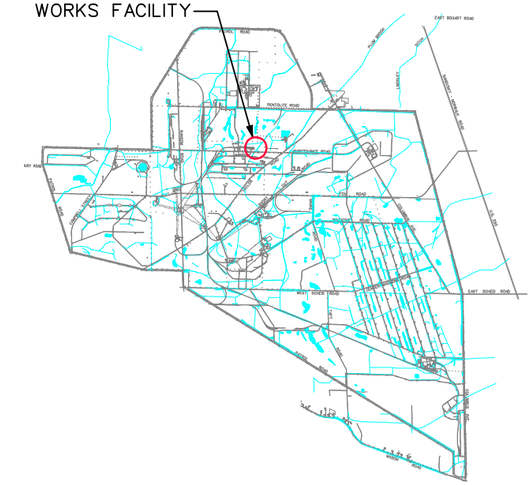


LEGEND:

- HEADWALL
- UTILITY POLE
- CREEK, DITCH, CONVEYANCE

LOCOMOTIVE BUILDING AREA
WITHIN PLUM BROOK ORDNANCE
WORKS FACILITY



NOTES:

1. ASH PIT 1 IS NOT PART OF THE LOCOMOTIVE BUILDING AREA, BUT IS AN ADJACENT STUDY AREA.

FIGURE 1-2
LOCATION MAP

LOCOMOTIVE BUILDING AREA BASELINE
HUMAN HEALTH RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

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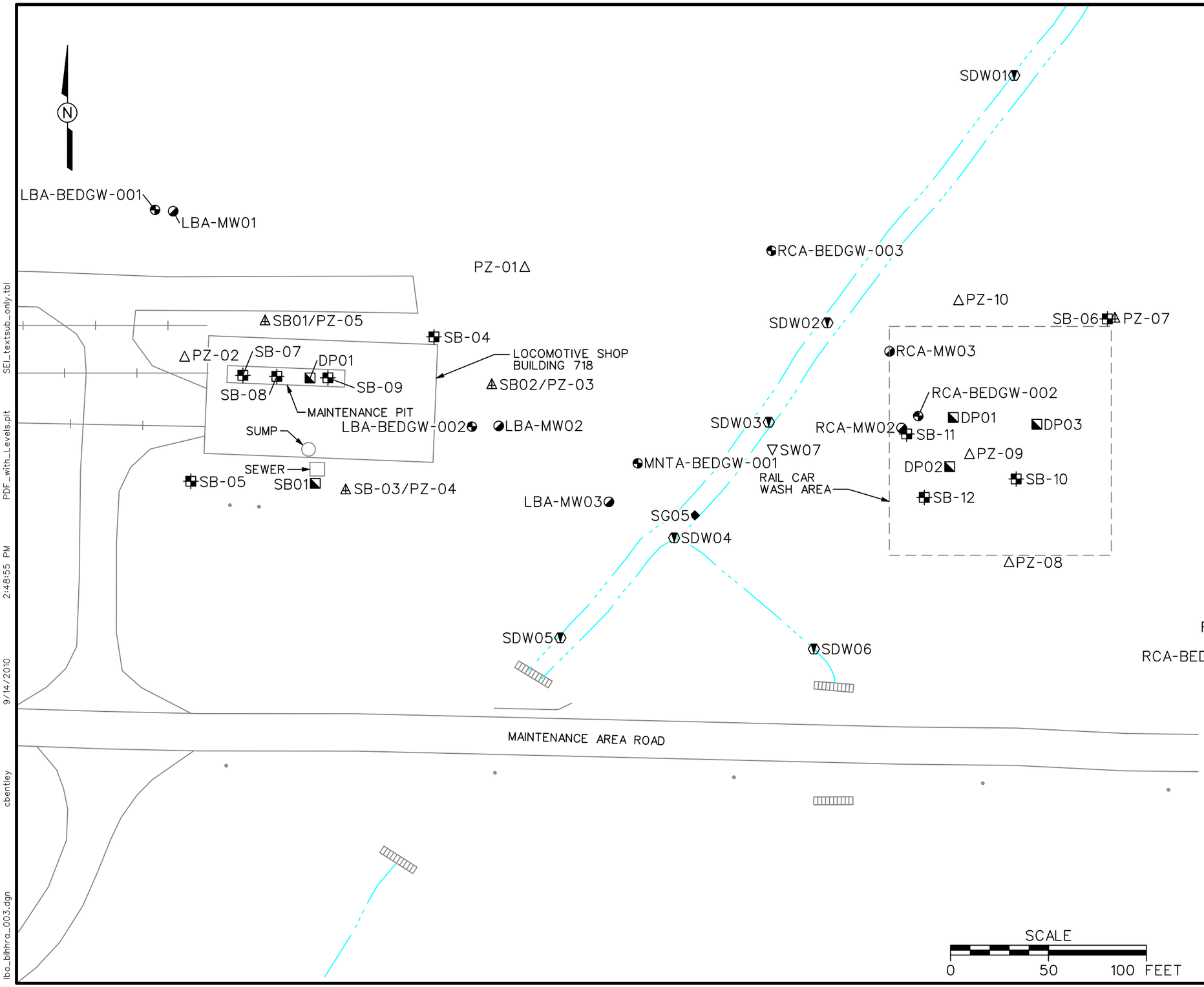

LEGEND:

- OVERBURDEN MONITORING WELL (SCREENED IN SHALE)
- BEDROCK MONITORING WELL (SCREENED IN LIMESTONE)
- SOIL BORING
- PIEZOMETER
- SOIL BORING/PIEZOMETER
- SURFACE WATER SAMPLE
- SURFACE WATER/SEDIMENT SAMPLE
- STAFF GAUGE
- HISTORICAL SOIL BORING

NOTES:

1. SAMPLES FROM THE HISTORICAL SOIL BORINGS WERE COLLECTED AND ANALYZED IN 1999 AS PART OF THE LIMITED SITE INVESTIGATION (USACE, 2000).

FIGURE 2-1
SITE MAP WITH SAMPLE LOCATIONS

LOCOMOTIVE BUILDING AREA BASELINE HUMAN HEALTH RISK ASSESSMENT
FORMER PLUM BROOK ORDNANCE WORKS
NASA PLUM BROOK STATION
SANDUSKY, OHIO

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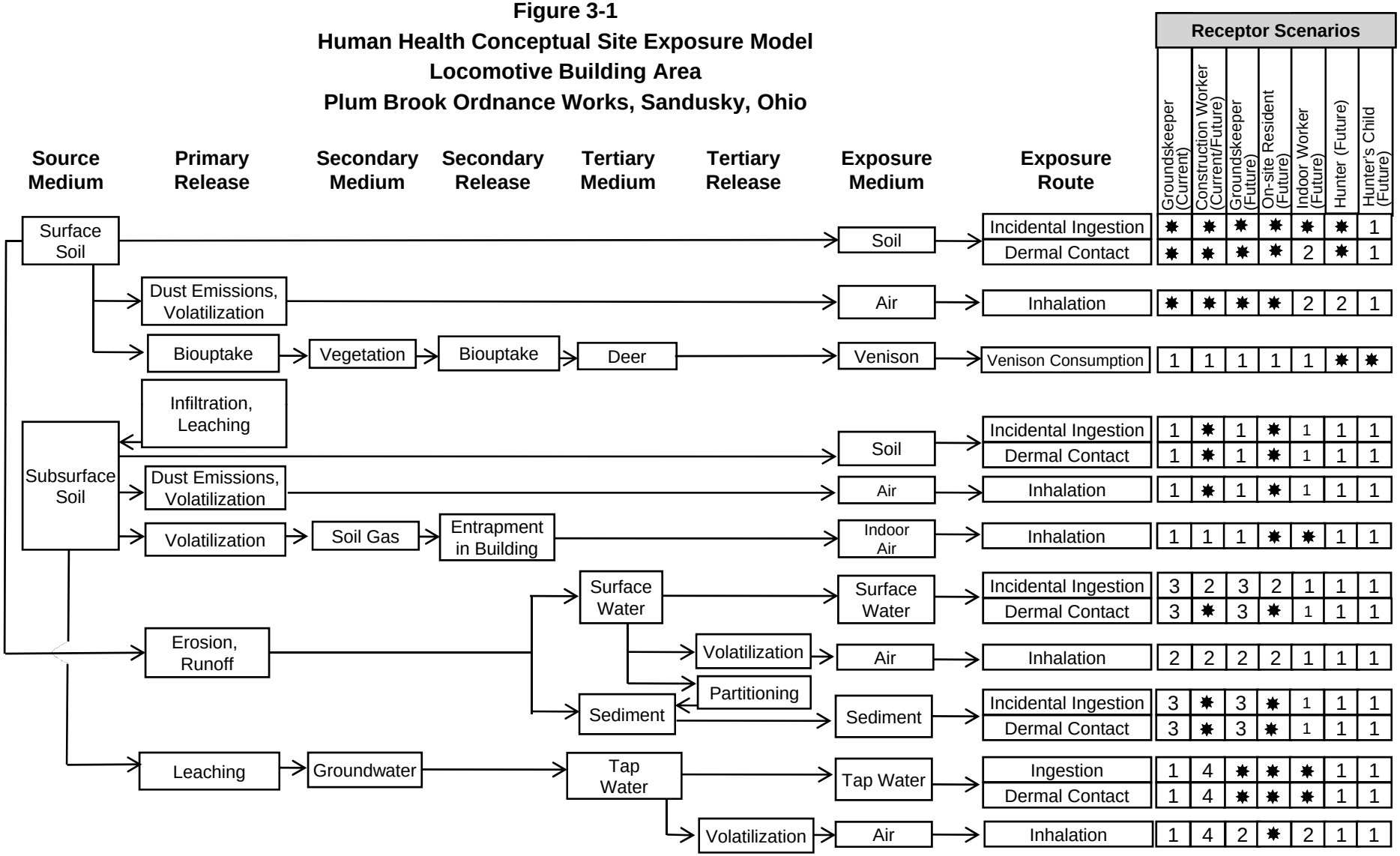
Figure 3-1
Human Health Conceptual Site Exposure Model
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio



✸= 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

DATA SUMMARY OF CHEMICALS DETECTED IN OVERBURDEN GROUNDWATER PIEZOMETERS

Table A-1

Summary of Samples for Human Health Risk Evaluation
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample No	Sample Purpose	Date	Analyses
Overburden Piezometer [a]				
LBA-PZ01	LBA3000	REG	31-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ05	LBA3001	FD	28-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ02	LBA3003	REG	29-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ03	LBA3004	REG	29-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ04	LBA3005	REG	30-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC
LBA-PZ05	LBA3006	REG	28-Jan-09	Cyanide, Exp, Gen Chem, Metals (f & uf), SVOC, VOC

[a] These samples are summarized and screened, but no exposure calculations are presented.

f & uf - Metals analysis performed on filtered and unfiltered samples, respectively.
FD - Field Duplicate sample; REG - regular sample
Gen Chem - General Chemistry
Metals - Metals analysis performed on unfiltered samples unless otherwise noted.
SVOC - Semivolatile organic compounds.
VOC - Volatile organic compounds.
ft bgs - Feet below ground surface.

Table A-2

Statistical Summary of Chemicals Detected in Overburden Piezometer Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	RBSC [b] µg/L		Exceeds RBSC? [c,d]
Inorganics												
Aluminum	5 / 5	100	1.62E+02		1.28E+04		8.89E+01	8.89E+01	6.02E+03	3700		Y
Arsenic	3 / 5	60	1.32E+01		4.27E+01		1.11E+01	1.11E+01	1.61E+01	0.045		Y
Barium	5 / 5	100	1.60E+01		3.23E+02		4.44E+00	4.44E+00	1.18E+02	730		N (a)
Beryllium	3 / 5	60	3.63E+00	JJ	1.37E+01		4.44E+00	4.44E+00	6.81E+00	7.3		Y
Cadmium	4 / 5	80	2.43E+00	J	4.63E+00		4.44E+00	4.44E+00	2.94E+00	1.8		Y
Calcium	5 / 5	100	7.53E+04		2.99E+05		1.11E+02	1.11E+02	1.72E+05	Nutrient		N (b)
Chromium	3 / 5	60	6.35E+00	J	2.04E+01		1.11E+01	1.11E+01	1.16E+01	0.043		Y
Cobalt	3 / 5	60	5.57E+00	JJ	1.86E+01		8.89E+00	8.89E+00	9.09E+00	1.1		Y
Copper	4 / 5	80	5.98E+00	J	6.60E+01		1.11E+01	1.11E+01	2.97E+01	150		N (a)
Iron	5 / 5	100	4.69E+02		4.07E+04		8.89E+01	8.89E+01	1.79E+04	2600		Y
Lead	3 / 5	60	7.66E+00		4.86E+01		5.56E+00	5.56E+00	1.77E+01	15	g	Y
Magnesium	5 / 5	100	1.63E+04		9.07E+04		4.44E+01	4.44E+01	4.85E+04	Nutrient		N (b)
Manganese	5 / 5	100	2.53E+01		1.45E+03		4.44E+00	4.44E+00	5.07E+02	88		Y
Nickel	3 / 5	60	6.42E+00	J	2.05E+01		6.67E+00	6.67E+00	1.02E+01	73		N (a)
Potassium	5 / 5	100	5.10E+02	J	4.80E+03		5.56E+02	5.56E+02	2.31E+03	Nutrient		N (b)
Sodium	5 / 5	100	2.82E+03		2.04E+04		2.22E+02	2.22E+02	9.75E+03	Nutrient		N (b)
Vanadium	3 / 5	60	1.04E+01		3.71E+01		4.44E+00	4.44E+00	1.76E+01	18		Y
Zinc	3 / 5	60	3.50E+01	JJ	1.36E+02		5.56E+01	5.56E+01	6.75E+01	1100		N (a)
Nitroaromatics												
Amino-2,6-dinitrotoluene, 4-	1 / 5	20	1.71E+00		1.71E+00		3.08E-01	3.43E-01	4.70E-01	7.3		N (a)
Amino-4,6-dinitrotoluene, 2-	1 / 5	20	1.60E+00		1.60E+00		3.08E-01	3.43E-01	4.48E-01	7.3		N (a)
Dinitrotoluene, 2,4-	1 / 5	20	1.72E+01		1.72E+01		3.08E-01	1.71E+00	3.57E+00	0.099		Y
Dinitrotoluene, 2,6-	1 / 5	20	1.51E+01		1.51E+01		3.08E-01	1.71E+00	3.15E+00	0.099		Y
Nitrotoluene, 2-	1 / 5	20	6.89E+00		6.89E+00		3.08E-01	1.71E+00	1.51E+00	0.31		Y
Nitrotoluene, 3-	1 / 5	20	3.91E+00		3.91E+00		3.08E-01	3.43E-01	9.10E-01	0.37		Y
Nitrotoluene, 4-	1 / 5	20	1.80E+00		1.80E+00		3.08E-01	3.43E-01	4.88E-01	4.2		N (a)
Semivolatile Organic Compounds												
Dinitrotoluene, 2,4-	1 / 5	20	1.33E+01		1.33E+01		1.00E+01	1.05E+01	6.71E+00	0.099		Y
Dinitrotoluene, 2,6-	1 / 5	20	7.11E+00	J	7.11E+00	J	1.00E+01	1.05E+01	5.47E+00	0.099		Y
Volatile Organic Compounds												
Acetone	1 / 1	100	3.46E+00	J	3.46E+00	J	1.00E+01	1.00E+01	3.46E+00	2200		N (a)
Dichloroethane, 1,1-	4 / 5	80	7.70E-01	J	1.00E+01		2.00E+00	2.00E+00	3.85E+00	2.4		Y
Dichloroethene, 1,1-	1 / 5	20	6.60E-01	J	6.60E-01	J	2.00E+00	2.00E+00	9.32E-01	34		N (a)
Dichloroethene, cis-1,2-	1 / 5	20	3.40E-01	J	3.40E-01	J	2.00E+00	2.00E+00	8.68E-01	37		N (a)
Naphthalene	1 / 5	20	4.33E+00		4.33E+00		2.00E+00	2.00E+00	1.67E+00	0.14		Y

Table A-2

Statistical Summary of Chemicals Detected in Overburden Piezometer Groundwater
Locomotive Building Area
Plum Brook Ordnance Works
Sandusky, Ohio

Chemical	Detection Frequency	Percent hits	Range of values, µg/L: Detected Concentrations Minimum	VQ	Maximum	VQ	Reporting Limits Minimum	Maximum	Arithmetic Mean µg/L	RBSC [b] µg/L		Exceeds RBSC? [c,d]
Trichloroethane, 1,1,1-	5 / 5	100	7.20E-01	J	5.84E+01		2.00E+00	2.00E+00	2.33E+01	910		N (a)
Trichloroethene	2 / 5	40	8.10E-01	J	7.03E+00		2.00E+00	2.00E+00	2.17E+00	2		Y
Trimethylbenzene, 1,2,4-	1 / 5	20	4.00E-01	J	4.00E-01	J	2.00E+00	2.00E+00	8.80E-01	1.5		N (a)
General Chemistry												
Chloride	5 / 5	100	3.24E+03	J	7.96E+03	J	2.50E+02	2.50E+02	5.54E+03	Nutrient		N (b)
Nitrate-Nitrite	4 / 5	80	2.60E+01	J	5.81E+02		4.00E+01	4.00E+01	1.97E+02	1000	f	N (a)
Sulfate	5 / 5	100	2.82E+04		1.93E+05		5.00E+02	5.00E+03	6.97E+04	250000	g	N (a)

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

[a] Shaw Environmental, Inc. (Shaw), 2005b, 2004 Groundwater Data Summary and Evaluation Report, Final, Former Plum Brook Ordnance Works, Sandusky, Ohio, April.
[b] Risk-Based Screening Concentrations based on USEPA Regional Screening Level Table (May 2010) tap water values and are based on a risk level of 1.0E-06 and a hazard index of 0.1.
[c] N = MDC of chemical does not exceed RBSC and (for applicable inorganics) BSC or chemical is an essential human macronutrient:
(a) = MDC is less than the RBSC.
(b) = essential nutrient.
[d] Y = MDC of chemical exceeds RBSC or BSC.
[e] Screening criteria for lead based on the action level of 15 µg/L (EPA, 1996, *Drinking Water Regulations and Health Advisories*, Office of Water, Washington, DC, October).
[f] Because no Regional Screening Level exists for nitrate-nitrite, 0.1 X the nitrate-nitrite MCL (10 mg/L) is used for screening.
[g] US Environmental Protection Agency (EPA), 2009, ***2009 Edition of the Drinking Water Standards and Health Advisories***, Office of Water, October, EPA 822-R-09-011.

APPENDIX B

ProUCL OUTPUT FOR UPPER CONFIDENCE LIMITS ON THE MEAN

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Aluminum	D_Aluminum	Chromium	D_Chromium	Vanadium	D_Vanadium	Nitrotoluene, 2-	D_Nitrotoluene, 2-
LBA-SB01	LBA0001	4400	1	10.4	1	26.5	1	0.3	0
LBA-SB01	LBA0002	7580	1	11.1	1	18.8	1	0.3	0
LBA-SB01	LBA0003	9330	1	13.6	1	22.9	1	0.291	0
LBA-SB02	LBA0004	7710	1	10.9	1	23.7	1	0.291	0
LBA-SB02	LBA0005	9960	1	17	1	24.7	1	0.3	0
LBA-SB02	LBA0006	8330	1	13.1	1	21.7	1	2.34	1
LBA-SB03	LBA0007LBA0008	6560	1	10.4	1	21.05	1	0.295	0
LBA-SB03	LBA0010	8700	1	13.8	1	20.9	1	0.296	0
LBA-SB03	LBA0011	7110	1	11.1	1	18.3	1	0.297	0
LBA-SB04	LBA0012	5800	1	11.8	1	27.5	1	0.299	0
LBA-SB04	LBA0013	5390	1	12	1	25.5	1	0.299	0
LBA-SB04	LBA0014	7750	1	11.9	1	19.2	1	0.294	0
LBA-SB05	LBA0015	8260	1	12.4	1	21.6	1	0.296	0
LBA-SB05	LBA0016	11000	1	18.5	1	27.6	1	0.297	0
LBA-SB05	LBA0017	6920	1	11	1	19	1	0.296	0
LBA-SB06	LBA0018	7020	1	11.5	1	24.4	1	0.288	0
LBA-SB06	LBA0019	7410	1	11.6	1	20.3	1	0.296	0
LBA-SB06	LBA0020LBA0021	7890	1	12.35	1	21.95	1	0.2965	0
LBA-SB10	LBA0026LBA0027	6520	1	12	1	23	1	0.293	0
LBA-SB10	LBA0029	7180	1	12.9	1	27.7	1	0.297	0
LBA-SB10	LBA0030	12000	1	19	1	27.9	1	0.294	0
LBA-SB11	LBA0031	6690	1	14.7	1	25.7	1	0.297	0
LBA-SB11	LBA0032	8940	1	16.4	1	28.2	1	0.291	0
LBA-SB11	LBA0033	10400	1	15.3	1	23.7	1	22.7	1
LBA-SB12	LBA0034	5950	1	11.7	1	23.5	1	0.294	0
LBA-SB12	LBA0035	7140	1	14.7	1	22.5	1	0.291	0
LBA-SB12	LBA0036	11800	1	17.6	1	24.3	1	0.294	0
PBOW99-DP001	PBOW99-DP001-0004	10100	1	16	1	31.2	1		
PBOW99-DP001	PBOW99-DP001-0008AA	10800	1	19.1	1	24.5	1		
PBOW99-DP002	PBOW99-DP002-0002	6290	1	11.7	1	22	1		
PBOW99-DP002	PBOW99-DP002-0004	8640	1	22.5	1	49.9	1		
PBOW99-DP002	PBOW99-DP002-0006	17200	1	28.6	1	33.7	1		
PBOW99-DP003	PBOW99-DP003-0004	9500	1	16.3	1	33.4	1		
PBOW99-DP003/-DP004	PBOW99-DP003-0006/DP004-0006	11550	1	20.4	1	28.15	1		
PBOW99-SB001	PBOW99-SB001-0005	9590	1	16.3	1	20.3	1		
PBOW99-SB001	PBOW99-SB001-001	7180	1	51	1	24.3	1		
PBOW99-SB001	PBOW99-SB001-003	8020	1	14.9	1	27.6	1		

All results are in mg/kg

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Nitrotoluene, 3-	D_Nitrotoluene, 3-	Benzo(a)anthracene	D_Benzo(a)anthracene	Benzo(a)pyrene	D_Benzo(a)pyrene
LBA-SB01	LBA0001	0.3	0	0.0426	1	0.38	0
LBA-SB01	LBA0002	0.3	0	0.414	0	0.414	0
LBA-SB01	LBA0003	0.291	0	0.403	0	0.403	0
LBA-SB02	LBA0004	0.291	0	2.06	0	2.06	0
LBA-SB02	LBA0005	0.3	0	0.409	0	0.409	0
LBA-SB02	LBA0006	0.308	1	0.417	0	0.417	0
LBA-SB03	LBA0007LBA0008	0.295	0	0.15025	1	0.1205	1
LBA-SB03	LBA0010	0.296	0	0.394	0	0.394	0
LBA-SB03	LBA0011	0.297	0	0.418	0	0.418	0
LBA-SB04	LBA0012	0.299	0	0.397	0	0.397	0
LBA-SB04	LBA0013	0.299	0	0.399	0	0.399	0
LBA-SB04	LBA0014	0.294	0	0.407	0	0.407	0
LBA-SB05	LBA0015	0.296	0	0.416	0	0.416	0
LBA-SB05	LBA0016	0.297	0	0.419	0	0.419	0
LBA-SB05	LBA0017	0.296	0	0.418	0	0.418	0
LBA-SB06	LBA0018	0.288	0	0.652	1	0.395	1
LBA-SB06	LBA0019	0.296	0	0.412	0	0.412	0
LBA-SB06	LBA0020LBA0021	0.2965	0	0.4115	0	0.4115	0
LBA-SB10	LBA0026LBA0027	0.293	0	0.4285	0	0.4285	0
LBA-SB10	LBA0029	0.297	0	0.383	0	0.383	0
LBA-SB10	LBA0030	0.294	0	0.421	0	0.421	0
LBA-SB11	LBA0031	0.297	0	0.407	0	0.407	0
LBA-SB11	LBA0032	0.291	0	0.395	0	0.395	0
LBA-SB11	LBA0033	1.52	1	0.434	0	0.434	0
LBA-SB12	LBA0034	0.294	0	0.423	0	0.423	0
LBA-SB12	LBA0035	0.291	0	0.384	0	0.384	0
LBA-SB12	LBA0036	0.294	0	0.399	0	0.399	0
PBOW99-DP001	PBOW99-DP001-0004			0.37	0	0.37	0
PBOW99-DP001	PBOW99-DP001-0008AA						
PBOW99-DP002	PBOW99-DP002-0002			0.97	0	0.97	0
PBOW99-DP002	PBOW99-DP002-0004			0.39	0	0.39	0
PBOW99-DP002	PBOW99-DP002-0006						
PBOW99-DP003	PBOW99-DP003-0004			0.49	0	0.49	0
PBOW99-DP003/-DP004	PBOW99-DP003-0006/DP004-0006						
PBOW99-SB001	PBOW99-SB001-0005			0.42	0	0.42	0
PBOW99-SB001	PBOW99-SB001-001			0.35	0	0.35	0
PBOW99-SB001	PBOW99-SB001-003			0.39	0	0.39	0

All results are in mg/kg

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Dinitrotoluene, 2,4- svoc	D_Dinitrotoluene, 2,4- svoc	Aroclor 1221	Dinitrotoluene, 2,6-exp	Benzo(b)fluoranthene
LBA-SB01	LBA0001	0.38	0	0.01905	0.6	0.19
LBA-SB01	LBA0002	0.414	0	0.02055	0.6	0.207
LBA-SB01	LBA0003	0.403	0		0.582	0.2015
LBA-SB02	LBA0004	0.268	1	0.02025	0.582	1.03
LBA-SB02	LBA0005	1.03	1	0.02025	0.6	0.2045
LBA-SB02	LBA0006	9.88	1		0.842	0.2085
LBA-SB03	LBA0007LBA0008	0.397	0	0.020275	0.59	0.1918
LBA-SB03	LBA0010	0.394	0	0.0197	0.592	0.197
LBA-SB03	LBA0011	0.418	0		0.594	0.209
LBA-SB04	LBA0012	0.416	0	0.01985	0.598	0.1985
LBA-SB04	LBA0013	0.399	0	0.0199	0.598	0.1995
LBA-SB04	LBA0014	0.407	0		0.588	0.2035
LBA-SB05	LBA0015	2.06	0	0.0209	0.592	0.208
LBA-SB05	LBA0016	0.419	0	0.02055	0.594	0.2095
LBA-SB05	LBA0017	0.418	0		0.592	0.209
LBA-SB06	LBA0018	1.29	1	0.0202	0.576	0.878
LBA-SB06	LBA0019	0.412	0	0.02085	0.592	0.206
LBA-SB06	LBA0020LBA0021	0.4115	0		0.593	0.20575
LBA-SB10	LBA0026LBA0027	0.423	0	0.021425	0.586	0.21425
LBA-SB10	LBA0029	0.383	0	0.01935	0.594	0.1915
LBA-SB10	LBA0030	0.352	1		0.588	0.2105
LBA-SB11	LBA0031	0.4095	0	0.02035	0.594	0.2035
LBA-SB11	LBA0032	0.866	1	0.0194	0.582	0.1975
LBA-SB11	LBA0033	5.82	1		4.12	0.217
LBA-SB12	LBA0034	0.4285	0	0.19	0.588	0.2115
LBA-SB12	LBA0035	0.23	1	0.0192	0.582	0.192
LBA-SB12	LBA0036	0.399	0		0.588	0.1995
PBOW99-DP001	PBOW99-DP001-0004	0.43	1	0.0185		0.185
PBOW99-DP001	PBOW99-DP001-0008AA					
PBOW99-DP002	PBOW99-DP002-0002	4	1	0.0195		0.485
PBOW99-DP002	PBOW99-DP002-0004	1.1	1	0.0195		0.195
PBOW99-DP002	PBOW99-DP002-0006					
PBOW99-DP003	PBOW99-DP003-0004	0.49	0	0.0245		0.245
PBOW99-DP003/-DP004	PBOW99-DP003-0006/DP004-0006					
PBOW99-SB001	PBOW99-SB001-0005	0.42	0	0.021		0.21
PBOW99-SB001	PBOW99-SB001-001	0.35	0	0.0175		0.175
PBOW99-SB001	PBOW99-SB001-003	0.39	0	0.0195		0.195

All results are in mg/kg

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Chromium	D_Chromium	Dinitrotoluene, 2,4-exp	D_Dinitrotoluene, 2,4-exp	Benzo(a)anthracene	D_Benzo(a)anthracene
LBA-SB01	LBA0001	11.7	1	0.3	0	0.0426	1
LBA-SB02	LBA0004	51	1	0.153	1	2.06	0
LBA-SB03	LBA0007LBA0008	10.4	1	0.295	0	0.15025	1
LBA-SB04	LBA0012	10.9	1	0.294	0	0.397	0
LBA-SB05	LBA0015	10.4	1	0.299	0	0.416	0
LBA-SB06	LBA0018	11.8	1	0.296	0	0.652	1
LBA-SB10	LBA0026LBA0027	12.4	1	0.288	0	0.4285	0
LBA-SB11	LBA0031	11.5	1	1.13	1	0.407	0
LBA-SB12	LBA0034	12	1	0.297	0	0.423	0
PBOW99-DP002	PBOW99-DP002-0002	14.7	1			0.485	0
PBOW99-SB001	PBOW99-SB001-001	11.7	1			0.175	0

All results are in mg/kg

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Benzo(a)pyrene	D_Benzo(a)pyrene	Dinitrotoluene, 2,4-	D_Dinitrotoluene, 2,4-	Aroclor 1221	Benzo(b)fluoranthene
LBA-SB01	LBA0001	0.38	0	0.38	0	0.01905	0.19
LBA-SB02	LBA0004	2.06	0	0.268	1	0.02025	1.03
LBA-SB03	LBA0007LBA0008	0.1205	1	0.397	0	0.020275	0.1918
LBA-SB04	LBA0012	0.397	0	0.416	0	0.01985	0.1985
LBA-SB05	LBA0015	0.416	0	2.06	0	0.0209	0.208
LBA-SB06	LBA0018	0.395	1	1.29	1	0.0202	0.878
LBA-SB10	LBA0026LBA0027	0.4285	0	0.423	0	0.021425	0.21425
LBA-SB11	LBA0031	0.407	0	0.4095	0	0.02035	0.2035
LBA-SB12	LBA0034	0.423	0	0.4285	0	0.19	0.2115
PBOW99-DP002	PBOW99-DP002-0002	0.485	0	4	1	0.0195	0.2425
PBOW99-SB001	PBOW99-SB001-001	0.175	0	0.175	0	0.0175	0.0875

All results are in mg/kg

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Arsenic	D_Arsenic	Iron	D_Iron	Manganese	D_Manganese	Thallium	D_Thallium	Methylnaphthalene, 2-
IT-MNTA-BEDGW-001	BEDGW-001-1109	10	0	180	1	85.8	1	10	0	256
LBA-BEDGW-002	LB3073	131	1	2940	1	1070	1	6.4	1	20.9
IT-MNTA-BEDGW-001	LB3074	10	0	591	1	95.5	1	3.8	1	71
RCA-BEDGW-002	RC3078	19.9	1	300	0	227	1	10	0	6.3
RCA-BEDGW-002	RC3084	28	1	385	1	158	1	10	0	8.1

All results are in µg/L

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	D_Methylnaphthalene, 2-	Naphthalene	D_Naphthalene	Benzene	D_Benzene	Chloroform	D_Chloroform	Chloromethane
IT-MNTA-BEDGW-001	BEDGW-001-1109	1	64.9	1	3.7	1	1	0	2
LBA-BEDGW-002	LB3073	1	25.6	1	1680	1	26.8	1	35.6
IT-MNTA-BEDGW-001	LB3074	1	17.7	1	20	0	66.7	1	40
RCA-BEDGW-002	RC3078	1	4.5	1	31.9	1	1	0	12.8
RCA-BEDGW-002	RC3084	1	7.2	1	114	1	1	0	2

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Chloromethane	D_Chloromethane	Ethylbenzene	D_Ethylbenzene	Tetrachloroethane, 1,1,2,2-	D_Tetrachloroethane, 1,1,2,2-	Toluene	D_Toluene
IT-MNTA-BEDGW-001	BEDGW-001-1109	0		9.9	1	1.2	1	10	1
LBA-BEDGW-002	LB3073	1		169	1	20	0	692	1
IT-MNTA-BEDGW-001	LB3074	0		16.7	1	20	0	14.2	1
RCA-BEDGW-002	RC3078	1		17	1	3.7	1	28.9	1
RCA-BEDGW-002	RC3084	0		43.4	1	1	0	93.6	1

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Xylenes, total	D_Xylenes, total	Cyanide, total	D_Cyanide, total	Methylene chloride
IT-MNTA-BEDGW-001	BEDGW-001-1109	136	1	0.01	0	2.5
LBA-BEDGW-002	LB3073	1130	1	0.014	1	50
IT-MNTA-BEDGW-001	LB3074	235	1	0.071	1	77.6
RCA-BEDGW-002	RC3078	97.2	1	0.11	1	2.5
RCA-BEDGW-002	RC3084	234	1	0.01	0	2.5

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Chromium	D_Chromium	Cobalt	D_Cobalt	Iron	D_Iron	Dinitrotoluene, 2,4-exp	D_Dinitrotoluene, 2,4-exp
LBA-MW01	LB3067	10	0	50	0	242	1	0.19	0
LBA-MW02	LB3068LB3069	10	0	50	0	300	0	8.1	1
LBA-MW03	LB3071	10	0	50	0	300	0	0.19	0
LBA-MW01	LB3075	10	0	50	0	684	1	0.2	0
LBA-MW02	LB3076LB3077	10	0	50	0	203.5	1	10.6	1
LBA-MW03	LB3080	10	0	50	0	300	0	0.19	0
RCA-MW02	RC3075	10	0	6.1	1	300	0	0.23	1
RCA-MW03	RC3076	10	0	50	0	300	0	0.19	0
RCA-MW02	RC3081	2.8	1	3.7	1	2830	1	0.2	0
RCA-MW03	RC3082	3.3	1	2.3	1	2510	1	0.2	0

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Dinitrotoluene, 2,6-exp	D_Dinitrotoluene, 2,6-exp	Nitrotoluene, 2-	D_Nitrotoluene, 2-	Nitrotoluene, 3-	D_Nitrotoluene, 3-
LBA-MW01	LB3067	0.19	0	0.19	0	0.19	0
LBA-MW02	LB3068LB3069	6.55	1	36.8	1	4.3	1
LBA-MW03	LB3071	0.19	0	0.19	0	0.19	0
LBA-MW01	LB3075	0.2	0	0.2	0	0.2	0
LBA-MW02	LB3076LB3077	9.15	1	41.4	1	5.15	1
LBA-MW03	LB3080	0.19	0	0.19	0	0.19	0
RCA-MW02	RC3075	0.29	1	2.1	1	0.19	0
RCA-MW03	RC3076	0.19	0	0.19	0	0.19	0
RCA-MW02	RC3081	0.2	0	1.8	1	0.2	0
RCA-MW03	RC3082	0.2	0	0.2	0	0.2	0

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Nitrotoluene, 4-	Nitrotoluene, 4-	Dichloroethane, 1,1-	D_Dichloroethane, 1,1-	Trichloroethene	D_Trichloroethene	Carbon tetrachloride
LBA-MW01	LB3067	0.19	0	2.2	1	1	0	0.5
LBA-MW02	LB3068LB3069	4.35	1	1.55	1	5.4	1	0.5
LBA-MW03	LB3071	0.19	0	6.3	1	1	0	0.5
LBA-MW01	LB3075	0.2	0	2	1	1	0	0.5
LBA-MW02	LB3076LB3077	4.58	1	3.35	1	4.55	1	0.5
LBA-MW03	LB3080	0.19	0	8.3	1	1	0	4
RCA-MW02	RC3075	1.2	1	1	0	1	0	0.5
RCA-MW03	RC3076	0.19	0	1	0	1	0	0.5
RCA-MW02	RC3081	0.61	1	1	0	1	0	0.5
RCA-MW03	RC3082	0.2	0	1	0	1	0	0.5

All results are in µg/L

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Dibromochloromethane
LBA-MW01	LB3067	0.5
LBA-MW02	LB3068LB3069	0.5
LBA-MW03	LB3071	0.5
LBA-MW01	LB3075	0.5
LBA-MW02	LB3076LB3077	0.5
LBA-MW03	LB3080	0.5
RCA-MW02	RC3075	0.5
RCA-MW03	RC3076	0.2
RCA-MW02	RC3081	0.5
RCA-MW03	RC3082	0.5

All results are in µg/L

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Arsenic	D_Arsenic	Chromium	D_Chromium	Cobalt	D_Cobalt	Manganese	D_Mangane	Thallium	D_Thallium
LBA-SD01	LBA1000	16	1	10.4	1	24.9	1	5360	1	1.9	1
LBA-SD02	LBA1001	9.4	1	10.7	1	17.5	1	1510	1	0.26	1
LBA-SD03	LBA1002	8.3	1	13.5	1	11.8	1	828	1	0.63	0
LBA-SD04	LBA1003	17.8	1	13.4	1	26	1	1610	1	0.67	0
LBA-SD05	LBA1004	9.5	1	15.9	1	10.8	1	737	1	0.66	0
LBA-SD06	LBA1005LBA1006	11.05	1	18.2	1	11.5	1	974.5	1	0.66	0

All results are in mg/kg

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

Location	Sample Number	Arsenic	Chromium	D_Chromium	Cobalt	D_Cobalt	Iron	D_Iron	Manganese	D_Manganese
LBA-SW01	LBA2000	5	2.6	1	50	0	2570	1	554	1
LBA-SW02	LBA2001	5	2.8	1	4.2	1	4020	1	1760	1
LBA-SW03	LBA2002LBA2003	5	3.05	1	2.35	1	4110	1	1715	1
LBA-SW04	LBA2005	5	4.1	1	2.5	1	4920	1	1050	1
LBA-SW05	LBA2006	5	4.6	1	3	1	5890	1	2220	1
LBA-SW06	LBA2007	8.8	39.8	1	16.3	1	43900	1	662	1
LBA-SW07	LBA2008	5	10	0	50	0	1090	1	381	1

All results are in µg/L

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Chromium

General Statistics

Number of Valid Observations	11	Number of Distinct Observations	10

Raw Statistics		**Log-transformed Statistics**	
Minimum	10.4	Minimum of Log Data	2.342
Maximum	51	Maximum of Log Data	3.932
Mean	15.32	Mean of log Data	2.593
Median	11.7	SD of log Data	0.454
SD	11.89		
Coefficient of Variation	0.776		
Skewness	3.26		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.427	Shapiro Wilk Test Statistic	0.526
Shapiro Wilk Critical Value	0.85	Shapiro Wilk Critical Value	0.85
Data not Normal at 5% Significance Level		**Data not Lognormal at 5% Significance Level**	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	21.82	95% H-UCL	20.09
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	23.58
95% Adjusted-CLT UCL (Chen-1995)	24.98	97.5% Chebyshev (MVUE) UCL	27.44
95% Modified-t UCL (Johnson-1978)	22.4	99% Chebyshev (MVUE) UCL	35.01

Gamma Distribution Test		**Data Distribution**
k star (bias corrected)	2.851	**Data do not follow a Discernable Distribution (0.05)**
Theta Star	5.372	
MLE of Mean	15.32	
MLE of Standard Deviation	9.072	
nu star	62.73	
Approximate Chi Square Value (.05)	45.51	**Nonparametric Statistics**
Adjusted Level of Significance	0.0278	95% CLT UCL 21.22
Adjusted Chi Square Value	43.13	95% Jackknife UCL 21.82
		95% Standard Bootstrap UCL 20.99
Anderson-Darling Test Statistic	2.586	95% Bootstrap-t UCL 88.37
Anderson-Darling 5% Critical Value	0.733	95% Hall's Bootstrap UCL 63.16
Kolmogorov-Smirnov Test Statistic	0.408	95% Percentile Bootstrap UCL 22.3
Kolmogorov-Smirnov 5% Critical Value	0.257	95% BCA Bootstrap UCL 25.84
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 30.95
		97.5% Chebyshev(Mean, Sd) UCL 37.71
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 50.99
95% Approximate Gamma UCL	21.11	
95% Adjusted Gamma UCL	22.28	

Potential UCL to Use	Use 95% Student's-t UCL 21.82
	or 95% Modified-t UCL 22.4

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

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

General Statistics

Number of Valid Data	11	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	8
		Percent Non-Detects	72.73%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.0426	Minimum Detected	-3.156
Maximum Detected	0.652	Maximum Detected	-0.428
Mean of Detected	0.282	Mean of Detected	-1.826
SD of Detected	0.325	SD of Detected	1.365
Minimum Non-Detect	0.175	Minimum Non-Detect	-1.743
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	11
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	0
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	100.00%

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Shapiro Wilk Test Statistic	0.878	Shapiro Wilk Test Statistic	0.998
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		**Data appear Lognormal at 5% Significance Level**	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.295	Mean	-1.561
SD	0.289	SD	0.852
95% DL/2 (t) UCL	0.453	95% H-Stat (DL/2) UCL	0.628
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-2.336
		SD in Log Scale	0.694
		Mean in Original Scale	0.135
		SD in Original Scale	0.173
		95% t UCL	0.23
		95% Percentile Bootstrap UCL	0.235
		95% BCA Bootstrap UCL	0.295
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		

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ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	0.152
SD	0.174
SE of Mean	0.077
95% KM (t) UCL	0.292
95% KM (z) UCL	0.279
95% KM (jackknife) UCL	0.307
95% KM (bootstrap t) UCL	0.388
95% KM (BCA) UCL	0.652
95% KM (Percentile Bootstrap) UCL	0.652
95% KM (Chebyshev) UCL	0.488
97.5% KM (Chebyshev) UCL	0.633
99% KM (Chebyshev) UCL	0.918

Potential UCLs to Use

95% KM (t) UCL	0.292
95% KM (Percentile Bootstrap) UCL	0.652

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.

Benzo(a)pyrene

General Statistics

Number of Valid Data	11	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	9
		Percent Non-Detects	81.82%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.121	Minimum Detected	-2.116
Maximum Detected	0.395	Maximum Detected	-0.929
Mean of Detected	0.258	Mean of Detected	-1.522
SD of Detected	0.194	SD of Detected	0.84
Minimum Non-Detect	0.175	Minimum Non-Detect	-1.743
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	11
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	0
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	100.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal 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	0.282	Mean	-1.491
SD	0.26	SD	0.631
95% DL/2 (t) UCL	0.424	95% H-Stat (DL/2) UCL	0.44
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
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	0.189
5% K-S Critical Value	N/A	SD	0.119
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.084
		95% KM (t) UCL	0.341
Assuming Gamma Distribution		95% KM (z) UCL	0.327
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	0.39
Minimum	N/A	95% KM (bootstrap t) UCL	N/A
Maximum	N/A	95% KM (BCA) UCL	0.395
Mean	N/A	95% KM (Percentile Bootstrap) UCL	0.395
Median	N/A	95% KM (Chebyshev) UCL	0.555
SD	N/A	97.5% KM (Chebyshev) UCL	0.714
k star	N/A	99% KM (Chebyshev) UCL	1.025
Theta star	N/A		
Nu star	N/A	**Potential UCLs to Use**	
AppChi2	N/A	95% KM (t) UCL	0.341
95% Gamma Approximate UCL	N/A	95% KM (% Bootstrap) UCL	0.395
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

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

Dinitrotoluene, 2,4-

General Statistics

Number of Valid Data	11	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	8
		Percent Non-Detects	72.73%

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.268	Minimum Detected	-1.317
Maximum Detected	4	Maximum Detected	1.386
Mean of Detected	1.853	Mean of Detected	0.108
SD of Detected	1.929	SD of Detected	1.357
Minimum Non-Detect	0.175	Minimum Non-Detect	-1.743
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

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

Number treated as Non-Detect	10
Number treated as Detected	1
Single DL Non-Detect Percentage	90.91%

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.936	Shapiro Wilk Test Statistic	0.991
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		**Data appear Lognormal at 5% Significance Level**	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.718	Mean	-1.055
SD	1.157	SD	1.12
95% DL/2 (t) UCL	1.35	95% H-Stat (DL/2) UCL	2.047
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-1.709
		SD in Log Scale	1.361
		Mean in Original Scale	0.576
		SD in Original Scale	1.19
		95% t UCL	1.226
		95% Percentile Bootstrap UCL	1.177
		95% BCA Bootstrap UCL	1.663

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 0.711
5% K-S Critical Value	N/A	SD 1.084
Data not Gamma Distributed at 5% Significance Level		SE of Mean 0.402
		95% KM (t) UCL 1.439

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Gamma Distribution		95% KM (z) UCL	1.372
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	1.518
Minimum	N/A	95% KM (bootstrap t) UCL	1.463
Maximum	N/A	95% KM (BCA) UCL	4
Mean	N/A	95% KM (Percentile Bootstrap) UCL	N/A
Median	N/A	95% KM (Chebyshev) UCL	2.464
SD	N/A	97.5% KM (Chebyshev) UCL	3.222
k star	N/A	99% KM (Chebyshev) UCL	4.712
Theta star	N/A		
Nu star	N/A	**Potential UCLs to Use**	
AppChi2	N/A	95% KM (t) UCL	1.439
95% Gamma Approximate UCL	N/A	95% KM (Percentile Bootstrap) UCL	N/A
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

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

General UCL Statistics for Full Data Sets

User Selected Options	
From File	ss UCL input.wst
Full Precision	OFF
Confidence Coefficient	95%
Number of Bootstrap Operations	2000

Aroclor 1221

General Statistics

Number of Valid Observations	11	Number of Distinct Observations	11

Raw Statistics		**Log-transformed Statistics**	
Minimum	0.0175	Minimum of Log Data	-4.046
Maximum	0.19	Maximum of Log Data	-1.661
Mean	0.0354	Mean of log Data	-3.712
Median	0.0203	SD of log Data	0.682
SD	0.0513		
Coefficient of Variation	1.449		
Skewness	3.314		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.364	Shapiro Wilk Test Statistic	0.417
Shapiro Wilk Critical Value	0.85	Shapiro Wilk Critical Value	0.85
Data not Normal at 5% Significance Level		**Data not Lognormal at 5% Significance Level**	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	0.0634	95% H-UCL	0.0522
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	0.058
95% Adjusted-CLT UCL (Chen-1995)	0.0773	97.5% Chebyshev (MVUE) UCL	0.0701
95% Modified-t UCL (Johnson-1978)	0.066	99% Chebyshev (MVUE) UCL	0.0938

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test

k star (bias corrected)	1.147
Theta Star	0.0308
MLE of Mean	0.0354
MLE of Standard Deviation	0.033
nu star	25.24
Approximate Chi Square Value (.05)	14.8
Adjusted Level of Significance	0.0278
Adjusted Chi Square Value	13.51
Anderson-Darling Test Statistic	3.381
Anderson-Darling 5% Critical Value	0.742
Kolmogorov-Smirnov Test Statistic	0.52
Kolmogorov-Smirnov 5% Critical Value	0.26

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	0.0604
95% Adjusted Gamma UCL	0.0661

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	0.0608
95% Jackknife UCL	0.0634
95% Standard Bootstrap UCL	0.0599
95% Bootstrap-t UCL	1.21
95% Hall's Bootstrap UCL	0.666
95% Percentile Bootstrap UCL	0.0662
95% BCA Bootstrap UCL	0.0817
95% Chebyshev(Mean, Sd) UCL	0.103
97.5% Chebyshev(Mean, Sd) UCL	0.132
99% Chebyshev(Mean, Sd) UCL	0.189

Potential UCL to Use — Use 95% Chebyshev (Mean, Sd) UCL 0.103

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)flouranthene

General Statistics

Number of Valid Observations	11	Number of Distinct Observations	11

Raw Statistics

Minimum	0.0875
Maximum	1.03
Mean	0.332
Median	0.208
SD	0.312
Coefficient of Variation	0.938
Skewness	1.903

Log-transformed Statistics

Minimum of Log Data	-2.436
Maximum of Log Data	0.0296
Mean of log Data	-1.376
SD of log Data	0.708

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.611
Shapiro Wilk Critical Value	0.85

Data not Normal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	0.503

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	0.544
95% Modified-t UCL (Johnson-1978)	0.512

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.763
Shapiro Wilk Critical Value	0.85

Data not Lognormal at 5% Significance Level

Assuming Lognormal Distribution

95% H-UCL	0.565
95% Chebyshev (MVUE) UCL	0.621
97.5% Chebyshev (MVUE) UCL	0.753
99% Chebyshev (MVUE) UCL	1.012

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test

k star (bias corrected)	1.494
Theta Star	0.222
MLE of Mean	0.332
MLE of Standard Deviation	0.272
nu star	32.87
Approximate Chi Square Value (.05)	20.76
Adjusted Level of Significance	0.0278
Adjusted Chi Square Value	19.21
Anderson-Darling Test Statistic	1.761
Anderson-Darling 5% Critical Value	0.739
Kolmogorov-Smirnov Test Statistic	0.388
Kolmogorov-Smirnov 5% Critical Value	0.259

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	0.526
95% Adjusted Gamma UCL	0.569

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	0.487
95% Jackknife UCL	0.503
95% Standard Bootstrap UCL	0.479
95% Bootstrap-t UCL	1.429
95% Hall's Bootstrap UCL	2.277
95% Percentile Bootstrap UCL	0.489
95% BCA Bootstrap UCL	0.545
95% Chebyshev(Mean, Sd) UCL	0.742
97.5% Chebyshev(Mean, Sd) UCL	0.919
99% Chebyshev(Mean, Sd) UCL	1.267

Potential UCL to Use Use 95% Chebyshev (Mean, Sd) UCL 0.742

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 B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Aluminum

General Statistics

Number of Valid Observations 37

Number of Distinct Observations 36

Raw Statistics

Minimum	4400
Maximum	17200
Mean	8449
Median	7890
SD	2375
Coefficient of Variation	0.281
Skewness	1.435

Log-transformed Statistics

Minimum of Log Data	8.389
Maximum of Log Data	9.753
Mean of log Data	9.007
SD of log Data	0.262

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.914
Shapiro Wilk Critical Value	0.936

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.987
Shapiro Wilk Critical Value	0.936

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 9108

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	9189
95% Modified-t UCL (Johnson-1978)	9123

Assuming Lognormal Distribution

95% H-UCL	9125
95% Chebyshev (MVUE) UCL	10046
97.5% Chebyshev (MVUE) UCL	10741
99% Chebyshev (MVUE) UCL	12107

Gamma Distribution Test

k star (bias corrected)	13.49
Theta Star	626.1
MLE of Mean	8449
MLE of Standard Deviation	2300
nu star	998.6
Approximate Chi Square Value (.05)	926.3
Adjusted Level of Significance	0.0431
Adjusted Chi Square Value	923.3
Anderson-Darling Test Statistic	0.336
Anderson-Darling 5% Critical Value	0.747
Kolmogorov-Smirnov Test Statistic	0.0844
Kolmogorov-Smirnov 5% Critical Value	0.145

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 9109

95% Adjusted Gamma UCL 9139

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	9091
95% Jackknife UCL	9108
95% Standard Bootstrap UCL	9080
95% Bootstrap-t UCL	9236
95% Hall's Bootstrap UCL	9322
95% Percentile Bootstrap UCL	9122
95% BCA Bootstrap UCL	9198
95% Chebyshev(Mean, Sd) UCL	10151
97.5% Chebyshev(Mean, Sd) UCL	10887
99% Chebyshev(Mean, Sd) UCL	12333

Potential UCL to Use

Use 95% Approximate Gamma UCL 9109

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 B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Chromium

General Statistics

Number of Valid Observations 37

Number of Distinct Observations 32

Raw Statistics

Minimum 10.4
Maximum 51
Mean 15.56
Median 13.6
SD 7.133
Coefficient of Variation 0.459
Skewness 3.733

Log-transformed Statistics

Minimum of Log Data 2.342
Maximum of Log Data 3.932
Mean of log Data 2.684
SD of log Data 0.317

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic 0.615
Shapiro Wilk Critical Value 0.936
Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.831
Shapiro Wilk Critical Value 0.936
Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 17.54

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 18.25
95% Modified-t UCL (Johnson-1978) 17.66

Assuming Lognormal Distribution

95% H-UCL 16.93
95% Chebyshev (MVUE) UCL 18.94
97.5% Chebyshev (MVUE) UCL 20.48
99% Chebyshev (MVUE) UCL 23.51

Gamma Distribution Test

k star (bias corrected) 7.718
Theta Star 2.015
MLE of Mean 15.56
MLE of Standard Deviation 5.599
nu star 571.1
Approximate Chi Square Value (.05) 516.7
Adjusted Level of Significance 0.0431
Adjusted Chi Square Value 514.5

Anderson-Darling Test Statistic 2.092
Anderson-Darling 5% Critical Value 0.749
Kolmogorov-Smirnov Test Statistic 0.165
Kolmogorov-Smirnov 5% Critical Value 0.145
Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 17.19
95% Adjusted Gamma UCL 17.27

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL 17.48
95% Jackknife UCL 17.54
95% Standard Bootstrap UCL 17.46
95% Bootstrap-t UCL 19.42
95% Hall's Bootstrap UCL 26.24
95% Percentile Bootstrap UCL 17.68
95% BCA Bootstrap UCL 18.46
95% Chebyshev(Mean, Sd) UCL 20.67
97.5% Chebyshev(Mean, Sd) UCL 22.88
99% Chebyshev(Mean, Sd) UCL 27.22

Potential UCL to Use

Use 95% Student's-t UCL 17.54
or 95% Modified-t UCL 17.66

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 B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Vanadium

General Statistics

Number of Valid Observations 37

Number of Distinct Observations 33

Raw Statistics

Minimum 18.3
Maximum 49.9
Mean 25.06
Median 24.3
SD 5.665
Coefficient of Variation 0.226
Skewness 2.491

Log-transformed Statistics

Minimum of Log Data 2.907
Maximum of Log Data 3.91
Mean of log Data 3.201
SD of log Data 0.194

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic 0.799
Shapiro Wilk Critical Value 0.936
Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.916
Shapiro Wilk Critical Value 0.936
Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 26.63

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 27
95% Modified-t UCL (Johnson-1978) 26.69

Assuming Lognormal Distribution

95% H-UCL 26.47
95% Chebyshev (MVUE) UCL 28.52
97.5% Chebyshev (MVUE) UCL 30.04
99% Chebyshev (MVUE) UCL 33.02

Gamma Distribution Test

k star (bias corrected) 23.09
Theta Star 1.085
MLE of Mean 25.06
MLE of Standard Deviation 5.215
nu star 1708
Approximate Chi Square Value (.05) 1613
Adjusted Level of Significance 0.0431
Adjusted Chi Square Value 1609

Anderson-Darling Test Statistic 0.851
Anderson-Darling 5% Critical Value 0.746
Kolmogorov-Smirnov Test Statistic 0.143
Kolmogorov-Smirnov 5% Critical Value 0.145
Data follow Appr. Gamma Distribution at 5% Significance Level

Data Distribution

Data Follow Appr. Gamma Distribution at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 26.59
95% Jackknife UCL 26.63
95% Standard Bootstrap UCL 26.63
95% Bootstrap-t UCL 27.35
95% Hall's Bootstrap UCL 29.13
95% Percentile Bootstrap UCL 26.62
95% BCA Bootstrap UCL 27.15
95% Chebyshev(Mean, Sd) UCL 29.12
97.5% Chebyshev(Mean, Sd) UCL 30.87
99% Chebyshev(Mean, Sd) UCL 34.33

Assuming Gamma Distribution

95% Approximate Gamma UCL 26.53
95% Adjusted Gamma UCL 26.6

Potential UCL to Use

Use 95% Approximate Gamma UCL 26.53

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 B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Nitrotoluene, 2-

General Statistics

Number of Valid Data	27	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	25
		Percent Non-Detects	92.59%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	2.34	Minimum Detected	0.85
Maximum Detected	22.7	Maximum Detected	3.122
Mean of Detected	12.52	Mean of Detected	1.986
SD of Detected	14.4	SD of Detected	1.607
Minimum Non-Detect	0.288	Minimum Non-Detect	-1.245
Maximum Non-Detect	0.3	Maximum Non-Detect	-1.204

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	25
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	2
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	92.59%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	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	1.064	Mean	-1.624
SD	4.344	SD	1.087
95% DL/2 (t) UCL	2.49	95% H-Stat (DL/2) UCL	0.51
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% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap 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	

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

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	3.094
5% K-S Critical Value	N/A	SD	3.845
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.046
		95% KM (t) UCL	4.879
Assuming Gamma Distribution		95% KM (z) UCL	4.815
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	16.47
Minimum	N/A	95% KM (bootstrap t) UCL	N/A
Maximum	N/A	95% KM (BCA) UCL	22.7
Mean	N/A	95% KM (Percentile Bootstrap) UCL	22.7
Median	N/A	95% KM (Chebyshev) UCL	7.656
SD	N/A	97.5% KM (Chebyshev) UCL	9.629
k star	N/A	99% KM (Chebyshev) UCL	13.51
Theta star	N/A		
Nu star	N/A	Potential UCLs to Use	
AppChi2	N/A	97.5% KM (Chebyshev) UCL	9.629
95% Gamma Approximate UCL	N/A		
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

Nitrotoluene, 3-

General Statistics

Number of Valid Data	27	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	25
		Percent Non-Detects	92.59%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.308	Minimum Detected	-1.178
Maximum Detected	1.52	Maximum Detected	0.419
Mean of Detected	0.914	Mean of Detected	-0.379
SD of Detected	0.857	SD of Detected	1.129
Minimum Non-Detect	0.288	Minimum Non-Detect	-1.245
Maximum Non-Detect	0.3	Maximum Non-Detect	-1.204

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	25
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	2
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	92.59%

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.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

UCL Statistics

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

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

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	0.204
SD	0.265
95% DL/2 (t) UCL	0.291
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	-1.799
SD	0.465
95% H-Stat (DL/2) UCL	0.201
Log ROS Method	
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% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	0.353
SD	0.229
SE of Mean	0.0623
95% KM (t) UCL	0.459
95% KM (z) UCL	0.455
95% KM (jackknife) UCL	1.149
95% KM (bootstrap t) UCL	0.353
95% KM (BCA) UCL	1.52
95% KM (Percentile Bootstrap) UCL	N/A
95% KM (Chebyshev) UCL	0.624
97.5% KM (Chebyshev) UCL	0.742
99% KM (Chebyshev) UCL	0.973
Potential UCLs to Use	
95% KM (BCA) UCL	1.52

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Benzo(a)anthracene

General Statistics

Number of Valid Data	34	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	31
		Percent Non-Detects	91.18%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.0426	Minimum Detected	-3.156
Maximum Detected	0.652	Maximum Detected	-0.428
Mean of Detected	0.282	Mean of Detected	-1.826
SD of Detected	0.325	SD of Detected	1.365
Minimum Non-Detect	0.35	Minimum Non-Detect	-1.05
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	34
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	0
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	100.00%

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.878	Shapiro Wilk Test Statistic	0.998
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.243	Mean	-1.54
SD	0.169	SD	0.47
95% DL/2 (t) UCL	0.292	95% H-Stat (DL/2) UCL	0.28
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-2.495
		SD in Log Scale	0.448
		Mean in Original Scale	0.0969
		SD in Original Scale	0.1
		95% t UCL	0.126
		95% Percentile Bootstrap UCL	0.131
		95% BCA Bootstrap UCL	0.147

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 0.114
5% K-S Critical Value	N/A	SD 0.11

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

SE of Mean	0.0498
95% KM (t) UCL	0.198
95% KM (z) UCL	0.196
95% KM (jackknife) UCL	0.24
95% KM (bootstrap t) UCL	0.224
95% KM (BCA) UCL	0.652
95% KM (Percentile Bootstrap) UCL	0.652
95% KM (Chebyshev) UCL	0.331
97.5% KM (Chebyshev) UCL	0.425
99% KM (Chebyshev) UCL	0.609

Potential UCLs to Use

95% KM (t) UCL	0.198
95% KM (Percentile Bootstrap) UCL	0.652

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.

Benzo(a)pyrene

General Statistics

Number of Valid Data	34	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	32
		Percent Non-Detects	94.12%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.121	Minimum Detected	-2.116
Maximum Detected	0.395	Maximum Detected	-0.929
Mean of Detected	0.258	Mean of Detected	-1.522
SD of Detected	0.194	SD of Detected	0.84
Minimum Non-Detect	0.35	Minimum Non-Detect	-1.05
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

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

Number treated as Non-Detect	34
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

UCL Statistics

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

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

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	0.239
SD	0.152
95% DL/2 (t) UCL	0.283
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	-1.517
SD	0.348
95% H-Stat (DL/2) UCL	0.26
Log ROS Method	
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

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	

Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	0.145
SD	0.0789
SE of Mean	0.0336
95% KM (t) UCL	0.202
95% KM (z) UCL	0.201
95% KM (jackknife) UCL	0.318
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	N/A
95% KM (Percentile Bootstrap) UCL	0.395
95% KM (Chebyshev) UCL	0.292
97.5% KM (Chebyshev) UCL	0.356
99% KM (Chebyshev) UCL	0.48

Potential UCLs to Use	
95% KM (t) UCL	0.202
95% KM (% Bootstrap) UCL	0.395

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Dinitrotoluene, 2,4- svoc

General Statistics

Number of Valid Data	34	Number of Detected Data	11
Number of Distinct Detected Data	11	Number of Non-Detect Data	23
		Percent Non-Detects	67.65%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.23	Minimum Detected	-1.47
Maximum Detected	9.88	Maximum Detected	2.291
Mean of Detected	2.297	Mean of Detected	0.0908
SD of Detected	3.075	SD of Detected	1.264
Minimum Non-Detect	0.35	Minimum Non-Detect	-1.05
Maximum Non-Detect	2.06	Maximum Non-Detect	0.723

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	31
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	3
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	91.18%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.713	Shapiro Wilk Test Statistic	0.928
5% Shapiro Wilk Critical Value	0.85	5% Shapiro Wilk Critical Value	0.85
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.905	Mean	-0.999
SD	1.96	SD	1.071
95% DL/2 (t) UCL	1.474	95% H-Stat (DL/2) UCL	1.049
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-0.974
		SD in Log Scale	1.036
		Mean in Original Scale	0.9
		SD in Original Scale	1.957
		95% t UCL	1.468
		95% Percentile Bootstrap UCL	1.503
		95% BCA Bootstrap UCL	1.698

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	0.643	Data appear Gamma Distributed at 5% Significance Level
Theta Star	3.571	
nu star	14.15	
A-D Test Statistic	0.631	**Nonparametric Statistics**
5% A-D Critical Value	0.76	Kaplan-Meier (KM) Method
K-S Test Statistic	0.76	Mean 0.932
5% K-S Critical Value	0.264	SD 1.918
Data appear Gamma Distributed at 5% Significance Level		SE of Mean 0.345
		95% KM (t) UCL 1.516
Assuming Gamma Distribution		95% KM (z) UCL 1.5
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL 1.498
Minimum	0.23	95% KM (bootstrap t) UCL 2.389
Maximum	9.88	95% KM (BCA) UCL 1.563
Mean	2.282	95% KM (Percentile Bootstrap) UCL 1.57
Median	2.267	95% KM (Chebyshev) UCL 2.438
SD	1.699	97.5% KM (Chebyshev) UCL 3.089

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

k star	2.046	99% KM (Chebyshev) UCL	4.369
Theta star	1.115		
Nu star	139.2	Potential UCLs to Use	
AppChi2	112.9	95% KM (t) UCL	1.516
95% Gamma Approximate UCL	2.812		
95% Adjusted Gamma UCL	2.842		

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.

General UCL Statistics for Full Data Sets

User Selected Options

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

Dinitrotoluene, 2,6-exp

General Statistics

Number of Valid Observations 27 | Number of Distinct Observations 12

Raw Statistics		Log-transformed Statistics	
Minimum	0.576	Minimum of Log Data	-0.552
Maximum	4.12	Maximum of Log Data	1.416
Mean	0.731	Mean of log Data	-0.442
Median	0.592	SD of log Data	0.378
SD	0.679		
Coefficient of Variation	0.93		
Skewness	5.155		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.225	Shapiro Wilk Test Statistic	0.269
Shapiro Wilk Critical Value	0.923	Shapiro Wilk Critical Value	0.923
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	0.954	95% H-UCL	0.793
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	0.912
95% Adjusted-CLT UCL	1.084	97.5% Chebyshev (MVUE) UCL	1.009
95% Modified-t UCL	0.975	99% Chebyshev (MVUE) UCL	1.199

Gamma Distribution Test		Data Distribution
k star (bias corrected)	3.645	Data do not follow a Discernable Distribution (0.05)
Theta Star	0.2	
MLE of Mean	0.731	
MLE of Standard Deviation	0.383	
nu star	196.9	

Approximate Chi Square Value (.05) 165.4
Adjusted Level of Significance 0.0401
Adjusted Chi Square Value 163.6

Anderson-Darling Test Statistic 8.903
Anderson-Darling 5% Critical Value 0.749
Kolmogorov-Smirnov Test Statistic 0.512
Kolmogorov-Smirnov 5% Critical Value 0.169
Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution
95% Approximate Gamma UCL 0.87
95% Adjusted Gamma UCL 0.879

Nonparametric Statistics
95% CLT UCL 0.946
95% Jackknife UCL 0.954
95% Standard Bootstrap UCL 0.938
95% Bootstrap-t UCL 16.4
95% Hall's Bootstrap UCL 5.125
95% Percentile Bootstrap UCL 0.991
95% BCA Bootstrap UCL 1.124
95% Chebyshev(Mean, Sd) UCL 1.3
97.5% Chebyshev(Mean, Sd) UCL 1.547
99% Chebyshev(Mean, Sd) UCL 2.031

Potential UCL to Use
Use 95% Student's-t UCL 0.954
or 95% Modified-t UCL 0.975

Aroclor 1221

General Statistics
Number of Valid Observations 25
Number of Missing Values 12
Number of Distinct Observations 21

Raw Statistics
Minimum 0.0175
Maximum 0.19
Mean 0.0269
Median 0.0202
SD 0.034
Coefficient of Variation 1.265
Skewness 4.989

Log-transformed Statistics
Minimum of Log Data -4.046
Maximum of Log Data -1.661
Mean of log Data -3.82
SD of log Data 0.454

Relevant UCL Statistics

Normal Distribution Test
Shapiro Wilk Test Statistic 0.233
Shapiro Wilk Critical Value 0.918
Data not Normal at 5% Significance Level

Assuming Normal Distribution
95% Student's-t UCL 0.0385
95% UCLs (Adjusted for Skewness)
95% Adjusted-CLT UCL (Chen-1995) 0.0453
95% Modified-t UCL (Johnson-1978) 0.0396

Lognormal Distribution Test
Shapiro Wilk Test Statistic 0.311
Shapiro Wilk Critical Value 0.918
Data not Lognormal at 5% Significance Level

Assuming Lognormal Distribution
95% H-UCL 0.0291
95% Chebyshev (MVUE) UCL 0.0341
97.5% Chebyshev (MVUE) UCL 0.0384
99% Chebyshev (MVUE) UCL 0.0468

Gamma Distribution Test
k star (bias corrected) 2.327
Theta Star 0.0116
MLE of Mean 0.0269
MLE of Standard Deviation 0.0176
nu star 116.3
Approximate Chi Square Value (.05) 92.43
Adjusted Level of Significance 0.0395
Adjusted Chi Square Value 90.97

Anderson-Darling Test Statistic 7.817
Anderson-Darling 5% Critical Value 0.753
Kolmogorov-Smirnov Test Statistic 0.477

Data Distribution
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics
95% CLT UCL 0.0381
95% Jackknife UCL 0.0385
95% Standard Bootstrap UCL 0.0379
95% Bootstrap-t UCL 0.312
95% Hall's Bootstrap UCL 0.168
95% Percentile Bootstrap UCL 0.0403

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Kolmogorov-Smirnov 5% Critical Value 0.176
Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution
95% Approximate Gamma UCL 0.0338
95% Adjusted Gamma UCL 0.0344

95% BCA Bootstrap UCL 0.0541
95% Chebyshev(Mean, Sd) UCL 0.0565
97.5% Chebyshev(Mean, Sd) UCL 0.0694
99% Chebyshev(Mean, Sd) UCL 0.0946

Potential UCL to Use

Use 95% Student's-t UCL 0.0385
or 95% Modified-t UCL 0.0396

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)flouranthene

General Statistics

Number of Valid Observations 34
Number of Missing Values 3
Number of Distinct Observations 30

Raw Statistics

Minimum 0.175
Maximum 1.03
Mean 0.255
Median 0.205
SD 0.185
Coefficient of Variation 0.724
Skewness 3.623

Log-transformed Statistics

Minimum of Log Data -1.743
Maximum of Log Data 0.0296
Mean of log Data -1.48
SD of log Data 0.397

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic 0.376
Shapiro Wilk Critical Value 0.933
Data not Normal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 0.309

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995) 0.329
95% Modified-t UCL (Johnson-1978) 0.312

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.46
Shapiro Wilk Critical Value 0.933
Data not Lognormal at 5% Significance Level

Assuming Lognormal Distribution

95% H-UCL 0.28
95% Chebyshev (MVUE) UCL 0.321
97.5% Chebyshev (MVUE) UCL 0.353
99% Chebyshev (MVUE) UCL 0.417

Gamma Distribution Test

k star (bias corrected) 4.129
Theta Star 0.0619
MLE of Mean 0.255
MLE of Standard Deviation 0.126
nu star 280.8
Approximate Chi Square Value (.05) 243
Adjusted Level of Significance 0.0422
Adjusted Chi Square Value 241.2

Anderson-Darling Test Statistic 8.638
Anderson-Darling 5% Critical Value 0.751
Kolmogorov-Smirnov Test Statistic 0.453
Kolmogorov-Smirnov 5% Critical Value 0.151
Data not Gamma Distributed at 5% Significance Level

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL 0.308
95% Jackknife UCL 0.309
95% Standard Bootstrap UCL 0.306
95% Bootstrap-t UCL 0.425
95% Hall's Bootstrap UCL 0.346
95% Percentile Bootstrap UCL 0.311
95% BCA Bootstrap UCL 0.341
95% Chebyshev(Mean, Sd) UCL 0.394
97.5% Chebyshev(Mean, Sd) UCL 0.454

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Total Soil
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Gamma Distribution

95% Approximate Gamma UCL 0.295

95% Adjusted Gamma UCL 0.297

99% Chebyshev(Mean, Sd) UCL 0.571

Potential UCL to Use

Use 95% Student's-t UCL 0.309

or 95% Modified-t UCL 0.312

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 B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Arsenic

General Statistics

Number of Valid Data	5	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	2
		Percent Non-Detects	40.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	19.9	Minimum Detected	2.991
Maximum Detected	131	Maximum Detected	4.875
Mean of Detected	59.63	Mean of Detected	3.733
SD of Detected	61.94	SD of Detected	1.004
Minimum Non-Detect	10	Minimum Non-Detect	2.303
Maximum Non-Detect	10	Maximum Non-Detect	2.303

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.804	Shapiro Wilk Test Statistic	0.881
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	37.78	Mean	2.883
SD	53.04	SD	1.363
95% DL/2 (t) UCL	88.35	95% H-Stat (DL/2) UCL	1942
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	19.6	Mean in Log Scale	2.525
SD	67.41	SD in Log Scale	1.836
95% MLE (t) UCL	83.87	Mean in Original Scale	36.71
95% MLE (Tiku) UCL	91.81	SD in Original Scale	53.89
		95% Percentile Bootstrap UCL	79.53
		95% BCA Bootstrap UCL	84.88

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	

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	43.74
SD	43.74
SE of Mean	23.96
95% KM (t) UCL	94.82
95% KM (z) UCL	83.15
95% KM (jackknife) UCL	89.46
95% KM (bootstrap t) UCL	361.9
95% KM (BCA) UCL	131
95% KM (Percentile Bootstrap) UCL	131
95% KM (Chebyshev) UCL	148.2
97.5% KM (Chebyshev) UCL	193.4
99% KM (Chebyshev) UCL	282.1

Potential UCLs to Use

95% KM (t) UCL	94.82
95% KM (Percentile Bootstrap) UCL	131

Iron

General Statistics

Number of Valid Data	5	Number of Detected Data	4
Number of Distinct Detected Data	4	Number of Non-Detect Data	1
		Percent Non-Detects	20.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	180	Minimum Detected	5.193
Maximum Detected	2940	Maximum Detected	7.986
Mean of Detected	1024	Mean of Detected	6.379
SD of Detected	1288	SD of Detected	1.179
Minimum Non-Detect	300	Minimum Non-Detect	5.704
Maximum Non-Detect	300	Maximum Non-Detect	5.704

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

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.749	Shapiro Wilk Test Statistic	0.951
5% Shapiro Wilk Critical Value	0.748	5% Shapiro Wilk Critical Value	0.748
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	849.2	Mean	6.105
SD	1182	SD	1.19
95% DL/2 (t) UCL	1976	95% H-Stat (DL/2) UCL	14294

Maximum Likelihood Estimate(MLE) Method	
Mean	450.9
SD	1484
95% MLE (t) UCL	1866
95% MLE (Tiku) UCL	2039

Log ROS Method	
Mean in Log Scale	6.088
SD in Log Scale	1.21
Mean in Original Scale	846.8
SD in Original Scale	1184
95% Percentile Bootstrap UCL	1828
95% BCA Bootstrap UCL	1918

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	0.426
Theta Star	2401
nu star	3.412
A-D Test Statistic	0.404
5% A-D Critical Value	0.666
K-S Test Statistic	0.666
5% K-S Critical Value	0.402

Data appear Gamma Distributed at 5% Significance Level

Data Distribution Test with Detected Values Only

Data appear Normal at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	97.22
Maximum	2940
Mean	838.6
Median	385
SD	1190
k star	0.465
Theta star	1805
Nu star	4.645
AppChi2	0.992
95% Gamma Approximate UCL	3927
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	855.2
SD	1053
SE of Mean	544
95% KM (t) UCL	2015
95% KM (z) UCL	1750
95% KM (jackknife) UCL	1968
95% KM (bootstrap t) UCL	6648
95% KM (BCA) UCL	1959
95% KM (Percentile Bootstrap) UCL	1877
95% KM (Chebyshev) UCL	3226
97.5% KM (Chebyshev) UCL	4253
99% KM (Chebyshev) UCL	6268

Potential UCLs to Use

95% KM (t) UCL	2015
95% KM (Percentile Bootstrap) UCL	1877

Manganese

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	85.8
Maximum	1070
Mean	327.3
Median	158
SD	419
Coefficient of Variation	1.28
Skewness	2.137

Log-transformed Statistics

Minimum of Log Data	4.452
Maximum of Log Data	6.975
Mean of log Data	5.295
SD of log Data	1.018

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

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

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

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	0.67
Shapiro Wilk Critical Value	0.762

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.858
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	726.8

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	826.9
95% Modified-t UCL	756.6

Assuming Lognormal Distribution

95% H-UCL	4231
95% Chebyshev (MVUE) UCL	853
97.5% Chebyshev (MVUE) UCL	1095
99% Chebyshev (MVUE) UCL	1570

Gamma Distribution Test

k star (bias corrected)	0.592
Theta Star	553
MLE of Mean	327.3
MLE of Standard Deviation	425.4
nu star	5.918
Approximate Chi Square Value (.05)	1.598
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	0.809
Anderson-Darling Test Statistic	0.616
Anderson-Darling 5% Critical Value	0.69
Kolmogorov-Smirnov Test Statistic	0.319
Kolmogorov-Smirnov 5% Critical Value	0.364

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	635.5
95% Jackknife UCL	726.8
95% Standard Bootstrap UCL	603.4
95% Bootstrap-t UCL	2742
95% Hall's Bootstrap UCL	2710
95% Percentile Bootstrap UCL	692.7
95% BCA Bootstrap UCL	719
95% Chebyshev(Mean, Sd) UCL	1144
97.5% Chebyshev(Mean, Sd) UCL	1498
99% Chebyshev(Mean, Sd) UCL	2192

Assuming Gamma Distribution

95% Approximate Gamma UCL	1212
95% Adjusted Gamma UCL	2395

Potential UCL to Use

Use 95% Approximate Gamma UCL 1212

Recommended UCL exceeds the maximum observation

Thallium

General Statistics

Number of Valid Data	5	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	3
		Percent Non-Detects	60.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	3.8	Minimum Detected	1.335
Maximum Detected	6.4	Maximum Detected	1.856
Mean of Detected	5.1	Mean of Detected	1.596
SD of Detected	1.838	SD of Detected	0.369
Minimum Non-Detect	10	Minimum Non-Detect	2.303
Maximum Non-Detect	10	Maximum Non-Detect	2.303

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.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	N/A
5% Shapiro Wilk Critical Value	N/A
Data not Normal at 5% Significance Level	
Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	5.04
SD	0.921
95% DL/2 (t) UCL	5.918
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	N/A
5% Shapiro Wilk Critical Value	N/A
Data not Lognormal at 5% Significance Level	
Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	1.604
SD	0.184
95% H-Stat (DL/2) UCL	16.38
Log ROS Method	
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% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	5.1
SD	1.3
SE of Mean	1.3
95% KM (t) UCL	7.871
95% KM (z) UCL	7.238
95% KM (jackknife) UCL	N/A
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	N/A
95% KM (Percentile Bootstrap) UCL	N/A
95% KM (Chebyshev) UCL	10.77
97.5% KM (Chebyshev) UCL	13.22
99% KM (Chebyshev) UCL	18.03
Potential UCLs to Use	
95% KM (t) UCL	7.871
95% KM (% Bootstrap) UCL	N/A

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Methylnaphthalene, 2-

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	6.3
Maximum	256
Mean	72.46
Median	20.9
SD	105.9
Coefficient of Variation	1.462
Skewness	1.925

Log-transformed Statistics

Minimum of Log Data	1.841
Maximum of Log Data	5.545
Mean of log Data	3.356
SD of log Data	1.55

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

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.731
Shapiro Wilk Critical Value	0.762

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.928
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	173.4

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	193.9
95% Modified-t UCL	180.2

Assuming Lognormal Distribution

95% H-UCL	28259
95% Chebyshev (MVUE) UCL	246.4
97.5% Chebyshev (MVUE) UCL	324.2
99% Chebyshev (MVUE) UCL	477.1

Gamma Distribution Test

k star (bias corrected)	0.396
Theta Star	182.8
MLE of Mean	72.46
MLE of Standard Deviation	115.1
nu star	3.964
Approximate Chi Square Value (.05)	0.708
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	0.29

Anderson-Darling Test Statistic	0.385
Anderson-Darling 5% Critical Value	0.703
Kolmogorov-Smirnov Test Statistic	0.255
Kolmogorov-Smirnov 5% Critical Value	0.368

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	150.4
95% Jackknife UCL	173.4
95% Standard Bootstrap UCL	144.4
95% Bootstrap-t UCL	896.8
95% Hall's Bootstrap UCL	815
95% Percentile Bootstrap UCL	156.8
95% BCA Bootstrap UCL	172

Data appear Gamma Distributed at 5% Significance Level

95% Chebyshev(Mean, Sd) UCL 278.9
97.5% Chebyshev(Mean, Sd) UCL 368.2
99% Chebyshev(Mean, Sd) UCL 543.7

Assuming Gamma Distribution
95% Approximate Gamma UCL 406
95% Adjusted Gamma UCL 989.9

Potential UCL to Use

Use 95% Approximate Gamma UCL 406

Recommended UCL exceeds the maximum observation

Naphthalene

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	4.5
Maximum	64.9
Mean	23.98
Median	17.7
SD	24.38
Coefficient of Variation	1.017
Skewness	1.615

Log-transformed Statistics

Minimum of Log Data	1.504
Maximum of Log Data	4.173
Mean of log Data	2.753
SD of log Data	1.053

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

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.836
Shapiro Wilk Critical Value	0.762

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.975
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution
95% Student's-t UCL 47.22

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	50.33
95% Modified-t UCL	48.54

Assuming Lognormal Distribution

95% H-UCL	408.3
95% Chebyshev (MVUE) UCL	70.31
97.5% Chebyshev (MVUE) UCL	90.44
99% Chebyshev (MVUE) UCL	130

Gamma Distribution Test

k star (bias corrected)	0.662
Theta Star	36.22
MLE of Mean	23.98
MLE of Standard Deviation	29.47
nu star	6.62

Data Distribution

Data appear Normal at 5% Significance Level

Approximate Chi Square Value (.05)	1.964	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0086	95% CLT UCL	41.91
Adjusted Chi Square Value	1.048	95% Jackknife UCL	47.22
		95% Standard Bootstrap UCL	39.61
Anderson-Darling Test Statistic	0.244	95% Bootstrap-t UCL	74.43
Anderson-Darling 5% Critical Value	0.688	95% Hall's Bootstrap UCL	118.3
Kolmogorov-Smirnov Test Statistic	0.2	95% Percentile Bootstrap UCL	41.32
Kolmogorov-Smirnov 5% Critical Value	0.363	95% BCA Bootstrap UCL	46.02
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	71.51
		97.5% Chebyshev(Mean, Sd) UCL	92.07
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	132.5
95% Approximate Gamma UCL	80.81		
95% Adjusted Gamma UCL	151.4		
Potential UCL to Use		Use 95% Student's-t UCL	47.22

Benzene

General Statistics

Number of Valid Data	5	Number of Detected Data	4
Number of Distinct Detected Data	4	Number of Non-Detect Data	1
		Percent Non-Detects	20.00%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	3.7	Minimum Detected	1.308
Maximum Detected	1680	Maximum Detected	7.427
Mean of Detected	457.4	Mean of Detected	4.233
SD of Detected	816.4	SD of Detected	2.556
Minimum Non-Detect	20	Minimum Non-Detect	2.996
Maximum Non-Detect	20	Maximum Non-Detect	2.996

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

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Shapiro Wilk Test Statistic	0.68	Shapiro Wilk Test Statistic	0.996
5% Shapiro Wilk Critical Value	0.748	5% Shapiro Wilk Critical Value	0.748
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	367.9	Mean	3.847
SD	734.8	SD	2.376
95% DL/2 (t) UCL	1068	95% H-Stat (DL/2) UCL	267600000
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	81.7	Mean in Log Scale	3.574
SD	940.9	SD in Log Scale	2.66
95% MLE (t) UCL	978.7	Mean in Original Scale	366.4
95% MLE (Tiku) UCL	1089	SD in Original Scale	735.7
		95% Percentile Bootstrap UCL	1009

95% BCA Bootstrap UCL	1032

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	0.255
Theta Star	1791
nu star	2.043
A-D Test Statistic	0.327
5% A-D Critical Value	0.698
K-S Test Statistic	0.698
5% K-S Critical Value	0.416
Data appear Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	1E-09
Maximum	1680
Mean	365.9
Median	31.9
SD	736
k star	0.181
Theta star	2022
Nu star	1.81
AppChi2	0.13
95% Gamma Approximate UCL	5092
95% Adjusted Gamma UCL	N/A

Data Distribution Test with Detected Values Only
Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	366.7
SD	657.9
SE of Mean	339.7
95% KM (t) UCL	1091
95% KM (z) UCL	925.5
95% KM (jackknife) UCL	1066
95% KM (bootstrap t) UCL	6953
95% KM (BCA) UCL	1021
95% KM (Percentile Bootstrap) UCL	1015
95% KM (Chebyshev) UCL	1848
97.5% KM (Chebyshev) UCL	2488
99% KM (Chebyshev) UCL	3747
Potential UCLs to Use	
95% KM (Chebyshev) UCL	1848

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Chloroform

General Statistics

Number of Valid Data	5	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	3
		Percent Non-Detects	60.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	26.8	Minimum Detected	3.288
Maximum Detected	66.7	Maximum Detected	4.2
Mean of Detected	46.75	Mean of Detected	3.744
SD of Detected	28.21	SD of Detected	0.645
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.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

UCL Statistics

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

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

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	19
SD	29
95% DL/2 (t) UCL	46.64
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	1.082
SD	2.452
95% H-Stat (DL/2) UCL	50290
Log ROS Method	
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% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	34.78
SD	15.96
SE of Mean	10.09
95% KM (t) UCL	56.3
95% KM (z) UCL	51.38
95% KM (jackknife) UCL	N/A
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	N/A
95% KM (Percentile Bootstrap) UCL	N/A
95% KM (Chebyshev) UCL	78.78
97.5% KM (Chebyshev) UCL	97.82
99% KM (Chebyshev) UCL	135.2
Potential UCLs to Use	
95% KM (t) UCL	56.3
95% KM (% Bootstrap) UCL	N/A

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Chloromethane

General Statistics

Number of Valid Data	5	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	3
		Percent Non-Detects	60.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	12.8	Minimum Detected	2.549
Maximum Detected	35.6	Maximum Detected	3.572
Mean of Detected	24.2	Mean of Detected	3.061
SD of Detected	16.12	SD of Detected	0.723
Minimum Non-Detect	2	Minimum Non-Detect	0.693
Maximum Non-Detect	40	Maximum Non-Detect	3.689

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	5
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	0
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	100.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	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	14.08	Mean	1.824
SD	14.51	SD	1.704
95% DL/2 (t) UCL	27.91	95% H-Stat (DL/2) UCL	4081
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% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap 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
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	18.5
SD	9.873
SE of Mean	6.981
95% KM (t) UCL	33.38
95% KM (z) UCL	29.98
95% KM (jackknife) UCL	38.58
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	35.6
95% KM (Percentile Bootstrap) UCL	N/A
95% KM (Chebyshev) UCL	48.93
97.5% KM (Chebyshev) UCL	62.1
99% KM (Chebyshev) UCL	87.96

Potential UCLs to Use

95% KM (t) UCL	33.38
95% KM (% Bootstrap) UCL	N/A

Ethylbenzene

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	9.9
Maximum	169
Mean	51.2
Median	17
SD	67.09
Coefficient of Variation	1.31
Skewness	2.046

Log-transformed Statistics

Minimum of Log Data	2.293
Maximum of Log Data	5.13
Mean of log Data	3.368
SD of log Data	1.12

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

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.699
Shapiro Wilk Critical Value	0.762

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.893
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Normal Distribution

95% Student's-t UCL 115.2

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL 129.9
95% Modified-t UCL 119.7

Assuming Lognormal Distribution

95% H-UCL 1132
95% Chebyshev (MVUE) UCL 141.8
97.5% Chebyshev (MVUE) UCL 183.1
99% Chebyshev (MVUE) UCL 264.3

Gamma Distribution Test

k star (bias corrected) 0.54
Theta Star 94.9
MLE of Mean 51.2
MLE of Standard Deviation 69.71
nu star 5.395
Approximate Chi Square Value (.05) 1.339
Adjusted Level of Significance 0.0086
Adjusted Chi Square Value 0.647

Anderson-Darling Test Statistic 0.544
Anderson-Darling 5% Critical Value 0.691
Kolmogorov-Smirnov Test Statistic 0.321
Kolmogorov-Smirnov 5% Critical Value 0.364
Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 100.6
95% Jackknife UCL 115.2
95% Standard Bootstrap UCL 96.25
95% Bootstrap-t UCL 710.5
95% Hall's Bootstrap UCL 582.5
95% Percentile Bootstrap UCL 106.8
95% BCA Bootstrap UCL 112.1
95% Chebyshev(Mean, Sd) UCL 182
97.5% Chebyshev(Mean, Sd) UCL 238.6
99% Chebyshev(Mean, Sd) UCL 349.7

Assuming Gamma Distribution

95% Approximate Gamma UCL 206.3
95% Adjusted Gamma UCL 427.1

Potential UCL to Use

Use 95% Approximate Gamma UCL 206.3

Recommended UCL exceeds the maximum observation

Tetrachloroethane, 1,1,2,2-

General Statistics

Number of Valid Data	5	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	3
		Percent Non-Detects	60.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	1.2	Minimum Detected	0.182
Maximum Detected	3.7	Maximum Detected	1.308
Mean of Detected	2.45	Mean of Detected	0.745
SD of Detected	1.768	SD of Detected	0.796
Minimum Non-Detect	1	Minimum Non-Detect	0
Maximum Non-Detect	20	Maximum Non-Detect	2.996

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

Number treated as Non-Detect	5
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

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

UCL Statistics

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

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

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	5.08
SD	4.646
95% DL/2 (t) UCL	9.51
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	1.081
SD	1.322
95% H-Stat (DL/2) UCL	1999
Log ROS Method	
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% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	2.033
SD	1.179
SE of Mean	0.962
95% KM (t) UCL	4.085
95% KM (z) UCL	3.616
95% KM (jackknife) UCL	4.515
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	3.7
95% KM (Percentile Bootstrap) UCL	3.7
95% KM (Chebyshev) UCL	6.228
97.5% KM (Chebyshev) UCL	8.043
99% KM (Chebyshev) UCL	11.61
Potential UCLs to Use	
95% KM (t) UCL	4.085
95% KM (% Bootstrap) UCL	3.7

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Toluene

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	10
Maximum	692
Mean	167.7
Median	28.9
SD	295
Coefficient of Variation	1.759
Skewness	2.166

Log-transformed Statistics

Minimum of Log Data	2.303
Maximum of Log Data	6.54
Mean of log Data	3.88
SD of log Data	1.715

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

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.641
Shapiro Wilk Critical Value	0.762

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.909
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	449

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	521.3
95% Modified-t UCL	470.3

Assuming Lognormal Distribution

95% H-UCL	217535
95% Chebyshev (MVUE) UCL	518.3
97.5% Chebyshev (MVUE) UCL	685.1
99% Chebyshev (MVUE) UCL	1013

Gamma Distribution Test

k star (bias corrected)	0.337
Theta Star	497.5
MLE of Mean	167.7
MLE of Standard Deviation	288.9
nu star	3.372
Approximate Chi Square Value (.05)	0.491
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	0.188
Anderson-Darling Test Statistic	0.531
Anderson-Darling 5% Critical Value	0.71
Kolmogorov-Smirnov Test Statistic	0.283
Kolmogorov-Smirnov 5% Critical Value	0.371

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	384.7
95% Jackknife UCL	449
95% Standard Bootstrap UCL	363.1
95% Bootstrap-t UCL	5044
95% Hall's Bootstrap UCL	2294
95% Percentile Bootstrap UCL	420
95% BCA Bootstrap UCL	435.9
95% Chebyshev(Mean, Sd) UCL	742.8
97.5% Chebyshev(Mean, Sd) UCL	991.6

Assuming Gamma Distribution

95% Approximate Gamma UCL 1153

95% Adjusted Gamma UCL 3014

99% Chebyshev(Mean, Sd) UCL 1480

Potential UCL to Use

Use 95% Approximate Gamma UCL 1153

Recommended UCL exceeds the maximum observation

Xylenes, total

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 5

Raw Statistics

Minimum	97.2
Maximum	1130
Mean	366.4
Median	234
SD	431.1
Coefficient of Variation	1.176
Skewness	2.126

Log-transformed Statistics

Minimum of Log Data	4.577
Maximum of Log Data	7.03
Mean of log Data	5.487
SD of log Data	0.941

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

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.679
Shapiro Wilk Critical Value	0.762

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.877
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 777.5

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL 879.4

95% Modified-t UCL 808

Assuming Lognormal Distribution

95% H-UCL 3353

95% Chebyshev (MVUE) UCL 934.8

97.5% Chebyshev (MVUE) UCL 1193

99% Chebyshev (MVUE) UCL 1701

Gamma Distribution Test

k star (bias corrected)	0.67
Theta Star	547
MLE of Mean	366.4
MLE of Standard Deviation	447.7
nu star	6.699

Data Distribution

Data Follow Appr. Gamma Distribution at 5% Significance Level

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Approximate Chi Square Value (.05)	2.007
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	1.077
Anderson-Darling Test Statistic	0.606
Anderson-Darling 5% Critical Value	0.688
Kolmogorov-Smirnov Test Statistic	0.371
Kolmogorov-Smirnov 5% Critical Value	0.363

Data follow Appr. Gamma Distribution at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	1223
95% Adjusted Gamma UCL	2280

Nonparametric Statistics

95% CLT UCL	683.6
95% Jackknife UCL	777.5
95% Standard Bootstrap UCL	639.2
95% Bootstrap-t UCL	2095
95% Hall's Bootstrap UCL	2693
95% Percentile Bootstrap UCL	724.6
95% BCA Bootstrap UCL	752.2
95% Chebyshev(Mean, Sd) UCL	1207
97.5% Chebyshev(Mean, Sd) UCL	1570
99% Chebyshev(Mean, Sd) UCL	2285

Potential UCL to Use — Use 95% Approximate Gamma UCL 1223

Recommended UCL exceeds the maximum observation

Cyanide, total

General Statistics

Number of Valid Data	5	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	2
		Percent Non-Detects	40.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.014	Minimum Detected	-4.269
Maximum Detected	0.11	Maximum Detected	-2.207
Mean of Detected	0.065	Mean of Detected	-3.04
SD of Detected	0.0483	SD of Detected	1.086
Minimum Non-Detect	0.01	Minimum Non-Detect	-4.605
Maximum Non-Detect	0.01	Maximum Non-Detect	-4.605

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.988	Shapiro Wilk Test Statistic	0.901
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.041	Mean	-3.944
SD	0.0474	SD	1.456
95% DL/2 (t) UCL	0.0862	95% H-Stat (DL/2) UCL	0.833

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Maximum Likelihood Estimate(MLE) Method	
Mean	0.0256
SD	0.061
95% MLE (t) UCL	0.0838
95% MLE (Tiku) UCL	0.0912

Log ROS Method	
Mean in Log Scale	-4.297
SD in Log Scale	1.922
Mean in Original Scale	0.0399
SD in Original Scale	0.0484
95% Percentile Bootstrap UCL	0.0731
95% BCA Bootstrap UCL	0.0731

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Data Distribution Test with Detected Values Only

Data appear Normal at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	0.0446
SD	0.0395
SE of Mean	0.0216
95% KM (t) UCL	0.0907
95% KM (z) UCL	0.0801
95% KM (jackknife) UCL	0.0974
95% KM (bootstrap t) UCL	0.0798
95% KM (BCA) UCL	0.11
95% KM (Percentile Bootstrap) UCL	0.11
95% KM (Chebyshev) UCL	0.139
97.5% KM (Chebyshev) UCL	0.18
99% KM (Chebyshev) UCL	0.26

Potential UCLs to Use

95% KM (t) UCL	0.0907
95% KM (Percentile Bootstrap) UCL	0.11

General UCL Statistics for Full Data Sets

User Selected Options

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

Methylene chloride

General Statistics

Number of Valid Observations 5

Number of Distinct Observations 3

Raw Statistics

Minimum	2.5
Maximum	77.6
Mean	27.02
Median	2.5
SD	34.96
Coefficient of Variation	1.294
Skewness	0.949

Log-transformed Statistics

Minimum of Log Data	0.916
Maximum of Log Data	4.352
Mean of log Data	2.202
SD of log Data	1.768

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Bedrock Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Warning: There are only 3 Distinct Values in this data
There are insufficient Distinct Values to perform some GOF tests and bootstrap methods.
Those methods will return a 'N/A' value on your output display!

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

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

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.77
Shapiro Wilk Critical Value	0.762

Data appear Normal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	60.35

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	59.83
95% Modified-t UCL	61.46

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.723
Shapiro Wilk Critical Value	0.762

Data not Lognormal at 5% Significance Level

Assuming Lognormal Distribution

95% H-UCL	68257
95% Chebyshev (MVUE) UCL	104
97.5% Chebyshev (MVUE) UCL	137.6
99% Chebyshev (MVUE) UCL	203.7

Gamma Distribution Test

k star (bias corrected)	0.361
Theta Star	74.85
MLE of Mean	27.02
MLE of Standard Deviation	44.97
nu star	3.61
Approximate Chi Square Value (.05)	0.574
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	0.225
Anderson-Darling Test Statistic	0.784
Anderson-Darling 5% Critical Value	0.707
Kolmogorov-Smirnov Test Statistic	0.394
Kolmogorov-Smirnov 5% Critical Value	0.37

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	169.9
95% Adjusted Gamma UCL	433

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	52.74
95% Jackknife UCL	60.35
95% Standard Bootstrap UCL	N/A
95% Bootstrap-t UCL	N/A
95% Hall's Bootstrap UCL	N/A
95% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A
95% Chebyshev(Mean, Sd) UCL	95.18
97.5% Chebyshev(Mean, Sd) UCL	124.7
99% Chebyshev(Mean, Sd) UCL	182.6

Potential UCL to Use

Use 95% Student's-t UCL 60.35

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Chromium

General Statistics

Number of Valid Data	10	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	8
		Percent Non-Detects	80.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	2.8	Minimum Detected	1.03
Maximum Detected	3.3	Maximum Detected	1.194
Mean of Detected	3.05	Mean of Detected	1.112
SD of Detected	0.354	SD of Detected	0.116
Minimum Non-Detect	10	Minimum Non-Detect	2.303
Maximum Non-Detect	10	Maximum Non-Detect	2.303

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	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	4.61	Mean	1.51
SD	0.831	SD	0.213
95% DL/2 (t) UCL	5.091	95% H-Stat (DL/2) UCL	9.369
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% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap 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	3.05
5% K-S Critical Value	N/A	SD	0.25
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.25
		95% KM (t) UCL	3.508
Assuming Gamma Distribution		95% KM (z) UCL	3.461
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	4.14
SD	N/A	97.5% KM (Chebyshev) UCL	4.611
k star	N/A	99% KM (Chebyshev) UCL	5.537
Theta star	N/A		
Nu star	N/A	Potential UCLs to Use	
AppChi2	N/A	95% KM (t) UCL	3.508
95% Gamma Approximate UCL	N/A	95% KM (% Bootstrap) UCL	N/A
95% Adjusted Gamma UCL	N/A		

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Cobalt

General Statistics

Number of Valid Data	10	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	7
		Percent Non-Detects	70.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	2.3	Minimum Detected	0.833
Maximum Detected	6.1	Maximum Detected	1.808
Mean of Detected	4.033	Mean of Detected	1.317
SD of Detected	1.922	SD of Detected	0.488
Minimum Non-Detect	50	Minimum Non-Detect	3.912
Maximum Non-Detect	50	Maximum Non-Detect	3.912

Warning: There are only 3 Distinct Detected Values in this data set

The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods.

Those methods will return a 'N/A' value on your output display!

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

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.977	Shapiro Wilk Test Statistic	1
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	

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Normal Distribution

DL/2 Substitution Method	
Mean	18.71
SD	10.17
95% DL/2 (t) UCL	24.6
Maximum Likelihood Estimate(MLE) Method	N/A

MLE method failed to converge properly

Assuming Lognormal Distribution

DL/2 Substitution Method	
Mean	2.648
SD	0.947
95% H-Stat (DL/2) UCL	289.7
Log ROS Method	
Mean in Log Scale	1.317
SD in Log Scale	0.521
Mean in Original Scale	4.205
SD in Original Scale	2.17
95% Percentile Bootstrap UCL	5.249
95% BCA Bootstrap UCL	5.421

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data appear Normal at 5% Significance Level

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	4.033
SD	1.569
SE of Mean	1.11
95% KM (t) UCL	6.067
95% KM (z) UCL	5.858
95% KM (jackknife) UCL	6.397
95% KM (bootstrap t) UCL	8.419
95% KM (BCA) UCL	6.1
95% KM (Percentile Bootstrap) UCL	6.1
95% KM (Chebyshev) UCL	8.87
97.5% KM (Chebyshev) UCL	10.96
99% KM (Chebyshev) UCL	15.07

Potential UCLs to Use

95% KM (t) UCL	6.067
95% KM (Percentile Bootstrap) UCL	6.1

Iron

General Statistics

Number of Valid Data	10	Number of Detected Data	5
Number of Distinct Detected Data	5	Number of Non-Detect Data	5
		Percent Non-Detects	50.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	203.5	Minimum Detected	5.316
Maximum Detected	2830	Maximum Detected	7.948
Mean of Detected	1294	Mean of Detected	6.622
SD of Detected	1275	SD of Detected	1.246
Minimum Non-Detect	300	Minimum Non-Detect	5.704
Maximum Non-Detect	300	Maximum Non-Detect	5.704

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.808
5% Shapiro Wilk Critical Value	0.762
Data appear Normal at 5% Significance Level	
Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	722
SD	1042
95% DL/2 (t) UCL	1326
Maximum Likelihood Estimate(MLE) Method	N/A
MLE yields a negative mean	

Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.862
5% Shapiro Wilk Critical Value	0.762
Data appear Lognormal at 5% Significance Level	
Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	5.816
SD	1.188
95% H-Stat (DL/2) UCL	2395
Log ROS Method	
Mean in Log Scale	6.005
SD in Log Scale	1.163
Mean in Original Scale	780.2
SD in Original Scale	1015
95% Percentile Bootstrap UCL	1297
95% BCA Bootstrap UCL	1515

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	0.555
Theta Star	2330
nu star	5.554
A-D Test Statistic	0.472
5% A-D Critical Value	0.691
K-S Test Statistic	0.691
5% K-S Critical Value	0.364
Data appear Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	203.5
Maximum	2830
Mean	1258
Median	1191
SD	858.4
k star	1.439
Theta star	874.3
Nu star	28.78
AppChi2	17.54
95% Gamma Approximate UCL	2065
95% Adjusted Gamma UCL	2259

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only	
Data appear Normal at 5% Significance Level	
Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	758.3
SD	968.3
SE of Mean	342.5
95% KM (t) UCL	1386
95% KM (z) UCL	1322
95% KM (jackknife) UCL	1351
95% KM (bootstrap t) UCL	2997
95% KM (BCA) UCL	1597
95% KM (Percentile Bootstrap) UCL	1477
95% KM (Chebyshev) UCL	2251
97.5% KM (Chebyshev) UCL	2897
99% KM (Chebyshev) UCL	4166
Potential UCLs to Use	
95% KM (t) UCL	1386
95% KM (Percentile Bootstrap) UCL	1477

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Dinitrotoluene, 2,4-exp

General Statistics

Number of Valid Data	10	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	7
		Percent Non-Detects	70.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.23	Minimum Detected	-1.47
Maximum Detected	10.6	Maximum Detected	2.361
Mean of Detected	6.31	Mean of Detected	0.994
SD of Detected	5.412	SD of Detected	2.138
Minimum Non-Detect	0.19	Minimum Non-Detect	-1.661
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	7
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	3
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	70.00%

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.918	Shapiro Wilk Test Statistic	0.802
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	1.961	Mean	-1.334
SD	3.939	SD	1.897
95% DL/2 (t) UCL	4.244	95% H-Stat (DL/2) UCL	8.002
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-4.532
		SD in Log Scale	4.434
		Mean in Original Scale	1.897
		SD in Original Scale	3.973
		95% Percentile Bootstrap UCL	4.014
		95% BCA Bootstrap UCL	4.801
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	2.054
5% K-S Critical Value	N/A	SD	3.691

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

SE of Mean	1.429
95% KM (t) UCL	4.674
95% KM (z) UCL	4.405
95% KM (jackknife) UCL	7.249
95% KM (bootstrap t) UCL	3.077
95% KM (BCA) UCL	10.6
95% KM (Percentile Bootstrap) UCL	10.6
95% KM (Chebyshev) UCL	8.284
97.5% KM (Chebyshev) UCL	10.98
99% KM (Chebyshev) UCL	16.28

Potential UCLs to Use

95% KM (t) UCL	4.674
95% KM (Percentile Bootstrap) UCL	10.6

Dinitrotoluene, 2,6-exp

General Statistics

Number of Valid Data	10	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	7
		Percent Non-Detects	70.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.29	Minimum Detected	-1.238
Maximum Detected	9.15	Maximum Detected	2.214
Mean of Detected	5.33	Mean of Detected	0.952
SD of Detected	4.554	SD of Detected	1.904
Minimum Non-Detect	0.19	Minimum Non-Detect	-1.661
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

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

Number treated as Non-Detect	7
Number treated as Detected	3
Single DL Non-Detect Percentage	70.00%

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.946	Shapiro Wilk Test Statistic	0.822
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	1.667	Mean	-1.347
SD	3.316	SD	1.823
95% DL/2 (t) UCL	3.589	95% H-Stat (DL/2) UCL	5.581

Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-4.044
		SD in Log Scale	4.005
		Mean in Original Scale	1.605
		SD in Original Scale	3.349
		95% Percentile Bootstrap UCL	3.407
		95% BCA Bootstrap UCL	4.087

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	1.802
5% K-S Critical Value	N/A	SD	3.079
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.193
		95% KM (t) UCL	3.988
Assuming Gamma Distribution		95% KM (z) UCL	3.764
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	5.965
Minimum	N/A	95% KM (bootstrap t) UCL	2.571
Maximum	N/A	95% KM (BCA) UCL	N/A
Mean	N/A	95% KM (Percentile Bootstrap) UCL	9.15
Median	N/A	95% KM (Chebyshev) UCL	7.001
SD	N/A	97.5% KM (Chebyshev) UCL	9.25
k star	N/A	99% KM (Chebyshev) UCL	13.67
Theta star	N/A		
Nu star	N/A	Potential UCLs to Use	
AppChi2	N/A	95% KM (t) UCL	3.988
95% Gamma Approximate UCL	N/A	95% KM (Percentile Bootstrap) UCL	9.15
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

Nitrotoluene, 2-

General Statistics

Number of Valid Data	10	Number of Detected Data	4
Number of Distinct Detected Data	4	Number of Non-Detect Data	6
		Percent Non-Detects	60.00%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	1.8	Minimum Detected	0.588
Maximum Detected	41.4	Maximum Detected	3.723
Mean of Detected	20.53	Mean of Detected	2.165
SD of Detected	21.53	SD of Detected	1.734
Minimum Non-Detect	0.19	Minimum Non-Detect	-1.661
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	6
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	4
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	60.00%

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

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

UCL Statistics

Normal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.785
5% Shapiro Wilk Critical Value	0.748
Data appear Normal at 5% Significance Level	

Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.77
5% Shapiro Wilk Critical Value	0.748
Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	8.268
SD	16.3
95% DL/2 (t) UCL	17.72
Maximum Likelihood Estimate(MLE) Method	N/A
MLE yields a negative mean	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	-0.536
SD	2.531
95% H-Stat (DL/2) UCL	815.3
Log ROS Method	
Mean in Log Scale	-1.204
SD in Log Scale	3.293
Mean in Original Scale	8.254
SD in Original Scale	16.31
95% Percentile Bootstrap UCL	16.46
95% BCA Bootstrap UCL	19.51

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	0.343
Theta Star	59.87
nu star	2.742
A-D Test Statistic	0.629
5% A-D Critical Value	0.672
K-S Test Statistic	0.672
5% K-S Critical Value	0.406
Data appear Gamma Distributed at 5% Significance Level	

Data Distribution Test with Detected Values Only
Data appear Normal at 5% Significance Level

Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	1.8
Maximum	46.78
Mean	19.98
Median	15.61
SD	17.45
k star	0.803
Theta star	24.88
Nu star	16.06
AppChi2	8.003
95% Gamma Approximate UCL	40.09
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	9.29
SD	14.94
SE of Mean	5.456
95% KM (t) UCL	19.29
95% KM (z) UCL	18.26
95% KM (jackknife) UCL	18.3
95% KM (bootstrap t) UCL	15.16
95% KM (BCA) UCL	41.4
95% KM (Percentile Bootstrap) UCL	37.72
95% KM (Chebyshev) UCL	33.07
97.5% KM (Chebyshev) UCL	43.36
99% KM (Chebyshev) UCL	63.57
Potential UCLs to Use	
95% KM (t) UCL	19.29
95% KM (Percentile Bootstrap) UCL	37.72

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Nitrotoluene, 3-

General Statistics

Number of Valid Data	10	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	8
		Percent Non-Detects	80.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	4.3	Minimum Detected	1.459
Maximum Detected	5.15	Maximum Detected	1.639
Mean of Detected	4.725	Mean of Detected	1.549
SD of Detected	0.601	SD of Detected	0.128
Minimum Non-Detect	0.19	Minimum Non-Detect	-1.661
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	8
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	2
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	80.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

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

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

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	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	1.023	Mean	-1.558
SD	1.962	SD	1.638
95% DL/2 (t) UCL	2.16	95% H-Stat (DL/2) UCL	2.505
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% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap UCL	N/A

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	4.385
SD	0.255
SE of Mean	0.114
95% KM (t) UCL	4.594
95% KM (z) UCL	4.573
95% KM (jackknife) UCL	4.978
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	5.15
95% KM (Percentile Bootstrap) UCL	5.15
95% KM (Chebyshev) UCL	4.882
97.5% KM (Chebyshev) UCL	5.097
99% KM (Chebyshev) UCL	5.52
Potential UCLs to Use	
95% KM (t) UCL	4.594
95% KM (% Bootstrap) UCL	5.15

Nitrotoluene, 4-

General Statistics

Number of Valid Data	10	Number of Detected Data	4
Number of Distinct Detected Data	4	Number of Non-Detect Data	6
		Percent Non-Detects	60.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.61	Minimum Detected	-0.494
Maximum Detected	4.58	Maximum Detected	1.522
Mean of Detected	2.685	Mean of Detected	0.67
SD of Detected	2.072	SD of Detected	0.993
Minimum Non-Detect	0.19	Minimum Non-Detect	-1.661
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

Note: Data have multiple DLs - Use of KM Method is recommended	Number treated as Non-Detect	6
For all methods (except KM, DL/2, and ROS Methods),	Number treated as Detected	4
Observations < Largest ND are treated as NDs	Single DL Non-Detect Percentage	60.00%

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.

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.824	Shapiro Wilk Test Statistic	0.867
5% Shapiro Wilk Critical Value	0.748	5% Shapiro Wilk Critical Value	0.748
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	1.132	Mean	-1.134
SD	1.794	SD	1.655
95% DL/2 (t) UCL	2.172	95% H-Stat (DL/2) UCL	5.585
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-1.307
		SD in Log Scale	1.928
		Mean in Original Scale	1.134
		SD in Original Scale	1.793
		95% Percentile Bootstrap UCL	2.042
		95% BCA Bootstrap UCL	2.374

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.597	Data appear Normal at 5% Significance Level	
Theta Star	4.497		
nu star	4.777		
A-D Test Statistic	0.463	**Nonparametric Statistics**	
5% A-D Critical Value	0.662	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.662	Mean	1.44
5% K-S Critical Value	0.399	SD	1.523
Data appear Gamma Distributed at 5% Significance Level		SE of Mean	0.556
		95% KM (t) UCL	2.46
Assuming Gamma Distribution		95% KM (z) UCL	2.355
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	2.314
Minimum	0.61	95% KM (bootstrap t) UCL	2.059
Maximum	5.101	95% KM (BCA) UCL	4.465
Mean	2.662	95% KM (Percentile Bootstrap) UCL	4.396
Median	2.479	95% KM (Chebyshev) UCL	3.865
SD	1.732	97.5% KM (Chebyshev) UCL	4.914
k star	1.561	99% KM (Chebyshev) UCL	6.975
Theta star	1.705		
Nu star	31.22	Potential UCLs to Use	
AppChi2	19.46	95% KM (t) UCL	2.46
95% Gamma Approximate UCL	4.272	95% KM (Percentile Bootstrap) UCL	4.396
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

Dichloroethane, 1,1-

General Statistics

Number of Valid Data	10	Number of Detected Data	6
Number of Distinct Detected Data	6	Number of Non-Detect Data	4
		Percent Non-Detects	40.00%

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Raw Statistics		Log-transformed Statistics	
Minimum Detected	1.55	Minimum Detected	0.438
Maximum Detected	8.3	Maximum Detected	2.116
Mean of Detected	3.95	Mean of Detected	1.181
SD of Detected	2.736	SD of Detected	0.671
Minimum Non-Detect	1	Minimum Non-Detect	0
Maximum Non-Detect	1	Maximum Non-Detect	0

Warning: There are only 6 Detected Values in this data
Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.852	Shapiro Wilk Test Statistic	0.917
5% Shapiro Wilk Critical Value	0.788	5% Shapiro Wilk Critical Value	0.788
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2.57	Mean	0.431
SD	2.708	SD	1.09
95% DL/2 (t) UCL	4.14	95% H-Stat (DL/2) UCL	2.6
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	1.728	Mean in Log Scale	0.424
SD	3.577	SD in Log Scale	1.138
95% MLE (t) UCL	3.801	Mean in Original Scale	2.586
95% MLE (Tiku) UCL	4.062	SD in Original Scale	2.699
		95% Percentile Bootstrap UCL	3.975
		95% BCA Bootstrap UCL	4.27

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.486	Data appear Normal at 5% Significance Level	
Theta Star	2.659		
nu star	17.83		
A-D Test Statistic	0.401	**Nonparametric Statistics**	
5% A-D Critical Value	0.702	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.702	Mean	2.99
5% K-S Critical Value	0.335	SD	2.264
Data appear Gamma Distributed at 5% Significance Level		SE of Mean	0.784
		95% KM (t) UCL	4.428
Assuming Gamma Distribution		95% KM (z) UCL	4.28
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	4.299
Minimum	0.553	95% KM (bootstrap t) UCL	7.371
Maximum	8.3	95% KM (BCA) UCL	4.7
Mean	3.366	95% KM (Percentile Bootstrap) UCL	4.435
Median	2.685	95% KM (Chebyshev) UCL	6.408
SD	2.362	97.5% KM (Chebyshev) UCL	7.888
k star	1.654	99% KM (Chebyshev) UCL	10.79
Theta star	2.035		

Nu star	33.08	Potential UCLs to Use	
AppChi2	20.93	95% KM (t) UCL	4.428
95% Gamma Approximate UCL	5.32	95% KM (Percentile Bootstrap) UCL	4.435
95% Adjusted Gamma UCL	5.779		

Note: DL/2 is not a recommended method.

Trichloroethene

General Statistics

Number of Valid Data	10	Number of Detected Data	2
Number of Distinct Detected Data	2	Number of Non-Detect Data	8
		Percent Non-Detects	80.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	4.55	Minimum Detected	1.515
Maximum Detected	5.4	Maximum Detected	1.686
Mean of Detected	4.975	Mean of Detected	1.601
SD of Detected	0.601	SD of Detected	0.121
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

Normal Distribution Test with Detected Values Only		Lognormal 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	1.395	Mean	-0.234
SD	1.897	SD	0.968
95% DL/2 (t) UCL	2.495	95% H-Stat (DL/2) UCL	1.711
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% Percentile Bootstrap UCL	N/A
		95% BCA Bootstrap UCL	N/A

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	4.635
SD	0.255
SE of Mean	0.114
95% KM (t) UCL	4.844
95% KM (z) UCL	4.823
95% KM (jackknife) UCL	N/A
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	N/A
95% KM (Percentile Bootstrap) UCL	N/A
95% KM (Chebyshev) UCL	5.132
97.5% KM (Chebyshev) UCL	5.347
99% KM (Chebyshev) UCL	5.77

Potential UCLs to Use

95% KM (t) UCL	4.844
95% KM (% Bootstrap) UCL	N/A

General UCL Statistics for Full Data Sets

User Selected Options

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

Carbon tetrachloride

General Statistics

Number of Valid Observations 10

Number of Distinct Observations 2

Raw Statistics

Minimum	0.5
Maximum	4
Mean	0.85
Median	0.5
SD	1.107
Coefficient of Variation	1.302
Skewness	3.162

Log-transformed Statistics

Minimum of Log Data	-0.693
Maximum of Log Data	1.386
Mean of log Data	-0.485
SD of log Data	0.658

Warning: There are only 2 Distinct Values in this data
There are insufficient Distinct Values to perform some GOF tests and bootstrap methods.
Those methods will return a 'N/A' value on your output display!

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.366
Shapiro Wilk Critical Value	0.842

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.366
Shapiro Wilk Critical Value	0.842

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	1.492

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	1.8
95% Modified-t UCL	1.55

Assuming Lognormal Distribution

95% H-UCL	1.31
95% Chebyshev (MVUE) UCL	1.44
97.5% Chebyshev (MVUE) UCL	1.74
99% Chebyshev (MVUE) UCL	2.331

Gamma Distribution Test

k star (bias corrected)	1.255
Theta Star	0.678
MLE of Mean	0.85
MLE of Standard Deviation	0.759
nu star	25.09
Approximate Chi Square Value (.05)	14.68
Adjusted Level of Significance	0.0267
Adjusted Chi Square Value	13.32
Anderson-Darling Test Statistic	3.357
Anderson-Darling 5% Critical Value	0.738
Kolmogorov-Smirnov Test Statistic	0.543
Kolmogorov-Smirnov 5% Critical Value	0.271

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	1.453
95% Adjusted Gamma UCL	1.602

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	1.426
95% Jackknife UCL	N/A
95% Standard Bootstrap UCL	N/A
95% Bootstrap-t UCL	N/A
95% Hall's Bootstrap UCL	N/A
95% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A
95% Chebyshev(Mean, Sd) UCL	2.376
97.5% Chebyshev(Mean, Sd) UCL	3.036
99% Chebyshev(Mean, Sd) UCL	4.332

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 2.376

Dibromochloromethane

General Statistics

Number of Valid Observations 10

Number of Distinct Observations 2

Raw Statistics

Minimum	0.2
Maximum	0.5
Mean	0.47
Median	0.5
SD	0.0949
Coefficient of Variation	0.202
Skewness	-3.162

Log-transformed Statistics

Minimum of Log Data	-1.609
Maximum of Log Data	-0.693
Mean of log Data	-0.785
SD of log Data	0.29

Warning: There are only 2 Distinct Values in this data
There are insufficient Distinct Values to perform some GOF tests and bootstrap methods.
Those methods will return a 'N/A' value on your output display!

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

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Overburden Groundwater
Plum Brook Ordnance Works, Sandusky, Ohio

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.366
Shapiro Wilk Critical Value	0.842

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.366
Shapiro Wilk Critical Value	0.842

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	0.525

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	0.487
95% Modified-t UCL	0.52

Assuming Lognormal Distribution

95% H-UCL	0.575
95% Chebyshev (MVUE) UCL	0.664
97.5% Chebyshev (MVUE) UCL	0.747
99% Chebyshev (MVUE) UCL	0.908

Gamma Distribution Test

k star (bias corrected)	11.95
Theta Star	0.0393
MLE of Mean	0.47
MLE of Standard Deviation	0.136
nu star	238.9
Approximate Chi Square Value (.05)	204.1
Adjusted Level of Significance	0.0267
Adjusted Chi Square Value	198.5
Anderson-Darling Test Statistic	3.305
Anderson-Darling 5% Critical Value	0.725
Kolmogorov-Smirnov Test Statistic	0.532
Kolmogorov-Smirnov 5% Critical Value	0.266

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	0.55
95% Adjusted Gamma UCL	0.566

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	0.519
95% Jackknife UCL	N/A
95% Standard Bootstrap UCL	N/A
95% Bootstrap-t UCL	N/A
95% Hall's Bootstrap UCL	N/A
95% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A
95% Chebyshev(Mean, Sd) UCL	0.601
97.5% Chebyshev(Mean, Sd) UCL	0.657
99% Chebyshev(Mean, Sd) UCL	0.768

Potential UCL to Use

Use 95% Student's-t UCL 0.525
or 95% Modified-t UCL 0.52

Recommended UCL exceeds the maximum observation

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Arsenic

General Statistics

Number of Valid Observations 6

Number of Distinct Observations 6

Raw Statistics

Minimum	8.3
Maximum	17.8
Mean	12.01
Median	10.28
SD	3.93
Coefficient of Variation	0.327
Skewness	0.852

Log-transformed Statistics

Minimum of Log Data	2.116
Maximum of Log Data	2.879
Mean of log Data	2.444
SD of log Data	0.311

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	0.849
Shapiro Wilk Critical Value	0.788

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.88
Shapiro Wilk Critical Value	0.788

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	15.24

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	15.24
95% Modified-t UCL	15.33

Assuming Lognormal Distribution

95% H-UCL	16.53
95% Chebyshev (MVUE) UCL	18.63
97.5% Chebyshev (MVUE) UCL	21.51
99% Chebyshev (MVUE) UCL	27.15

Gamma Distribution Test

k star (bias corrected)	6.166
Theta Star	1.947
MLE of Mean	12.01
MLE of Standard Deviation	4.836
nu star	73.99
Approximate Chi Square Value (.05)	55.18
Adjusted Level of Significance	0.0122
Adjusted Chi Square Value	49.36

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	14.65
95% Jackknife UCL	15.24
95% Standard Bootstrap UCL	14.42

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

Anderson-Darling Test Statistic 0.5
Anderson-Darling 5% Critical Value 0.698
Kolmogorov-Smirnov Test Statistic 0.254
Kolmogorov-Smirnov 5% Critical Value 0.332
Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution
95% Approximate Gamma UCL 16.1
95% Adjusted Gamma UCL 18

95% Bootstrap-t UCL 21.79
95% Hall's Bootstrap UCL 32.37
95% Percentile Bootstrap UCL 14.63
95% BCA Bootstrap UCL 14.69
95% Chebyshev(Mean, Sd) UCL 19
97.5% Chebyshev(Mean, Sd) UCL 22.03
99% Chebyshev(Mean, Sd) UCL 27.97

Potential UCL to Use

Use 95% Student's-t UCL 15.24

Chromium

General Statistics

Number of Valid Observations 6
Number of Distinct Observations 6

Raw Statistics
Minimum 10.4
Maximum 18.2
Mean 13.68
Median 13.45
SD 3.004
Coefficient of Variation 0.22
Skewness 0.455

Log-transformed Statistics
Minimum of Log Data 2.342
Maximum of Log Data 2.901
Mean of log Data 2.596
SD of log Data 0.218

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 0.932
Shapiro Wilk Critical Value 0.788
Data appear Normal at 5% Significance Level

Lognormal Distribution Test
Shapiro Wilk Test Statistic 0.935
Shapiro Wilk Critical Value 0.788
Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution
95% Student's-t UCL 16.15
95% UCLs (Adjusted for Skewness)
95% Adjusted-CLT UCL 15.94
95% Modified-t UCL 16.19

Assuming Lognormal Distribution
95% H-UCL 16.85
95% Chebyshev (MVUE) UCL 18.99
97.5% Chebyshev (MVUE) UCL 21.29
99% Chebyshev (MVUE) UCL 25.8

Gamma Distribution Test
k star (bias corrected) 12.76
Theta Star 1.072
MLE of Mean 13.68
MLE of Standard Deviation 3.83
nu star 153.2

Data Distribution
Data appear Normal at 5% Significance Level

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

Approximate Chi Square Value (.05) 125.6
Adjusted Level of Significance 0.0122
Adjusted Chi Square Value 116.5

Anderson-Darling Test Statistic 0.292
Anderson-Darling 5% Critical Value 0.697
Kolmogorov-Smirnov Test Statistic 0.202
Kolmogorov-Smirnov 5% Critical Value 0.332
Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution
95% Approximate Gamma UCL 16.69
95% Adjusted Gamma UCL 17.99

Nonparametric Statistics
95% CLT UCL 15.7
95% Jackknife UCL 16.15
95% Standard Bootstrap UCL 15.49
95% Bootstrap-t UCL 16.83
95% Hall's Bootstrap UCL 17.49
95% Percentile Bootstrap UCL 15.48
95% BCA Bootstrap UCL 15.48
95% Chebyshev(Mean, Sd) UCL 19.03
97.5% Chebyshev(Mean, Sd) UCL 21.34
99% Chebyshev(Mean, Sd) UCL 25.88

Potential UCL to Use

Use 95% Student's-t UCL 16.15

Cobalt

General Statistics

Number of Valid Observations 6
Number of Distinct Observations 6

Raw Statistics
Minimum 10.8
Maximum 26
Mean 17.08
Median 14.65
SD 6.919
Coefficient of Variation 0.405
Skewness 0.559

Log-transformed Statistics
Minimum of Log Data 2.38
Maximum of Log Data 3.258
Mean of log Data 2.771
SD of log Data 0.399

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 0.823
Shapiro Wilk Critical Value 0.788
Data appear Normal at 5% Significance Level

Assuming Normal Distribution
95% Student's-t UCL 22.78

95% UCLs (Adjusted for Skewness)
95% Adjusted-CLT UCL 22.42
95% Modified-t UCL 22.88

Lognormal Distribution Test
Shapiro Wilk Test Statistic 0.837
Shapiro Wilk Critical Value 0.788
Data appear Lognormal at 5% Significance Level

Assuming Lognormal Distribution
95% H-UCL 26.6
95% Chebyshev (MVUE) UCL 29.16
97.5% Chebyshev (MVUE) UCL 34.39
99% Chebyshev (MVUE) UCL 44.68

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test

k star (bias corrected)	3.91
Theta Star	4.369
MLE of Mean	17.08
MLE of Standard Deviation	8.639
nu star	46.92
Approximate Chi Square Value (.05)	32.2
Adjusted Level of Significance	0.0122
Adjusted Chi Square Value	27.87
Anderson-Darling Test Statistic	0.57
Anderson-Darling 5% Critical Value	0.698
Kolmogorov-Smirnov Test Statistic	0.298
Kolmogorov-Smirnov 5% Critical Value	0.333

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	24.89
95% Adjusted Gamma UCL	28.76

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	21.73
95% Jackknife UCL	22.78
95% Standard Bootstrap UCL	21.25
95% Bootstrap-t UCL	25.1
95% Hall's Bootstrap UCL	23.57
95% Percentile Bootstrap UCL	21.57
95% BCA Bootstrap UCL	21.5
95% Chebyshev(Mean, Sd) UCL	29.4
97.5% Chebyshev(Mean, Sd) UCL	34.72
99% Chebyshev(Mean, Sd) UCL	45.19

Potential UCL to Use

Use 95% Student's-t UCL 22.78

Manganese

General Statistics

Number of Valid Observations 6

Number of Distinct Observations 6

Raw Statistics

Minimum	737
Maximum	5360
Mean	1837
Median	1242
SD	1763
Coefficient of Variation	0.96
Skewness	2.228

Log-transformed Statistics

Minimum of Log Data	6.603
Maximum of Log Data	8.587
Mean of log Data	7.249
SD of log Data	0.727

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	0.675
Shapiro Wilk Critical Value	0.788

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.851
Shapiro Wilk Critical Value	0.788

Data appear Lognormal at 5% Significance Level

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Normal Distribution

95% Student's-t UCL 3287

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL 3720

95% Modified-t UCL 3396

Assuming Lognormal Distribution

95% H-UCL 5282

95% Chebyshev (MVUE) UCL 3986

97.5% Chebyshev (MVUE) UCL 4956

99% Chebyshev (MVUE) UCL 6863

Gamma Distribution Test

k star (bias corrected) 1.124

Theta Star 1634

MLE of Mean 1837

MLE of Standard Deviation 1732

nu star 13.49

Approximate Chi Square Value (.05) 6.223

Adjusted Level of Significance 0.0122

Adjusted Chi Square Value 4.561

Anderson-Darling Test Statistic 0.661

Anderson-Darling 5% Critical Value 0.704

Kolmogorov-Smirnov Test Statistic 0.311

Kolmogorov-Smirnov 5% Critical Value 0.336

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 3020

95% Jackknife UCL 3287

95% Standard Bootstrap UCL 2921

95% Bootstrap-t UCL 6629

95% Hall's Bootstrap UCL 7976

95% Percentile Bootstrap UCL 3193

95% BCA Bootstrap UCL 3362

95% Chebyshev(Mean, Sd) UCL 4974

97.5% Chebyshev(Mean, Sd) UCL 6332

99% Chebyshev(Mean, Sd) UCL 8998

Assuming Gamma Distribution

95% Approximate Gamma UCL 3981

95% Adjusted Gamma UCL 5431

Potential UCL to Use

Use 95% Approximate Gamma UCL 3981

Thallium

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	0.26	Minimum Detected	-1.347
Maximum Detected	1.9	Maximum Detected	0.642
Mean of Detected	1.08	Mean of Detected	-0.353
SD of Detected	1.16	SD of Detected	1.406
Minimum Non-Detect	0.63	Minimum Non-Detect	-0.462
Maximum Non-Detect	0.67	Maximum Non-Detect	-0.4

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

Number treated as Non-Detect	5
Number treated as Detected	1
Single DL Non-Detect Percentage	83.33%

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!

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Sediment
Plum Brook Ordnance Works, Sandusky, Ohio

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

UCL Statistics

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

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

Assuming Normal Distribution	
DL/2 Substitution Method	
Mean	0.578
SD	0.648
95% DL/2 (t) UCL	1.111
Maximum Likelihood Estimate(MLE) Method	N/A
MLE method failed to converge properly	

Assuming Lognormal Distribution	
DL/2 Substitution Method	
Mean	-0.862
SD	0.743
95% H-Stat (DL/2) UCL	0.855
Log ROS Method	
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% Percentile Bootstrap UCL	N/A
95% BCA Bootstrap UCL	N/A

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	N/A
Theta Star	N/A
nu star	N/A
A-D Test Statistic	N/A
5% A-D Critical Value	N/A
K-S Test Statistic	N/A
5% K-S Critical Value	N/A
Data not Gamma Distributed at 5% Significance Level	

Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	N/A
Maximum	N/A
Mean	N/A
Median	N/A
SD	N/A
k star	N/A
Theta star	N/A
Nu star	N/A
AppChi2	N/A
95% Gamma Approximate UCL	N/A
95% Adjusted Gamma UCL	N/A

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only
Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	0.533
SD	0.611
SE of Mean	0.353
95% KM (t) UCL	1.244
95% KM (z) UCL	1.114
95% KM (jackknife) UCL	1.815
95% KM (bootstrap t) UCL	N/A
95% KM (BCA) UCL	1.9
95% KM (Percentile Bootstrap) UCL	1.9
95% KM (Chebyshev) UCL	2.071
97.5% KM (Chebyshev) UCL	2.737
99% KM (Chebyshev) UCL	4.044
Potential UCLs to Use	
95% KM (BCA) UCL	1.9

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

General UCL Statistics for Data Sets with Non-Detects

User Selected Options

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

Arsenic

General Statistics

Number of Valid Observations 7

Number of Distinct Observations 2

Raw Statistics

Minimum	5
Maximum	8.8
Mean	5.543
Median	5
SD	1.436
Coefficient of Variation	0.259
Skewness	2.646

Log-transformed Statistics

Minimum of Log Data	1.609
Maximum of Log Data	2.175
Mean of log Data	1.69
SD of log Data	0.214

Warning: There are only 2 Distinct Values in this data
There are insufficient Distinct Values to perform some GOF tests and bootstrap methods.
Those methods will return a 'N/A' value on your output display!

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

Warning: A sample size of 'n' = 7 may not adequate enough to compute meaningful and reliable test statistics and estimates!

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

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.453
Shapiro Wilk Critical Value	0.803

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.453
Shapiro Wilk Critical Value	0.803

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 6.598

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	7.016
95% Modified-t UCL	6.688

Assuming Lognormal Distribution

95% H-UCL	6.612
95% Chebyshev (MVUE) UCL	7.475
97.5% Chebyshev (MVUE) UCL	8.318
99% Chebyshev (MVUE) UCL	9.974

Gamma Distribution Test

k star (bias corrected)	12.99
Theta Star	0.427
MLE of Mean	5.543
MLE of Standard Deviation	1.538
nu star	181.9
Approximate Chi Square Value (.05)	151.7
Adjusted Level of Significance	0.0158
Adjusted Chi Square Value	143.4
Anderson-Darling Test Statistic	2.112

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	6.436
95% Jackknife UCL	N/A
95% Standard Bootstrap UCL	N/A
95% Bootstrap-t UCL	N/A

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

Anderson-Darling 5% Critical Value	0.707	95% Hall's Bootstrap UCL	N/A
Kolmogorov-Smirnov Test Statistic	0.516	95% Percentile Bootstrap UCL	N/A
Kolmogorov-Smirnov 5% Critical Value	0.311	95% BCA Bootstrap UCL	N/A
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	7.909
		97.5% Chebyshev(Mean, Sd) UCL	8.933
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	10.94
95% Approximate Gamma UCL	6.646		
95% Adjusted Gamma UCL	7.033		
Potential UCL to Use		Use 95% Student's-t UCL	6.598
		or 95% Modified-t UCL	6.688

Chromium

General Statistics

Number of Valid Data	7	Number of Detected Data	6
Number of Distinct Detected Data	6	Number of Non-Detect Data	1
		Percent Non-Detects	14.29%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	2.6	Minimum Detected	0.956
Maximum Detected	39.8	Maximum Detected	3.684
Mean of Detected	9.492	Mean of Detected	1.62
SD of Detected	14.87	SD of Detected	1.035
Minimum Non-Detect	10	Minimum Non-Detect	2.303
Maximum Non-Detect	10	Maximum Non-Detect	2.303

Warning: There are only 6 Detected Values in this data
Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.544	Shapiro Wilk Test Statistic	0.688
5% Shapiro Wilk Critical Value	0.788	5% Shapiro Wilk Critical Value	0.788
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	8.85	Mean	1.619
SD	13.68	SD	0.945
95% DL/2 (t) UCL	18.9	95% H-Stat (DL/2) UCL	17.24
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	1.584
		SD in Log Scale	0.95
		Mean in Original Scale	8.697
		SD in Original Scale	13.74
		95% Percentile Bootstrap UCL	18.96
		95% BCA Bootstrap UCL	19.49

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

Gamma Distribution Test with Detected Values Only	
k star (bias corrected)	0.573
Theta Star	16.55
nu star	6.881
A-D Test Statistic	1.218
5% A-D Critical Value	0.717
K-S Test Statistic	0.717
5% K-S Critical Value	0.342
Data not Gamma Distributed at 5% Significance Level	
Assuming Gamma Distribution	
Gamma ROS Statistics using Extrapolated Data	
Minimum	2.6
Maximum	39.8
Mean	9.499
Median	4.1
SD	13.57
k star	0.702
Theta star	13.54
Nu star	9.823
AppChi2	3.832
95% Gamma Approximate UCL	24.35
95% Adjusted Gamma UCL	33.45

Data Distribution Test with Detected Values Only	
Data do not follow a Discernable Distribution (0.05)	
Nonparametric Statistics	
Kaplan-Meier (KM) Method	
Mean	8.626
SD	12.75
SE of Mean	5.28
95% KM (t) UCL	18.88
95% KM (z) UCL	17.31
95% KM (jackknife) UCL	18.74
95% KM (bootstrap t) UCL	120.8
95% KM (BCA) UCL	19.1
95% KM (Percentile Bootstrap) UCL	18.92
95% KM (Chebyshev) UCL	31.64
97.5% KM (Chebyshev) UCL	41.6
99% KM (Chebyshev) UCL	61.16
Potential UCLs to Use	
97.5% KM (Chebyshev) UCL	41.6

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Cobalt

General Statistics

Number of Valid Data	7	Number of Detected Data	5
Number of Distinct Detected Data	5	Number of Non-Detect Data	2
		Percent Non-Detects	28.57%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	2.35	Minimum Detected	0.854
Maximum Detected	16.3	Maximum Detected	2.791
Mean of Detected	5.67	Mean of Detected	1.419
SD of Detected	5.987	SD of Detected	0.8
Minimum Non-Detect	50	Minimum Non-Detect	3.912
Maximum Non-Detect	50	Maximum Non-Detect	3.912

Warning: There are only 5 Detected Values in this data

Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

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

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.654	Shapiro Wilk Test Statistic	0.78
5% Shapiro Wilk Critical Value	0.762	5% Shapiro Wilk Critical Value	0.762
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

Assuming Normal Distribution

DL/2 Substitution Method	
Mean	11.19
SD	10.62
95% DL/2 (t) UCL	19
Maximum Likelihood Estimate(MLE) Method	N/A

MLE method failed to converge properly

Assuming Lognormal Distribution

DL/2 Substitution Method	
Mean	1.933
SD	1.094
95% H-Stat (DL/2) UCL	269.9
Log ROS Method	
Mean in Log Scale	1.419
SD in Log Scale	0.693
Mean in Original Scale	5.328
SD in Original Scale	5.021
95% Percentile Bootstrap UCL	8.595
95% BCA Bootstrap UCL	10.4

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	0.825
Theta Star	6.87
nu star	8.253
A-D Test Statistic	0.773
5% A-D Critical Value	0.685
K-S Test Statistic	0.685
5% K-S Critical Value	0.361

Data follow Appr. Gamma Distribution at 5% Significance Level

Data Distribution Test with Detected Values Only

Data Follow Appr. Gamma Distribution at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	2.35
Maximum	16.3
Mean	5.628
Median	3
SD	5.203
k star	1.204
Theta star	4.673
Nu star	16.86
AppChi2	8.574
95% Gamma Approximate UCL	11.07
95% Adjusted Gamma UCL	13.82

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	5.67
SD	5.355
SE of Mean	2.677
95% KM (t) UCL	10.87
95% KM (z) UCL	10.07
95% KM (jackknife) UCL	11.06
95% KM (bootstrap t) UCL	63.8
95% KM (BCA) UCL	9.788
95% KM (Percentile Bootstrap) UCL	10.25
95% KM (Chebyshev) UCL	17.34
97.5% KM (Chebyshev) UCL	22.39
99% KM (Chebyshev) UCL	32.31
Potential UCLs to Use	
95% KM (Chebyshev) UCL	17.34

Warning: Recommended UCL exceeds the maximum observation

Note: DL/2 is not a recommended method.

Iron

General Statistics

Number of Valid Observations 7

Number of Distinct Observations 7

Raw Statistics

Minimum	1090
Maximum	43900
Mean	9500
Median	4110
SD	15249
Coefficient of Variation	1.605
Skewness	2.587

Log-transformed Statistics

Minimum of Log Data	6.994
Maximum of Log Data	10.69
Mean of log Data	8.477
SD of log Data	1.124

Warning: A sample size of 'n' = 7 may not adequate enough to compute meaningful and reliable test statistics and estimates!

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

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 7 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 0.555
Shapiro Wilk Critical Value 0.803
Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic 0.88
Shapiro Wilk Critical Value 0.803
Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL 20700

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL 25002
95% Modified-t UCL 21639

Assuming Lognormal Distribution

95% H-UCL 58087
95% Chebyshev (MVUE) UCL 22798
97.5% Chebyshev (MVUE) UCL 29204
99% Chebyshev (MVUE) UCL 41787

Gamma Distribution Test

k star (bias corrected) 0.588
Theta Star 16168
MLE of Mean 9500
MLE of Standard Deviation 12393
nu star 8.226
Approximate Chi Square Value (.05) 2.867
Adjusted Level of Significance 0.0158
Adjusted Chi Square Value 2.002

Anderson-Darling Test Statistic 0.908
Anderson-Darling 5% Critical Value 0.732
Kolmogorov-Smirnov Test Statistic 0.372
Kolmogorov-Smirnov 5% Critical Value 0.321
Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL 27261
95% Adjusted Gamma UCL 39035

Data Distribution

Data appear Lognormal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL 18980
95% Jackknife UCL 20700
95% Standard Bootstrap UCL 18128
95% Bootstrap-t UCL 85663
95% Hall's Bootstrap UCL 83148
95% Percentile Bootstrap UCL 20546
95% BCA Bootstrap UCL 21913
95% Chebyshev(Mean, Sd) UCL 34623
97.5% Chebyshev(Mean, Sd) UCL 45494
99% Chebyshev(Mean, Sd) UCL 66847

Potential UCL to Use

Use 95% Chebyshev (MVUE) UCL 22798

Manganese

General Statistics

Number of Valid Observations 7
Number of Distinct Observations 7

Appendix B

ProUCL Input for Upper Confidence Limits on the Mean
Locomotive Building Area Surface Water
Plum Brook Ordnance Works, Sandusky, Ohio

Raw Statistics

Minimum	381
Maximum	2220
Mean	1192
Median	1050
SD	709.3
Coefficient of Variation	0.595
Skewness	0.308

Log-transformed Statistics

Minimum of Log Data	5.943
Maximum of Log Data	7.705
Mean of log Data	6.905
SD of log Data	0.671

Warning: A sample size of 'n' = 7 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 7 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	0.912
Shapiro Wilk Critical Value	0.803

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.928
Shapiro Wilk Critical Value	0.803

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	1713

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL	1666
95% Modified-t UCL	1718

Assuming Lognormal Distribution

95% H-UCL	2714
95% Chebyshev (MVUE) UCL	2544
97.5% Chebyshev (MVUE) UCL	3123
99% Chebyshev (MVUE) UCL	4262

Gamma Distribution Test

k star (bias corrected)	1.791
Theta Star	665.3
MLE of Mean	1192
MLE of Standard Deviation	890.4
nu star	25.08
Approximate Chi Square Value (.05)	14.67
Adjusted Level of Significance	0.0158
Adjusted Chi Square Value	12.33
Anderson-Darling Test Statistic	0.34
Anderson-Darling 5% Critical Value	0.712
Kolmogorov-Smirnov Test Statistic	0.233
Kolmogorov-Smirnov 5% Critical Value	0.314

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	2037
95% Adjusted Gamma UCL	2423

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	1633
95% Jackknife UCL	1713
95% Standard Bootstrap UCL	1606
95% Bootstrap-t UCL	1806
95% Hall's Bootstrap UCL	1554
95% Percentile Bootstrap UCL	1605
95% BCA Bootstrap UCL	1600
95% Chebyshev(Mean, Sd) UCL	2360
97.5% Chebyshev(Mean, Sd) UCL	2866
99% Chebyshev(Mean, Sd) UCL	3859

Potential UCL to Use

Use 95% Student's-t UCL 1713

APPENDIX C

TOXICOLOGICAL PROFILES

Introduction to the Toxicological Profiles

1.0 Purpose and Use of the Toxicological Profiles

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

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

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

2.0 Chemical Identity

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

3.0 Physical Properties

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

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

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

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

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

where:

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

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

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

where:

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

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

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

where:

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

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

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

where:

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

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

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

where:

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

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

4.0 Environmental Release, Fate and Transport

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

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

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

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

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

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

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

where:

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

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

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

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

where:

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

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

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

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

where:

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

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

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

where:

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

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

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

5.0 Toxicokinetics

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

6.0 Dermal Exposure

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

7.0 Toxicity Evaluation

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

7.1 Noncancer Evaluation

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

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

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

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

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

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

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

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

7.2 Carcinogenicity Evaluation

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

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

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

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

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

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

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

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

7.3 Hierarchy for Selecting Toxicity Data

Toxicity values generally are chosen using the following hierarchy:

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

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

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

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

8.0 References

Bintein, S. and J. Devillers, 1993, "Nonlinear Dependence of Fish Bioconcentration on n-Octanol/Water Partition Coefficient," ***SAR and QSAR in Environmental Research***, 1: 29-39.

Devillers, J., S. Bintein and D. Domine, 1996, "Comparison of BCF Models Based on Log P," ***Chemosphere***, 33(6): 1047-1065.

Hart, R.W. and L.T. Frame, 1996, "Toxicological Defense Mechanisms and How They May Affect the Nature of Dose-Response Relationships," ***Biological Effects of Low Level Exposure (BELLE) Newsletter***, 5(1): 1-16.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Lyman, W. J., W. F. Reehl and D. H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods, Environmental Behavior of Organic Compounds***, American Chemical Society, Washington, DC.

National Institute of Standards and Technology (NIST), 2010, ***NIST Chemistry WebBook***, on line.

Travis, C.C. and A.D. Arms, 1988, "Bioconcentration of Organics in Beef, Milk and Vegetation," ***Environmental Science and Technology***, 22(3): 271-274.

U.S. Environmental Protection Agency (EPA), 1986, "Guidelines for Carcinogen Risk Assessment," ***Federal Register***, 51(185): 33992-34003.

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

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: 9285.6-03, 25 March.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 1998, ***Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Volume Two***, Peer Review Draft, Office of Solid Waste and Emergency Response, Washington, DC, EPA530-D-98-001B, July.

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, DC, 9355.4-24, December.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2005, ***Guidelines for Carcinogen Risk Assessment***, Risk Assessment Forum, Washington, D.C., March, EPA/630/P-03/001B.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

METALS

ALUMINUM (7429-90-5)

1.0 Introduction and Physical Properties

Aluminum is a naturally occurring **metal** (ATSDR, 2008). It is the third most abundant element in nature, constituting approximately 8 percent of the minerals in the earth's crust. Aluminum is highly reactive; thus, is always found in combination with other substances such as oxygen, fluoride or silica. Aluminum is used in a wide variety of metallurgical applications, including packaging (particularly food items), kitchen utensils, auto bodies and components, airplanes, building components, electrical transmission, and to adsorb occluded gases in making steel (ATSDR, 2008; Elinder and Sjogren, 1986; HSDB, 2010). Aluminum compounds are used in testing for gold, arsenic and mercury, in metal alloys, in protective coatings, paints and printer inks, in cosmetics, food additives and some pharmaceuticals, as coagulating agents in the treatment of drinking water, and in photography flash bulbs and fireworks. Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
26.98[a] (Element)	NA	NA	1500[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-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 29 November.

[b]Baes, C.F., R.D. Sharp, A.L. Sjoreen, R.W. Shor, 1984, ***A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture***, Health and Safety Division, Oak Ridge National Laboratory, ORNL-5786, September.

[c]Solubility of aluminum compounds varies from essentially insoluble in hot or cold water to highly soluble to highly reactive (e.g., aluminum chloride reacts explosively with water liberating heat and hydrogen chloride gas). Agency for Toxic Substances and Disease Registry (ATSDR), 2008, ***Update Toxicological Profile for Aluminum***, U.S. Department of Health and Human Services, Atlanta, Georgia, July, on line.

2.0 Environmental Fate and Transport

Aluminum is one of the few elements for which natural release far outweighs anthropogenic release (ATSDR, 2008; HSDB, 2010). The predominant natural process is the weathering and release of dust from soil and rocks into the air. Major anthropogenic sources include aluminum mining and smelting, coal combustion, and other mining or industrial activities that process crustal materials and release aluminum to the atmosphere. Aluminum exists in the air as particulates that presumably are subject to wet and dry deposition. Large particulates predominate, which suggests that deposition occurs not far from the point of release.

Aluminum is deposited on soil from the atmosphere (ATSDR, 2008). Other anthropogenic sources to soil include wastes from mines, smelters and coal combustion. Aluminum in soil exists in several soluble and insoluble forms, depending on pH and the nature of the minerals and organic material present in the soil. Generally, increasing acidity (decreasing pH) mobilizes aluminum and facilitates leaching.

Aluminum exists naturally in water in several hydrated and complexed forms (ATSDR, 2008). Sources include solution from surrounding soil, precipitation, and runoff and erosion from soil. The extent to which aluminum in water settles out is highly variable and depends on pH and the other constituents present in the water with which aluminum may form compounds and complexes. Generally an equilibrium is established between soluble and insoluble forms.

Bioconcentration in fish is largely a function of water quality (e.g., pH and total organic carbon). Highest concentrations of aluminum are generally found in the gills and lowest concentrations are found in edible muscle tissue. Concentrations decrease as the fish pass through the larval and juvenile stages. Therefore, the bioconcentration of aluminum in edible fish tissues is not expected to represent a toxicologically significant source to humans (EPA, 1995).

Acid conditions in soil mobilize aluminum for uptake by plants, but the extent of uptake and the distribution between the roots and the aerial parts is highly dependent on the species of plant (ATSDR, 2008). Teas and some herbs may bioconcentrate aluminum; however, these items are unlikely to contribute significantly to the diet. Aluminum does not accumulate significantly in cow's milk or beef, and is not expected to undergo biomagnification in terrestrial food chains. For these reasons biotransfer is not expected to contribute significantly to human exposure and biotransfer factors are not compiled or estimated.

3.0 Toxicokinetics

Aluminum is poorly absorbed following inhalation exposure (ATSDR, 2008). Evidence suggests that the epithelium of the upper and lower respiratory tract can absorb aluminum, and that absorption is somewhat more efficient for aluminum fumes (occupational exposure) rather than dust. Aluminum can be retained in the lungs for long periods. Also, it has been shown that aluminum absorbed by the olfactory epithelium can reach the brain directly via axonal transfer.

Dietary aluminum is absorbed from the GI tract in the range of 0.1 to 0.3 or perhaps 0.5 percent (ATSDR, 2008). Unusually bioavailable forms such as carboxylic acid complexes may be absorbed at 1 percent of the administered dose. Ingestion of large amounts of aluminum in antacid preparations can increase total absorption, but factors other than the amount ingested appear to reduce the proportion of the total dose that is absorption.

ATSDR (2008) concluded that aluminum is "essentially not absorbed dermally," although the data are confusing and one study in mice verifies that dermal uptake occurs. Quantitative data were not located.

Normal humans have a body burden of aluminum in the 30 to 50 mg range, with approximately one-half located in the skeleton and one-fourth in the lungs (ATSDR, 2008). Body burden generally increases with age. The amount in the lungs probably reflects accumulation from inhalation of aluminum in dust. Levels in brain tissue range from 0.25 to 0.75 mg/kg wet weight in the white matter and approximately twice that in the gray matter.

Aluminum following inhalation exposure is associated largely with the lungs and thoracic lymph nodes (ATSDR, 2008). In humans, increased levels are seen also in the liver and spleen. Increased levels in these tissues have not been seen in animals exposed via inhalation. Some inhaled

particulates are cleared from the respiratory tract by mucocilliary elevation and are swallowed, thereby becoming available for GI absorption.

Animal studies with ingested aluminum suggest that distribution is preferentially to various regions of the brain (ATSDR, 2008). Prolonged studies with rats indicate that distribution also occurs to bone, muscle and kidneys, but that tissue levels return to normal shortly after exposure ceases. Limited evidence in animals suggests that transplacental distribution and excretion via lactation also occur. Vitamin D and related compounds may increase uptake and tissue retention. Distribution studies following administration by various parenteral routes yielded quite variable results, suggesting that the target tissues may be specific to the form of aluminum administered. The brain was clearly not reported as the most predictable target tissue for all forms of aluminum administered to all species by parenteral routes.

Aluminum in the body may occur in the form of free ions (Al^{+3}), low-molecular-weight complexes (particularly stable chelates), macromolecular complexes, or covalently bound macromolecular complexes (ATSDR, 2008). All forms may be metabolically active. Essentially irreversible complexes may be formed in the nucleus and chromatin material of the cell.

The kidney is the primary organ for excretion of absorbed aluminum (ATSDR, 2008). Urinary excretion patterns after inhalation exposure (i.e., prolongation of elevated urinary levels of aluminum) indicate that body burden and excretion half-life both increase with prolonged exposure. Excretion is lower in premature infants than full-term infants, probably because glomerular filtration is underdeveloped before birth. This suggests that the unborn and premature may be unusually sensitive to the effects of aluminum.

4.0 Dermal Exposure

As noted above, ATSDR (2008) concluded that aluminum is essentially not absorbed dermally. Therefore, the likelihood of systemic toxic effects arising from dermal exposure to aluminum is very small, and no attempt is made to quantify dermal uptake or risk.

5.0 Noncancer Effects Evaluation

Aluminum is relatively nontoxic via ingestion to physiologically normal humans, but recently concern has arisen regarding neurological effects, including Alzheimer's disease and similar forms of dementia (EPA, 2006). Much of this concern stems from studies with large oral doses of soluble aluminum salts (e.g., aluminum hydroxide) and from medical procedures that bypass the GI absorption barrier (i.e., parenteral administration). A review of several animal studies with many different forms of aluminum suggests that the developing infant is potentially the most sensitive member of the human population. A provisional chronic oral RfD of 1E+0 mg/kg-day was derived from a LOAEL of 100 mg/kg-day for minimal and equivocal neurological effects in the offspring of mice. The mice were fed diets fortified with aluminum lactate throughout gestation and lactation. An uncertainty factor of 100 (factor of 10 for interspecies extrapolation, and factors of 3 each for a minimal LOAEL and for intraspecies variation) was used. Confidence in the oral RfD is low. The nervous system is the target organ for oral exposure to aluminum. (The nervous system rather than the CNS is chosen as the target organ because it cannot be stated unequivocally that some of the neurologic effects observed do not involve the peripheral nerves.)

EPA (1997) provides no subchronic oral RfD for aluminum, and one cannot be derived from the key study for the chronic oral RfD. Therefore, the chronic oral RfD of 1E+0 mg/kg-day is adopted as sufficiently protective for subchronic oral exposure.

Occupational exposure to aluminum metal dust and aluminum oxide is associated with pulmonary fibrosis and related respiratory effects such as cough, emphysema, pneumonia and decreased pulmonary function (ACGIH, 1991). The toxicity of occupational exposure to dusts of aluminum pyro powders is somewhat higher, and occupational exposure to salts such as the sulfate and chloride may yield hydrolysis to the corresponding acids, which are irritants in their own right. Pulmonary effects were also observed in animals exposed to powders and dusts of metallic aluminum and various aluminum compounds.

Results of testing Canadian gold and uranium miners prophylactically treated with aluminum oxide powders to prevent silicosis associated aluminum exposure with impaired cognitive performance (EPA, 2006). The likelihood of cognitive impairment was directly related to the duration of exposure. Neurological performance was also impaired in aluminum foundry workers exposed to fumes and aluminum dust (4.6 to 11.5 mg aluminum/m^3) for a mean of 12 years. Other studies also implicate aluminum as a cause of neurological impairment and neurophysical effects in exposed smelter workers and aluminum welders. A provisional inhalation RfC is based on the LOAEL of 4.6 mg aluminum/m^3 in the study of foundry workers described above. The lower end of the exposure concentration adjusted for intermittent exposure is 1.64 mg/m^3. Application of an uncertainty factor of 300 (factors of 10 each to estimate a NOAEL from a LOAEL and for interspecies extrapolation, and a factor of 3 to address deficiencies in the inhalation toxicity data base) yields an RfC of 5E-3 mg/m^3, which is equivalent to a chronic inhalation RfD of 1.4E-3 mg/kg-day. Confidence in the inhalation RfC is medium. The nervous system is the target organ for inhalation exposure to aluminum.

EPA (1997) provides no subchronic inhalation RfC for aluminum, nor can one be estimated from the key study used for the chronic RfC. Therefore, the chronic inhalation RfC of 5E-3 mg/m^3 (1.4E-3 mg/kg-day) is adopted as sufficiently protective for subchronic oral exposure.

6.0 Carcinogenicity Evaluation

EPA (2006) reviewed the human and laboratory animal data regarding the potential carcinogenicity of aluminum and concluded that the carcinogenicity cannot be determined. The human data are insufficient for drawing conclusions and the animal data are largely negative. Potency factors are not derived.

7.0 Toxicity Summary

Toxicity values for aluminum are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1E+0	1E+0	NS	5E-3/ 1.4E-3	5E-3/ 1.4E-3	NS	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

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
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: NS = nervous system. [a]Dermal exposure is not evaluated.										

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2008, ***Update Toxicological Profile for Aluminum***, U.S. Department of Health and Human Services, Atlanta, Georgia, September.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 46-47.

Elinder, C-G. and B. Sjogren, 1986, "Aluminum," in Friberg, L., G.F. Nordberg and V.B. Vouk, 1986, ***Handbook on the Toxicology of Metals,*** Second Edition, Volume II: Specific Metals, Elsevier Science Publishers, New York, NY, pp. 1-25.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 2006, ***Draft Provisional Peer-Reviewed Toxicity Values for Aluminum (CASRN 7429-90-5)***, National Center for Environmental Assessment, Cincinnati, OH, 23 October.

ARSENIC (7440-38-2)

1.0 Introduction and Physical Properties

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

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

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.

Note 1: Variable: inorganic arsenic compounds are not likely to volatilize; some organic arsenic compounds are low-boiling liquids or gases at normal temperatures.

Note 2: Variable: inorganic arsenic compounds range from practically insoluble to freely miscible in water; most organic arsenic compounds are not readily soluble, most arsenic acid compounds are soluble to freely miscible.

[a] Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 21 April.

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

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

2.0 Environmental Fate and Transport

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

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

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

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

3.0 Toxicokinetics

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

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

4.0 Dermal Exposure

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

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

5.0 Noncancer Effects Evaluation

Inorganic arsenic may be an essential nutrient, at least for food-producing domestic animals, exerting beneficial effects on growth, health and feed conversion efficiency (Underwood, 1977). A lethal dose of arsenic trioxide in humans is 70-180 mg, approximately 50 to 140 mg arsenic (Ishinishi et al., 1986). Acute oral exposure of humans to high doses of arsenic produces liver swelling, skin lesions, disturbed heart function and neurological effects. The only noncancer effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyper pigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (EPA, 2010). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico. EPA (2010) verified an RfD of 3E-4 mg/kg-day for chronic oral exposure, based on a NOAEL of 8E-4 mg/kg-day for hyper pigmentation and kertatosis of the skin from the Chinese data. An uncertainty factor of 3 was applied. An increased incidence of Blackfoot disease was also observed, which may not be related to arsenic alone. Goyer (1991) describes black-foot disease

as a peripheral vascular disorder manifested as acrocyanosis and Raynaud's disease, which may progress to gangrene. EPA (2010) notes that the skin is the more sensitive target organ. Nonetheless, in keeping with EPA (1989) guidance regarding selection of target organ, both the skin and peripheral vascular system are selected as target organs for prolonged oral exposure to arsenic. Confidence in the RfD is medium.

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

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

6.0 Carcinogenicity Evaluation

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

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

7.0 Toxicity Summary

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

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

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
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; PVS = peripheral vascular system. [a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.										

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Arsenic***, Draft for Public Comment, U.S. Public Health Service, Atlanta, Georgia, September, on line.

Goyer, R.A., 1991, "Toxic Effects of Metals," in ***Casarett and Doull's Toxicology, The Basic Science of Poisons***, Fourth Edition, Amdur, M.O., J. Doull and C.D. Klaassen, Eds., Pergamon Press, New York, pp. 623-680.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Ishinishi, N., K. Tsuchiya, M. Vahter and B.A. Fowler, 1986, "Arsenic," In: Friberg, L., G.F. Nordberg and V.B. Vouk, Eds., ***Handbook on the Toxicology of Metals***, Volume II, Second Edition, New York: Elsevier Science Publishers B.V., pp. 43-83.

Lewis, R.J., Sr., 1997, ***Hawley's Condensed Chemical Dictionary***, Thirteenth Edition, John Wiley & Sons, Inc., New York, pp. 92-93.

Opresko, D.M., R.A. Young, R.A. Faust, et al., 1998, "Chemical Warfare Agents: Estimating Oral Reference Doses," ***Reviews in Environmental Contamination and Toxicology***, 156: 1-183.

Underwood, E.J., 1977, ***Trace Elements in Human and Animal Nutrition***, Fourth Edition, New York: Academic Press, pp. 424-429.

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

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

CHROMIUM (7440-47-3)

1.0 Introduction and Physical Properties

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

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

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

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

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

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

2.0 Environmental Fate and Transport

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

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

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

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

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

3.0 Toxicokinetics

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

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

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

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

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

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

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

4.0 Dermal Exposure

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

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

5.0 Noncancer Effects Evaluation

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

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

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

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

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

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

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

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

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

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

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

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

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

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

6.0 Carcinogenicity Evaluation

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

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

7.0 Toxicity Summary

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

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

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

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

8.0 References

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

Goyer, R.A., 1991, "Toxic Effects of Metals," in ***Casarett and Doull's Toxicology The Basic Science of Poisons***, Fourth Edition, M.O Amdur, J. Doull and C.D. Klaassen, eds., Pergamon Press, New York, pp. 623-680.

U.S. Environmental Protection Agency (EPA), 1980, "Appendix C – Guidelines and Methodology Used in the Preparation of Health Effect Assessment Chapters of the Consent Decree Water Criteria Documents," ***Federal Register***, 45(231): 79347-79357.

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 1998a, ***Toxicological Review of Hexavalent Chromium (CAS No. 18540-29-9) In Support of Summary Information on the Integrated Risk Information System (IRIS)***, August, on line.

U.S. Environmental Protection Agency (EPA), 1998b, ***Combustion Technical Assistance Center (CTAC) Request No. 70: Chromium Values***, National Center for Environmental Assessment, Cincinnati, OH, 22 September.

U.S. Environmental Protection Agency (EPA), 1998c, ***Combustion Technical Assistance Center (CTAC) Request No. 77: Information Regarding Chromium (III) and (VI) for Site-Specific Risk Assessment***, National Center for Environmental Assessment, Cincinnati, OH, 5 October.

U.S. Environmental Protection Agency (EPA), 2003, Telephone conversation between Ted Simon, EPA Region IV, and P. Goetchius, Shaw E & I, 10 October.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

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, 2010). 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-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable; ND = no data.
Note 1: Cobaltous chloride as a typical cobalt salt: soluble.
[a]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line.
[b]Baes, 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*** .
[c]Budavari, 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, 2010). 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, 2010).

HSDB (2010) 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, 2010). 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, 2010). 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 cardiomyopthy. 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 3E-3 to 1.1E-2 mg/day (4E-5 to 2E-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 1.6E-1 to 3.2E-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 1.8E-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 3E-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 3E-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. There 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 $\mu g/m^3$ was identified by EPA (2008). A subchronic RfC of 2E-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 5.7E-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/m^3. Using a benchmark response of 10 percent in extra risk, a provisional URF of 9E-3$(\mu g/m^3)^{-1}$ was derived for cobalt sulfate. This equals an inhalation SF of 3.2E+1 $(mg/kg\text{-}day)^{-1}$.

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

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: RT = respiratory tract.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2004, ***Toxicological Profile for Cobalt***, Draft for Public Comment, U.S. Department of Health and Human Services, Atlanta, Georgia, April.

Elinder, C.-G and L. Friberg, 1986, "Cobalt," In: Friberg, L., G.F. Nordberg and V.B. Vouk, Eds., ***Handbook on the Toxicology of Metals,*** Second Edition, Volume II: Specific Metals, Elsevier Science Publishers, New York, NY, pp. 211-232.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Roche, M., and M. Layrisse, 1956, "The Effect of Cobalt on Thyroidal Uptake of I^{131}", ***Journal of Clinical Endocrinology and Metabolism***, Volume 16, pp. 831-833.

U.S. Environmental Protection Agency (EPA), 2002, ***Risk Assessment Issue Paper for: Derivation of a Provisional RfD for Cobalt and Compounds (CASRN 7440-48-4)***, National Center for Environmental Assessment, Cincinnati, OH, 15 January.

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 Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2008, ***Provisional Peer-Reviewed Toxicity Values for Cobalt (CASRN 7440-48-4)***, National Center for Environmental Assessment, Cincinnati, OH, 25 August.

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, 2010). 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-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable; ND = no data.
Note 1: Ferrous sulfate as a typical iron salt: soluble.
[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
[b] Baes, 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, 2010). 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, 2010). 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, 2010). 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 sequellae 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 hemochromotosis, 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 hemochromotosis. Hemochromotosis 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.

8.0 References

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 803-809.

Elinder, C.-G., 1986, "Iron," in Friberg, L., G.F. Nordberg and V.B. Vouk, eds., ***Handbook on the Toxicology of Metals,*** Second Edition, Volume II: Specific Metals, Elsevier Science Publishers, New York, NY, pp. 276-297.

Goyer, R.A., 1991, "Toxic Effects of Metals," in ***Casarett and Doull's Toxicology, The Basic Science of Poisons, Fourth Edition***, M.O. Amdur, J. Doull and C.D. Klaassen, eds., Pergamon Press, New York, pp 623-680.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

National Academy of Sciences (NAS), 1989, ***Recommended Daily Allowances, 10th Edition***, National Academy Press, Washington, DC. (Cited in EPA, 1996).

Spivey Fox, M.R. and J.I. Rader, 1988, "Iron," in Seiler, H.G. and H. Sigel, eds., ***Handbook on Toxicity of Inorganic Compounds***, Marcel Dekker, Inc., New York, pp. 345-354.

U.S. Environmental Protection Agency (EPA), 1996, ***Risk Assessment Issue Paper for: Derivation of a Provisional RfD for Iron (CASRN 7439-89-6)***, National Center for Environmental Assessment, Cincinnati, OH, 23 July. 2008

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 Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2005a, ***Provisional Peer-Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6), Derivation of a Carcinogenicity Assessment***, National Center for Environmental Assessment, Cincinnati, OH, 29 July.

U.S. Environmental Protection Agency (EPA), 2005b, ***Provisional Peer-Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6), Derivation of n Inhalation RfC***, National Center for Environmental Assessment, Cincinnati, OH, 16 June.

U.S. Environmental Protection Agency (EPA), 2006, ***Provisional Peer-Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6), Derivation of Chronic and Subchronic Oral RfDs***, National Center for Environmental Assessment, Cincinnati, OH, 29 July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

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, 2000; HSDB, 2010; 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-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.

Note 1: Metallic manganese decomposes in the presence of water. Manganous chloride as a typical manganese salt: 7.23E+5 mg/L (25˚ C).

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[b]Baes, C.F., R.D. Sharp, A.L. Sjoreen, R.W. Shor, 1984, ***A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture***, Health and Safety Division, Oak Ridge National Laboratory, ORNL-5786, September.

[c]Agency 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, 2010). 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, 2000; HSDB, 2010). 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, 2000). 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, 2000; HSDB, 2010). 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, 2000). Stable tissue levels are maintained by homeostatic regulation of GI absorption and biliary excretion.

Manganese is absorbed by the respiratory tract (ATSDR, 2000), 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 mucocilliary 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, 2000). 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, 2000). 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 (2000),

however, notes that the uptake of manganese across intact skin is "extremely limited," 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 (2010) 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 (2010) 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 (2010) presents a verified chronic inhalation RfC of 5E-5 mg/m^3 (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 (2010) 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.

8.0 References

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

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 876-878.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Keen and Leach, 1988, "Manganese," in Seiler, H.G. and H. Sigel, eds., ***Handbook on Toxicity of Inorganic Compounds***, Marcel Dekker, Inc., New York, pp. 405-415.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

THALLIUM (7440-28-0)

1.0 Introduction and Physical Properties

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

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

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

2.0 Environmental Fate and Transport

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

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

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

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

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

3.0 Toxicokinetics

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

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

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

4.0 Dermal Exposure

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

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

5.0 Noncancer Effects Evaluation

Thallium is highly toxic. Acute oral exposure to thallium and its compounds may cause death preceded by neurological disturbances and effects on the lungs, heart and liver (ATSDR, 1992). Formerly, thallium was used medicinally to induce alopecia to facilitate treatment of ringworm of the scalp, sometimes with disastrous results. The critical effect of chronic oral exposure to low levels of thallium compounds in animals and humans is alopecia (ACGIH, 1992; EPA, 2010). EPA (2010) 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 (EPA, 2010). 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) and liver (altered blood biochemistry) are considered the target organs for oral exposure to thallium, based on other data reviewed by EPA (2010). Effects on the testis and kidney were observed in rats exposed to higher dose rates. The chronic oral RfD for thallium sulfate of 8E-5, when adjusted for differences in molecular weight, is equivalent to a chronic oral RfD for thallium of 6.5E-5 mg/kg-day. This adjustment is based on the assumption that the toxicity of thallium sulfate is due entirely to thallium, rather than to the sulfate moiety. The oral RfD for thallium of 6.5E-5 mg/kg-day will be used in this evaluation.

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

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

6.0 Carcinogenicity Evaluation

Several thallium compounds (thallium oxide, thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium sulfate) are classified as cancer weight-of-evidence Group D substances (not classifiable as to carcinogenicity to humans) (EPA, 2010). No weight-of-evidence classification was located for thallium alone, but the Group D classification can be applied to thallium. Quantitative cancer risk estimates are not derived for Group D chemicals.

7.0 Toxicity Summary

Toxicity values for thallium are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
6.5E-4	6.5E-5	S, L	ND	ND	NA	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.

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
Target organ or critical effect abbreviations: L = liver; S = skin. [a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.										

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1992, ***Toxicological Profile for Thallium***, U.S. Public Health Service, Atlanta, Georgia, July, on line.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

VANADIUM (7440-62-2)

1.0 Introduction and Physical Properties

Vanadium is a widespread **metal**, comprising about 0.014 percent of the earth's crust (Wennig and Kirsch, 1988) with an average concentration of approximately 150 mg/kg (ATSDR, 1992). It may exist in six oxidation states (-1, 0, +2, +3, +4 and +5); +3, +4 and +5 are most important in environmental and biological media. Vanadium is always found combined in nature, generally with oxygen, and occurs in upwards of 50 different mineral ores. It is also associated with fossil fuels. Vanadium is used predominantly in the production of special steels and alloys, some of which have unique application in nuclear power technology (Lagerkvist et al., 1986; HSDB, 2010). Relevant physical properties are compiled below:

MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
50.942[a] (Element)	NA	NA	155[b, c]	NA	NA	NA	Note 1[b]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable; ND = no data.
Note 1: Vanadium pentoxide as a typical vanadium compound: 8E+3 mg/L (reference temperature not given).
[a]Agency for Toxic Substances and Disease Registry (ATSDR), 1992, ***Toxicological Profile for Vanadium***, U.S. Department of Health and Human Services, Atlanta, Georgia, July, on line.
[b]From EPA (2010) EPISuite™ data base.

2.0 Environmental Fate and Transport

Vanadium compounds are released naturally to air through the formation of continental dust, marine aerosols, and volcanic emissions (ATSDR, 1992; HSDB, 2010). Natural release greatly exceeds anthropogenic release. The most important anthropogenic contribution is from the combustion of fuel oil and other fossil fuels, with significant local contributions also from metallurgical and other industrial operations. Most anthropogenic releases to the atmosphere occur in the form of simple or complex vanadium oxides. Vanadium exists as particulates in the atmosphere. Small particles may remain in air for long periods of time and travel long distances. Removal is limited to wet and dry deposition.

The natural release of vanadium to water and soils occurs primarily as a result of weathering of rocks and soil erosion (ATSDR, 1992; HSDB, 2010). Anthropogenic sources include deposition from the atmosphere and the production and industrial use of vanadium and its compounds. Application of mineral fertilizers may lead to the accumulation of vanadium compounds in soil. Natural releases to water and sediment greatly exceed anthropogenic sources, although water pollution in the area of heaped-up or landfilled mine waste can be extensive in mining areas.

The behavior of vanadium in soils is not yet fully understood; however, it is known that the mobility of vanadium in soils is affected by pH (ATSDR, 1992; HSDB, 2010). Vanadium is fairly mobile in neutral or alkaline soils, relative to other metals, but its mobility decreases in acidic soils. Leaching is a potentially significant process, but great variability is expected based on soil characteristics as indicated by the range for K_d given above.

Vanadium in surface water exists largely as vanadyl and vanadate species (ATSDR, 1992; HSDB, 2010). Both forms are known to bind strongly to mineral or biogenic surfaces by adsorption or complexation, reducing the amount of soluble vanadium found in water. Biochemical removal may occur in marine environments. Some marine organisms particularly the ascidians (sea squirts) bioconcentrate vanadium, attaining body concentrations approximately 10,000 times greater than the ambient seawater. The potential for bioconcentration appears to be greater among the lower phyla.

Vanadium in freshwater has some potential for bioconcentration. A study in bluegills estimated BCFs of 51 to 92 L/kg; however, details of the study were not located (HSDB, 2010). Although empirical data are limited, it appears that biomagnification in terrestrial food chains is unlikely (ATSDR, 1992). The potential for bioconcentration in terrestrial plants appears to be greater among the lower phyla than among the seed producers. Since vanadium is unlikely to participate significantly in aquatic and terrestrial food-chain pathways relevant to humans, biotransfer factors are not compiled or estimated.

3.0 Toxicokinetics

Excretion studies with occupationally exposed humans and intratracheal administration studies with vanadyl chloride in rats suggest that pulmonary absorption occurs (ATSDR, 1992). Although quantitative data are not available, it appears that water soluble forms may be more readily absorbed from the lung. Vanadium is poorly absorbed from the GI tract (Lagerkvist et al., 1986). GI uptake of vanadium pentoxide in rats was estimated at about 2.6 percent of the administered dose. GI uptake of oxytartarovanadate by humans was estimated at 0.1 to 1 percent of the administered dose. GI absorption may be somewhat higher in the young than in adults. The International Commission on Radiological Protection (ICRP) (1960) estimate for GI absorption by humans of soluble vanadium compounds is 2 percent. Data regarding dermal uptake of vanadium were not located.

Absorbed vanadium is transported primarily in the blood plasma rather than in the cellular elements (ATSDR, 1992). Distribution following inhalation or oral exposure is generally widespread, with highest levels initially found in the lung, liver and kidney. The kidney is the primary excretory organ, which probably accounts for the relatively high levels observed in this organ during or shortly after administration. Vanadium that is not excreted eventually partitions to the skeleton where it may displace the phosphate radical and persist.

The kinetics of vanadium metabolism are not well understood. Metabolism is limited to interconversion between the vanadyl form (valence of +4) and the vanadate form (valence of +5) that presumably reaches equilibrium (ATSDR, 1992). This interconversion may occur within the GI tract as well as systemically following absorption (EPA, 2000). Vanadate is considered to be the more toxic form because it reacts with several enzymes and is a potent inhibitor of energy metabolism in cell membranes. This is a moot point, however, because of the equilibrium that would be reached following exposure to vanadium of either valence.

The kidney is the main excretory organ for systemically absorbed vanadium (ATSDR, 1992).

4.0 Dermal Exposure

The estimate of GI absorption of 2.6 percent by rats described above is recommended by EPA (2004) as the GAF of 0.026 for vanadium.

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

5.0 Noncancer Effects Evaluation

EPA (2010a) reported the results of an unpublished study in which rats were exposed to dietary levels of 10 or 100 ppm vanadium as vanadium pentoxide (approximately 17.9 or 179 ppm vanadium pentoxide) for 2.5 years. The criteria used to evaluate vanadium toxicity were limited to growth rate, survival, and hair cystine content. The only significant change reported was a decrease in the amount of cystine in the hair of rats in the high dose group. Therefore, the lower dose level (17.9 ppm vanadium pentoxide) was designated a NOAEL. The NOAEL is equivalent to a dose rate of 0.895 mg vanadium pentoxide/kg-day, assuming rats eat food equivalent to 5 percent of their body weight each day. Application of an uncertainty factor of 100 yields the verified chronic oral RfD of 9E-3 mg vanadium pentoxide/kg-day. The oral RfD of 9E-3 mg/kg-day should be applied only to vanadium pentoxide. Confidence in the oral RfD is low. An oral chronic RfD for vanadium of 5E-3 mg/kg-day is derived from the vanadium pentoxide RfD. This is done by multiplying the vanadium pentoxide RfD (9E-3 mg/kg-day) by the molecular weight of vanadium (101.9) in the vanadium pentoxide molecule (V_sO_5) divided by the molecular weight of vanadium pentoxide (181.9) as follow: 9E-3(mg/kg-day) × 101.9/181.0 = 5E-3(mg/kg-day). This approach is also used by EPA (2010b). the chronic RfD is also used as the subchronic RfD.

The only effect observed from exposure to vanadium pentoxide was reduced cystine content of the hair (EPA, 200a). It is unclear if this resulted in fragile or otherwise damaged hair shafts, and it is unclear whether it reflects altered hair follicle function or a more central effect on protein metabolism. Hair is assumed to be the target organ for vanadium.

Effects in humans exposed by inhalation include upper and lower respiratory tract irritation (ATSDR, 1992). The respiratory tract is the target organ for inhalation exposure, but the data are inadequate for derivation of an inhalation RfC or RfD.

6.0 Carcinogenicity Evaluation

No empirical information was located regarding the carcinogenicity of vanadium.

7.0 Toxicity Summary

Toxicity values for vanadium and unspeciated vanadium compounds, and for vanadium pentoxide are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfc/ sRfD	cRfC/ cRfD	TO	WOE	SFo	WOE	URFi	SFi
Vanadium and Compounds										
5E-3	5E-3	hair	ND	ND	NA	ND	ND	ND	ND	ND
Vanadium Pentoxide										
9E-3	9E-3	hair	ND	ND	NA	ND	ND	ND	ND	ND

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfc/ sRfD	cRfC/ cRfD	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 milligram per cubic meter); SFi = inhalation cancer slope factor (risk per microgram per kilogram-day); ND = no data; NA = not applicable.
Target organ abbreviations: K = kidney.
[a]sRfDo and cRfDo should by multiplied by the GAF of 0.026 when applied to dermal exposure.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1992, ***Toxicological Profile for Vanadium***, U.S. Department of Health and Human Services, Atlanta, Georgia, July, on line.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

International Commission on Radiological Protection (ICRP), 1960, ***Report of Committee II on Permissible Dose for Internal Radiation. Recommendations of the ICRP***, Publ. No. 2, Pergamon Press, Oxford, England, p. 163.

Lagerkvist, B., G.F. Nordberg and V. Vouk, 1986, "Vanadium," in: Friberg, L., G.F. Nordberg and V.B. Vouk, eds., ***Handbook on the Toxicology of Metals***, Volume II, Second Edition, New York: Elsevier Science Publishers B.V., pp. 638-663.

U.S. Environmental Protection Agency (EPA), 2000, ***Provisional Toxicity Value Assessment for HWIR: RfD for Vanadium (CASRN 7440-62-2)***, National Center for Environmental Assessment, Cincinnati, OH, May.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

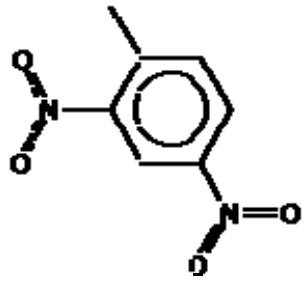
U.S. Environmental Protection Agency (EPA), 2010a, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line, accessed.

U.S. Environmental Protection Agency (EPA), 2010b, ***Regional Screening Level Table, User's Guide (May 2010)***, on line at www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/usersguide.htm.

Wennig, R. and N. Kirsch, 1988, "Vanadium," in Seiler, H.G. and H. Sigel, ***Handbook on Toxicity of Inorganic Compounds***, Marcel Dekker, Inc., New York, pp. 749-765.

EXPLOSIVES

DINITROTOLUENE (DNT)



(2,4-Dinitrotoluene)

1.0 Introduction and Physical Properties

The dinitrotoluenes (DNT) are **SVOCs** that occur as yellow to orange to red needles or crystals at ambient temperatures (HSDB, 2010). They are used predominantly as intermediates in the production of polyurethanes, dyes, rubber, plastics, toluidines, toluenediisocyanates, flexible and rigid foams, surface coatings, and other organic chemicals (ATSDR, 1998; HSDB, 2010). The DNTs are also used as gelatinizing and waterproofing agents in the production of explosives. The DNTs may appear in environmental media as break down products of 2,4,6-trinitrotoluene (TNT). Isomers include 2,3-, 2,4-, 2,5-, 2,6-, 3,4- and 3,5-DNT. Analysis of technical grade DNT yielded the following results: 76.49 percent 2,4-DNT, 18.83 percent 2,6-DNT, 0.65 percent 2,5-DNT, 2.43 percent 3,4-DNT, 1.54 percent 2,3-DNT, 0.040 percent 3,5-DNT, 0.050 percent trinitrotoluene, 0.005 percent cresols, 0.003 percent mononitrobenzene, and 0.003 percent, 0.0005 percent, and 0.006 percent ortho-, meta-, and para-, mononitrotoluenes, respectively. The compounds of most interest in environmental exposure are limited to 2,4- and 2,6-DNT. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
2,3-Dinitrotoluene (602-01-7)										
182.14[a]	2.2[a]	9.3E-8[a] (25°C)[b]	2.57[a]	5.91E-2[c] (25°C)[b]	6.85E-6[c] (25°C)[b]	5.26E-7[a] (25°C)	2.2E+2[d] (25°C)[b]	ND	ND	ND
2,4-Dinitrotoluene (121-14-2)										
182.1[e]	1.98[e]	8.40E-8[i] (25°C)	1.98[e]	2.03E-1[f] (25°C)	7.06E-6[f] (25°C)	1.93E-7[a] (22°C)	2.70E+2[f] (20-25°C)	590.0[g]	814.0[g]	1.35E+4[g]
2,5-Dinitrotoluene (619-15-8)										
182.14[a]	2.2[a]	9.3E-8[a] (25°C)[b]	2.56[a]	5.91E-2[c] (25°C)[b]	6.85E-6[c] (25°C)[b]	5.26E-7[a] (25°C)	2.2E+2[d] (25°C)[b]	ND	ND	ND
2,6-Dinitrotoluene (606-20-2)										
182.1[e]	1.72[e]	7.47E-8[i] (25°C)[b]	1.72[e]	5.91E-2[c] (25°C)[b]	6.85E-6[c] (25°C)[b]	7.46E-7[a] (25°C)	1.82E+2[h] (20-25°C)	558.0[g]	770.0[g]	1.29E+4[g]
3,4-Dinitrotoluene (610-39-9)										
182.14[a]	2.08[a]	9.3E-8[a] (25°C)[b]	2.56[a]	5.91E-2[c] (25°C)[b]	6.85E-6[c] (25°C)[b]	5.26E-7[a] (25°C)	1.00E+2[a] (25°C)	ND	ND	ND

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
3,5-Dinitrotoluene (618-85-9)										
182.14[a]	2.2[a]	9.3E-8[a] (25°C)[b]	2.56[a]	5.91E-2[c] (25°C)[b]	6.85E-6[c] (25°C)[b]	2.50E-6[a] (25°C)	ND	ND	ND	ND

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[b]Assumed.

[c]Calculated as described in Introduction to Toxicity Profiles.

[d]Syracuse Research Corporation (SRC), 2004, *CHEMFATE Data Base*, on line.

[e]U.S. Environmental Protection Agency (EPA), 2003, *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Interim*, Office of Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP March.

[f]U.S. Environmental Protection Agency (EPA), 2001, *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.

[g]U.S. Environmental Protection Agency (EPA), 2003, *Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings*, Office of Emergency and Remedial Response, Washington, DC, 19 June.

[h]U.S. Environmental Protection Agency (EPA), 1996, *Soil Screening Guidance: Technical Background Document*, Office of Emergency and Remedial Response, Washington, DC, EPA/540/R-95/128, Publication 9355.4-17A, PB96-963502, May.

[I i]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

The physical properties of 2,4-DNT can be used for the DNT mixture (25321-14-6) because the 2,4-isomer predominates in the technical grade product.

2.0 Environmental Fate and Transport

The DNTs are released into the environment in waste streams from processes in which they are manufactured or used (ATSDR, 1998; HSDB, 2010). Also, they may form from degradation of TNT in gunpowder or explosives, particularly at TNT manufacturing or military sites. DNTs released to soil are expected to exhibit moderate mobility and may leach to groundwater. No information on biodegradation in soil was found; however, biodegradation may be an important fate process in soil based on the observation that all DNT isomers in surface water to which yeast extract was added were metabolized. DNTs are not expected to hydrolyze in soils.

DNTs released to water, may exhibit weak to moderate adsorption to sediment (ATSDR, 1998; HSDB, 2010). Evaporation from water is not expected to be an important removal process, based on the low Henry's Law constants for these compounds. Biodegradation may be an important fate process in water, as indicated above. DNTs in water may be susceptible to photolysis, based on the behavior of 2,4-DNT, which had photolytic half-lives ranging from 2.7 to 9.6 hours in natural water environments and 43 hours in distilled water.

DNTs released to the atmosphere are expected to exist solely in the vapor phase (HSDB, 2010). They may be susceptible to direct and indirect photolysis. The half-life for reaction with photochemically produced hydroxyl radicals is 84 days.

Bioconcentration of the DNTs is expected to be negligible (ATSDR, 1998; EPA, 1995; HSDB, 2010); therefore, biotransfer factors are not estimated.

3.0 Toxicokinetics

The rapid disappearance of radioactivity from the small intestine of rats following oral administration of radiolabeled 2,4- or 2,6-DNT suggests that absorption from the GI tract is rapid and essentially complete (ATSDR, 1998; EPA, 1992). Species differences in the excretion of the DNTs, however, confound quantification of the extent of GI absorption. Fecal excretion and the GI tract (tissue + contents) accounted for 9.2 to 17.3 percent of the dose of radioactivity 24 hours after oral administration of radiolabeled 2,4-DNT to rats, rabbits, dogs and monkeys (EPA, 1992). Fecal excretion and the GI tract accounted for 83.1 to 85 percent of an oral dose in mice in the same study. Biliary excretion appears to be more important in mice than in the other species tested. Together, the data for the 2,4-isomer suggest that the GI absorption of the DNTs is practically complete.

Body distribution of radioactivity was followed after administration of a ^{14}C-labeled oral dose of 2,4-DNT in male and female rats (ATSDR, 1998). Levels of radioactivity in the liver and kidneys exceeded levels in the plasma or erythrocytes by 5- to 10-fold; levels in other tissues were lower than those in blood. The greater concentrations in the liver and kidneys probably represent metabolites rather than parent compound.

Metabolism of the DNTs involves both oxidative and reductive reactions as well as alkylation (ATSDR, 1998). Oxidative reactions occur primarily in the liver and involve stepwise enzymatic oxidation of the methyl group to the corresponding dinitrobenzyl alcohol, dinitrobenzylaldehyde and dinitrobenzoic acid. Dinitrobenzyl alcohol is subject to conjugation, particularly with gluconuric acid, before excretion via the urine or bile. Acetylation of one of the nitro groups of the dinitrobenzoic acid may also occur, yielding the corresponding (n-acetyl)amino nitrobenzoic acid, which is observed as a minor urinary metabolite.

Two types of reduction reactions involving metabolites excreted via the bile or GI tract are accomplished anaerobically by intestinal microflora (ATSDR, 1998). The first reaction involves lysis of the glucuronide conjugate described above to release the corresponding benzyl alcohol. The second reaction involves nitroreduction, resulting in transforming one or both of the nitro groups to amine groups, probably through nitroso and hydroxylamino intermediates. The amino and hydroxylamino benzyl alcohols are reabsorbed, completing the enterohepatic cycle.

The amine group on the reabsorbed benzyl alcohol is subject to partial oxidation in the liver to yield the corresponding hydroxylamino compound, potentially providing a second pathway for formation of these compounds (ATSDR, 1998). The hydroxylamino compounds may conjugate with sulfate, forming an unstable product that decomposes to yield carbonium or nitrenium ions that bond covalently with hepatic macromolecules, giving rise to the first stages of hepatic carcinogenesis. From the foregoing, it appears that microbial transformation and hepatic recirculation are involved in the carcinogenicity observed in laboratory rodents (Section 6.0).

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for the DNTs and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Urinary excretion data obtained from workers exposed to the DNTs indicates that dermal absorption occurs (ATSDR, 1998) but multiple exposure routes were involved and the extent of dermal uptake cannot be quantified. Empirical data regarding dermal uptake from soil were not located. Lacking empirical data, the EPA (2004) default ABS of 0.1 for SVOCs is adopted for the DNTs for this evaluation. t*, K_p, τ, FA and B values are provided by EPA (2003) or estimated by the EPA (2003) methods as follows:

Chemical	t*	K_p	τ	FA	B
2,3-Dinitrotoluene[a]	2.64	4.29E-3	1.10	1	0.0
2,4-Dinitrotoluene[b]	2.69	3.1E-3	1.12	1	0.0
2,5-Dinitrotoluene[a]	2.64	4.29E-3	1.10	1	0.0
2,6-Dinitrotoluene[b]	2.69	2.1E-3	1.12	1	0.0
3,4-Dinitrotoluene[a]	2.64	3.57E-3	1.10	1	0.0
3,5-Dinitrotoluene[a]	2.64	4.84E-3	1.10	1	0.0

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Estimated as described in Introduction to Toxicity Profiles.
[b]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 Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

The estimated values for K_p for all the DNTs fell within the effective prediction domain.

5.0 Noncancer Effects Evaluation

Data regarding the toxicity of oral exposure to 2,3-DNT are limited to the statement that the 2,4-, 2,6- and 3,4-isomers are more toxic to rats than the 2,3- or 2,5-isomers (HSDB, 2010).

Acute oral exposure to 2,4-DNT induces methemoglobinemia; dogs appear to be more sensitive than rats, which are more sensitive than mice (ATSDR, 1998). Oral LD_{50} values include 270 mg/kg for rats, 1630 mg/kg for mice and 1300 mg/kg for guinea pigs (ATSDR, 1998; HSDB, 2010), establishing the rat as the species most sensitive to acute oral exposure. Subchronic or chronic oral exposure to 2,4-DNT in rats is associated with depressed weight gain, reticulocytosis and splenic hemosiderosis, and decreased spermatogenesis (HSDB, 2010). Some rats had widespread and stiff-legged gaits and demyelination in the cerebellum and brain stem. A verified chronic oral RfD of 2E-3 mg/kg-day for 2,4-DNT is based on neurotoxicity, hematologic effects and biliary tract hyperplasia in dogs given capsules containing the test substance for 2 years (EPA, 2010). Progressive paralysis, accompanied by degenerative lesions in the central nervous system, were observed in dogs treated with 10 mg/kg-day. Hematologic effects, including methemoglobinemia and its sequelae, were observed in dogs treated with 1.5 mg/kg-day, which is the LOAEL for this study. No adverse effects were observed in dogs treated with 0.2 mg/kg-day. Application of an uncertainty factor of 100 to the NOAEL of 0.2 mg/kg-day yields an RfD of 2E-3 mg/kg-day. Confidence in the RfD is high. The CNS, erythrocyte and liver appear to be the target organs for prolonged oral exposure, at least in dogs. EPA (1997) adopted the verified chronic oral RfD as the preliminary RfD for subchronic oral exposure.

HSDB (2010) reports oral LD_{50} values for 2,5-DNT of 707 mg/kg for rats and 1231 mg/kg for mice. Data regarding the effects of prolonged oral exposure were not reported.

Oral LD_{50} values for 2,6-DNT for rats (177 to 665 mg/kg) are similar to those for mice (621 to 714 mg/kg) (ATSDR, 1998; HSDB, 2010). Dogs, however, appear to be more sensitive than rodents in subchronic studies. A provisional chronic oral RfD of 1E-3 mg/kg-day for 2,6-DNT is based on a 13-week dietary study with 2,6-DNT in dogs (EPA, 1997). The NOAEL in this study was 4 mg/kg-day, somewhat higher than the LOAEL for 2,4-DNT. An uncertainty factor of 3000 was applied. The CNS, erythrocyte, liver and kidney appear to be the target organs for prolonged oral exposure to 2,6-DNT. The oral data in dogs suggest that the 2,6-isomer is somewhat less toxic than the 2,4-isomer for prolonged oral exposure. The oral RfD for the 2,6-isomer, however, is less than the oral RfD for the 2,4-isomer. This apparent contradiction is an artifact of the lesser quality data base for 2,6-DNT and, consequently, the higher uncertainty factor used to derive the RfD for the 2,6-isomer. EPA (1997) derived a provisional subchronic oral RfD of 1E-2 mg/kg-day by applying an uncertainty factor of 300 to the NOAEL of 4 mg/kg-day in the 13-week dog study.

The verified chronic oral RfD of 2E-3 mg/kg-day and the provisional subchronic oral RfD of 2E-3 mg/kg-day for 2,4-DNT can be used for DNT mixtures and the other DNT isomers for which oral exposure data sufficient for RfD derivation are not available. The RfD for 2,4-DNT is chosen over the RfD for the 2,6-isomer because the 2,4-isomer predominates in the technical product and because the RfD for the 2,4-isomer is compromised by much less uncertainty.

Occupational studies indicate that acute exposure to the DNTs produces methemoglobinemia, which causes headache, irritability, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, unconsciousness and, in extreme cases, death (ACGIH, 1991). Occupational exposure may lead to increased risk of ischemic heart disease, possibly arising from decreased oxygen-carrying capacity of the blood. The data are insufficient to develop inhalation RfCs for the DNT isomers.

6.0 Carcinogenicity Evaluation

Carcinogenicity data for the DNTs are somewhat confusing. Treatment of rats and mice with purified 2,4-DNT generally yields no increased incidence of neoplasia, but treatment with technical grade DNT, 2,4-DNT containing as little as 2 percent 2,6-DNT, or with 2,6-DNT itself generally yields a positive neoplastic response (EPA, 2010). Initiation/promotion tests suggest that 2,6-DNT may be a complete carcinogen and 2,4-DNT may be a promoter.

EPA (2010) classified the 2,4-/2,6-DNT mixture in EPA cancer weight-of-evidence Group B2 (probable human carcinogen) on the basis of multiple tumor types in male and female rats and renal tumors in male mice in several dietary studies. Occupational data provide no evidence of carcinogenicity in humans, but the studies reviewed lack the power to reveal small differences in cancer risk (ACGIH, 1991). EPA (2010) verified an oral SF of 6.8E-1 per mg/kg-day for exposure to the 2,4-/2,6-DNT mixture based on an increased incidence of a variety of tumors in female rats. The test material contained approximately 98 percent 2,4-DNT and 2 percent 2,6-DNT. The B2 classification and the oral SF of 6.8E-1 per mg/kg-day are used in this evaluation for 2,6-DNT and the 2,4-/2,6-DNT mixture. Given the uncertainty about to potential carcinogenicity of the 2,4-isomer alone, and the likelihood that small amounts of the 2,6-isomer may be present, it is deemed prudent to apply the B2 classification and the oral SF of 6.8E-1 per mg/kg-day to the 2,4-isomer as well. The

data are insufficient for development of potency factors for inhalation exposure. Data regarding the carcinogenicity of the other DNT isomers were not located.

7.0 Toxicity Summary

Toxicity values for the DNTs are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a, b]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2,4-Dinitrotoluene										
2E-3	2E-3	E, CNS, L	ND	ND	NA	B2	6.8E-1	ND	ND	ND
2,6-Dinitrotoluene										
1E-2	1E-3	E, CNS, L	ND	ND	NA	B2	6.8E-1	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 abbreviations: E = erythrocyte; CNS = central nervous system; L = liver.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.
[b]The cancer evaluation for the 2,4-/2,6-DNT mixture is applied to any DNT mixture containing 2,4- and 2,6-DNT (e.g., the technical grade material) as well as the individual 2,4- and 2,6-isomers.

The oral RfD values for 2,4-DNT should be applied to the technical grade material, any 2,4-/2,6 mixture, and any isomers other than pure 2,6-DNT. The cancer weight-of-evidence group and the verified oral SF for the 2,4-/2,6-DNT mixture should not be applied to isomers other than 2,4- and 2,6-DNT because the potential for the other isomers to cause cancer has not been evaluated.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1998, ***Update Toxicological Profile for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene***, U.S. Department of Health and Human Services, Atlanta, Georgia, December, PB/99/122004, December.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 506-511.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 17 February.

U.S. Environmental Protection Agency (EPA), 1992, ***Health Advisory for 2,4- and 2,6-Dinitrotoluene (DNT)***, Health and Ecological Criteria Division, Office of Water, Washington, DC.

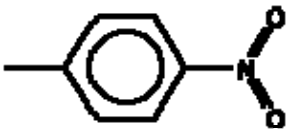
U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

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 Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

NITROTOLUENES



p-Nitrotoluene

1.0 Introduction and Physical Properties

The nitrotoluenes consist of three isomers: 2-nitrotoluene (o-nitrotoluene, 1-methyl-2-nitrobenzene), 3-nitrotoluene (m-nitrotoluene, 1-methyl-3-nitrobenzene) and 4-nitrotoluene (p-nitrotoluene, 1-methyl-4-nitrobenzene) (Lewis, 1997). 2-Nitrotoluene occurs as a yellowish liquid at ambient temperatures below 25 °F, and 3-nitrotoluene occurs as a yellowish liquid at ambient temperatures below 59 °F (HSDB, 2010). 4-Nitrotoluene occurs as colorless rhombic needles to yellowish crystals at ambient temperatures. All behave in the environment as **VOCs**. The nitrotoluenes, especially the 2- and 4-isomers, are used as intermediates in the synthesis of other organic chemicals, particularly toluidine, tolidine, fuchsine and other synthetic dyes, explosives, petrochemicals, pesticides, pharmaceuticals, rubber and agricultural chemicals, dyes and pigments (HSDB, 2010). Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
2-Nitrotoluene (88-72-2)										
137.13[a]	2.30[a]	1.25E-5[e] (25°C)	2.44[a]	7.14E-2[b] (25°C)[c]	8.27E-6[b] (25°C)[c]	1.32E-4[a] (20°C)	6.52E+2[a] (30°C)	495.2[a]	720[d]	1.22E+4[a]
3-Nitrotoluene (99-08-1)										
137.15[a]	2.45[a]	9.3E-6[e] (25-30°C)	2.16[a]	7.14E-2[b] (25°C)[c]	8.27E-6[b] (25°C)[c]	2.72E-4[a] (25°C)	4.98E+2[a] (30°C)	505.1[a]	734[d]	1.18E+4[a]
4-Nitrotoluene (99-99-0)										
137.14[a]	2.37[a]	.63E-6[e] (25°C)	2.49[a]	7.14E-2[b] (25°C)[c]	8.27E-6[b] (25°C)[c]	1.32E-4[a] (20°C)	4.42E+2[a] (30°C)	511.5[a]	743[d]	1.19E+4[a]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[b]Calculated as described in Introduction to the Toxicity Profiles.

[c]Assumed.

[d]Calculated as described by Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.

[e]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

The major sources of release of nitrotoluenes to the environment appear to be facilities and plants that produce these compounds as by-products (HSDB, 2010). Nitrotoluenes may also enter the environment from the disposal of waste products which contain these compounds, and they may form from degradation of higher explosives.

Nitrotoluenes released to soil are expected to be resistant to oxidation and chemical hydrolysis (HSDB, 2010). The nitrotoluenes are reported to biodegrade under anaerobic conditions to form toluidines (nitroreduction products); one study under aerobic conditions in a mixed culture of soil microorganisms resulted in persistence exceeding 64 days. The nitrotoluenes are expected to be moderately to highly mobile in soil and volatilize slowly from dry soil surfaces.

Nitrotoluenes released to water are expected to be susceptible to photolysis, volatilization and aerobic biodegradation, provided suitable acclimation has been achieved (HSDB, 2010). Oxidation, chemical hydrolysis, adsorption to suspended solids and sediments and bioaccumulation in aquatic organisms are not expected to be significant removal processes. Insufficient data were available to indicate the significance of anaerobic biodegradation as a removal mechanism.

Nitrotoluenes released to the atmosphere are expected to exist entirely in the vapor phase (HSDB, 2010). The dominant removal mechanism is expected to be reaction with photochemically generated hydroxyl radicals (estimated half-life 8 hours) and direct photolysis. Photoproducts include 2-methyl-6-nitrophenol and 2-methyl-4-nitrophenol (from 2-nitrotoluene), 3-methyl-4-nitrophenol, 3-methyl-2-nitrophenol and 3-methyl-6-nitrophenol (from 3-nitrotoluene) and 2-methyl-6-nitrophenol (from 4-nitrotoluene).

The nitrotoluenes are not expected to bioconcentrate or bioaccumulate significantly (HSDB, 2010); therefore, biotransfer factors are not estimated.

3.0 Toxicokinetics

Excretion data indicate that the nitrotoluenes are rapidly and extensively absorbed from the GI tract (EPA, 2009; 2008; 2007). Fecal excretion of radioactivity in rats treated with radiolabeled 2-, 3- or 4-nitrotoluene did not exceed 4, 12, or 7 percent of the administered dose, respectively. Biliary excretion has been shown to be important for the nitrotoluenes, so that GI uptake is probably greater than indicated by the fecal excretion data (EPA, 2002a,b,c). The nitrotoluenes are also absorbed from the respiratory tract and through intact skin, although quantitative data were not available (HSDB, 2010; EPA, 2009; 2008; 2007).

Data regarding tissue distribution following absorption were not located. Metabolites identified in the urine of the rats discussed above include the corresponding 2-, 3- or 4-nitrobenzoic alcohol or nitrobenzoic acid derivatives of 2-, 3- and 4-nitrotoluene, respectively, indicating oxidation of the methyl group as an initial step in biotransformation of the nitrotoluenes (EPA, 2009; 2008; 2007). The nitrobenzoic acid derivatives were excreted unchanged or as glucuronide, sulfate, cysteine or hippuric acid conjugates, depending on the nitrotoluene isomer given.

Excretion of radioactivity after oral administration of radiolabeled compound occurs rapidly primarily through the urine, but biliary excretion is significant, particularly for male rats, and pulmonary excretion accounts for small amounts. Biliary excretion is followed by microbial nitroreduction followed by re-absorption of products that bind to hepatic macromolecules (EPA, 2002a,b,c).

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. As noted in Section 3.0, GI absorption is extensive. Therefore, no GAF is developed for the nitrotoluenes and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of the nitrotoluenes from soil is not evaluated. t*, K_p, τ, FA and B values are estimated as described by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
2-Nitrotoluene	1.48	8.91E-3	0.62	1	0[a]
3-Nitrotoluene	1.48	1.12E-2	0.62	1	0.1
4-Nitrotoluene	1.48	9.91E-3	0.62	1	0[a]

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Sufficiently small to be rounded to zero.

5.0 Noncancer Effects Evaluation

2-Nitrotoluene. Limited occupational exposure studies suggested mild changes in intestinal activity associated with exposure to mixed nitrotoluenes. However, these studies lacked exposure data and other details that limited the usefulness of the studies (EPA, 2009). No human data was found concerning subchronic or chronic exposure to 2-nitrotoluene. The toxic effects in animal studies associated with 2-nitrotoluene include elevated methemoglobinemic levels, anemia, hemosiderin deposition in the spleen (an indication of erythrocyte damage), hematopoiesis in the spleen, splenic lesions, and indication of liver toxicity. Reproductive effects were noted at higher concentrations (EPA, 2008).

Based on the anemia as the critical effect in a 13-week study in rats (NTP, 1992), a LOAEL of 44 mg/kg-day was identified as the point of departure for the provisional subchronic RfD. EPA (2008) derived a provisional subchronic RfD of 1E-2 mg/kg-day, using the LOAEL and a composite UF of 3,000, including a factor of 3 for the use of a LOAEL with limited biological significance and 10 each for interspecies extrapolation, intraspecies differences, and limited date on reproductive and development toxicity. Confidence in the key subchronic study is regarded as medium.

A provisional chronic RfD was derived from a 2-year study in rats (NTP, 2002). This study did not include hematological analysis or clinical chemistry parameters. Effects in this chronic study include atrophy in the germinal epithelium of the testes, liver lesions, pigmentation of kidney tubules, hematopoietic cell proliferation in the liver, bone marrow, and spleen in males (25 mg/kg-day) and the spleens of females (30 mg/kg-day). A benchmark dose level, assuming 10 percent lower confidence limit, resulted in an estimated NOAEL of 0.94 mg/kg-day for bone marrow hyperplasia in male rats and liver cell necrosis in male mice. A provisional chronic RfD or 9E-4 mg/kg-day was derived, based on this estimated NOAEL and a composite UF of 1,000, including a factor of 10 each

for interspecies extrapolation, intraspecies differences, and limited date on reproductive and development toxicity (EPA, 2008). Confidence in the key chronic study is regarded as medium.

Provisional RfCs could not be derived for 2-nitrotoluene due to insufficient human data, the lack of chronic/subchronic inhalation toxicity data for animals, and insufficient data on the other nitrotoluenes which eliminated the possibility of deriving a provisional RfC by analogy to these compounds.

3-Nitrotoluene. Limited occupational exposure studies suggested mild changes in intestinal activity associated with exposure to mixed nitrotoluenes. However, these studies lacked exposure data and other details that limited the usefulness of the studies (EPA, 2009). The toxic effects in animal studies associated with 3-nitrotoluene include extravascular hemolysis, hemosiderin deposition in the spleen (an indication of erythrocyte damage), splenic lesions, and various hematological effects. Based on hematological effects in female rats, including hemosiderin deposition in the spleen, EPA (2009) identified a subchronic LOAEL of 46 mg/kg-day based on hematologic effects. This LOAEL was used to derive a benchmark dose level of 1.1 mg/kg-day. A provisional subchronic RfD of 1E-3 mg/kg-day was derived assuming a composite UF of 1,000, including a factor of 10 each for interspecies extrapolation, intraspecies differences, and uncertainty of the database. Confidence is regarded as medium.

The derivation of a provisional chronic RfD was attempted. However, no chronic RfD was derived because extrapolation of the subchronic data would have resulted in a UF of 10,000, which is regarded by EPA (2009) and unacceptable for purposes other than screening. Therefore, the provisional subchronic of 1E-3 mg/kg-day is used as the chronic RfD to enable quantitative assessment of noncancer hazards. However, it is possible that use of the provisional subchronic RfD for chronic exposure may underestimate potential noncancer effects. It is noted that this value is 20 times more conservative than the previous provisional chronic RfD of 2E-2 mg/kg-day (EPA, 2002b) and is similar to the chronic RfD values for 2-nitrotoluene (as discussed above) and 4-nitrotoluene (as discussed below). Also note each of the nitrotoluenes elicits hemosiderosis, indicating that mechanism of toxicity is similar.

Provisional RfCs could not be derived for 3-nitrotoluene due to insufficient human data, the lack of chronic/subchronic inhalation toxicity data for animals, and insufficient toxicokinetic data for inter-route extrapolalation.

4-Nitrotoluene. Limited occupational exposure studies suggested mild changes in intestinal activity associated with exposure to mixed nitrotoluenes. However, these studies lacked exposure data and other details that limited the usefulness of the studies (EPA, 2009). No human data was found concerning subchronic or chronic exposure to 4-nitrotoluene (EPA, 2007). The toxic effects in animal studies associated with 4-nitrotoluene include elevated methemoglobinemic levels, erythropoiesis, splenic effects, testes lesions, increased liver weight, lung lesions, and kidney lesions.

A LOAEL of 42 mg/kg-day was identified in a 13-week rat study (NTP, 1992; Dunnick et al, 1994) for the effects of splenic hematopoiesis and spleen pigmentation. Model-based benchmark dose levels were derived for both of these effects. EPA (2007) selected a benchmark dose level of 1.1 mg/kg-day for spleen pigmentation as the point of departure, which is interpreted as a modeled NOAEL. A provisional subchronic RfD of 4E-3 mg/kg-day was derived using the benchmark dose

level and a composite UF of 300, including a factor of 3 for data base limitations and 10 each for interspecies extrapolation and intraspecies differences. Confidence in the key subchronic study is regarded as medium.

A provisional chronic RfD was derived from a 2-year study in rats (NTP, 2002). Kidney lesions were observed at the lowest doses levels. Splenic lesions, including hemosiderin deposition and hemopoietic proliferation, were observed at a higher dose level. This study did not include hematological analysis or clinical chemistry parameters. Benchmark dose level modeling was performed, assuming 10 percent lower confidence limit, based on the kidney lesions. The resultant value of 2.7 mg/kg-day was used as a point of departure. A provisional chronic RfD of 8E-3 mg/kg-day was derived based on this value and a composite UF of 300, including a factor of 3 for data base limitations and 10 each for interspecies extrapolation and intraspecies differences. Confidence in the key subchronic study is regarded as medium. However, because this value is higher than the provisional subchronic RfD, the subchronic RfD value of 4E-3 mg/kg-day is used as the chronic RfD.

Provisional RfCs could not be derived for 4-nitrotoluene due to insufficient human data, the lack of chronic/subchronic inhalation toxicity data for animals, and insufficient data on the other nitrotoluenes which eliminated the possibility of deriving a provisional RfC by analogy to these compounds.

6.0 Carcinogenicity Evaluation

Various *in vitro* and *in vivo* tests show that 2-nitrotoluene is not genotoxic to bacteria, but is genotoxic to mammalian cells following activation (EPA, 2002a). Studies in intact and bile-duct cannulated rats showed that intestinal microfloral reduction of hepatic metabolites is required for binding to various macromolecules including DNA. A wide variety of tumors, many of mesothelial origin, were observed in rats in the subchronic dietary study (as well as other studies) discussed in Section 5.0. Clear evidence of carcinogenicity in rats and mice was observed in a chronic (2-year) dietary study. EPA (2008) classified 2-nitrotoluene in cancer weight-of-evidence as *likely to be carcinogenic to humans,* based on no data in humans and sufficient evidence of carcinogenicity in animals. Potency factors were estimated for the most prevalent tumor types observed in rats and mice in the 2-year dietary study. An oral SF of 2.3E-1 per mg/kg-day based on the increased incidence of skin fibromas and fibrosarcomas in male rats was selected as the provisional oral SF for 2-nitrotoluene. Data are insufficient for estimation of an inhalation URF for 2-nitrotoluene.

Data regarding the carcinogenicity of 3-nitrotoluene were not located in the available literature (EPA, 2008). Enterohepatic recirculation results in the production of metabolites that bind to some cellular macromolecules but apparently not to DNA (EPA, 2008), reducing concern for a carcinogenic role for 3-nitrotoluene. EPA (2008) classified 3-nitrotoluene as Inadequate Information to Assess Carcinogenic Potential. SFs are not developed for Group D chemicals.

EPA (2002c) classified 4-nitrotoluene in cancer weight-of-evidence as Suggestive Evidence of Carcinogenic Potential, based on no data in humans and limited evidence of carcinogenicity in animals. The animal data consist of a 2-year dietary study with 4-nitrotoluene that yielded equivocal evidence for variety of tumor types in male and female rats and male mice. Studies in intact and bile-duct cannulated rats showed that intestinal microfloral reduction of hepatic metabolites is required for binding to various macromolecules (proteins and RNA). Binding to DNA has not been observed.

Potency factors were estimated for the most prevalent tumor types observed in male and female rats and male mice in the 2-year dietary study. An oral SF of 1.6E-2 per mg/kg-day based on the increased incidence of clitoral tumors in female rats was selected as the provisional oral SF for 4-nitrotoluene. Data are insufficient for estimation of an inhalation URF for 4-nitrotoluene.

7.0 Toxicity Summary

Toxicity values for the nitrotoluenes are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2-Nitrotoluene										
1E-2	9E-4	E	ND	ND	NA	B2[b]	2.3E-1	B2[b]	ND	ND
3-Nitrotoluene										
1E-3	1E-3	E	ND	ND	NA	D[c]	ND	D[c]	ND	ND
4-Nitrotoluene										
4E-3	4E-3	E, K	ND	ND	NA	C[d]	1.6E-2	C[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; NA = not applicable.
Target organ abbreviations: E = erythrocyte; K = kidney; NA = not applicable
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.
[b]*Likely to be carcinogenic to humans.*
[c]*Data are inadequate for assessment of human carcinogenic potential.*
[d]*Suggestive evidence of carcinogenicity to humans.*

8.0 References

Dunnick, J.K., M.R. Elwell, and J.R. Bucher, "Comparative Toxicities of o-, m-, and p-Nitrotoluenes in 13-Week Study Feed Studies in F344/N Rats and B6C3F$_1$ Mice," ***Fundamentals of Applied Toxicology***, Volume 23, pp. 411-421.`
Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 17 December.

National Toxicology Program NTP, 1992, ***NTP Technical Report on Toxicity Studies of o-, m-, and p-Nitrotoluenes (CAS Nos. 8872-2, 99-08-1, 99-99-0) Administered in Dosed Feed to F344/N Rats and B6C3F$_1$ Mice***. NTP Tox Report 23.

National Toxicology Program NTP, 2002, ***Toxicology and Carcinogenicity Studies o-Nitrotoluene (88-72-2) in F344/N Rats and B6C3F$_1$ Mice (Feed Studies)***. NTP Tox Report 504.

U.S. Environmental Protection Agency (EPA), 2002a, ***PPRTV Derivation Support Document for o-Nitrotoluene (2-Nitrotoluene) (CASRN 88-72-2)***, National Center for Environmental Assessment, Cincinnati, OH, 19 September.

U.S. Environmental Protection Agency (EPA), 2002b, ***PPRTV Derivation Support Document for 1-Methyl-3-Nitrobenzene (m-Nitrotoluene) (CASRN 99-08-1)***, National Center for Environmental Assessment, Cincinnati, OH, 10 September.

U.S. Environmental Protection Agency (EPA), 2002c, ***PPRTV Derivation Support Document for p-Nitrotoluene (4-Nitrotoluene) (CASRN 99-99-0)***, National Center for Environmental Assessment, Cincinnati, OH, 19 September.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2007, ***Provisional Peer-Reviewed Toxicity Values for p-Nitrotoluene (4-Nitrotoluene) (CASRN 99-99-0)***, National Center for Environmental Assessment, Cincinnati, OH, 28 September.

U.S. Environmental Protection Agency (EPA), 2007, ***Provisional Peer-Reviewed Toxicity Values for o-Nitrotoluene (2-Nitrotoluene) (CASRN 88-72-2)***, National Center for Environmental Assessment, Cincinnati, OH, 15 August.

U.S. Environmental Protection Agency (EPA), 2007, ***Provisional Peer-Reviewed Toxicity Values for m-Nitrotoluene (3-Nitrotoluene) (CASRN 99-089-1)***, National Center for Environmental Assessment, Cincinnati, OH, 16 June.

POLYCHLORINATED BIPHENYLS (PCBs)

1.0 Introduction and Physical Properties

The nomenclature of the PCBs is somewhat unique, including the terms homologue and congener. The PCB molecule consists of two joined phenyl groups. The carbon atoms are numbered 1 through 6 on one ring and 1′ through 6′ on the other ring. Numbering proceeds consecutively starting with the carbon atom attached to the other ring. The term homologue refers to the degree of chlorination. Since one carbon atom in each phenyl group is bonded to the other ring, five carbon atoms on each phenyl group – a total of 10 carbon atoms – are available for bonding to chlorine atoms. Consequently, there are ten possible *homologues* of PCBs – monochlorinate biphenyls, dichlorinated biphenyls, through decachlorinated biphenyls. Congener refers to the exact placement of each chlorine atom. For example, there are three congeners of monochlorinated biphenyls: 2-PCB, 3-PCB and 4-PCB. The term isomer is often incorrectly used instead of congener with the PCBs. Free rotation around the C-C bond, however, precludes stereoisomerism, which limits the number of theoretically possible congeners.

The PCBs are a class of chemicals generally considered to be **SVOC**s including 209 possible individual congeners (ATSDR, 2000). The PCBs formerly manufactured and used in the U.S. are called Aroclors. The Aroclors are mixtures of several PCB congeners and related compounds. Many individual PCB congeners are common to all or many of the different Aroclors. The Aroclors differ from one another primarily in the relative proportions of the different congeners. ATSDR (2000) provides a summary of the proportion of each homologue in commercial mixtures of the various Aroclors. Of interest to this evaluation is the observation that Aroclor-1016 and Aroclor-1221 contain no homologues with 5 or more chlorines. Most Aroclors occur as oily or viscous liquids at room temperatures, although Aroclor-1260 occurs as a sticky resin. Aroclors were used as dielectric and heat exchange agents in several open and closed systems, but since the middle 1970s, use has been restricted largely to electrical transformers and capacitors.

Analysis of PCBs in environmental media is often performed using EPA Method SW8082. Method SW8082 is usually performed as a "finger printing" procedure and the results are reported as the commercial Aroclor(s) that most closely reflect the fingerprint(s) (ATSDR, 2000). Recently, however, concern has arisen that some individual PCB congeners may behave toxicologically in the same manner as the polychlorinated dibenzo-p-dioxins (PCDD), and that they may have greater toxic potency than is reflected in the toxicity assessment of the commercial Aroclors. This problem may be exacerbated by weathering, which may preferentially remove the less toxic (generally less highly chlorinated) congeners, leading to a relative concentration of the more highly chlorinated congeners. The more highly chlorinated congeners are generally the more persistent and the more toxic (ATSDR, 2000; EPA, 2010). For this reason, Method SW8082 has been expanded so that the analytical results can be reported in terms of selected specific congeners. In addition, EPA Method E1668 is a very high-resolution protocol (highly sensitive and highly selective) that reports individual congeners as well as totals for each of the homologues. Method E1668 is seldom used for environmental media, with the exception of incinerator emissions testing, because of its unusually high cost.

EPA (2000) identified 12 individual PCB congeners with dioxin-like activity. The PCB mixtures and dioxin-like congeners analyzed by the EPA methods mentioned above are compiled below:

PCB Mixture or Congener	CASRN	IUPAC No.	Method 8082	Method 1668
Aroclor-1016	12674-11-2	NA	Yes	No
Aroclor-1221	11104-28-2	NA	Yes	No
Aroclor-1232	11141-16-5	NA	Yes	No
Aroclor-1242	53469-21-9	NA	Yes	No
Aroclor-1248	12672-29-6	NA	Yes	No
Aroclor-1254	11097-69-1	NA	Yes	No
Aroclor-1260	11096-82-5	NA	Yes	No
3,3′,4,4′-Tetrachlorobiphenyl	**32598-13-3**	**77**	**Probably[a]**	**Yes**
3,4,4′,5-Tetrachlorobiphenyl	**70362-50-4**	**81**	**Probably[a]**	**Yes**
2,3,3′,4,4′-Pentachlorobiphenyl	**32598-14-4**	**105**	**Probably[a]**	**Yes**
2,3,4,4′,5-Pentachlorobiphenyl	**74472-37-0**	**114**	**Probably[a]**	**Yes**
2,3′,4,4′,5-Pentachlorobiphenyl	**31508-00-6**	**118**	**Probably[a]**	**Yes**
2′,3,4,4′,5-Pentachlorobiphenyl 2,3′,4,4′,5′-Pentachlorobiphenyl)	**65510-44-3**	**123**	**Probably[a]**	**Yes**
3,3′,4,4′,5-Pentachlorobiphenyl	**57465-28-8**	**126**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5-Hexachlorobiphenyl	**38380-08-4**	**156**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5′-Hexachlorobiphenyl	**69782-90-7**	**157**	**Probably[a]**	**Yes**
2,3′,4,4′,5,5′-Hexachlorobiphenyl	**52663-72-6**	**167**	**Probably[a]**	**Yes**
3,3′,4,4′,5,5′-Hexachlorobiphenyl	**32774-16-6**	**169**	**Probably[a]**	**Yes**
2,3,3′,4,4′,5,5′-Heptachlorobiphenyl	**39635-31-9**	**189**	**Probably[a]**	**Yes**
Total Monochlorobiphenyls	NA	NA	No	Yes
Total Dichlorobiphenyls	NA	NA	No	Yes
Total Trichlorobiphenyls	NA	NA	No	Yes
Total Tetrachlorobiphenyls	NA	NA	No	Yes
Total Pentachlorobiphenyls	NA	NA	No	Yes
Total Hexachlorobiphenyls	NA	NA	No	Yes
Total Heptachlorobiphenyls	NA	NA	No	Yes
Total Octachlorobiphenyls	NA	NA	No	Yes
Total Nonachlorobiphenyls	NA	NA	No	Yes
Total Decachlorobiphenyls	NA	NA	No	Yes

PCB = polychlorinated biphenyl; CASRN = Chemical Abstract Service Registry Number; IUPAC No. = congener-specific identification number assigned by the International Union of Pure and Applied Chemists. The specific congeners are sometimes called PCB-77, PCB-81, etc., and are sometimes *inappropriately* called Aroclor-77, Aroclor-81, etc. Technically, the term Aroclor should be reserved for chemical mixtures, where the name Aroclor is followed by a four-digit identifier code.

Dioxin-like congeners are bolded.

[a]Although the congeners listed have not been tested by Method 8082, it is likely that they could be quantified by the method. Other non-dioxin-like congeners can routinely be analyzed by this method.

Data regarding the physical properties of the individual PCB congeners are very limited. The physical characteristics of the Aroclor mixtures have been much more thoroughly evaluated. Although the Aroclors described below are classified as SVOCs, largely on the basis of MW, their H values suggest that volatilization could be important. Furthermore, the MW values provided are proportional averages based on the composition of the various Aroclor mixtures. Several individual PCB congeners have MW values below the limit of 200 g/mole ordinarily used to distinguish VOCs from SVOCs. For these reasons it is recommended that the Aroclors

should be evaluated in exposure pathways (such as vapor intrusion into indoor air) that involve volatilization. Relevant physical properties are compiled below:

MW[a]	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Aroclor-1016 (12674-11-2)										
257.9[b]	5.6[b]	2.9E-4[b] (25°C)	4.96[c]	4.69E-2[d] (25°C)[e]	6.8E-6[c] (25°C)	5.3E-7[b] (25°C)	4.2E-1[b] (25°C)	340.50[f]	475.22[f]	1.8E+4[f]
Aroclor-1221 (11104-28-2)										
200.7[b]	4.7[b]	7.36E-4[b] (25°C)	2.44[c]	5.54E-2[d] (25°C)[e]	7.5E-6[c] (25°C)	8.8E-6[b] (25°C)	5.9E-1[b] (24°C)	571[b]	ND	ND
Aroclor-1232 (11141-16-5)										
232.2[B]	5.1[B]	8.64E-4[c] (25°C)[e]	2.83[c]	5.03E-2[d] (25°C)[e]	7.2E-6[c] (20°C)	5.34E-6[b] (25°C)	4.5E-1[b] (25°C)	581[b]	ND	ND
Aroclor-1242 (53469-21-9)										
266.5[b]	5.6[b]	5.2E-4[b] (25°C)	4.59[c]	4.59E-2[d] (25°C)[e]	6.1E-6[c] (20°C)	5.34E-7[b] (25°C)	2.4E-1[b] (25°C)	345.5[f]	482.20[f]	1.8E+4[f]
Aroclor-1248 (12672-29-6)										
299.5[g]	6.2[g]	2.8E-3[g] (25°C)	5.64[c]	4.24E-2[d] (25°C)[e]	6.6E-6[c] (20°C)	6.5E-7[g] (25°C)	5.7E-2[g] (24°C)	631[g]	ND	ND
Aroclor-1254 (11097-69-1)										
328[b]	6.5[b]	2.0E-3[b] (25°C)	5.0[c]	3.99E-2[d] (25°C)[e]	5.6E-6[c] (20°C)	1.01E-7[b] (25°C)	3.5E-2[b] (24°C)	377.50[f]	512.27[f]	1.9E+4[f]
Aroclor-1260 (11096-82-5)										
375.7[b]	6.8[b]	4.6E-3[b] (25°C)	6.42[c]	3.65E-2[d] (25°C)[e]	5.3E-6[c] (20°C)	5.33E-8[b] (25°C)	8.0E-2[b] (24°C)	402.50[f]	539.37[f]	1.9E+4[f]
Aroclor-1262 (1336-36-3)										
389[b]	ND	ND	ND	3.57E-2[d] (25°C)[e]	4.13E-6[d] (25°C)[e]	ND	5.2E-2[b] (24°C)	680.7[b]	ND	ND

MW[a]	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Aroclor-1268 (11100-14-4)										
453[b]	ND	ND	ND	3.22E-2[d] (25°C)[e]	3.73E-6[d] (25°C)[e]	ND	3.0E-1[b] (24°C)	ND	ND	ND

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K); $\Delta H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); I = insoluble; ND = no data.
[a]Average molecular mass for the proportions of individual congeners in the commercial product.
[b]Agency for Toxic Substances and Disease Registry (ATSDR), 2000, ***Update Toxicological Profile for Polychlorinated Biphenyls***, U.S. Department of Health and Human Services, Atlanta, Georgia, November, on line.
[c]Montgomery, J.H., 1996, ***Groundwater Chemicals Desk Reference, Second Edition***, Lewis Publishers, New York.
[d]Calculated as described in Introduction to Toxicological profiles.
[e]Assumed.
[f]U.S. Environmental Protection Agency (EPA), 1997, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings***, prepared by Environmental Quality Management, Inc. for the Office of Emergency and Remedial Response, Washington, DC, September.
[g]Earlier version (1995 Draft for Public Comment) of ATSDR Toxicological profile.
[h]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

Former PCB releases occurred as a result of their manufacture, use, disposal, and leakage from damaged PCB-containing equipment (ATSDR, 2000). Currently the major source of PCB release to the environment is recycling of PCBs previously introduced into the environment, which involves volatilization from ground surfaces (water, soil) into the atmosphere with subsequent removal from the atmosphere via wet/dry deposition, followed by volatilization (HSDB, 2010). PCBs are also currently released to the environment from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, and improper (or illegal) disposal of PCB materials, such as waste transformer fluid, to open areas.

Major sources to air include emissions from the overhaul, repair or reuse of PCB-containing items, and the recycling of previously released PCBs described above (ATSDR, 2000; HSDB, 2010). The vapor pressures of the PCBs indicate that they would exist primarily in the vapor-phase in the atmosphere; monitoring data have shown that between 87 and 100% of the PCBs in air are in the vapor-phase. Vapor pressure of the PCBs generally decreases with an increase in the extent of chlorination; therefore, the more highly chlorinated PCBs are more likely to be associated with the particulate phase in air than are the lesser chlorinated PCBs. Physical removal of PCBs in the atmosphere is accomplished by wet and dry deposition; dry deposition occurs only for PCBs in the particulate phase.

Major sources to soil include leaks or discharges from PCB-containing items and deposition from the atmosphere (ATSDR, 2000). PCBs sorb tightly to constituents of soil with adsorption

generally increasing with the degree of chlorination. Although the lesser chlorinated biphenyls may exhibit some mobility in soil, PCBs generally will not leach significantly in most aqueous soil systems. In the presence of organic solvents, PCBs may leach quite rapidly through soil. Vapor loss of PCBs from soil surfaces appears to be an important loss mechanism with the rate of volatilization decreasing with increasing chlorination. Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of the PCBs. In general, persistence of the PCBs increases with an increase in the degree of chlorination (ATSDR, 2000; HSDB, 2010). Mono-, di- and trichlorinated biphenyls (Aroclors-1221 and -1232) biodegrade relatively rapidly, tetrachlorinated biphenyls (Aroclors-1016 and -1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors-1248, -1254 and -1260) are quite resistant to biodegradation. The position of chlorination within a given homologue also determines the extent of biodegradation. Although biodegradation of the more highly chlorinated congeners may occur very slowly on an environmental basis, no other degradation mechanisms have been shown to be important in soil systems; therefore, biodegradation may be the ultimate degradation process in soil.

Former sources of PCBs to surface water include waste streams and discharges from waste water treatment plants (ATSDR, 2000). Currently, direct deposition, runoff and erosion probably predominate. Removal from water involves sorption to particles and sedimentation, as well as volatilization.

The PCBs are among the compounds that bioaccumulate in food chain pathways and are of special concern for biomagnification from sediment in benthic fish. Biotransfer factors for the PCBs are compiled below:

BSAF	Bpr[a,b]	Bpv[a,b]	Bb[a]	Bm[a]
Aroclor-1016				
See below	2.24E-2	2.24E-2	1.00E-2	3.16E-3
Aroclor-1221				
See below	7.44E-2	7.44E-2	1.26E-3	3.98E-4
Aroclor-1232				
See below	4.37E-2	4.37E-2	3.16E-3	1.00E-3
Aroclor-1242				
See below	2.24E-2	2.24E-2	1.00E-2	3.16E-3
Aroclor-1248				
See below	1.01E-2	1.01E-2	3.98E-2	1.26E-2
Aroclor-1254				
See below	6.78E-3	6.78E-3	7.94E-2	2.51E-2
Aroclor-1260				

BSAF	Bpr[a,b]	Bpv[a,b]	Bb[a]	Bm[a]
See below	4.55E-3	4.55E-3	1.58E-1	5.01E-2
Aroclor-1262				
See below	ND	ND	ND	ND
Aroclor-1268				
See below	ND	ND	ND	ND

BSAF = biota-sediment accumulation factor, unitless; Bpr = soil-to-plant (reproductive portions) biotransfer factor, unitless; Bpv = soil-to-plant (vegetative portions) biotransfer factor, unitless; Bb = soil-to-beef biotransfer factor, days/kg; Bm = soil-to-milk biotransfer factor, days/kg; ND = no data.
[a]Calculated as described in Introduction to the Toxicological profiles.
[b]The methodology for estimating biotransfer of organic chemicals to plants does not differentiate vegetative and reproductive portions.

EPA (1995) reported a biota/sediment absorption factor (BSAF) for total PCBs of 1.85 for trout in the Great Lakes ecosystem. The BSAF of 1.85 is applied to all PCBs in this evaluation.

3.0 Data for Evaluating Dermal Exposure

Toxicokinetic data from laboratory animals suggest that the efficiency of GI absorption is roughly inversely related to the degree of chlorination (ATSDR, 2000). The GI absorption of mono- to hexachlorinated biphenyls exceeds 90 percent. Dichlorobiphenyl GI absorption efficiency is approximately 95 percent, but the absorption efficiency of octachlorobiphenyl approximates only 75 percent. GI absorption efficiency of Aroclor-1254 approximates 85.4 percent in ferrets and greater than 90 percent in monkeys. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for the PCBs and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

The PCBs appear to be readily absorbed by the skin when applied as neat compound or mixed with a suitable vehicle (ATSDR, 2000), but efficiency falls when soil is the medium of exchange. The EPA (2004) recommended ABS of 0.14 for PCBs is used in this evaluation. EPA (2004) provides values for K_p, τ, FA and B for 4-chlorobiphenyl and hexachlorobiphenyl. Generally, the more highly chlorinated PCB congeners are the more persistent in the environment; therefore, the values for hexachlorobiphenyl provided below are applied to all PCBs in this evaluation.

Chemical	K_p	τ	FA	B
Hexachlorobiphenyl	4.3E-1	1.13E+1	0.5	3.2

K_p = permeability coefficient (cm/hour); τ= lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).

4.0 Noncancer Effects Evaluation

The acute oral toxicity of the PCBs is low to moderate, as indicated by LD_{50} values in laboratory animals ranging from 750 mg/kg (mink) to 4250 mg/kg (rats) (ATSDR, 2000). Mink appear to be unusually sensitive, which is also true for prolonged oral exposure. Death appears to be due to respiratory depression and dehydration from diarrhea.

The best known incident involving oral exposure of humans to PCBs is the "Yusho" incident in Japan, in which persistent chloracne, gastrointestinal irritation and central nervous symptoms followed ingestion of cooking oil contaminated with PCBs (Gaffey, 1983). Further investigation, however, revealed that concentrations of polychlorinated dibenzofurans (PCDF) and polychlorinated quaterphenyls in the cooking oil were similar to those of PCBs, which confounds interpretation of the reported observations and symptoms.

Prolonged oral exposure of laboratory animals to various Aroclors leads to liver damage, signs of chloracne, immunological effects, and neurological impairment, particularly of the young (EPA, 2010). A verified oral RfD for Aroclor-1254 of 2E-5 mg/kg-day for chronic oral exposure is based on a LOAEL of 5E-3 mg/kg-day associated with chloracne and related signs and immunological effects in monkeys treated with the test material in gelatin capsules for over five years. An uncertainty factor of 300 was applied. Confidence in the RfD is medium. The immune system and skin are considered the target organs for prolonged oral exposure to Aroclor-1254.

EPA (1997) derived a provisional RfD for subchronic oral exposure to Aroclor-1254 of 5E-5 by applying an uncertainty factor of 100 to the LOAEL from the monkey study described above.

A verified oral RfD of 7E-5 mg/kg-day for Aroclor-1016 is based on a NOAEL of 7E-3 mg/kg-day in a perinatal and neurobehavioral toxicity study in monkeys (EPA, 2010). An uncertainty factor of 100 was applied to the NOAEL. The uncertainty factor of 100 consists of factors of 3 each to extrapolate from monkeys to humans, to provide greater protection for more sensitive humans, to account for deficiencies in the database, and to expand from a subchronic study to chronic exposure. The LOAEL (2.8E-2 mg/kg-day) was associated with low birth weights. The fetus is considered to be the most sensitive member of the population. The fetus and nervous system are selected as “target organs” for prolonged oral exposure to Aroclor-1016. Confidence in the oral RfD is medium.

There is no EPA-derived subchronic oral RfD for exposure to Aroclor-1016. However, a preliminary RfD can be derived from the NOAEL of 7E-3 mg/kg-day in the monkey study described above by application of an uncertainty factor of 30. The uncertainty factor of 30 includes all the factors described above except the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 2.3E-4 mg/kg-day, which is expressed as 2E-4 mg/kg-day when rounded to one significant figure.

The similarities in the nature of the adverse effects and the thresholds for these effects for various Aroclors suggest that the adverse effects and thresholds across all the Aroclors may be similar. That this may be true is supported by the fact that many of the same PCB congeners are present in the different Aroclors, which differ from one another primarily only in the relative proportions of the individual congeners. Therefore, it is reasonable to apply the RfDs described above to any of the Aroclor mixtures, or to total PCBs. The chronic and subchronic oral RfDs

developed for Aroclor-1016 rather than those developed for Aroclor-1254 are selected as surrogates for evaluating the adverse effects of the other Aroclors or total PCBs. The RfDs developed for Aroclor-1016 are preferred because they include less uncertainty than the RfDs developed for Aroclor-1254. Therefore, the verified chronic oral RfD of 7E-5 mg/kg-day and the preliminary subchronic oral RfD of 2E-4 mg/kg-day developed for Aroclor-1016 are applied to the Aroclors other than Aroclor-1254 or to total PCBs.

Occupational exposure to PCBs, which involved both inhalation and dermal exposure, was associated with upper respiratory tract and ocular irritation, loss of appetite, liver enlargement and increased serum concentrations of liver enzymes, skin irritation, rashes and chloracne, and, in heavily exposed female workers, decreased birth weight of their infants (ATSDR, 2000). Concurrent exposure to PCB contaminants, such as polychlorinated dibenzofurans, confounds the interpretation of the occupational exposure studies. Rats, mice, rabbits and guinea pigs intermittently exposed to Aroclor-1254 vapors exhibit moderate liver degeneration, decreased body weight gain and slight renal tubular degeneration; however, the accuracy of the reported exposure concentration is in doubt. Neither verified nor provisional chronic inhalation RfC values are available.

5.0 Carcinogenicity Evaluation

EPA (2010) declined to evaluate the carcinogenicity of the individual Aroclor mixtures. Instead, EPA (2010) classified PCBs in cancer weight-of-evidence Group B2 (probable human carcinogen) based on adequate evidence for liver tumors in laboratory animals and inadequate data in humans. EPA (2010) established a three-tiered approach for estimating the cancer potency of exposure to PCBs.

<u>High-Risk Tier</u> A SF of 2.0E+0 per mg/kg-day is verified as an upper-bound potency factor for exposure to PCBs in the high-risk tier (EPA, 2010). The high-risk tier includes ingestion in the food chain, incidental ingestion of soil or sediment, inhalation of dust or aerosol, or dermal contact with soil or sediment if a dermal absorption factor is applied. The high-risk SF of 2.0E+0 per mg/kg-day is used for mixtures containing any congeners considered to be persistent or acting in a dioxin-like manner, and for any early life exposures (e.g., residential scenario, which includes both adults and children). In this context, persistent congeners are those with five or more chlorines. The 12 PCB congeners considered to act in a dioxin-like manner were identified above. The SF of 2.0E+0 per mg/kg-day is equivalent to a URF (for dust or aerosol inhalation) of 5.7E-4 per $\mu g/m^3$.

The high-risk tier SF for central tendency (CT) assessment is 1.0E+0 per mg/kg-day. The SF of 1.0E+0 per mg/kg-day is equivalent to a URF (for dust or aerosol inhalation) of 2.9E-4 per $\mu g/m^3$.

<u>Low-Risk Tier</u> EPA (2010) verified an upper-bound SF of 4E-1 per mg/kg-day for the low-risk tier, which includes ingestion of water-soluble congeners (assumed to be PCBs detected in drinking water), inhalation of evaporated congeners, and dermal exposure if no absorption factor is applied. The SF of 4E-1 per mg/kg-day is equivalent to a URF for vapor inhalation of 1E-4 per $\mu g/m^3$. A SF of 3E-1 per mg/kg-day is recommended for the low-risk CT evaluation. The SF of 3E-1 per mg/kg-day is equivalent to a URF (for vapor inhalation) of 9E-5 per $\mu g/m^3$.

Lowest-Risk Tier EPA (2010) verified an upper-bound SF of 7E-2 per mg/kg-day for the lowest-risk tier, which is restricted to data sets for which homologue-or congener-specific data verify that congeners containing 5 or more chlorines constitute less than 0.5 percent of the total PCBs. The SF of 7E-2 per mg/kg-day is equivalent to a URF for inhalation of 2E-5 per $\mu g/m^3$. These values are used in for Aroclor 1016. An SF of 4E-2 per mg/kg-day is recommended for the low-risk CT evaluation. The SF of 4E-2 per mg/kg-day is equivalent to a URF for vapor inhalation of 1E-5 per $\mu g/m^3$.

An alternative method of PCB cancer risk assessment was proposed by EPA (2000, 2010) for sites where congener-specific analytical results are available. This method involves application of toxicity equivalency factors (TEF) adopted by EPA (2000) to the 12 dioxin-like congeners identified above. TEFs are factors that express the toxicity of the dioxin-like congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD or "dioxin"), the reference compound. EPA (1997) presented a provisional oral and inhalation SF for 2,3,7,8-TCDD of 1.5E+5 per mg/kg-day. As explained in the toxicological profile for polychlorinated dibenzo-p-dioxins and dibenzofurans, the inhalation SF is more accurately expressed as 1.1E+5 per mg/kg-day, which is equivalent to 3.3E+1 per $\mu g/m^3$, or 3.3E+4 per mg/m^3. Application of the EPA (2000) TEFs to the dioxin-like PCB congeners yields the cancer potency factors presented below:

Dioxin-Like PCB Congener	IUPAC No.	TEF	SFo	URFi	SFi
3,3′,4,4′-Tetrachlorobiphenyl	77	0.0001	1.5E+1	3.3E+0	1.1E+1
3,4,4′,5-Tetrachlorobiphenyl	81	0.0001	1.5E+1	3.3E+0	1.1E+1
2,3,3′,4,4′-Pentachlorobiphenyl	105	0.0001	1.5E+1	3.3E+0	1.1E+1
2,3,4,4′,5-Pentachlorobiphenyl	114	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3′,4,4′,5-Pentachlorobiphenyl	118	0.0001	1.5E+1	3.3E+0	1.1E+1
2′,3,4,4′,5-Pentachlorobiphenyl 2,3′,4,4′,5′-Pentachlorobiphenyl)	123	0.0001	1.5E+1	3.3E+0	1.1E+1
3,3′,4,4′,5-Pentachlorobiphenyl	126	0.1	1.5E+4	3.3E+3	1.1E+4
2,3,3′,4,4′,5-Hexachlorobiphenyl	156	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3,3′,4,4′,5′-Hexachlorobiphenyl	157	0.0005	7.5E+1	1.7E+1	5.5E+1
2,3′,4,4′,5,5′-Hexachlorobiphenyl	167	0.00001	1.5E+0	3.3E-1	1.1E+0
3,3′,4,4′,5,5′-Hexachlorobiphenyl	169	0.01	1.5E+3	3.3E+2	1.1E+3
2,3,3′,4,4′,5,5′-Heptachlorobiphenyl	189	0.0001	1.5E+1	3.3E+0	1.1E+1

IUPAC No. = unique sequentially numbered congener identifier proposed by the International Union of Pure and Applied Chemists; TEF = toxicity equivalency factor (EPA, 2000); SFo = oral slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per milligram per cubic meter); SFi = inhalation slope factor (risk per milligram per kilogram-day).

6.0 Toxicity Summary

Cancer potency factors for the dioxin-like congeners are summarized above. Toxicity values for PCBs other than the dioxin-like congeners, are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Aroclor-1016[b]										
2E-4	7E-5	F	ND	ND	NA	Please see below.				
Aroclor-1254										
5E-5	2E-5	Is, S	ND	ND	NA	Please see below.				
PCBs, High-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote "b."						B2	2.0E+0	B2	5.7E-1[c]	2.0E+0[c]
PCBs, High-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote "b."						B2	1.0E+0	B2	2.9E-1[c]	1.0E+0[c]
PCBs, Low-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote "b."						B2	4E-1	B2	1E-1[d]	4E-1[d]
PCBs, Low-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote "b."						B2	3E-1	B2	9E-2[d]	3E-1[d]
PCBs, Lowest-Risk, Upper Bound (Reasonable Maximum Exposure) Assumptions										
Please see above, including footnote "b."						B2	7E-2	B2	2E-2	7E-2
PCBs, Lowest-Risk, Central Tendency Exposure Assumptions										
Please see above, including footnote "b."						B2	4E-2	B2	1E-2	4E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per milligram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data.
Target organ abbreviations: F = unborn child; Is = immune system; S = skin; NA = not applicable.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.
[b]sRfDo and cRfDo derived for Aroclor-1016 should be applied to all other Aroclor mixtures except Aroclor-1254. They should also be applied to total PCBs, but not to specific PCB homologues congeners.

[c]Reasonable maximum exposure evaluation of inhalation pathways including dusts or aerosols.
[d]Reasonable maximum exposure evaluation of inhalation pathways limited to vapors, excluding dusts or aerosols.

7.0 PCB Risk Assessment

The approach to risk assessment of PCB-contaminated sites depends on the analytical data available. Typically, PCB analysis is limited to Method 8082 for the seven commonly encountered Aroclors. When this is the case, the hazard associated with noncancer effects is evaluated with the oral RfDs provided in the table above. Cancer risk is evaluated with the upper bound (reasonable maximum exposure) SFo and URFi or SFi from the "High-Risk" or "Low-Risk" scenarios, depending on the Aroclors identified, and the receptors and exposure pathways evaluated.

If homologue-specific analytical results are available in addition to results for the seven common Aroclors, it will be necessary to decide which is the better data set. This decision will depend, at least in part, on the samples included in each data set. Generally, the data set that better represents contamination across the area of investigation or a given exposure unit should be used in the risk assessment. If the same samples were analyzed for both Aroclors and specific homologues so that representativeness is not an issue, EPA (2010) generally considers that the homologue data are preferable because they are based on greater sensitivity (lower reporting limits) and are more likely to reflect selective removal of the more labile and mobile congeners due to weathering. Homologue-specific analytical results in the absence of data for the seven common Aroclors can be evaluated directly in the risk assessment.

The homologues should not be evaluated individually for noncancer hazard using the oral RfDs provided above. However, their sum (total PCBs) can be evaluated with the oral RfD for Aroclor-1016, assuming that the total PCBs reflect the characteristics of a mixture not unlike one or more of the Aroclors. If this assumption is not reasonable, the reason should be clearly stated, total PCBs should not be evaluated for noncancer hazard, and the uncertainty imparted to the risk assessment (probably a non-conservative bias) should be discussed. For example, it might be that only the most highly chlorinated homologues persist and are identified from an "old" release.

Cancer risk is estimated for total PCBs rather than the individual homologues. If the penta-, hexa-, hepta-, octa-, nona- and deca-chlorinated homologues together constitute not more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "Lowest-Risk" scenario should be used. If the penta-, hexa-, hepta-, nona- and deca-chlorinated homologues together constitute more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "High-Risk" or "Low-Risk" scenario should be used, depending on the receptors and exposure pathways evaluated.

It is unlikely that congener-specific data will be the only PCB data available. Should that be the case, however, the noncancer hazard can be estimated for total PCBs as described in the previous paragraph. For cancer risk assessment, the dioxin-like congeners should be assessed as explained below. The non-dioxin-like congeners should be summed. If the penta-, hexa-, hepta-, nona- and deca-chlorinated congeners together constitute not more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "Lowest-Risk" scenario should be used. If the penta-, hexa-, hepta-, nona- and deca-chlorinated congeners together constitute more than 0.5 percent of the total PCB, the SFo and URFi or SFi for the "High-Risk" or "Low-Risk" scenario should be used, depending on the receptors and exposure pathways evaluated. The cancer risks from the non-dioxin-like and the dioxin-like congeners (see next paragraph) should be summed.

It is more likely that limited congener-specific data will be available *in addition to* data for the Aroclors or homologues. (Limited in the number of congeners analyzed and/or the number of samples analyzed for individual congeners.) For cancer risk assessment, EPA (2010) recommends evaluating the Aroclors or homologues as described above, after reducing their exposure-point concentrations by the contribution from the dioxin-like congeners. In most cases the concentrations of the dioxin-like congeners will be so much lower than the concentrations of the Aroclors or homologues that their subtraction would make little difference. The cancer risk for the dioxin-like congeners is assessed by using the TEF-based SFo and URFi or SFi values presented above. The two sets of risk estimates are then summed.

The newer trends in PCB analysis and risk assessment introduce additional sources of uncertainty to the assessment. PCBs tend to be somewhat ubiquitous because of their once-common use, potential for widespread dispersion, and persistence in environmental media (ATSDR, 2000). Method 1668 is a high-resolution method that is likely to identify very low "anthropogenic background" levels of PCBs that may not be reflected in the results of Aroclor analysis using Method 8082. This may impart an overly conservative bias to the risk assessment. Another source of uncertainty may arise from analyzing only a limited number or proportion of PCB-bearing samples for individual congeners. The limited analysis could impart either a conservative or non-conservative bias to the risk assessment, depending on the relative level of contamination in the samples selected for analysis of individual congeners.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Polychlorinated Biphenyls***, U.S. Public Health Service, Atlanta, Georgia, November, on line.

Gaffey, W.R., 1983, "The epidemiology of PCBs," In: ***PCBs: Human and Environmental Hazards***, F.M. D'itri and M.A. Kamrin, Eds., Boston: Butterworth Publishers.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

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

U.S. Environmental Protection Agency (EPA), 1995, ***Great Lakes Water Quality Initiative, Technical Support Document for the Procedure to Determine Bioaccumulation Factors***, EPA.820.8.95.CC5, PB95-187290.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 2000, ***Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Part***

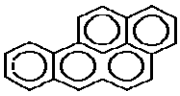
II: Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds, Draft Final, National Center for Environmental Assessment, May.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

Semivolatile Organic Compounds

*POLYNUCLEAR AROMATIC HYDROCARBONS a.k.a. POLYCYCLIC AROMATIC HYDROCARBONS a.k.a. POLYAROMATIC HYDROCARBONS (PAH) (*130498-29-2)



Benzo(a)Pyrene
(A representative and the most studied member of this class of compounds.)

1.0 Introduction and Physical Properties

The PAHs regularly observed in environmental media and addressed in this profile include acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, naphthalene, phenanthrene and pyrene. All exist as solids at room temperature. Classification as **SVOC** or **VOC** is made in the following table. PAHs are the products of incomplete combustion of fossil fuels or other organic matter, hence include both natural and anthropogenic sources (ATSDR, 1995a; HSDB, 2010). Several are also components of crude oil. Naphthalene is used in the synthesis of phthalic anhydride, the insecticide carbaryl, leather tanning agents and surface active agents, and is a component of diesel and other fuels (ATSDR, 1995b). Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
Acenaphthene (83-32-9) **(VOC)**										
154.21[a]	3.92[a]	1.55E-4[b] (25°C)	3.85[b]	4.21E-2[b] (25°C)	7.69E-6[b] (25°C)	3.29E-6[a] (25°C)	4.24E+0[b] (20-25°C)	550.54[c]	803.15[c]	1.22E+4[c]
Acenaphthylene (208-96-8) **(VOC)**										
152.20[a]	4.07[a]	1.13E-5[a] (25°C)	3.42[a]	6.67E-2[d] (25°C)[e]	7.72E-6[d] (25°C)[e]	1.20E-6[a] (25°C)	3.93E+0[a] (25°C)	543.2[a]	797[f]	1.1E+4[g]
Anthracene (120-12-7) **VOC)**										
178.23[a]	4.45[a]	6.51E-5[b] (25°C)	4.47[b]	3.24E-2[b] (25°C)	7.74E-6[b] (25°C)	3.51E-9[a] (25°C)	4.34E-2[b] (20-25°C)	615.18[c]	873.00[c]	1.31E+4[c]
Benzo(a)anthracene (56-55-3) **(SVOC)**										
228.3[h]	5.66[h]	3.34E-6[b] (25°C)	5.60[b]	5.10E-2[b] (25°C)	9.00E-6[b] (25°C)	2.50E-9[a] (25°C)	9.40E-3[b] (20-25°C)	708.15[c]	1004.8[c]	1.60E+4[c]
Benzo(a)pyrene (50-32-8) **(SVOC)**										
252.3[a]	6.10[h]	4.57E-7[k] (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[k] (25°C)	6.09[b]	2.26E-2[b] (25°C)	5.56E-6[b] (25°C)	6.58E-10[a] (20°C)	1.50E-3[b] (20-25°C)	715.90[c]	969.27[c]	1.70+4[c]
Benzo(k)fluoranthene (207-08-9) **(SVOC)**										
252.32[a]	6.84[a]	8.29E-7[b] (25°C)	6.09[b]	2.26E-2[b] (25°C)	5.56E-6[b] (25°C)	1.28E-12[a] (25°C)	8.00E-4[b] (20-25°C)	753.15[c]	1019.7[c]	1.80E+4[c]
Benzo(g,h,i)perylene (191-24-2) **(SVOC)**										
276.34[a]	6.63[a]	2.66E-7[a] (20°C)	4.98[a]	4.48E-2[d] (25°C)[e]	5.19E-6[d] (25°C)[e]	1.3E-13[a] (25°C)	2.6E-4[a] (25°C)	823[a]	1097[f]	1.96E+4[g]
Chrysene (218-01-9) **(VOC)**										
228.3[h]	5.66[h]	9.46E-5[b] (25°C)	5.60[b]	2.48E-2[b] (25°C)	6.21E-6[b] (25°C)	8.20E-12[a] (25°C)	1.60E-3[b] (20-25°C)	714.15[c]	979.0[c]	1.65E+4[c]
Dibenz(a,h)anthracene (53-70-3) **(SVOC)**										
		1.47E-8[b]		2.02E-2[b]	5.18E-6[b]	1.32E-13[a]	2.49E-3[b]			

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
278.4[h]	6.84[h]	(25°C)	6.58[b]	(25°C)	(25°C)	(20°C)	(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]
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]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 23 April.

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

[c]U.S. Environmental Protection Agency (EPA), 2000, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings, Revised***, Office of Emergency and Remedial Response, Washington, DC, December.

[d]Calculated as described in Introduction to Toxicological profiles.

[e]Assumed.

[f]Estimated from Equation 12-4 in Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.

[g]Estimated from Equation 13-5 in Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.

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

[i]Syracuse Research Corporation (SRC), 2004, ***CHEMFATE Search Results***, on line, accessed 23 April.

[j]National Institute of Standards and Technology (NIST), 2004, Standard Reference Data Program, Online Data Bases, accessed 23 April.

[k]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

The PAHs are ubiquitous products of incomplete combustion; natural sources include volcanoes and forest fires (ATSDR, 1995a, b; HSDB, 2010). There is some evidence for biosynthesis by plants, bacteria and algae. Some of the PAHs occur naturally in fossil fuels. Anthropogenic releases to the environment, primarily to the atmosphere, greatly outweigh the natural sources and include any processes that involve incomplete combustion of fossil fuels and organic matter, including wood-burning for home heat (the predominant source), cigarette smoke, internal combustion engine exhaust, and fuel oil emissions. Industrial sources include coal mining, processing and storing, wood treatment (creosote), manufactured gas plants (coal tar), power generation, production of coal tar, coke and asphalt, petroleum cracking and industrial and municipal incineration. Other sources include various crude oils, fresh and used motor oils, gasolines, charcoal-broiled foods, processed foods, various oils, margarine, butter and fats, fruits, vegetables, and cereals, roasted coffee and tea. Indoor sources include unvented kerosene heaters

and gas cooking and heating appliances. Naphthalene is released during its manufacture and processes that involve its use (e.g., vaporization from moth balls).

Although PAHs generally occur in mixtures, certain chemicals tend to predominate in different sources so that the PAH pattern observed in a given medium may provide a clue to the source. The following compilation relates individual PAHs and sources:

Source[a]	Individual PAHs
Residential wood burning	Acenaphthylene
Auto emissions	Benzo(g,h,i)perylene, pyrene
Diesel exhaust particulates	Fluoranthene, phenanthrene, pyrene
Diesel exhaust vapors	Phenanthrene, anthracene
Diesel total emissions	Acenaphthene, fluorene, phenanthrene
Fly ash & bottom ash from U.S. municipal waste incinerators	Phenanthrene
Fly ash from U.K. municipal waste incinerators	Benzo(g,h,i)perylene
Particulate emissions from municipal waste incinerator	Benzo(a)fluoranthene, benzo(g,h,i)perylene, chrysene, fluoranthene, indeno(1,2,3-cd)pyrene, phenanthrene
Municipal/medical waste incinerator	Benzo(a)anthracene, benzo(g,h,i)perylene
Rotary kiln incinerator charged with polyethylene, no afterburner	Fluoranthene, phenanthrene, pyrene (see next entry)
Rotary kiln incinerator charged with polyethylene with afterburner	Benzo(a)anthracene, phenanthrene (total PAH emissions reduced 100-fold compared with no afterburner)
Coal tar pitch emissions	Phenanthrene and pyrene 20 to 80 X > benzo(a)pyrene, benzo(g,h,i)perylene
Natural gas home appliances – fine particulate emissions	Chrysene, fluoranthene, pyrene, triphenylene
Groundwater near coal & oil gasification plant	Acenaphthylene, acenaphthene, fluorene, phenanthrene, fluoranthene, pyrene, chrysene
Groundwater near wood treatment facilities	Benzo(a)pyrene, phenanthrene
[a]Agency for Toxic Substances and Disease Registry, (ATSDR), 1995, ***Update Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)***, U.S. Public Health Service, Atlanta, Georgia, August, on line.	

Data compiled for soils are not included in the table above because the lists are long and the variation from one site to another is great, so that the information is not useful for identifying sources. Similarly, sediment is a sink for PAHs from all sources including atmospheric deposition from far distant locations, and the patterns observed in sediment do not necessarily reflect a nearby source (ATSDR, 1995a). Generally, PAHs associated with combustion exhibit highly condensed ring structures and little alkylation (i.e., benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, benzo(e)pyrene, chrysene, fluoranthene, pyrene).

Crude petroleum and products made from crude petroleum (e.g., asphalts, fuels, oils) contain PAHs, but the patterns observed depend on the location of the source (ATSDR, 1995a; Potter and Simmons, 1998). Phenanthrene and alkylated forms of PAHs, particularly the methylnaphthalenes, often predominate in petroleum products.

The partitioning and fate of the PAHs in environmental media depend largely on VP (tendency to exist in air as a vapor), H (indicator of partitioning between air and water) and K_{oc} (indicator of affinity to bind to organic matter in soil and sediment). ATSDR (1995a) noted that H and K_{oc} are roughly directly related to MW, and that VP is roughly inversely related to MW. Therefore, they grouped the PAHs into the following categories to facilitate understanding their behavior in the environment:

- Low MW (152 to 178 g/mole) – acenaphthene, acenaphthylene, anthracene, fluorene, naphthalene, phenanthrene

- Medium MW (approximately 202 g/mole) – fluoranthene, pyrene

- High MW (228 to 278 g/mole) – benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene.

PAHs exist in the atmosphere as vapors or adsorbed to particulates, the proportion depending on the vapor pressure of the individual chemical and atmospheric conditions such as temperature and humidity (ATSDR, 1995a, b; HSDB, 2010). Generally, the two- and three-ring compounds (low and medium MW except pyrene) exist predominantly as vapors; the four-ring compounds exist in both the vapor and particulate phase, and compounds with 5 or more rings (benzo[a]pyrene, benzo[g,h,i]perylene exist predominantly as particulates. Increasing atmospheric temperature and relative humidity favors existence in the vapor phase. PAHs may travel short or long distances before removal from the air. Vapor forms are subject to chemical oxidation processes in the air, which reduces the distance they may travel. Residence time and transport distance is inversely related to the size of the particles on which the PAHs are adsorbed. Small particles may have residence times of weeks, permitting transport for hundreds or upwards of a thousand miles. Wet and dry deposition accounts for removal of the particulates.

The predominant sources of PAHs in surface water are deposition from the atmosphere, industrial and sewage effluent and oil spills (ATSDR, 1995a, b). Runoff and erosion can also contribute PAHs to surface water bodies. Volatilization is a significant fate process for PAHs with H values of 1E-5 atm-m^3/mole or greater (generally the low and medium MW compounds). Volatilization is a very limited removal process for most of the high MW compounds. Volatilization is enhanced by high temperature, turbulence and high wind. Sorption to benthic or suspended sediment and biodegradation are competing removal processes. Generally, volatilization and biodegradation are the predominant removal processes for the low MW compounds, and volatilization and sorption are the predominant removal processes for medium and high MW compounds. Naphthalene is relatively water soluble and may remain largely in solution. PAHs in sediment may biodegrade, recycle back to the water column (lower MW compounds) or accumulate in the lower trophic levels of living organisms.

Deposition from the atmosphere is the principal source of PAHs in soil (ATSDR, 1995a, b). Other sources include industrial activities, disposal of sewage sludge, and leaching from coal storage sites. Most PAHs sorb to soil constituents because of their low solubility and high affinity for organic matter. Volatilization is an important removal process for the low molecular weight compounds. Some of the low molecular weight compounds, particularly naphthalene, may leach fairly rapidly to groundwater.

The high lipophilicity of many PAHs evidenced by high log K_{ow} values suggest that the PAHs might cross biologic membranes and participate significantly in food-chain pathways involving fish, fruit and vegetable, and meat and milk consumption. This, however, is not the case. For example, the PAHs are efficiently metabolized and excreted by fin fish, greatly reducing the likelihood of bioconcentration from water or bioaccumulation from sediment (ATSDR, 1995a). Consequently, there are often great discrepancies between modeled BCF values and empirical BCF values, where care was taken to identify the parent compound rather than PAH metabolites in fish tissue. This distinction is necessary because aromatic fragments from PAH metabolism are readily assimilated as normal endogenous components of biological tissue. PAH concentrations in aquatic food-chain systems usually decrease with increasing trophic level, reflecting the generally greater efficiency of metabolism in the higher trophic organisms. Approximate tissue-to-sediment PAH concentration ratios were reported to be 0.6 to 1.2 for amphipods, 0.1 for clams, and 0.05 for fin fish and shrimp.

It has been reported that terrestrial plants can accumulate PAHs from soil by uptake through the roots (ATSDR, 1995a). More careful investigation, however, reveals that root uptake is quite low, and there is no evidence of bioconcentration or biomagnification. For example, PAH ratios in vegetation to soil in one survey ranged from 0.001 to 0.18 for total PAHs, and from 0.002 to 0.33 for benzo(a)pyrene. It is likely that most of the PAH contamination on the vegetation resulted from atmospheric deposition. Increased concentrations in vegetation have not been observed when soil PAH concentrations were greatly increased by amendment with sewage sludge. Data from carrots (*Daucus carotu*) showed that PAHs adhere to the outer skin of the root but show little tendency to penetrate to deeper layers, and even less tendency to translocate to aerial parts.

The inability of plants to bioconcentrate PAHs from soil reduces concern for bioconcentration in agricultural products such as meat and milk. It should be noted, however, that measurable concentrations of PAHs may accumulate on the aerial parts of plants because of deposition from the atmosphere (ATSDR, 1995a). Also, food-producing animals ingest a substantial amount of soil while grazing or consuming mechanically harvested forage crops. Mammals, however, efficiently metabolize the PAHs to polar compounds that are readily excreted, reducing concern for bioconcentration.

In summary, the PAHs are highly lipophilic, which implies significance in food-chain exposure pathways. Empirical data, however, show that fish and mammals efficiently metabolize and eliminate these compounds. Furthermore, bioconcentration in plants because of uptake from soil is not significant. Therefore, it is unlikely that the PAHs would participate significantly in food-chain pathways, and biotransfer factors are not estimated.

3.0 Toxicokinetics

The PAHs are absorbed by all routes of exposure, but the rate and extent depends on the compound, the species of animal and the vehicle (or nature of the particulates) (ATSDR, 1995a). PAHs are readily absorbed during inhalation exposure; however, intratracheal instillation studies indicate that particle size is the most important determiner of the extent of pulmonary uptake.

Toxicokinetic studies of several PAHs summarized by ATSDR (1995a) provide limited quantitative information regarding the extent of GI absorption. Limitations arise largely because biliary excretion complicates quantification of uptake from the GI tract. A study of benzo(a)pyrene in rats suggests that GI absorption ranges from 38 to 58 percent. A study in rats reported absorption efficiency for anthracene ranging from 53 to 74 percent. GI absorption of pyrene, chrysene and dibenzo(a,h)anthracene is described as high. Administration of the test compound in oil or in a high-fat diet appears to increase the extent of GI absorption.

Empirical data with pure compound dissolved or suspended in various vehicles suggest that dermal uptake of benzo(a)pyrene is extensive (ATSDR, 1995a). One occupational study reported that approximately 75 percent of systemically absorbed pyrene entered the body through dermal uptake rather than through inhalation exposure. Combining PAHs with soil appears to significantly reduce the extent of dermal uptake compared with oleaginous or acetone vehicles. Anecdotal evidence from using cloth diapers stored in contact with naphthalene indicates that naphthalene is absorbed by the skin, but quantitative data are not available (ATSDR, 1995b).

Distribution of absorbed PAHs is generally widespread, with highest levels located initially in lipid-rich tissues (ATSDR, 1995a). Highest levels of metabolites (radioactivity following administration of radiolabeled compounds) are located in the liver and GI tract, even after inhalation exposure, probably reflecting extensive metabolism in the liver followed by biliary excretion. Ciliary clearance and deglutition probably contribute to levels associated with the GI tract. Concentrations of radioactivity following administration of radiolabeled compound reveal fetal levels approximately 2- to 10-fold lower than maternal levels, although this depends on the specific compound administered.

Metabolism of the PAHs, particularly benzo(a)pyrene, has been extensively studied (ATSDR, 1995a). Metabolism proceeds rapidly, yielding products that are more water soluble and readily excreted than the parent compound. All tissues have the ability to metabolize the PAHs, potentially to carcinogenic intermediates, which probably accounts for the observation that cancers occur at the point of contact. There is considerable variability in tissue metabolic activity; however, the liver is probably the most active in most cases.

ATSDR (1995a) distinguishes between “alternant” and “nonalternant” PAHs, based on the nature of the electron density associated with the molecule, which influences how that compound is metabolized to its ultimate carcinogen. Alternant PAHs (e.g., benzo[a]pyrene, benzo[a]anthracene, chrysene, dibenz[a,h]anthracene) exhibit a uniformly distributed electron density. Nonalternant PAHs (e.g., fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene) behave more like two separate molecules because of uneven electron distribution.

Alternant PAHs (based on data for benzo[a]pyrene) are initially metabolized by the microsomal cytochrome P-450 system to several arene oxides (ATSDR, 1995a). These may re-arrange

spontaneously to phenols or undergo hydration to form the corresponding trans-dihydrodiols. Further oxidation of the dihydrodiols results in formation of quinones, phenol diols and dihydrodiol epoxides. Phenols may be formed by direct insertion of oxygen into a ring. Many of the metabolites resulting from the reactions described above are subject to conjugation with various substrates followed rapidly by excretion. The dihydrodiol epoxides that form in the "bay" region (the three-sided concave region formed by the fusion of three benzene rings) are most likely the ultimate carcinogens that covalently bind to macromolecules such as DNA, resulting in alkylation or other adducts that yield genetic errors.

Metabolism of the nonalternant PAHs differs from the alternant PAHs in that more extensive oxidation to hydroxy-epoxy-diols may be important to achieve genotoxicity (ATSDR, 1995a). Dihydrodiol epoxide formation in the bay region is associated with carcinogenicity of the PAHs generally; however, some of the nonalternant PAHs yield reactive metabolites that deviate from the classical bay region model.

PAH metabolites are readily excreted, largely through the bile, although some metabolites are excreted by the kidney (ATSDR, 1995a). The extent of elimination is species specific. In one inhalation study rats excreted metabolites much more efficiently than dogs or monkeys.

4.0 Dermal Exposure

EPA (2004) recommends that oral toxicity values for the PAHs should not be adjusted when applied to dermal exposure because GI absorption probably exceeds 50 percent. Therefore, no GAF is estimated and the oral toxicity values described below should be used for dermal exposure without adjustment.

Empirical data with pure compound dissolved or suspended in various vehicles suggest that dermal uptake of benzo(a)pyrene is extensive (ATSDR, 1995a). Anecdotal evidence from using cloth diapers stored in contact with naphthalene indicates that naphthalene is absorbed by the skin, but quantitative data are not available (ATSDR, 1995b). EPA (2004) reviewed empirical data regarding dermal uptake of benzo(a)pyrene and recommended an ABS of 0.13 for the PAHs. However, EPA (2004) also notes that VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels. Therefore, the ABS of 0.13 is applied to all the PAHs identified above as SVOCs. Dermal uptake of the PAHs identified as VOCs is not quantified.

Values for t*, K_p, τ, FA and B are provided or estimated as follows:

Chemical	t*	K_p	τ	FA	B
Acenaphthene[a]	1.84	8.39E-2	7.67E-1	1	0.4
Acenaphthylene[a]	1.79	1.08E-1	7.47E-1	1	0.5
Anthracene[a]	4.06	1.38E-1	1.05E+0	1	0.7
Benzo(a)anthracene[b]	8.53	4.7E-1	2.03E+0	1.0	2.8
Benzo(a)pyrene[b]	11.67	7.0E-1	2.69E+0	1.0	4.3
Benzo(b)fluoranthene[b]	12.03	7.0E-1	2.77E+0	1.0	4.3
Benzo(k)fluoranthene[a]	12.40	2.00E+0	2.72E+0	0.4	12
Benzo(g,h,i)perylene[a]	8.89	1.07E+0	3.70E+0	0.7	6.8
Chrysene[b]	8.53	4.7E-1	2.03E+0	1.0	2.8
Dibenz(a,h)anthracene[b]	17.57	1.5E+0	3.88E+0	0.6	9.7
Fluoranthene[b]	5.68	2.2E-1	1.45E+0	1.0	1.2

Chemical	t*	K_p	τ	FA	B
Fluorene[a]	2.15	1.07E-1	8.95E-1	1	0.5
Indeno(1,2,3-cd)pyrene[b]	16.83	1.0E+0	3.78E+0	0.6	6.7
Naphthalene[b]	1.34	4.7E-2	5.6E-1	1	0.2
Phenanthrene[b]	4.11	1.4E-1	1.06E+0	1	0.7
Pyrene[a]	3.42	1.94E-1	1.43E+0	1	1.1

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Estimated as described in Introduction to Toxicological profiles.
[b]Provided by U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)***, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.

5.0 Noncancer Effects Evaluation

The PAHs are generally divided into two EPA cancer weight-of-evidence groups: Group D – not classifiable as to carcinogenicity to humans, and Group B2 – probable human carcinogens. The Group D PAHs have not been evaluated for carcinogenicity by toxicity testing; therefore, cancer SFs or URFs cannot be estimated for these compounds (EPA, 1986, 2010). Risk evaluation of these compounds is limited to noncancer effects. It is theoretically possible that cancer risk could be the "driver" for some of these compounds. However, the mechanism of carcinogenicity of the PAHs is fairly well understood to be correlated with molecular morphology and the propensity to form certain active metabolites (ATSDR, 1995a). The Group D PAHs either do not fit the morphologic mold or have been shown empirically to be unlikely to cause cancer, reducing greatly the uncertainty that significant cancer risk is being overlooked.

Cancer SFs and URFs are available for the Group B2 compounds, but noncancer RfDs or RfCs are not. Therefore, risk evaluation of these compounds is limited to cancer risk. These compounds have the morphologic requirements for carcinogenicity; therefore, it has been thought that cancer risk is the driver, and that noncancer effects are relatively insignificant, although empirical data were lacking until recently.

Recent data support this assumption. De Jong et al. (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 LD_{50} values for naphthalene include 533 to 710 mg/kg for mice and 2200 to 2400 mg/kg for rats, establishing the mouse as more sensitive to the lethal effects of acute oral exposure (ATSDR, 1995b). Decreased terminal body weights, accompanied by a remarkable

absence of hematological and histopathological effects, were observed in rats treated by gavage for 13 weeks (EPA, 2010). The LOAEL in this study was 142 mg/kg-day associated with greater than 10 percent reduction in terminal body weights. The NOAEL was 71 mg/kg-day. The high dose rate, 286 mg/kg-day was a FEL associated with increased mortality. Application of an uncertainty factor of 3000 (10 to extrapolate from rats to humans, 10 to protect sensitive humans, 10 to extrapolate from subchronic to chronic exposure, and 3 for database deficiencies) to the NOAEL of 71 mg/kg-day yields the verified chronic oral RfD of 2E-2 mg/kg-day. Confidence in the RfD is low. A preliminary subchronic oral RfD can be derived for naphthalene by applying an uncertainty factor of 300 to the NOAEL of 71 mg/kg-day described above. The uncertainty factor of 300 reflects the chronic uncertainty factor of 3000 without the factor of 10 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 2E-1 mg/kg-day.

The key study described above is not sufficient to identify target organs for prolonged oral exposure to naphthalene. Adults, children and neonates exposed to moth balls exhibit hemolytic anemia, evidence of liver disease and neurological deficits (EPA, 1993a, 1998). The liver effects and neurological deficits may be secondary to hemolytic anemia and reduced oxygen-carrying capacity of the blood. Hemolytic anemia and cataract formation have been seen also in orally exposed humans (EPA, 1998). The data suggest that the erythrocyte may be the most sensitive tissue in humans. Among common species of laboratory mammals, hemolytic anemia is seen only in dogs (EPA, 1998). Cataracts are seen in several laboratory mammals, but only at relatively high doses. The key study identifies reduced body weight as the critical effect in rats. Rats, however, do not exhibit hemolytic anemia, and exhibit cataracts only at very high doses, suggesting that rats may not be a totally acceptable model for the toxicity of naphthalene to humans. Therefore, based on the effects observed in humans, the erythrocyte and eye are selected as target organs for prolonged oral exposure to naphthalene. Reduced body weight is also included as a critical effect because this was the only endpoint observed in rats in the key study.

EPA (1993a, 2010) reported a 2-year study in which mice were exposed to naphthalene vapors for 6 hours/day on 5 days/week. Inflammation of the nasal and olfactory epithelium was the most consistently observed sign; granulomatous lesions in the lungs were also observed. There was equivocal evidence of hematologic involvement. The lowest exposure concentration, 10 ppm, was a LOAEL for the nasal effects, which are considered the critical effects of inhalation exposure. The LOAEL is equivalent to a human equivalent concentration of 9.3 mg/m^3 (EPA, 2010). Application of an uncertainty factor of 3000 yields a verified chronic inhalation RfC of 3E-3 mg/m^3, which is equivalent to a chronic inhalation RfD of 8.6E-4 mg/kg-day. The nasal and olfactory epithelia are the target organs for inhalation exposure to naphthalene. Confidence in the RfC is medium. The chronic inhalation RfC of 3E-3 mg/m^3, equivalent to an inhalation RfD of 8.6E-4 mg/kg-day, is adopted as sufficiently protective for subchronic inhalation exposure as well.

Subchronic exposure to pyrene induces mild renal tubular degeneration and reduced kidney weight in orally treated mice (EPA, 2010). A verified RfD of 3E-2 mg/kg-day for chronic oral exposure was derived from a NOAEL of 75 mg/kg-day in a 13-week gavage study. The LOAEL was 125 mg/kg-day in this study. An uncertainty factor of 3000 was applied. Confidence in the oral RfD is low. The kidney tubule is chosen as the target organ for chronic oral exposure to

pyrene. EPA (1997) derived a provisional subchronic oral RfD for pyrene of 3E-1 mg/kg-day from the same mouse study using an uncertainty factor of 300.

Data regarding prolonged oral exposure are not available for several of the Group D PAHs, which hinders estimation of an RfD or RfC and compromises evaluation of the potential for noncancer effects. Therefore, surrogates are used to develop toxicity values for the noncancer effects.

Generally surrogates are chosen on the basis of the following hierarchy:

- Toxicological similarity (effects and dose-response relationship).
- Toxicokinetic similarity, assuming that likeness in absorption, distribution and especially products of biotransformation suggests toxicological similarity.
- Structural similarity, assuming that likeness in structure suggests similarity in toxicokinetics.

Selection of defensible surrogates for the PAHs is compromised because toxicological and toxicokinetic data are virtually non-existent (except for benzo[a]pyrene), and the structural similarities often are not very convincing, which imparts a great deal of uncertainty to the effort. Therefore, the most defensible approach for some of the PAHs is to select the most conservative surrogate; i.e., the PAH with the smallest verified oral RfD, which happens to be pyrene.

Data regarding the effects of chronic or subchronic exposure to acenaphthylene were not located in the available literature. Acenaphthene is adopted as a reasonable surrogate for acenaphthylene based on structural similarity, since the surrogate differs from the principal chemical only in the presence of two hydrogen atoms and the absence of a double bond. Therefore, the verified chronic oral RfD of 6E-2 mg/kg-day for acenaphthene is adopted as the RfD for chronic oral exposure to acenaphthylene. The liver, which is the target organ for acenaphthene, is adopted for oral exposure to acenaphthylene. Similarly, the provisional subchronic oral RfD of 6E-1 mg/kg-day for acenaphthene is adopted as the RfD for subchronic oral exposure to acenaphthylene.

Data regarding the effects of chronic or subchronic exposure to benzo(g,h,i)perylene were not located in the available literature. Pyrene is adopted as a reasonable surrogate for benzo(g,h,i)perylene based somewhat on structural similarity, but more on the selection of a conservative approach as justified above. Therefore, the verified oral RfD of 3E-2 mg/kg-day for pyrene is adopted as the oral RfD for chronic exposure to benzo(g,h,i)perylene. Similarly, the provisional subchronic oral RfD of 3E-1 mg/kg-day for pyrene is adopted as the RfD for subchronic oral exposure to benzo(g,h,i)perylene. The kidney tubule, which is the target organ for pyrene, is adopted for oral exposure to benzo(g,h,i)perylene.

Relevant data regarding chronic or subchronic exposure to phenanthrene were not located. Potential surrogates based on similarity in chemical structure include anthracene and pyrene. Pyrene is selected as the surrogate only because it is the more conservative choice. Therefore, the verified oral RfD of 3E-2 mg/kg-day for pyrene is adopted as the oral RfD for chronic exposure to phenanthrene. Similarly, the provisional subchronic oral RfD of 3E-1 mg/kg-day for pyrene is

adopted as the RfD for subchronic oral exposure to phenanthrene. The kidney tubule, which is the target organ for pyrene, is adopted for oral exposure to phenanthrene.

Data regarding inhalation exposure sufficient for development of inhalation RfCs were not located for any of the PAHs with the exception of naphthalene as noted above.

6.0 Carcinogenicity Evaluation

Acenaphthylene, anthracene, benzo(g,h,i)perylene, fluoranthene, fluorene, phenanthrene and pyrene are classified in EPA cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) because of a lack of human data and inadequate animal data (EPA, 2010). Data regarding the carcinogenicity of acenaphthene were not located. Benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, , chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene are classified in EPA weight-of-evidence Group B2 (probable human carcinogens) (EPA, 1997, 2010). Benzo(a)pyrene is the most extensively studied member of the class, inducing tumors in tissues at the point of contact of practically all laboratory species tested by all routes of exposure.

Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans, the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR, 1995a). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke and coke oven emissions accounted for only 0.1-8% of the total mutagenic activity in *Salmonella* of the unfractionated complex mixture (Lewtas, 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounts for the majority of the mutagenicity of coke oven emissions and cigarette smoke. Furthermore, coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR, 1995a).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups is based largely on the results of animal studies with large doses of purified compound (EPA, 2010). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Although the carcinogenicity of benzo(a)pyrene in animals managed in an unnatural manner in laboratory conditions has been well established, the potential for carcinogenicity to humans in environmental settings involving exposure to low concentrations remains unclear.

EPA (200) verified a SF for oral exposure to benzo(a)pyrene of 7.3E+0 per mg/kg-day, based on several dietary studies in mice and rats. Recent reevaluations of the carcinogenicity and mutagenicity of the Group B2 PAHs suggest that there are large differences between individual PAHs in cancer potency (Krewski et al., 1989). Based on the available cancer and mutagenicity data, and assuming that there is a constant relative potency between different potential carcinogens across different bioassay systems and that the PAHs under consideration have similar dose-response curves, EPA (1993b) adopted relative potency values for several PAHs.

These values and the corresponding oral SFs, based on a relative potency for benzo(a)pyrene of 1.0, are presented below:

Relative Potency Estimates for PAHs				
			Inhalation	
PAH	Relative Potency	Oral Slope Factor (per mg/kg-day)	Unit Risk Factor (per $\mu g/m^3$)	Slope Factor (per mg/kg-day)
Benzo(a)pyrene	1.0	7.3E+0	8.8E-4	3.1E+0
Benzo(a)anthracene	0.1	7.3E-1	8.8E-5	3.1E-1
Benzo(b)fluoranthene	0.1	7.3E-1	8.8E-5	3.1E-1
Benzo(k)fluoranthene	0.01	7.3E-2	8.8E-6	3.1E-2
Chrysene	0.001	7.3E-3	8.8E-7	3.1E-3
Dibenz(a,h)anthracene	1.0	7.3E+0	8.8E-4	3.1E+0
Indeno(1,2,3-cd)pyrene	0.1	7.3E-1	8.8E-5	3.1E-1

Although the EPA has not verified SFs for Group B2 PAHs other than benzo(a)pyrene, the SFs above represent reasonable estimates based on the data available. The relative potency approach employed here meets criteria considered to be desirable for this type of analysis (Lewtas, 1988). For example, the chemicals compared have similar chemical structures and would be expected to have similar toxicokinetic fate in mammalian systems. In addition, the available data suggest that the Group B2 PAHs have a similar mechanism of action, inducing frameshift mutations in *Salmonella* and tumor initiation in the mouse skin painting assay. Similar noncancer effects (minor changes in the blood, liver, kidneys) of the Group D PAHs support the hypothesis of a common mechanism of toxicity. Finally, the same endpoints of toxicity, i.e., potency in various cancer assays, and related data, were used to derive the relative potency values (Krewski et al., 1989). The oral SF for benzo(a)pyrene of 7.3E+0 per mg/kg-day, and the SFs presented above for the other Group B2 PAHs are adopted for the purposes of this evaluation.

An EPA (1994) evaluation of the inhalation cancer data suggests adoption of an inhalation SF for benzo(a)pyrene of 3.1E+0 per mg/kg-day, based on the incidence of upper respiratory and digestive tract tumors in hamsters. Applying the relative potency estimates presented above yield the inhalation URFs and SFs for the other Group B2 PAHs presented above.

EPA (2010) classified naphthalene in EPA cancer weight-of-evidence group C – possible human carcinogen – based on benign respiratory tumors and one carcinoma in female mice exposed to naphthalene by inhalation. Cancer potency factors are not available because the data are not sufficient.

7.0 Toxicity Summary

Toxicity values for the PAHs are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
Acenaphthene										
6E-1	6E-2	L	ND	ND	NA	ND	NA	ND	NA	NA
Acenaphthylene										
6E-1	6E-2	L	ND	ND	NA	D	NA	D	NA	NA
Anthracene										

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+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

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.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1995a, ***Update Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)***, U.S. Public Health Service, Atlanta, Georgia, August, on line.

Agency for Toxic Substances and Disease Registry, (ATSDR), 1995b, ***Update Toxicological Profile for Naphthalene, 1-Methylnaphthalene, 2-Methylnaphthalene***, U.S. Public Health Service, Atlanta, Georgia, August, on line.

De Jong, W.H., E.D. Kroes, J.G. Vos and H. Van Loveren, 1999, “Detection of Immunotoxicity of Benzo[a]pyrene in a Subacute Toxicity Study after Oral Exposure in Rats,” ***Toxicological Sciences*** 50: 214-220.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Krewski, D., T. Thorslund and J. Withey, 1989, "Carcinogenic Risk Assessment of Complex Mixtures," ***Toxicology and Industrial Health***, 5: 851-867.

Lewtas, J., 1988, "Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources," ***Fundamental and Applied Toxicology***, 10: 571-589.

Potter, T.L. and K.E. Simmons, 1998, ***Composition of Petroleum Mixtures***, Volume 2, Total Petroleum Hydrocarbon Criteria Working Group Series, University of Massachusetts, Amherst, May.

U.S. Environmental Protection Agency (EPA), 1986, “Guidelines for Carcinogen Risk Assessment,” ***Federal Register*** 51(185): 33992-34003.

U.S. Environmental Protection Agency (EPA), 1990, “National Oil and Hazardous Substances Pollution Contingency Plan,” ***Federal Register*** 55(46): 8666-8865.

U.S. Environmental Protection Agency (EPA), 1993a, ***Risk Assessment Issue Paper for: Derivation of a Subchronic RfC for Naphthalene (CASRN 91-20-3)***, National Center for Environmental Assessment, Cincinnati, Ohio, March 12.

U.S. Environmental Protection Agency (EPA), 1993b, ***Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons***, Office of Health and Environmental Assessment, Cincinnati, OH, EPA/600/R-93/089, March.

U.S. Environmental Protection Agency (EPA), 1994, ***Risk Assessment Issue Paper: Status of Inhalation Cancer Unit Risk for Benzo(a)Pyrene (CAS No. 50-32-8)***, National Center for Environmental Assessment, Cincinnati, OH, November 18.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 1998, ***Toxicological Review of Naphthalene (CAS No. 91-20-3) In Support of Summary Information on the Integrated Risk Information System (IRIS)***, August.

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.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

VOLATILE ORGANIC COMPOUNDS

BENZENE (71-43-2)

1.0 Introduction and Physical Properties

Benzene is a naturally occurring **VOC**, present in low levels in many plants and animals, as well as a synthetic compound derived from petroleum (ATSDR, 1997). It occurs as a colorless or slightly yellow liquid, or as a solid at temperatures below approximately 5.5 EC (HSDB, 2010). Benzene is widely used as a solvent and in the manufacturing of other organic compounds such as styrene, phenol, detergents, pesticides, explosives, polychlorinated biphenyls, flavors, perfumes, paints and coatings, nylon intermediates and photographic chemicals, among others. It is also used in food processing and leather tanning and occurs as a component of gasoline. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
78.1[a]	2.13[a]	5.56E-3[b] (25°C)	1.77[b]	8.80E-2[b] (25°C)	9.80E-6[b] (25°C)	1.25E-1[c] (25°C)	1.75E+3[b] (20-25°C)	353.24[d]	562.16[d]	7.34E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

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

[b]U.S. Environmental Protection Agency (EPA), 2001, *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.

[c]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 21 April.

[d]U.S. Environmental Protection Agency (EPA), 2003, *Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings*, Office of Emergency and Remedial Response, Washington, DC, 19 June.

2.0 Environmental Fate and Transport

Benzene enters the atmosphere primarily from fugitive emissions and automobile exhaust because of its presence in gasoline (ATSDR, 1997; HSDB, 2010). Other important sources are emissions associated with its production and use as a solvent, as an industrial intermediate in the production of the materials mentioned above, and coke oven blast furnaces and coke by-product recovery plants. In addition, there are discharges into water from industrial effluents and losses during spills. Sources of release to soil include land disposal of benzene-containing wastes, industrial discharge, and leaks from underground storage tanks and associated plumbing.

Benzene released to the atmosphere will exist almost exclusively in the vapor phase (ATSDR, 1997; HSDB, 2010). It is not subject to direct photolysis but it will react with photochemically produced hydroxyl radicals with a half-life of approximately 13 days. The reaction time in polluted atmospheres containing nitrogen oxides or sulfur dioxide is accelerated with the half-life being reported as 4 to 6 hours. Products of photooxidation include phenol, nitrophenols,

nitrobenzene, formic acid, and peroxyacetyl nitrate. Benzene is fairly soluble in water and is removed from the atmosphere in rain.

Benzene released to soil will be subject to rapid volatilization near the surface; that which does not evaporate will be highly to very highly mobile in the soil and may leach to groundwater (ATSDR, 1997; HSDB, 2010). Benzene may be subject to biodegradation under aerobic conditions. Microbial transformation proceeds through formation of cis-dihydrodiols to catechols, which lead to destruction of the aromatic ring.

Benzene may be subject to photooxidation and biodegradation in shallow, aerobic groundwaters, but probably not significantly under deeper or anaerobic conditions (ATSDR, 1997; HSDB, 2010). Benzene released to surface water will be subject to rapid volatilization. It is not expected to sorb to sediment or to hydrolyze significantly. Biodegradation is unlikely to be a significant fate process in sediment.

Benzene is not expected to participate significantly in food-chain pathways (ATSDR, 1997; EPA, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Benzene is rapidly and readily absorbed during inhalation exposures of humans or laboratory animals to vapors (ATSDR, 1997). However, the extent of uptake and retention decrease with increasing concentration or time of exposure, reflecting saturation of the metabolic processes that remove the substance from the blood. Excretion data from laboratory animals show that absorption is rapid and essentially complete following oral administration. Dermal uptake of liquid benzene through intact skin has been demonstrated in humans and may be toxicologically significant. The extent of dermal uptake of benzene vapor has not been quantified in humans, but experiments in laboratory animals suggest this exposure route also may be toxicologically significant.

Benzene is widely distributed following any route of exposure (ATSDR, 1997). Highest levels were located in adipose and other tissues high in fat such as the brain following inhalation exposure. Highest levels following oral administration were located in liver, kidney and bone marrow.

Quantitative aspects of the metabolism of benzene are somewhat species-, exposure route- and dose-dependent (ATSDR, 1997), but certain relevant generalizations pertain. The first metabolic step appears to be cytochrome P-450-catalyzed oxidation of benzene to benzene epoxide in the liver, which rearranges to phenol. Phenol is transformed to hydroxyquinone, which is further oxidized to benzoquinone, considered to be a major toxicant in bone marrow. Alternatively, phenol may be further oxidized to catechol and possibly to trihydroxybenzene. Phenol and all its derivatives except benzoquinone have been shown to undergo detoxication by conjugation with glucuronide or sulfate, followed by excretion.

A second general pathway involves the further oxidation of benzene oxide to benzene dihydrodiol, which leads to ring-opening and the formation of muconic dialdehyde, a known hematotoxin, primarily in the liver (ATSDR, 1997).

Absorbed benzene is excreted through the lungs as unchanged compound, and through the urinary tract as conjugated metabolites (ATSDR, 1997). Urinary excretion predominates.

Nothing in this review of toxicokinetics suggests that laboratory animals would not serve as appropriate models for the toxicity of benzene to humans.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for benzene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

As mentioned above, benzene in the liquid and vapor form are absorbed through the skin (ATSDR, 1997). Combining benzene with soil slows dermal uptake. EPA (2004) notes that VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels. Therefore, dermal uptake of benzene from soil is not evaluated. EPA (2003) provides t*, K_p, τ, B and FA values as follows:

Chemical	t*	K_p	τ	FA	B
Benzene	0.70	1.5E-2	0.29	1	0.1
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Short-term exposure to benzene induces central nervous system (CNS) effects such as drowsiness, dizziness and headaches; long-term exposure may induce anemia (ATSDR, 1997). Oral dosing in animals induces hematopoietic and immune effects. EPA (2010) verified a chronic oral RfD of 4E-3 mg/kg-day, based on hematological effects (reduced lymphocyte count) in a cross-sectional occupational study of 44 workers exposed to benzene. The workers were exposed for 0.7 to 16 years, with a mean of 6.3 years. Because the mean fell below 7 years, the minimal length considered to be chronic, the study is judged to be subchronic in duration. The control group consisted of an equal number of age- and gender-matched unexposed workers. The exposed workers were divided into two equal groups of 22: those exposed to airborne concentrations below and those exposed to airborne concentrations above the median concentration for all workers of 99 mg/m^3. An exposure-adjusted benchmark concentration (BMC) for minimal hematologic effects of 8.2 mg/m^3 was estimated. Assuming that uptake from the respiratory tract is approximately 50 percent of that from the GI tract allowed application of inhalation-to-oral extrapolation to estimate a benchmark dose (BMD) of 1.2 mg/kg-day. Application of an uncertainty factor of 300 to the BMD yielded the verified chronic oral RfD of 4E-3 mg/kg-day. The uncertainty factor of 300 consists of factors of 3 each for extrapolation from a BMC for minimal adverse effects to a BMC for no adverse effects, to expand from subchronic to chronic exposure, and to reflect deficiencies in the data base; a factor of 10 was used to provide additional protection for more sensitive human subpopulations. The blood cells (specifically inhibited hematopoiesis) are the target for prolonged oral exposure to benzene. Confidence in the chronic oral RfD is medium.

A preliminary subchronic oral RfD can be developed from the BMD described above. The subchronic oral RfD is derived by applying an uncertainty factor of 100 to the BMD of 1.2 mg/kg-day. The uncertainty factor of 100 reflects the uncertainty factor of 300 developed by EPA (2010) without the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 1.2E-2 mg/kg-day, which is rounded to 1E-2 mg/kg-day.

A verified chronic inhalation RfC of 3E-2 mg/m^3 for benzene was based on the exposure-adjusted BMC of 8.2 mg/m^3 in the occupational exposure study from which the oral RfD values were derived (EPA, 2010). Application of the uncertainty factor of 300 (as described for the verified chronic oral RfD) to the BMC of 8.2 mg/m^3 yielded the chronic RfC of 3E-2 mg/m^3. The RfC is equivalent to an inhalation RfD of 8.6E-3 mg/kg-day. Confidence in the chronic inhalation RfC is medium. The blood cells (specifically inhibited hematopoiesis) are the target for prolonged inhalation exposure to benzene. A preliminary subchronic inhalation RfC can be derived by application of an uncertainty factor of 100 to the BMC of 8.2 mg/m^3 in the same manner as was done to derive the subchronic oral RfD. The subchronic inhalation RfC so derived is 8.2E-2 mg/m^3, rounded to 8E-2 mg/m^3. The preliminary subchronic inhalation RfC is equivalent to an inhalation RfD of 2.3E-2 mg/kg-day.

6.0 Carcinogenicity Evaluation

Benzene is classified as an EPA cancer weight-of-evidence Group A chemical – a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies (EPA, 2010). Epidemiologic studies and case studies provide clear evidence of a causal association between exposure to benzene and acute nonlymphocytic leukemia, and also provide supportive evidence for chronic nonlymphocytic leukemia and chronic lymphocytic leukemia. Other neoplastic conditions in humans that are associated with benzene exposure include hematologic neoplasms, blood disorders such as preleukemia and aplastic anemia, Hodgkin's lymphoma, and myelodysplastic syndrome. Experimental animal data add to the weight of evidence that exposure to benzene increases the risk of cancer in multiple species at multiple organ sites (hematopoietic, oral and nasal, liver, forestomach, preputial gland, lung, ovary, and mammary gland). A verified URF range for inhalation exposure of 2.2E-6 to 7.8E-6 per µg/m^3 was based on the incidence of leukemia in humans exposed to benzene while employed in the Pliofilm industry (EPA, 2010). A low-dose linearity model utilizing maximum likelihood estimates was used to estimate the URF. The range reflects differences in the exposure assumptions estimated from the study. EPA (2010) concluded that neither any specific URF value nor either end of the range is more defensible than the other. Therefore, the upper end of the range, 7.8E-6 per µg/m^3, is conservatively selected for this evaluation. The URF of 7.8E-6 per µg/m^3 is equivalent to an inhalation SF of 2.7E-2 per mg/kg/day.

The occupational data that serve as the basis for the inhalation URF also serve as the basis for a verified oral SF for benzene (EPA, 2010). The inhalation URF range was mathematically converted to an equivalent oral SF range by applying standard inhalation rate and body weight assumptions, and by adjusting for route-specific differences in absorption as described for the chronic oral RfD derivation. The oral SF range so derived is 1.5E-2 to 5.5E-2 per mg/kg-day.

The upper end of the range, 5.5E-2 per mg/kg-day, is conservatively selected as the oral SF for this evaluation.

7.0 Toxicity Summary

Toxicity values for benzene are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1E-2	4E-3	Bc	8E-2/ 2.3E-2	3E-2/ 8.6E-3	Bc	A	5.5E-2	A	7.8E-6	2.7E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day).
Target organ abbreviations: Bc = blood cells.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1997, ***Update Toxicological Profile for Benzene***, U.S. Department of Health and Human Services, Atlanta, Georgia, September, on line.

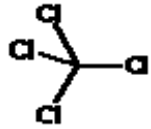
Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 21 April.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

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

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

CARBON TETRACHLORIDE (56-23-5)



1.0 Introduction and Physical Properties

Carbon tetrachloride is a **VOC** that occurs as a clear, colorless heavy liquid at ambient temperatures (HSDB, 2010). Its most recent major use has been in the production of various chlorofluorocarbons used as refrigerants (ATSDR, 2005). Other less recent uses include widespread application as a solvent and cleaning fluid. The production of carbon tetrachloride for most uses has been phased out due to Clean Air Act legislation; it is currently used in a relatively small number of applications where no satisfactory substitute has been found. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
153.8[a]	2.83[a]	2.76E-2[e] (25°C)	2.24[b]	7.80E-2[b] (25°C)	8.80E-6[b] (25°C)	1.51E-1[c] (25°C)	7.93E+2[b] (25°C)	349.90[d]	556.60[d]	7.13E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

[b]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[c]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, DC, 9355.4-24, December.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

[e]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

Carbon tetrachloride's production and use as a powerful solvent for asphalt, benzyl resin, bitumens, chlorinated rubber, ethylcellulose, gums, and rosin, as a cleaning agent for machinery and electrical equipment, and in the synthesis of nylon-7 and other chlorination processes has resulted in its release to the environment as a fugitive and through various waste streams (ATSDR, 2005; HSDB, 2010). Waste water from iron and steel manufacturing, foundries, metal finishing, paint and ink formulations, petroleum refining and nonferrous metal manufacturing industries may have contained carbon tetrachloride. Its use has been a major contributor to atmospheric concentrations.

Based on its K_{oc} value, carbon tetrachloride is expected to be moderately to highly mobile in soil, depending in part on the soil content of organic carbon, and may leach to groundwater (ATSDR, 2005; HSDB, 2010). Volatilization of carbon tetrachloride from moist soil surfaces is expected to be an important removal process; volatilization from dry surfaces is probably less significant. Biodegradation is another significant removal process, particularly in methanogenic conditions associated with landfills.

Carbon tetrachloride is not expected to adsorb to suspended solids and sediment in surface water, unless the organic carbon content of the sediment is high (ATSDR, 2005; HSDB, 2010). Volatilization from water surfaces is expected to be the predominant removal mechanism. Biodegradation is also expected to be significant.

Carbon tetrachloride is expected to exist solely as a vapor in the ambient atmosphere, which acts as a sink for the compound (ATSDR, 2005; HSDB, 2010). The compound is very stable in the troposphere with residence times of 30 to 50 years. A half-life of 366 years has been estimated for removal by reaction with photochemically-produced hydroxyl radicals. Its main loss mechanism is diffusion to the stratosphere where it is subject to photolysis.

The K_{ow} for carbon tetrachloride suggests that bioconcentration could be significant (ATSDR, 2005; HSDB, 2010). Empirical data with aquatic organisms, however, indicate that bioconcentration is not important (EPA, 1995). This is supported by the observation that most animals metabolize and eliminate carbon tetrachloride quite efficiently. It is concluded that food-chain pathways are not significant for this compound and biotransfer factors are not estimated.

3.0 Toxicokinetics

Carbon tetrachloride is rapidly absorbed by the lungs, as indicated by the measurement of near peak blood levels within 5 minutes of the beginning of exposure in rats (ATSDR, 2005). Net pulmonary uptake approximates 60 percent of the inhaled concentration in humans. Carbon tetrachloride is also absorbed rapidly from the GI tract, as indicated by peak blood levels observed within 3 to 6 minutes of oral administration in several species of laboratory animals. The recovery of 80 to 85 percent of an oral dose in exhaled air suggests that GI absorption is extensive. GI absorption occurs less rapidly following gavage in corn oil than administration in aqueous vehicles. Dermal uptake of neat carbon tetrachloride occurs rapidly and readily in humans and laboratory animals; dermal uptake of vapors is far less significant.

Absorbed carbon tetrachloride equivalents (parent compound and biotransformation products) are found in highest concentrations in fat and other lipid-containing tissues (ATSDR, 2005). Generally fat acts as a temporary sink, so that carbon tetrachloride equivalents are found in highest concentrations initially in high lipid-containing tissues with re-distribution resulting in higher levels in fat. At least in rats, unusually high levels initially have been reported in the brain, spinal cord and spinal nerves following inhalation exposure, and in the liver following oral exposure, the latter probably because of the first-pass phenomenon (all venous blood from the GI tract passes through the liver on its way to the *vena cava*).

The metabolism of carbon tetrachloride in laboratory animals has been studied extensively because the compound is a classical hepatotoxicant (ATSDR, 2005). The hepatotoxicity of carbon tetrachloride appears to be due entirely to the cytochrome P-450 enzyme CYP2E1, which is destroyed during metabolism of the compound. Bioactivation begins with reductive dehalogenation to form the trichlormethyl radical, which may undergo several subsequent reactions:

- Direct binding to microsomal lipids and proteins (see below).
- Accept a hydrogen atom to form chloroform, a toxicant in its own right.
- Dimerization to form hexachloroethane.
- Further reductive dechlorination to form carbon monoxide.

- Oxygenation to form trichloromethanol, a precursor to phosgene.

The trichloromethyl radical bound to liposomal components may remove a hydrogen atom from methylene groups on polyenic fatty acids, resulting in the release of an organic free radical, which is subsequently oxygenated to form a peroxy radical. Peroxy radicals rapidly transform to organic peroxides that attack neighboring methylene groups on polyenic fatty acids, forming a self-catalyzing reaction that rapidly destroys liver tissue.

Animal studies indicate that excretion following inhalation exposure is predominantly through the feces (32 to 60 percent of the inhaled dose), with 30 to 40 percent through expired air (ATSDR, 2005). Most of the expired material is parent compound; very little appears as carbon dioxide. Excretion following inhalation exposure of humans or animals is generally biphasic, with an initial phase half-life in humans of less than 1 hour and the second phase half-life approximating 40 hours. Studies in orally exposed animals indicate that saturation of biotransformation pathways occurs and storage in body fat depots increases with increasing dose, which lengthens the time for excretory mechanisms to effectively clear body residues.

4.0 Dermal Exposure

GI absorption is rapid and extensive as noted above. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for carbon tetrachloride and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data were not located for dermal uptake of carbon tetrachloride from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of carbon tetrachloride from soil is not evaluated. t*, K_p, τ, FA and B values are provided by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
Carbon Tetrachloride	1.86	1.6E-2	0.78	1.0	0.1
t* = Time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Carbon tetrachloride is a classic hepatotoxicant in humans and animals exposed by any route (ATSDR, 2005). High exposure levels also induced kidney effects in animals. Occupational exposure is associated with CNS and liver effects (ACGIH, 1991). EPA (2010) presents a verified chronic oral RfD of 7E-4 mg/kg-day based on a NOAEL for liver lesions in a 12-week gavage study in rats and an uncertainty factor of 1000. The NOAEL was 1 mg/kg in rats that were treated on five days each week, which is equivalent to a continuous exposure dose rate of 0.71 mg/kg-day. The LOAEL, 10 mg/kg (equivalent to 7.1 mg/kg-day) was associated with mild centrilobular vacuolization of the liver and accompanying blood chemistry indications of mild hepatocellular damage. EPA (2010) used a benchmark dose level approach to include a 95 percent confidence limit on the LOAEL, resulting a value of 3.09 mg/kg-day. An RfD of 4E-3 was derived by including a

composite UF of 1,000. The composite UF consists of factors of 10 each for interspecies variation, intrahuman variability, and extrapolation from subchronic to chronic duration of exposure. The principal target organ for the toxicity of carbon tetrachloride is the liver. Confidence in the oral RfD is medium.

No EPA subchronic oral RfD for carbon tetrachloride is currently available. The chronic RfD is used as a conservative estimate for subchronic toxicity.

EPA (2010) derived an RfC of 0.1 mg/m^3 based on a study by the Japan Bioassay Research Center (JBRC, 1998; Nagano et al., 2007; JBRC) where groups of F344/DuCrj rats (50/sex/group) were exposed (whole-body) to 0, 5, 25, or 125 ppm (0, 31.5, 157, or 786 mg/m^3) of carbon tetrachloride (99.8% pure) vapor for 6 hours/day, 5 days/week for 104 weeks. The critical effect is fatty change in the liver. Animals were observed daily for clinical signs, behavioral changes, and mortality. Body weights were measured once a week for the first 14 weeks and every 2 weeks thereafter. Urinalysis, hematology, and clinical chemistry tests were conducted at study termination.

Exposure levels studied in the 2-year JBRC rat bioassay were converted to estimates of internal doses by application of a physiologically based pharmacokinetic model. Benchmark dose modeling methodology was used to analyze the relationship between the estimated internal doses and response (i.e., fatty change of the liver). The resulting benchmark dose level values were converted to estimates of equivalent human exposure concentrations by applying a human physiologically based pharmacokinetic model.

A human physiologically based pharmacokinetic model was used to estimate continuous human equivalent concentrations (in mg/m^3) that would result in values for the internal dose metric equal to the benchmark dose level (at 10 percent risk) values for fatty changes of the liver. Dosing was normalized to a constant exposure concentration. The resultant human equivalent concentration calculated from male rat data of 14.3 mg/m^3 was used as the point of departure for RfC derivation. Dividing this point-of-departure concentration by a composite UF of 100 results in the RfC of 0.1 mg/m^3. The composite UF includes a UF of 10 each for interspecies extrapolation and intraspecies differences. Further details are provided in IRIS (EPA, 2010). An inhalation RfD of 2.9E-2 mg/kg-day is derived using the equation described in the introduction to this appendix.

6.0 Carcinogenicity Evaluation

Carbon tetrachloride is classified in cancer weight-of-evidence Likely to be Carcinogenic to Humans, based on increased incidence of liver tumors in rats, mice, and hamsters treated orally or by subcutaneous injection (EPA, 2010). A verified oral slope factor of 7E-2 per mg/kg-day was based on liver tumor data from inhalation studies in mice (JBRC, 1998; Nagano et al., 2007). Suitable data from oral studies were not available. However, because liver tumors have been observed to result for both oral and inhalation exposure, EPA (2010) judged it appropriate to derive an oral SF using route-to-route extrapolation. An inhalation unit risk of 6E-6 per µg/m^3, equivalent to 2.9E-2 per mg/kg-day, was derived from the same data. Refer to IRIS (2010) for further details.

7.0 Toxicity Summary

Toxicity values for carbon tetrachloride are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfc/ sRfD	cRfC/ cRfD	TO	WOE	SFo	WOE	URFi	SFi
4E-3	4E-3	L	1E-1/ 2.8E-2	1E-1/ 2.8E-2	L	B	7E-2	B	6E-6	2.1E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day).
Target organ abbreviations: L = liver.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2005, ***Update Toxicological Profile for Carbon Tetrachloride***, Draft for Public Comment, U.S. Department of Health and Human Services, Atlanta, Georgia, August.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices, Sixth Edition,*** Cincinnati, OH, pp. 232-236.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Japan Bioassay Research Center (JBRC), 1998, Subchronic Inhalation Toxicity and Carcinogenicity Studies of Carbon Tetrachloride in F344 Rats and BDF1 Mice (Studies Nos. 0020, 0021, 0043, and 0044), Kanagawa, Japan Industrial Safety and Health Association, Japan Bioassay Research Center, Kanagawa, Japan. Unpublished report to the Ministry of Labor. Hirasawa Hadano Kanagawa, 257 Japan.

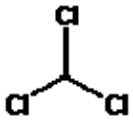
Nagano, K; Sasaki, T; Umeda, Y; et al.,2007, Inhalation Carcinogenicity and Chronic Toxicity of Carbon Tetrachloride in Rats and Mice. Inhal Tox 19:1089-1103.

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

CHLOROFORM (67-66-3)



1.0 Introduction and Physical Properties

Chloroform is a **VOC** that occurs as a clear, colorless, heavy volatile liquid at ambient temperatures (HSDB, 2010). It occurs naturally in the environment, being made by certain forms of algae, seaweed, and the ecological community in peat bogs. Chloroform has been used as a solvent or extraction solvent, dry cleaning fluid, in fire extinguishers, and as an intermediate in the manufacture of dyes and pesticides (ATSDR, 1997). Major current uses include organic synthesis, particularly fluorocarbon-22. It is also formed in the chlorination of drinking water. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
119.4[b]	1.97[a]	3.67E-3[b] (25°C)	1.60[b]	1.04E-1[b] (25°C)	1.00E-5[b] (25°C)	2.59E-1[c] (25°C)	7.92E+3[b] (20-25°C)	334.32[d]	536.40[d]	6.99E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K);)$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).
data.

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.

[c]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 9 January.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

2.0 Environmental Fate and Transport

Chloroform has been shown to occur naturally in the environment, and its release to the atmosphere from tropical oceans amounts to hundreds of thousands of tons per year (HSDB, 2010). Chloroform also enters the environment associated with its use as an industrial solvent, extractant and chemical intermediate as well as from its inadvertent production in the chlorination of drinking water, municipal sewage and cooling water (ATSDR, 1997; HSDB, 2010). Most environmental releases from industrial uses occur directly to the atmosphere.

Chloroform is expected to exhibit moderate mobility in soil (HSDB, 2010), potentially leaching to groundwater. Volatilization from moist soil surfaces is expected to be an important removal process, given its relatively large Henry's Law constant. Volatilization from dry soil surfaces may also be an important removal process. In one study of chloroform residence time in soils, the compound was found to have a half-life of 0.3 days when applied 1 cm deep into soil, and 1.4 days when applied 10 cm deep. It is classified as a "very short-lived" chemical in soil matrices primarily due to its high volatility. Under normal environmental conditions, chloroform is not expected to undergo

biodegradation in soils, although the compound has been shown to be anaerobically degraded by methanogenic bacteria in the presence of a primary substrate such as acetic acid, and even more readily under sulfate reducing conditions.

Chloroform is not expected to adsorb significantly to sediment or suspended solids in water (HSDB, 2010). Volatilization from water surfaces is expected to be the major process for removing chloroform from surface water. Volatilization half-lives for a model river and model lake have been estimated at 1.3 hours and 4.4 days, respectively. A field study found that the volatilization half-life from the Rhine River was 1.2 days, while in a lake located in the Rhine basin the half-life was 31 days. In another study, chloroform from a municipal treatment plant injected into an estuarine arm of Chesapeake Bay entirely disappeared within 4 km in the spring and within 11 km in the winter under ice. The decrease in concentration could not be attributed entirely to dilution. Based on available experimental data, aquatic degradation is not expected to be a major removal process, although the data are somewhat conflicting. Although base-catalyzed hydrolysis of chloroform is expected to occur, the estimated kinetics predict that this will not be an environmentally important degradation process.

Chloroform is expected to exist solely as a vapor in the ambient atmosphere (HSDB, 2010). Vapor-phase chloroform is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 151 days. Data regarding the potential for other mechanisms to play an important role in chloroform removal were not located. It is likely that wet deposition or washout may occur, but volatilization would probably readily return the compound to the atmosphere. The tropospheric half-life for chloroform has been estimated at 3 years.

The relatively low log K_{ow} value for chloroform suggests that its participation in food-chain pathways is unlikely to be significant (HSDB, 2010). Measured values for BCF, as well as the observation that the compound is rapidly metabolized confirm this suggestion (ATSDR, 1997; HSDB, 2010; EPA, 1995). Therefore, biotransfer factors are not derived for chloroform.

3.0 Toxicokinetics

Several toxicokinetic studies in animals and humans suggest that chloroform is virtually completely absorbed from the GI tract (ATSDR, 1997). Peak blood levels were higher and attained more quickly from an aqueous vehicle than from an oleaginous vehicle. Although several physiological factors (blood/air partition coefficient, ventilation rate, cardiac output, total body weight and fat content) influence the kinetics of uptake from the respiratory tract, generally arterial blood concentrations are directly proportional to the exposure concentration, suggesting that uptake occurs readily, particularly at concentrations below the saturation point. Experiments with humans indicate that dermal uptake of chloroform in water or ethanol readily occurs.

Absorbed chloroform is rapidly and widely distributed throughout the body, readily crossing the blood-brain and placental barriers (ATSDR, 1997). Highest levels are located in tissues with higher fat content, including adipose tissue, nervous system and liver.

Excretion data suggest that chloroform metabolism is quite species-dependent. Following oral dosing with radiolabeled compound, monkeys excreted most of the dose as unchanged compound in expired air, mice excreted most of the dose as carbon dioxide, and rats excreted both unchanged

compound and carbon dioxide (HSDB, 2010). Urinary metabolites included urea (probably formed from carbon dioxide).

The predominant pathway for chloroform metabolism involves the generation of phosgene in the liver, kidney and other tissues as an intermediate in the formation of carbon dioxide (ATSDR, 1997). Phosgene is a toxicant in its own right, combining covalently with microsomal proteins to disrupt cellular integrity and induce necrosis. Humans appear to be less efficient than laboratory rodents in metabolizing chloroform to phosgene, suggesting that a comparable dose might be less toxic to humans. Trihalomethanes as a group are metabolized to carbon monoxide by most species as a result of reductive dechlorination and hydrolysis (ATSDR, 1997; HSDB, 2010). This, however, represents a relatively minor metabolic pathway, and chloroform is somewhat less efficient in forming carbon monoxide than the other trihalomethanes. Carbon monoxide is also toxic, resulting in the formation of carboxyhemoglobin, which is incapable of releasing oxygen to the tissues and transporting carbon dioxide from the tissues.

The routes and kinetics of chloroform excretion depend on the species, percent of body fat, route of exposure and dose. Excretion occurs primarily as carbon dioxide or unchanged compound via exhaled air; minor metabolites are eliminated through the urine and, to a lesser extent, the feces.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for chloroform and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

A hypothetical dermal absorption rate of 29.7 mg/minute was modeled for human hands immersed in liquid chloroform, based on absorption data measured in mice (ATSDR, 1997). Data were not located for dermal uptake from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of chloroform from soil is not evaluated. t*, K_p, τ, FA and B values are provided by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
Chloroform	1.19	6.8E-3	0.50	1.0	0
t* = Time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Oral or inhalation exposure of animals to chloroform is associated with liver and kidney damage (ACGIH, 1991; ATSDR, 1997; EPA, 2010). The case for kidney effects associated with prolonged oral exposure in humans, however, is equivocal. In humans, acute inhalation exposure to high levels induces narcosis, ventricular fibrillation and death (ACGIH, 1991). Limited occupational data associate chronic exposure to chloroform with CNS depression, digestive disturbances and enlarged livers, but not with olfactory epithelial involvement, which has been seen in laboratory animals.

Chloroform has been the subject of continued investigation and reevaluation because of its nearly ubiquitous nature as an anthropogenic chemical and the potential for widespread exposure. An earlier EPA (2004b) evaluation derived a chronic oral RfD of 1E-2 mg/kg-day based on a LOAEL for fatty cyst formation in the livers and biochemical evidence of liver damage in a 1979 study in which dogs were treated orally for 7.5 years. An uncertainty factor of 1000 was applied. In this dog study, the chemical was given orally in a toothpaste base in gelatin capsules in an effort to test the long-term effects of chloroform in toothpaste. Confidence in this earlier oral RfD was medium to low. The liver was considered the target organ for chronic oral exposure to chloroform.

Other studies analyzed in a recent reevaluation include a 1995 drinking water study in mice, a 1995 gavage study in rats, and a 2000 drinking water study in rats. Subtle effects on the kidney appear to be the critical effects of prolonged exposure in rodents. The liver lesions in the 1979 dog study remain the more sensitive endpoint of chloroform exposure and the more appropriate basis for an oral RfD. EPA (2010) developed a benchmark dose limit associated with a 10 percent extra risk for the liver lesions and biochemical evidence described above ($BMDL_{10}$) of 1.2 mg/kg-day. Application of an uncertainty factor of 100 and rounding to one significant figure yields the more recently verified oral RfD of 1E-2 mg/kg-day. The uncertainty factor of 100 consists of factors of 10 each for inter- and intra-species variation. Confidence in the new oral RfD is medium; slightly higher than the level of confidence in the earlier derivation because the BMDL approach is considered somewhat more reliable than the LOAEL/NOAEL approach to RfD development. The rodent studies provide sufficient information to identify both the liver and the kidney as target organs for prolonged oral exposure to chloroform.

Previously, EPA (1997a) adopted the earlier chronic oral RfD of 1E-2 mg/kg-day as the provisional RfD for subchronic oral exposure. As noted above, the earlier chronic oral RfD derivation has been revised, although the value of the RfD remains the same. Therefore, the new verified chronic oral RfD of 1E-2 mg/kg-day is adopted as sufficiently protective for subchronic exposure, but EPA (1997a) is no longer cited as the source.

Prolonged inhalation exposure of rats is associated with atrophy of the ethmoid epithelium at concentrations lower than those associated with liver or kidney effects (EPA, 1997b, 2003). An earlier provisional subchronic inhalation RfC was derived from a 13-week intermittent exposure study in rats based on effects on the ethmoid epithelium. More recently, EPA (2003) determined that atrophy of the nasal epithelium in rats is not an appropriate model for human toxicity, in part because this effect has not been observed in humans. The effects on the liver and kidney appear consistently to be the critical effects in humans following inhalation or oral exposure. Subchronic studies in rats and mice showed that mice were somewhat more sensitive than rats to these effects.

A large study of B6C3F1 strain mice was conducted in which groups of 15 mice of each sex were exposed to chloroform in air at concentrations of 0 (control), 0.3, 2, 10, 30 or 90 ppm on 6 hours per day, 7 days per week, for a total of 13 weeks (EPA, 2003). Other groups of 13 to 15 mice of each sex were exposed to 10 or 90 ppm 6 hours per day, 5 days per week, for a total of 13 weeks to see if there were differences between exposure on 5 or 7 days per week in the intensity of the response. Still other groups of 5 to 13 female mice were exposed to 0, 0.3, 2, 10, 30 or 90 ppm on 6 hours per day for 4, 21 or 42 continuous days to investigate the time-course development of chloroform-induced liver lesions. Finally, groups of 8 mice of each sex were exposed to 30 or 90 ppm 6 hours

per day for 6 weeks followed by a 6-week recovery period in which they were held without exposure, to test the reversibility of the liver and kidney effects.

Kidney lesions were observed only in the male mice exposed to 30 or 90 ppm chloroform (EPA, 2003). There appeared to be little difference between mice exposed on 5 days per week and those exposed for 7 days per week. The kidney lesions did not appear to be reversible, at least during the 6-week recovery period afforded certain groups. Liver lesions were observed in both male and female mice exposed to 30 or 90 ppm chloroform. Severity was greater in those exposed on 7 days per week compared with those exposed on 5 days per week. The liver lesions appeared to be largely reversible during the 6-week recovery period. This study identified 10 ppm as the NOAEL and 30 ppm as the LOAEL for liver and kidney effects in mice. However, a "labeling index" was slightly increased in the kidneys of male mice exposed to 10 ppm. The labeling index suggests that chloroform metabolites may have bound to macromolecules in the kidney cells at levels below those with discernable pathological change. A smaller study in BDF1 mice identified 5 ppm as a NOAEL and 30 ppm as the LOAEL for kidney and liver effects in a second strain of mice.

EPA (2003) chose the 5 ppm NOAEL in mice described above as the basis for developing subchronic and chronic RfCs for chloroform. The 5 ppm concentration, equivalent to 25 mg/m^3, was adjusted to a human equivalent concentration for continuous exposure of 4.5 mg/m^3. Application of an uncertainty factor of 100 yields a provisional inhalation RfC, rounded to one significant figure, of 5E-2 mg/m^3. The uncertainty factor consists of a factor of 10 to provide additional protection to unusually sensitive members of the population, and factors of 3 each to account for physiological differences between mice and humans, and to reflect uncertainty about the data base. The major uncertainty surrounds the potential for neurological effects to occur at levels lower than the NOAEL for kidney and liver effects. The provisional RfC is equivalent to 1.4E-2 mg/kg-day. The provisional RfC is appropriate for either subchronic or chronic exposure, because the large study in B6C3F1 mice did not indicate increased incidence or severity of effects with increased duration of exposure. The liver and kidney are the target organs for prolonged inhalation exposure to chloroform. Confidence in the RfC is low to medium.

6.0 Carcinogenicity Evaluation

The carcinogenicity of chloroform has recent undergone reevaluation and updating on the IRIS record (EPA, 2010). Chloroform was formerly classified as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on increased incidence of several tumor types in rats and liver tumors in mice. Human carcinogenicity data are inadequate. A verified oral SF of 6.1E-3 per mg/kg-day was derived from the incidence of kidney tumors in rats treated with chloroform in drinking water for two years. A verified inhalation URF of 2.3E-5 per ug/m^3, developed in 1987, was based on the incidence of hepatocellular carcinomas in mice treated by gavage for 78 weeks. The inhalation URF is equivalent to 8.1E-2 per mg/kg-day.

The more recent cancer evaluation describes chloroform *as likely to be carcinogenic to humans by all routes of exposure under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues (i.e., liver and kidney)* (EPA, 2010). On the other hand, chloroform is unlikely to be carcinogenic to humans by any route of exposure under exposure conditions that do not cause cytotoxicity and cell regeneration. Chloroform appears to exert its carcinogenicity through epigenetic mechanism(s) that are inoperative unless cellular damage in susceptible tissues is sufficient to result in cell regeneration. Since there are thresholds for cytotoxicity, the carcinogenic

expression of chloroform is generally assessed as a threshold phenomenon. In other words, the chronic oral RfD of 1E-2 mg/kg-day is considered to be sufficiently protective against carcinogenicity by oral or dermal exposure, and the inhalation RfC of 5E-2 mg/m^3 (1.4E-2 mg/kg-day) is considered to be sufficiently protective against carcinogenicity for inhalation exposure. It should be noted that the previously verified oral SF has been removed from the IRIS record. The previously verified inhalation URF still appears on the IRIS record, but it is preceded by the caveat that it is based on a 1987 evaluation and does not incorporate newer data or reflect updated cancer assessment guidelines. The inhalation carcinogenicity assessment is currently under revision (EPA, 2010).

In most cases it is not necessary to evaluate the carcinogenicity of chloroform using either the oral or inhalation SFs described above, because the noncancer effects would drive decision-making. In fact, it would be inappropriate to base selection of chloroform as a chemical of concern, or to develop remedial goal options for chloroform, based on cancer risk. However, it may be useful to estimate the cancer risk associated with chloroform for the baseline condition. This could be done using the oral and inhalation SFs described above, but it should only be done if the route-specific exposure dose on a daily basis exceeds the route-specific RfD.

7.0 Toxicity Summary

Toxicity values for chloroform are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfc/ sRfD	cRfC/ cRfD	TO	WOE	SFo	WOE	URFi	SFi
1E-2	1E-2	L, K	5E-2/ 1.4E-2	5E-2/ 1.4E-2	L, K	See Section 6	ND[b]	See Section 6	ND[b]	ND[b]

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data.
Target organ abbreviations: L = liver; K = kidney.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.
[b]Chloroform is determined to be a threshold carcinogen more appropriately evaluated by the RfD and RfC values derived for protection against threshold phenomena.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1997, ***Update Toxicological Profile for Chloroform***, U.S. Department of Health and Human Services, Atlanta, Georgia, September.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 289-291.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 9 January.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 1997a, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, PB97-921199, July.

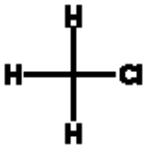
U.S. Environmental Protection Agency (EPA), 1997b, ***Risk Assessment Issue Paper for: Derivation of Provisional Subchronic and Chronic RfCs for Chloroform (CASRN 67-66-3)***, National Center for Environmental Assessment, Cincinnati, OH, 1 December.

U.S. Environmental Protection Agency (EPA), 2003, ***Risk Assessment Issue Paper for: Derivation of Provisional Subchronic and Chronic RfCs for Chloroform (CASRN 67-66-3)***, National Center for Environmental Assessment, Cincinnati, OH, 22 January.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*** Final, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

CHLOROMETHANE (METHYL CHLORIDE) (74-87-3)



1.0 Introduction and Physical Properties

Chloromethane is a **VOC** that exists as a colorless gas at ambient temperatures (HSDB, 2010). It is used largely as a methylating agent in the production of silicones, tetramethyl lead, methyl cellulose, methylene chloride, methyl mercaptan, plastics, pesticides, pharmaceuticals, dyes, perfumes, ethers, resins, agricultural chemicals, quaternary amines and butyl rubber (ACGIH, 1991; ATSDR, 1998; HSDB, 2010). It is also used as an extractant for fats, oils and resins, as a blowing agent and as a refrigerant. It was formerly used as a propellant in various aerosol products. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
50.5[a]	0.91[a]	8.82E-3[b] (25□C)	1.15[b]	1.39E-1[c] (25□C)[d]	1.49E-5[e] (20□C)	5.66E+0[b] (25□C)	5.32E+3[b] (25 □C)	247[f]	416[f]	5.15E+3[f]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

[b]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[c]Calculated as described in Introduction to Toxicological profiles.

[d]Assumed.

[e]Montgomery, J.H., 1996, ***Groundwater Chemicals Desk Reference, Second Edition***, Lewis Publishers, New York, pp. 664-666.

[f]National Institute of Standards and Technology (NIST), 2010, ***NIST Chemistry WebBook***, on line.

2.0 Environmental Fate and Transport

Chloromethane is one of the few halogenated organic chemicals for which natural sources are significant. Natural sources include volcanoes, volatilization from plants, and forest fires (HSDB, 2010). It is produced in seawater by the reaction of methyl iodide with chloride ions. Methyl iodide is produced photosynthetically by several marine organisms, and there may be other oceanic sources as well. The mean air flux of 1.3E-6 g/cm^2-year for the eastern Pacific Ocean, when extrapolated to global waters, provides an adequate source to explain the atmospheric reservoir of chloromethane. Chloromethane is released from brush and forest fires. The estimated 0.6 g of chloromethane released per kg of vegetation burned is believed to make a significant impact on the global burden of the compound.

The production and many uses of chloromethane may result in its release to the environment through various waste streams (HSDB, 2010). It is also released in tobacco smoke and turbine exhaust. Just as forest fires contribute to natural sources, wood burning, field burning, and backyard burning also contribute to atmospheric burdens. Chloromethane is formed in the

chlorination of drinking water and sewage effluent, and is found in the effluent of some publically owned treatment works.

Most chloromethane is released to the atmosphere where it is expected to exist solely as a gas (HSDB, 2010). Gas-phase chloromethane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 310 days, which provides opportunity for widespread dispersion. The dominant process for removal from the troposphere is upward diffusion, although washout by rain may also be important. From the tropopause to about 30 km, both upward diffusion and reaction with hydroxyl radicals will be of approximately equal importance, and above 30 km in the stratosphere, diffusion, reaction with hydroxyl radicals, and photodissociation will have approximately equal importance. The surface half-life resulting from upward diffusion is approximately 80 days.

Based on its very low estimated K_{oc} value, chloromethane is expected to have very high mobility in soil (HSDB, 2010). Its relatively high water solubility suggests the compound released in sufficient quantity in the liquid phase or dissolved in water could leach readily to groundwater. Volatilization from moist soil surfaces is expected to be an important removal process for small quantities released to the surface. Volatilization from dry soil surfaces may also be important. Field and laboratory studies demonstrate that several halogenated aliphatics may biodegrade slowly under anaerobic conditions, but not under aerobic conditions. Biodegradation is not expected to be a significant removal process.

Chloromethane is not expected to adsorb to suspended solids and sediment in surface water (HSDB, 2010). Volatilization from water surfaces is expected to be a significant removal mechanism; volatilization half-lives for a model river and model lake are 46 minutes and 3 days, respectively. Field and laboratory studies demonstrate that several halogenated aliphatics may biodegrade slowly under anaerobic conditions, but not under aerobic conditions. Biodegradation is not expected to be a significant removal process. Hydrolysis may occur, but very slowly, with a half-life of approximately 2 years (ATSDR, 1998). Hydrolysis will not be a significant removal process for chloromethane in surface water, but may be the only significant removal process for the compound in groundwater, where it has been shown to persist.

Chloromethane is water soluble, volatile and labile, and is not expected to participate significantly in food-chain pathways (ATSDR, 1998; HSDB, 2010). Therefore, biotransfer factors are not estimated for this compound.

3.0 Toxicokinetics

Studies regarding the toxicokinetics of chloromethane are limited to inhalation exposure. Chloromethane is readily absorbed by the respiratory system and equilibrium conditions are reached in approximately one hour in exposed humans and laboratory animals (ATSDR, 1998). Data regarding the extent of respiratory absorption were not located. Although data were not located for GI absorption, it is expected that GI absorption would be rapid and extensive because the compound is a low MW organic substance that is readily absorbed by the respiratory system. HSDB (2010) stated that dermal absorption can occur, but no other information was provided.

Radioactivity is rapidly and extensively distributed throughout the body following inhalation exposure to radiolabeled chloromethane, with highest levels of radioactivity measured in liver, kidney, testis and lung (ATSDR, 1998). Approximately 20 percent of the radiolabel in liver and testis was found to have been metabolically incorporated into the cellular structure, reflecting uptake and utilization from the one-carbon atom pool of the body.

The primary and predominant metabolic pathway of chloromethane involves enzymatic conjugation with glutathione to form S-methylglutathione (ATSDR, 1998). Further metabolism involves deamination to yield S-methylcysteine and other sulfur-containing conjugation derivatives, which are excreted in the urine. Another metabolic pathway involves dehalogenation of chloromethane to form formaldehyde (a toxicant in its own right) and formic acid. These intermediates contribute to the one-carbon atom pool that is available for incorporation into the tissues or for mineralization to carbon dioxide followed by excretion through the lungs.

Studies have shown that adult humans, unlike laboratory animals, may be divided into two groups – fast or slow metabolizers – depending on the activity in their erythrocytes of glutathione-S-transferase, which is responsible for the first conjugation reaction (ATSDR, 1998). Fast metabolizers clear chloromethane from the body more rapidly than slow metabolizers. Whether the unborn, new born or young children exhibit this characteristic is unknown. The implications of glutathione-S-transferase activity regarding population differences in toxic potency are unclear. On the one hand, it may appear that fast metabolizers should be more tolerant of chloromethane because of their enhanced ability to clear the compound from the body. On the other hand, experiments in laboratory animals suggest that a metabolite of S-methylglutathione may be the ultimate toxicant, in which case fast metabolizers may be more sensitive to chloromethane toxicity.

Chloromethane residues are rapidly cleared from the body following the cessation of inhalation exposure (ATSDR, 1998). Clearance in laboratory animals and humans is biphasic, consistent with a linear 2-compartment model. The half-lives for both phases were measured in minutes, indicating the rapidity with which clearance occurs. The exception is that the β-phase in some humans (slow metabolizers) approximated 1.5 hours.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Chloromethane is assumed to be absorbed readily from the GI tract; therefore, no GAF is developed.

Data regarding the dermal uptake of chloromethane were not located, except that HSDB (2010) stated that dermal absorption could occur. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of chloromethane from soil is not quantified. EPA (2004a) provides t*, K_p, τ, FA and B values as follows:

Chemical	t*	K_p	τ	FA	B
Chloromethane	0.49	3.3E-3	0.20	1	0

Chemical	t*	K_p	τ	FA	B
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Data regarding the toxicity of oral exposure to chloromethane consist of the statement that liver effects were not observed in rats given a single oral dose of 420 mg/kg (ATSDR, 1998; EPA, no date). EPA (no date) derived a provisional chronic oral RfD of 4E-3 mg/kg-day based on a LOAEL of 69.32 mg/m^3 for subtle neurobehavioral effects from an occupational (inhalation) study. Subsequently, EPA (2010) reviewed the data and determined that ingestion exposure is not applicable because the compound exists as a gas at ambient temperatures. They also noted that the oral exposure data are insufficient to develop an oral RfD. Therefore, the EPA (no date) derivation is considered to be not applicable and no oral RfD is offered herein.

EPA (2010) verified a chronic inhalation RfC of 9E-2 mg/m^3 based on a NOAEL of 50 ppm (103.2 mg/m^3) from an 11-day continuous exposure study in mice. The NOAEL corresponds to a human equivalent concentration (HEC) of 94.6 mg/m^3. The LOAEL in this study, 100 ppm (206.4 mg/m^3 or a HEC of 189.2 mg/m^3) was associated with slight to moderate degenerative changes in the granule cells of the cerebellum. EPA (2010) applied an uncertainty factor of 1000 to the NOAEL HEC of 94.6 mg/m^3 to derive the RfC of 9E-2 mg/m^3. The uncertainty factor consists of factors of 10 each to provide additional protection for unusually sensitive humans and to expand from a short-term to lifetime exposure, and factors of 3 each to extrapolate from mice to humans and to address the uncertainty regarding the potential for CNS effects in the developing young. Confidence in the verified chronic inhalation RfC is medium. The verified inhalation RfC is equivalent to an inhalation RfD of 2.6E-2 mg/kg-day. The CNS is the target organ for prolonged inhalation exposure to chloromethane.

EPA (1996) developed a provisional chronic RfC based on subtle neurobehavioral effects from an occupational (inhalation) study. They adopted the provisional chronic RfC for subchronic application, noting no significant differences in response between chronic and subchronic exposure. However, given the EPA (2010) reevaluation and verification of a more conservative chronic RfC, it is deemed prudent to adopt the verified chronic RfC rather than the provisional chronic RfC as sufficiently protective for subchronic exposure.

6.0 Carcinogenicity Evaluation

Previous EPA (1997, 1998) evaluations classified chloromethane in EPA weight-of-cancer Group C (possible human carcinogen), and developed an oral SF and an inhalation URF. More recently, EPA (2010) reevaluated the data, noting that chloromethane is a naturally occurring compound, and concluded that it is more appropriately classified in Group D (not classifiable as to its human carcinogenicity). Applying more recent cancer assessment guidelines, EPA (2010) concluded that the available data suggest that chloromethane would be classified as an agent whose carcinogenic potential *cannot be determined*. Previously developed potency factors are considered to be not applicable; no potency factors are offered herein for chloromethane.

7.0 Toxicity Summary

Toxicity values for chloromethane are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
ND	ND	NA	9E-2/ 2.6E-2	9E-2/ 2.6E-2	CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.
Target organ or critical effect abbreviations: CNS = central nervous system.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1998, ***Update Toxicological Profile for Chloromethane***, U.S. Department of Health and Human Services, Atlanta, GA, December, on line.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, Cincinnati, OH, pp. 953-957.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), no date, ***Risk Assessment Issue Paper for: Derivation of a Provisional RfD for Chloromethane (CASRN 74-87-3)***, National Center for Environmental Assessment, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1996, ***Risk Assessment Issue Paper for: Derivation of a Provisional Chronic and Subchronic RfC for Chloromethane (CASRN 74-87-3)***, National Center for Environmental Assessment, Cincinnati, OH, 23 July.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

U.S. Environmental Protection Agency (EPA), 1998, ***Draft Risk Assessment Issue Paper for: Updating the Quantitative Carcinogenicity Evaluation of Chloromethane (CASRN 74-87-3)***, National Center for Environmental Assessment, Cincinnati, OH, 18 December.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*** Final, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

DIBROMOCHLOROMETHANE (CHLORODIBROMOMETHANE) (124-48-1)

1.0 Introduction and Physical Properties

Dibromochloromethane is a liquid (Lewis, 1997) generally considered to be a **VOC** (EPA, 2002). It is used as an intermediate in the organic chemical synthesis of fire extinguishing agents, aerosol propellants, refrigerants and pesticides (EPA, 1980; HSDB, 2010; Lewis, 1997). It is one of the trihalogenated methanes generated in potable water or cooling water by treatment with chlorination (HSDB, 2010). Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
208.3[a]	2.23[a]	7.83E-4[b] (25 °C)	1.80[b]	1.96E-2[b] (25 °C)	1.05E-5[b] (25 °C)	4.10E-2[c] (25 °C)	2.60E+3[b] (20-25 °C)	416.14[d]	678.2[d]	8.0E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K); $\Delta H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.
[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.
[c]U.S. Environmental Protection Agency (EPA), 1994, ***Technical Background Document for Soil Screening Guidance***, Review Draft, Office of Solid Waste and Emergency Response, Washington, DC, Publication No. 9355.4-17, EPA540/R-94/106, PB95-963532, November.
[d]U.S. Environmental Protection Agency (EPA), 1997, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings***, prepared by Environmental Quality Management, Inc. for the Office of Emergency and Remedial Response, Washington, DC, September.

2.0 Environmental Fate and Transport

The predominant anthropogenic source of dibromochloromethane release to the environment is its inadvertent formation during chlorination of water (HSDB, 2010). It may also be released as a fugitive or in waste streams from its production or use as a chemical intermediate. In addition to anthropogenic sources, it is biosynthesized and released to the environment by various species of marine macroalgae, which are abundant in various locations of the world's oceans.

Volatilization is the dominant removal process for dibromochloromethane in surface water (HSDB, 2010). The volatilization half-life from rivers and streams has been estimated to range from 43 minutes to 16.6 days with a typical half-life being 46 hours. It is not expected to adsorb to sediment or solids. In confined aquatic media where volatilization is not plausible (e.g., groundwater), anaerobic biodegradation may be the major removal process. Aquatic hydrolysis, oxidation and direct photolysis are not environmentally important.

Volatilization is the dominant removal process for dibromochloromethane released to soil where exposure to air is possible (HSDB, 2010). Dibromochloromethane is moderately to highly mobile in soil and will leach to groundwater. The compound is resistant to aerobic biodegradation but degrades anaerobically within 2 weeks to 8 weeks.

Dibromochloromethane is expected to exist solely as a vapor in the ambient atmosphere (HSDB, 2010). Vapor-phase dibromochloromethane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to

be 278 days. Direct photolysis does not occur beneath the ozone layer. Dibromochloromethane is expected to persist in air and potentially travel long distances.

Dibromochloromethane is a VOC for which participation in food-chain pathways is unlikely to be significant (ATSDR, 1989; HSDB, 2010). EPA (1995) identified dibromochloromethane as a chemical that poses no concern for bioaccumulation in fish. Therefore, biotransfer factors are not developed.

3.0 Data for Evaluating Dermal Exposure

Data were not located regarding the toxicokinetics of dibromochloromethane. Studies in mice and monkeys suggest that GI absorption of bromodichloromethane, however, is virtually complete (ATSDR, 1989). EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for dibromochloromethane and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data regarding the dermal uptake of dibromochloromethane were not located. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of dibromochloromethane from soil is not quantified. EPA (2004) provides K_p, τ, FA and B values as follows:

Chemical	K_p	τ	FA	B
Dibromochloromethane	3.2E-3	1.57E+0	1.0	0
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).				

4.0 Noncancer Effects Evaluation

Oral LD_{50} values for dibromochloromethane range from 800 to 1200 mg/kg in rats and mice (HSDB, 2010). Acute oral exposure is associated with sedation and anesthesia upon administration and with liver and kidney effects upon necropsy (EPA, 1980; Lewis, 1992). Liver lesions appear to be the most sensitive effect of long-term oral exposure. A 13-week gavage study in rats identified a dose rate of 21.4 mg/kg-day as a NOEL for fatty liver changes and hepatic necrosis (EPA, 2010). Kidney lesions were also observed. The LOAEL was 43 mg/kg-day. Application of an uncertainty factor of 1000 to the NOEL of 21.4 mg/kg-day yields a verified RfD for chronic oral exposure of 2E-2 mg/kg-day. The uncertainty factor consists of factors of 10 each for intra- and inter-species variation, and to expand from subchronic to chronic exposure. Confidence in the RfD is medium. The liver and kidney are considered to be the target organs for prolonged oral exposure to dibromochloromethane.

EPA (1997) derived a provisional subchronic oral RfD for dibromochloromethane of 2E-1 mg/kg-day by applying an uncertainty factor of 100 to the NOEL of 21.4 mg/kg-day in the same study. The uncertainty factor was adjusted to remove the component for subchronic to chronic expansion.

Data regarding inhalation exposure to dibromochloromethane were not located in the available literature; an inhalation RfC is not available.

5.0 Carcinogenicity Evaluation

EPA (2010) classified dibromochloromethane as a cancer weight-of-evidence Group C compound (possible human carcinogen) on the basis of inadequate evidence of carcinogenicity in humans and limited evidence of carcinogenicity in animals. The human data are restricted to several ecological and case-control studies that associate a variety of human tumors with exposure to chlorinated drinking water; however, exposure in this scenario involves a mixture of trihalogenated methanes. The animal data include two-year gavage studies in rats and mice; no evidence of carcinogenicity was observed in the rats, but increased incidences of hepatocellular adenomas and carcinomas was observed in the mice. Dibromochloromethane is structurally similar to other known animal carcinogens. A verified oral SF of 8.4E-2 per mg/kg-day is estimated for the incidence of liver tumors in female mice in the two-year gavage study. The data are inadequate for estimating cancer potency for inhalation exposure.

6.0 Toxicity Summary

Toxicity values for dibromochloromethane 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
2E-1	2E-2	L, K	ND/ ND	ND/ ND	NA	C	8.4E-2	C	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 abbreviations: K = kidney; L = liver; NA = not applicable.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

7.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1989, ***Toxicological Profile for Bromodichloromethane***, U.S. Department of Health and Human Services, Atlanta, Georgia, December, on line.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

Lewis, R.J., Sr., 1992, ***Sax's Dangerous Properties of Industrial Materials***, Eighth Edition, New York: Van Nostrand Reinhold Co. p. 784.

Lewis, R.J., Sr., 1997, ***Hawley's Condensed Chemical Dictionary***, Thirteenth Edition, John Wiley and Sons, New York, p. 354.

U.S. Environmental Protection Agency (EPA), 1980, ***Health and Environmental Effects Profiles***, Office of Solid Waste, Washington, DC, NTIS No. PB81-190019.

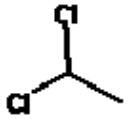
U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

1,1-DICHLOROETHANE (75-34-3)



1.0 Introduction and Physical Properties

1,1-Dichloroethane is a **VOC** that occurs in the pure state as a colorless oily liquid at ambient temperatures (EPA, 2002; HSDB, 2010). It is used primarily in the synthesis of other organic chemicals such as vinyl chloride and 1,1,1-trichloroethane (ATSDR, 1990; HSDB, 2010). It is also used as a solvent for plastics, oils and fats, as a cleaning and degreasing agent, in rubber cement, as a fumigant and insect spray, in fabric spreading, in fire extinguishers, as an extractant for heat-sensitive substances, in the manufacture of high vacuum rubber, as a coupling agent in antiknock gasoline, in paint, varnish and paint removers, and in ore flotation. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
99[a]	1.79[a]	5.62E-3[b] (25°C)	1.50[b]	7.42E-2[b] (25°C)	1.05E-5[b] (25°C)	2.99E-1[c] (25°C)	5.06E+3[b] (20-25°C)	330.55[d]	523[d]	6.90E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);)$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP July.

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

[c]Hazardous Substance Data Bank (HSDB), 2007, National Library of Medicine, on line, accessed 1 May.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

2.0 Environmental Fate and Transport

1,1-Dichloroethane may be released to the atmosphere as fugitive emissions during its production and use (ATSDR, 1990; HSDB, 2010). Its largest industrial use is in the production of 1,1,1-trichloroethane. Another source of environmental release is the reduction of 1,1,1-trichloroethane to 1,1-dichloroethane in groundwater or landfill wastes. 1,1-Dichloroethane also may be released from ethylene dichloride tars, which are by-products of vinyl chloride synthesis.

Most industrial releases occur directly to the atmosphere (ATSDR, 1990; HSDB, 2010). Also, volatilization is the most important fate mechanism for 1,1-dichloroethane released to soil or surface water; therefore, most 1,1-dichloroethane released to the environment finds its way to the atmosphere. 1,1-Dichloroethane will exist entirely in the vapor phase in the atmosphere, where it will degrade by reaction with photochemically produced hydroxyl radicals to form products including monochloroacetyl chloride, chloroacetic acid, hydrochloric acid and chlorine gas. The half-life for this reaction, approximately 44 to 62 days, results in the compound being fairly persistent in ambient air and able to travel considerable distance before removal. 1,1-Dichloroethane is moderately soluble and will be scavenged by rain, but wet deposition is not expected to be a significant removal process.

Small amounts of 1,1-dichloroethane released to the surface of the soil will be rapidly lost by evaporation (ATSDR, 1990; HSDB, 2010). The compound is not expected to sorb significantly to soil unless the organic content of the soil is high; therefore, releases of large amounts may leach into the groundwater. No abiotic degradation of 1,1-dichloroethane was observed in experiments lasting over 1 year. Both aerobic and anaerobic biodegradation are expected to be insignificant removal processes.

1,1-Dichloroethane released to surface water is expected to volatilize rapidly to the atmosphere (ATSDR, 1990; HSDB, 2010). The volatilization half-life is highly dependent on wind speed and mixing conditions. Half lives of 22 minutes to 6-9 days have been reported. 1,1-Dichloroethane is not expected to partition significantly to suspended or benthic sediment. Biodegradation has been observed in surface water, but this removal process will be far less significant than volatilization. 1,1-Dichloroethane in groundwater is part of a progressive anaerobic biodegradation chain in which 1,1,1-trichloroethane is degraded to 1,1-dichloroethane, which is further degraded to chloroethane, then to ethanol, and finally to carbon dioxide. This process, however, is expected to be slow.

1,1-Dichloroethane is not expected to participate significantly in food-chain pathways (ATSDR, 1990; HSDB, 2010). Based on data from the Great Lakes, EPA (1995) concluded that 1,1-dichloroethane does not bioconcentrate sufficiently in fish to represent a threat to human health. Rapid excretion of systemic 1,1-dichloroethane by rats and mice (ATSDR, 1990) suggest that significant participation in terrestrial food chains is unlikely. Biotransfer factors are not provided for these reasons.

3.0 Toxicokinetics

It's former use as an anesthetic agent indicates that 1,1-dichloroethane is readily absorbed by the respiratory tract (ATSDR, 1990). A study in humans reported that 59 percent of inhaled 1,1-dichloroethane was metabolized and excreted as urinary metabolites, indicating that respiratory uptake was at least 59 percent. An oral study with rats and mice reported that more than 90 percent of a single oral dose was excreted as carbon dioxide or unchanged compound by the lungs within 48 hours, indicating that GI absorption is extensive. Preliminary data indicate that 1,1-dichloroethane is absorbed through the intact skin of rabbits but the rate and extent of dermal uptake were not quantified.

Data regarding distribution of 1,1-dichloroethane after inhalation, oral or dermal exposure are limited. The former use of the compound as an inhalation anesthetic indicates that distribution to the CNS occurs. Rats and mice given single oral doses of 700 and 1800 mg/kg, respectively, retained only 1.4 and 2.4 percent, respectively, of the administered dose in the carcass after 48 hours. Studies in rats and mice following intraperitoneal injection of radiolabeled compound reported that the label was found bound to proteins, RNA and DNA of the liver, kidney, lungs and stomach (these were the only tissues analyzed in the study) (ATSDR, 1990). Tissue binding was greatest to proteins and least to DNA.

Metabolism is probably saturable at higher concentrations or doses (ATSDR, 1990). Rats and mice given single oral doses of 700 and 1800 mg/kg, respectively, excreted unchanged compound in expired air accounting for 86.1 and 70.4 percent, respectively, of the administered dose. Metabolism of 1,1-dichloroethane is mediated by inducible cytochrome P-450, which may cause hydroxylation

of either carbon atom. Hydroxylation of the # 1 carbon would yield an unstable α-haloalcohol that would spontaneously eject hydrogen chloride to form acetyl chloride. Alternatively, the α-haloalcohol may undergo a halide shift to yield chloroacetyl chloride. The acyl chlorides may bind directly to cellular macromolecules, or may undergo hydrolysis to yield hydrochloric acid and the corresponding free acid. Hydroxylation of the # 2 carbon would yield 2,2-dichloroethanol, which would likely undergo further oxidation to dichloroacetic acid. 1,1-Dichloroethane may also form free radicals (possibly through an epoxide intermediate) that directly induce toxic effects including initial stages of cancer, but this pathway is thought to be relatively insignificant for 1,1-dichloroethane compared with other chlorinated hydrocarbons.

Data regarding excretion are limited. A study in humans reported that 59 percent of the amount of 1,1-dichloroethane inhaled was excreted as metabolites in urine (ATSDR, 1990). An oral study with rats and mice reported that more than 90 percent of a single oral dose was excreted as carbon dioxide or unchanged compound by the lungs within 48 hours.

4.0 Dermal Exposure

As indicated above, excretion data suggest that GI absorption of 1,1-dichloroethane is extensive. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for 1,1-dichloroethane and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data regarding the dermal uptake of 1,1-dichloroethane from soil were not located in the available literature. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of 1,1-dichloroethane from soil is not evaluated. K_p, τ, FA and B values are provided by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
1,1-Dichloroethane	0.92	6.7E-3	0.38	1.0	0.0

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).

5.0 Noncancer Effects Evaluation

ATSDR (1990) reported oral LD_{50} values in rats of 725 mg/kg and 14.1 g/kg. Since these values were taken from a secondary source, their quality and the reasons for their marked differences cannot be determined.

An earlier provisional chronic oral RfD of 1E-1 mg/kg-day was based on a NOEL of 115 mg/kg-day in a 13-week inhalation study in rats and an uncertainty factor of 1000 (EPA, 1997). Adverse effects were not observed in the study. EPA (1997) provided a provisional subchronic oral RfD for 1,1-dichloroethane of 1E+0 mg/kg-day based on the same study and an uncertainty factor of 100. Presumably, the uncertainty factor of 100 is equivalent to the uncertainty factor used for the chronic RfD without a factor of 10 to expand from subchronic to chronic exposure.

More recently, EPA (2006) derived a provisional subchronic oral RfD of 2E+0 mg/kg-day by applying an uncertainty factor of 300 to a NOAEL of 1000 mg/kg (714.3 mg/kg-day) from a 13-week gavage study in male rats. The uncertainty factor of 300 consisted of factors of 10 each for inter- and intra-species variation, and a factor of 3 for an incomplete data base. The LOAEL in this study, 2000 mg/kg (1429 mg/kg-day), was associated with mild changes in urine chemistry suggestive of renal injury. Mild liver lesions were observed at 4000 mg/day, showing the kidney to be slightly more sensitive than the liver, at least in rats. EPA (2006) derived a provisional chronic oral RfD of 2E-1 mg/kg-day by applying an additional uncertainty factor of 10 (to expand from subchronic to chronic exposure) to the NOAEL of 714.3 mg/kg-day. Confidence in the provisional RfDs is low. The kidney is the most sensitive target organ for prolonged oral exposure to 1,1-dichloroethane.

Acute inhalation exposure to high (anesthetic) concentrations induces cardiac arrhythmias in humans (ATSDR, 1990). The only effect reported in animals from prolonged inhalation exposure is renal injury in cats (ATSDR, 1990; EPA, 1997). The key study involved small numbers of rats, guinea pigs, rabbits and cats exposed by inhalation to 0 or 500 ppm (2024 mg/m^3) 1,1-dichloroethane on 6 hours per day, 5 days per week for 13 weeks. No adverse effects were observed; therefore, the exposure concentration was then increased to 1000 ppm (4047 mg/m^3) for an additional 10 to 13 weeks. The only adverse effect noted was biochemical evidence of impaired kidney function in the cats exposed to 1000 ppm; however, histopathological evaluation of cats during the 500 ppm exposure period was not performed to identify subtle kidney effects. A provisional chronic inhalation RfC of 5E-1 mg/m^3 was derived from the NOEL of 500 ppm (138 mg/kg-day) by applying an uncertainty factor of 1000 (EPA, 1997). The provisional chronic RfC is equivalent to an inhalation RfD of 1.4E-1 mg/kg-day.

EPA (2006) re-evaluated the available information for 1,1-dichloroethane and concluded that the data were insufficient for derivation of provisional inhalation RfC values.

6.0 Carcinogenicity Evaluation

EPA (2007) classified 1,1-dichloroethane in cancer weight-of-evidence Group C (possible human carcinogen) based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice) as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice. EPA (2010) concluded that the data are insufficient for derivation of oral or inhalation potency factors.

EPA (2006) re-evaluated data regarding carcinogenicity and confirmed the foregoing. As a more descriptive weight of evidence statement, they noted that 1,1-dichloroethane shows “suggestive evidence of carcinogenic potential.” EPA (2006) also noted that, although IRIS (EPA, 2010) deemed the cancer date inadequate for quantitative evaluation, the California Environmental Protection Agency (Cal EPA) estimated a SF of 5.7E-3 per mg/kg-day and a URF of 1.6E-6 per µg/m^3 from the incidence of mammary gland tumors in female rats. EPA (2006), however, did not adopt the Cal EPA estimates for use outside of California.

7.0 Toxicity Summary

Toxicity values for 1,1-dichloroethane are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2E+0	2E-1	K	ND	ND	K	C	ND	C	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: K = kidney.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1990, ***Toxicological Profile for 1,1-Dichloroethane***, U.S. Department of Health and Human Services, Atlanta, Georgia, December, on line.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

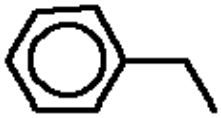
U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2006, ***Provisional Peer Reviewed Toxicity Values for 1,1-Dichloroethane (CASRN 75-34-3)***, National Center for Environmental Assessment, Cincinnati, OH, 27 September.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

ETHYLBENZENE (PHENYLETHANE) (100-41-4)



1.0 Introduction and Physical Properties

Ethylbenzene, also known as phenylethane, is a **VOC** that exists in the pure state as a colorless liquid at ambient temperatures (HSDB, 2010). It is used primarily as a precursor in the manufacture of styrene (ATSDR, 1999). It is also used as a solvent and diluent, and as an intermediate in the manufacturing of cellulose acetate, rubber and other organic compounds (ATSDR, 1999; HSDB, 2010). It is also a constituent of asphalt, naphtha, and automotive and aviation fuels. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
106.2[a]	3.15[a]	7.88E-3[b] (25°C)	2.56[b]	7.50E-2[b] (25°C)	7.80E-6[b] (25°C)	1.26E-2[c] (25°C)	1.69E+2[b] (20-25°C)	409.34[d]	617.20[d]	8.50E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

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

[c]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***User's Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, June 19.

2.0 Environmental Fate and Transport

Ethylbenzene enters the atmosphere primarily from fugitive emissions and exhaust connected with its use in gasoline (ATSDR, 1999; HSDB, 2010). More localized sources to the air and other environmental media include fugitive emissions, waste water and spills from its production and industrial use, leaking underground storage tanks, land disposal of ethylbenzene-containing waste, and municipal and hazardous waste combustion.

Ethylbenzene exists in the air in the vapor phase (ATSDR, 1999; HSDB, 2010). It is somewhat water soluble and may be removed by precipitation; however, it is likely to reenter the air by volatilization. Ethylbenzene is transformed in air by various oxidative processes to yield ethylphenols, benzaldehyde, acetophenone, and m- and p-nitroethylbenzene. Reaction with photolytically generated hydroxyl radicals is probably the most significant reaction, with a half-life estimated to be 55 hours, suggesting that ethylbenzene will not persist in the air or travel long distances.

Ethylbenzene released to soil will volatilize rapidly (ATSDR, 1999; HSDB, 2010). That which infiltrates is expected to be quite mobile in soil and may leach to groundwater because sorption to soil particles is not sufficiently strong to retard movement. Biodegradation in soil in aerobic conditions involves oxidation of the ethyl side chain, ring hydroxylation and ring cleavage, and may be a significant source of removal. Anaerobic degradation is expected to be insignificant.

Ethylbenzene released to surface water will volatilize rapidly to air (ATSDR, 1999; HSDB, 2010). Biodegradation in aerobic conditions will significantly reduce water-borne concentrations. Photolysis and hydrolysis are unlikely to be significant. Ethylbenzene partitions weakly to sediment.

Ethylbenzene is not expected to participate significantly in food-chain pathways (ATSDR, 1999; EPA, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Ethylbenzene is rapidly absorbed during inhalation exposure, with retention near or exceeding 50 percent of the inhaled concentration (ATSDR, 1999). The compound is similarly rapidly absorbed following oral administration. Excretion data in rabbits suggest that a minimum of 72 to 92 of an oral dose is absorbed. Liquid ethylbenzene applied to the skin of humans is rapidly absorbed, but ethylbenzene vapor is poorly absorbed.

Ethylbenzene appears to be efficiently and widely distributed throughout the body following inhalation exposure (ATSDR, 1999). The same is expected following oral and dermal exposure, but data are lacking.

Metabolism in humans of absorbed ethylbenzene following either inhalation or oral exposure predominantly involves successive oxidation of the ethyl side-chain through 1-phenylethanol, acetophenone, ω-hydroxyacetophenone, and 1-phenyl-1,2-ethanediol to form mandelic acid and phenylglyoxylic acid (ATSDR, 1999). Small quantities of the first 4 intermediates are conjugated and excreted in the urine. Mandelic acid and phenylglyoxylic acid are excreted without conjugation and comprise the majority of excreted metabolites. A minor metabolic pathway involves epoxide formation and hydroxylation of the phenyl ring with or without oxidation of the ethyl group to yield small quantities of metabolites that are conjugated and excreted. Experiments with laboratory animals reveal species differences in the predominance of certain metabolites, but the pathways described above appear to hold. There is no evidence that the metabolites are more toxic than the parent compound.

Excretion of metabolites, primarily through the urine, is rapid and extensive in humans and laboratory animals (ATSDR, 1999). Pulmonary excretion of carbon dioxide is a minor pathway.

Nothing in this review of toxicokinetics suggests that laboratory animals would not serve as appropriate models for the toxicity of ethylbenzene to humans.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. GI

absorption of ethylbenzene is extensive as discussed in Section 3.0. Therefore, no GAF is developed for ethylbenzene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data were not located for dermal uptake of ethylbenzene from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of ethylbenzene from soil is not evaluated. Values for t*, K_p, τ, FA and B are provided by EPA (2004) as follows:

Chemical	t*	K_p	τ	FA	B
Ethylbenzene	1.01	4.9E-2	0.42	1.0	0.2
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

An oral LD_{50} in rats of 5.46 g/kg (HSDB, 2010) suggests that the acute toxicity of ingested ethylbenzene is low. Subchronic to chronic oral exposure of laboratory animals to ethylbenzene induces mild liver and kidney lesions (EPA, 2010). EPA (2010) presents a verified chronic oral RfD for ethylbenzene of 1E-1 mg/kg-day based on a NOEL for liver and kidney lesions in female rats of 136 mg/kg-day in a six-month gavage study. The LOAEL in this study was 408 mg/kg-day. An uncertainty factor of 1000 was applied to the NOEL to derive the chronic oral RfD. The uncertainty factor consists of factors of 10 each to extrapolate from animals to humans, to provide additional protection for more sensitive humans, and to expand from subchronic to chronic exposure. Confidence in the oral RfD is low. The liver and kidney are the target organs for prolonged oral exposure to ethylbenzene.

No EPA-derived subchronic oral RfD is available for ethylbenzene. A preliminary subchronic oral RfD can be made, however, by applying an uncertainty factor of 100 to the NOEL of 136 mg/kg-day in the 6-month rat study described above. The uncertainty factor of 100 reflects the chronic uncertainty factor of 1000 without the factor of 10 for expansion from subchronic to chronic exposure. The preliminary subchronic oral RfD so derived is 1E+0 mg/kg-day.

Acute inhalation exposure induces irritation of the mucous membranes in animals and humans (ACGIH, 1991). Prolonged inhalation exposure induces liver enzymes and results in elevated liver and kidney weights; prolonged exposure to relatively high levels is associated with mild histopathological lesions of the liver, kidney and testes (ACGIH, 1991; EPA, 2010). EPA (2010) considered that the effects on the liver at the lower exposure concentrations are adaptive rather than adverse. EPA (2010) presented a verified chronic inhalation RfC of 1E+0 mg/m^3 derived from a human equivalent adjusted NOAEL of 434 mg/m^3 from studies on developmental effects in rats and rabbits. An uncertainty factor of 300 was used. The human equivalent adjusted LOAEL of 4340 mg/m^3 was associated with equivocal evidence of reduced fetal size in rabbits, an increased incidence of extra ribs in rat fetuses, and increased liver, kidney and spleen weights in the rats. The inhalation RfC is equivalent to 2.9E-1 mg/kg-day. Confidence in the RfC is low. The chronic inhalation RfC is adopted as being sufficiently conservative for subchronic as well as chronic inhalation exposure.

The key study from which the inhalation RfC is derived does not clearly identify target organ(s) for inhalation exposure to ethylbenzene. The entire inhalation exposure data base, however, suggests that the liver, kidneys and fetus may be the most significant target organs for prolonged inhalation exposure.

6.0 Carcinogenicity Evaluation

Ethylbenzene is currently verified as a cancer weight-of-evidence Group D compound (not classifiable as carcinogenicity to humans) (EPA, 2010) based on an absence of human or animal cancer studies. Cancer potency factors are not estimated for Group D compounds.

7.0 Toxicity Summary

Toxicity values for ethylbenzene are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
1E+0	1E-1	L, K	1E+0/ 2.9E-1	1E+0/ 2.9E-1	L, K, F	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: L = liver; K = kidney; F = fetus.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1999, ***Update Toxicological Profile for Ethylbenzene***, U.S. Department of Health and Human Services, Atlanta, Georgia, July, on line.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, Cincinnati, OH, p. 581-584.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*** Final, Office of Superfund Remediation and Technology Innovation, EPA/540/R/99/005, OSWER 9285.7-02EP, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

METHYLENE CHLORIDE (DICHLOROMETHANE) (75-09-2)

1.0 Introduction and Physical Properties

Methylene chloride is a **VOC** used predominantly as a paint remover and as a solvent and flammability depressant in aerosol products such as coatings, paint removers, hair sprays, room deodorants, herbicides and insecticides (ATSDR, 2000; HSDB, 2010). Methylene chloride is also used in the foam blowing of polyurethanes, in metal degreasing, stripping and degreasing of electronic components, in resin production, photographic film splicing, adhesive production, and other solvent, cleaning and thinning applications. It occurs as a colorless liquid at ambient temperatures. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
84.9[a]	1.25[a]	3.25E-3[e] (25EC)	1.07[b]	1.01E-1[b] (25EC)	1.17E-5[b] (25EC)	5.72E-1[c] (25EC)	1.30E+4[b] (20-25EC)	313.0[d]	510.0[d]	6.71E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry′s Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (EK); T_c = critical temperature (EK); $\Delta H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.[a]U.S 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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.
[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.
[c]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
[d] U.S. Environmental Protection Agency (EPA), 1997, ***User′s Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings***, prepared by Environmental Quality Management, Inc. for the Office of Emergency and Remedial Response, Washington, DC, September.
[e]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

Methylene chloride is highly volatile; therefore, most environmental releases are to the atmosphere (ATSDR, 2000; HSDB, 2010). Large amounts (greater than a hundred million pounds) are released each year during its industrial and consumer use. Methylene chloride exists solely as a vapor in air and may travel long distances before removal. It is subject to reaction with photochemically generated hydroxyl radicals with a half-life of a few months. It is also subject to direct photolysis.

Methylene chloride released to soil will be highly mobile and will infiltrate or leach to greater depths or to groundwater (ATSDR, 2000; HSDB, 2010). Some will also volatilize. Methylene chloride released to water will be rapidly lost primarily by volatilization. It is not expected to partition to sediment. It is subject to hydrolysis and biodegradation, but these processes are less significant than volatilization. Methylene chloride is not expected to participate significantly in food-chain pathways; therefore, biotransfer factors are not provided.

3.0 Data for Evaluating Dermal Exposure

Animal studies indicate that methylene chloride is readily absorbed from the GI tract (ATSDR, 2000). EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract,

and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for methylene chloride and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of methylene chloride from soil is not evaluated. K_p, τ, FA and B values are provided by EPA (2001) as follows:

Chemical	K_p	τ	FA	B
Methylene chloride	3.5E-3	0.32	1.0	0

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).

4.0 Noncancer Effects Evaluation

Oral LD_{50} values in rats and mice approximate 2 g/kg, suggesting that the acute toxicity of methylene chloride is moderate (ATSDR, 2000). Prolonged oral exposure induces liver lesions in laboratory animals. EPA (2010) verified an RfD for chronic oral exposure of 6E-2 mg/kg-day by applying an uncertainty factor of 100 to a NOAEL of about 6 mg/kg-day in a two-year drinking water study in rats. The LOAEL, about 55 mg/kg-day, was associated with histopathologic lesions in the liver. Confidence in the RfD is medium. EPA (1997) adopted the verified chronic oral RfD of 6E-2 mg/kg-day as the provisional RfD for subchronic oral exposure.

Occupational exposure to high concentrations of methylene chloride is associated with a wide variety of neurological dysfunctions and elevated blood levels of carbon monoxide (a metabolite of methylene chloride) and carboxyhemoglobin (ACGIH, 1991). Chronic inhalation exposure of laboratory animals induces liver effects. EPA (1997) derived a provisional chronic inhalation RfC of 3E+0 mg/m^3 based on a NOAEL of 695 mg/m^3 in a two-year intermittent exposure study in rats. ATSDR (2000) lists a chronic inhalation minimum risk level (MRL) of 0.3 ppm. A concentration analogous to an RfC can be calculated if this MRL is multiplied by the ratio of the molecular weight of methylene chloride (84.9) to the average molecular weight of air (~29). This result in an "RfC" of 9E-1 mg/m^3. This is equivalent to an inhalation RfC of 0.3 mg/kg-day. The values are also used for subchronic exposure.

5.0 Carcinogenicity Evaluation

Methylene chloride is classified in EPA cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient evidence of carcinogenicity in animals (EPA, 2010). Animal inhalation studies showed increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, mammary tumors in rats of either sex, salivary gland sarcomas in male rats and leukemia in female rats. Oral studies were inconclusive. An oral SF of 7.5E-3 per mg/kg-day was based on the incidence of liver tumors in two inhalation studies in mice. An inhalation URF of 4.7E-7 per µg/m^3 was based on the incidence of liver and lung tumors in one inhalation study. The inhalation URF is equivalent to 1.6E-3 per mg/kg-day.

6.0 Toxicity Summary

Toxicity values for methylene chloride 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
6E-2	6E-2	L	9E-1/ 3E-1	9E-1/ 3E-1	L	B2	7.5E-3	B2	4.7E-7	1.6E-3

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); URF = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data.
Target organ abbreviations: L = liver; K = kidney; NA = not applicable.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

7.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Methylene Chloride***, U.S. Department of Health and Human Services, Atlanta, Georgia, September, on line.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, ACGIH, Cincinnati, OH, pp. 981-987.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 7 February.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

1,1,2,2-TETRACHLOROETHANE (79-34-5)

1.0 Introduction and Physical Properties

1,1,2,2-Tetrachloroethane is a **VOC** that exists in the pure state as a colorless to pale yellow liquid at ambient temperatures (HSDB, 2010). It was once used as a non-flammable solvent in cleaning and degreasing metals, in paint removers, varnishes and lacquers, in photographic film, as an extractant for oils and fats, and as an intermediate in the synthesis of unsaturated chlorinated solvents (ATSDR, 2008; HSDB, 2010). All these uses have declined markedly. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
167.9[a]	2.39[a]	3.67E-4[e] (25 °C)	1.97[b]	7.10E-2[b] (25 °C)	7.90E-6[b] (25 °C)	8.03E-3[c] (25 °C)	297E+3[b] (20-25 °C)	419.60[d]	661.15[d]	9.00E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry s Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (°K); T_c = critical temperature (°K); $\Delta H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Review Draft, Office of Solid Waste and Emergency Response, Washington, DC, 9355.4-24, March.

[c]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[d]U.S. Environmental Protection Agency (EPA), 1997, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, September.

[e]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

Releases of 1,1,2,2-tetrachloroethane include process and fugitive emissions from its production and use as a chemical intermediate and industrial solvent, petroleum refining, incineration of chlorinated solvents, and evaporation from waste water streams (ATSDR, 2008). Most of the released 1,1,2,2-tetrachloroethane enters the atmosphere where it exists solely as a vapor and is very stable (half-life > 2 years) (HSDB, 2010). The chemical eventually diffuses to the stratosphere where it rapidly photodegrades. Wet deposition may remove some 1,1,2,2-tetrachloroethane from air, but volatilization from soil or surface water quickly returns it to the air.

1,1,2,2-Tetrachloroethane released to soil will be highly mobile and will infiltrate or leach to greater depths or to groundwater (ATSDR, 2008; HSDB, 2010). Some will also volatilize. Various hydrolytic, oxidative and biodegradation processes slowly transform the parent compound to 1,1,2-trichloroethane, trichloroethene, dichloroethene and vinyl chloride, all of which are relatively stable and each of which is a toxicant in its own right. The biotransformation products are also subject to leaching to groundwater.

1,1,2,2-Tetrachloroethane released to water will be lost primarily by volatilization within days to weeks (ATSDR, 2008; HSDB, 2010). The compound is not expected to partition to sediment. Hydrolysis slowly converts the parent compound to trichloroethene.

Theoretical and empirical considerations suggest that participation in food-chain pathways is unlikely to be significant (HSDB, 2010; EPA, 1995); therefore, biotransfer factors are not provided.

3.0 Data for Evaluating Dermal Exposure

ATSDR (2008) summarized several studies that suggest that GI absorption of 1,1,2,2-tetrachloroethane is nearly complete in rats and mice. The most useful study reported excretion in rats and mice given a 150 mg/kg oral dose. Excretion was followed for 72 hours after treatment. Rats excreted 42 percent of the dose in expired air, 23 percent in urine and 4 percent in the feces. Mice excreted 51 percent in the breath, 22 percent in the urine and 6 percent in the feces. These data suggest that a minimum of approximately 95 percent of the oral dose was absorbed in both species, assuming that material recovered from the feces represents unabsorbed compound. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for 1,1,2,2-tetrachloroethane and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

1,1,2,2-Tetrachloroethane is readily absorbed through the skin (HSDB, 2010), but data regarding the dermal uptake of 1,1,2,2-tetrachloroethane from soil were not located in the available literature. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of 1,1,2,2-tetrachloroethane from soil is not evaluated. K_p, τ, FA and B values are provided by EPA (2004) as follows:

Chemical	K_p	τ	FA	B
1,1,2,2-Tetrachloroethane	6.9E-3	0.93	1.0	0

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).

4.0 Noncancer Effects Evaluation

Single dose oral LD_{50} values in rats are grouped at 250 to 800 mg/kg (ATSDR, 2007). It appears that death is due largely to narcosis (probably accompanied by respiratory arrest and/or cardiovascular collapse). Prolonged oral exposure of laboratory animals to 1,1,2,2-tetrachloroethane is associated with liver and kidney effects (ATSDR, 2008).
Acute occupational exposure to high levels is associated with CNS effects; prolonged exposure to more moderate levels is associated with GI disturbances and liver damage (ATSDR, 2008).

The results of numerous subchronic studies were reviewed to derive a subchronic oral pRfD (EPA, 2008) for 1,1,2,2-tetrachloroethane. The lowest LOAEL identified was 40 mg/kg-day, in

which increases in liver weights were observed (NTP, 2004). The results of several endpoints from this NTP study were evaluated using benchmark dose modeling, assuming 95% lower confidence limits, to identify potential points of departure to derive a subchronic pRfD. This modeling resulted in the identification of 10.8 mg/kg-day point of departure, based on increases in lever weights of male rats. This value can be regarded as an estimated NOAEL. An oral subchronic pRfD of 1E-2 mg/kg-day was derived by dividing this value by a composite UF of 1,000. This assumes a UF of 10 for each of the following: interspecies variability, intraspecies sensitivity, and deficiencies in the database (EPA, 2008).

Because of deficiencies in chronic animal studies, the subchronic NTP (2004) study used to derive the subchronic pRfD was also selected as the most appropriate to derive a chronic RfD. The chronic oral RfD of 4E-3 mg/kg-day was derived as described for the subchronic RfD, with an additional UF of 3 (for a composite UF of 3,000) to extrapolate from subchronic to chronic duration added to extrapolate from subchronic to chronic exposure for a composite UF of 3,000 (EPA, 2008).

Adequate studies for the derivation of inhalation pRfDs could not be located (EPA 2008)

5.0 Carcinogenicity Evaluation

Oral treatment with 1,1,2,2-tetrachloroethane is associated with a highly significant dose-related increase in hepatocellular carcinomas in rats (ATSDR, 2008). Occupational data regarding carcinogenicity in humans are inadequate. EPA (2010) classified 1,1,2,2-tetrachloroethane as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on liver tumors in mice in a 78-week gavage study in which the test compound was dissolved in corn oil. EPA (2002) verified an oral SF of 2.0E-1 per mg/kg-day from the mouse study. The same data serve as the basis for an inhalation URF of 5.8E-5 per $\mu g/m^3$, which is equivalent to 2.0E-1 per mg/kg-day.

6.0 Toxicity Summary

Toxicity values for 1,1,2,2-tetrachloroethane 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
1E-2	4E-3	L	ND	ND	NA	C	2.0E-1	C	5.8E-5	2.0E-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.
Target organ abbreviations: L = liver; NA = not applicable.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

7.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2008, ***Toxicological Profile for 1,1,2,2-Tetrachloroethane***, U.S. Department of Health and Human Services, Atlanta, Georgia, September.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

National Toxicology Program (NCP), 2004, ***NTP Technical Report on the Toxicity Studies of 1,1,2,2 Tetrachloroethane Administered in Microcapsules in Feed to F344/N Rats and B6C3F1Mice***, U.S. DHHS, Public Health Service, National Institute of Health. Toxicity Report Series, Number 49.

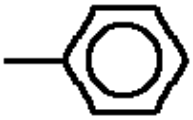
U.S. Environmental Protection Agency (EPA), 1995, ″Final Water Quality Guidance for the Great Lakes System; Final Rule,″ (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2008, ***Provisional Peer Reviewed Toxicity Values for 1,1,2,2-Tetrachloroethane (CASRN 79-34-5)***, National Center for Environmental Assessment, Cincinnati, OH, 24 August 14.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

TOLUENE (METHYLBENZENE) (108-88-3)



1.0 Introduction and Physical Properties

Toluene is a **VOC** that occurs as a colorless liquid at ambient temperatures (HSDB, 2010). It is back-blended into gasoline to improve octane rating, used to produce benzene, used as a solvent in paints, coatings, adhesives, inks and cleaning agents, and used in the synthesis of a wide range of organic chemicals, particularly benzene and its derivatives (ATSDR, 2000; HSDB, 2010). Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
92.1[a]	2.73[a]	6.64E-3[b] (25°C)	2.26[b]	8.70E-2[b] (25°C)	8.60E-6[b] (25°C)	3.74E-2[c] (25°C)	5.26E+2[b] (20-25°C)	383.78[d]	591.79[d]	7.93E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final,*** Office of Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP July.

[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.

[c]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 23 April.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

2.0 Environmental Fate and Transport

Large amounts of toluene are released into the atmosphere each year, principally from the production, transport and use of gasoline (ATSDR, 2000; HSDB, 2010). Other major sources include volatilization of toluene-based solvents and thinners, and motor vehicle exhaust. Considerable amounts are discharged into waterways or spilled on land during the storage, transport and disposal of fuels and oils. Natural sources include emissions from volcanoes, forest fires and crude oil.

Toluene in the atmosphere will exist predominantly or entirely in the vapor phase, where it is subject to reaction with photochemically produced hydroxyl radicals (ATSDR, 2000; HSDB, 2010). It is also subject to reaction with various oxidizing agents in the atmosphere, but these reactions are far less significant. Wet deposition is another important process for removal of toluene from the atmosphere.

Toluene is expected to be moderately to highly mobile in soil and may leach to groundwater (ATSDR, 2000; HSDB, 2010). Volatilization is an important removal process for toluene near the surface of the soil. Biodegradation may also be significant in soil. Toluene in water will be lost by

both volatilization to the atmosphere and biodegradation. Which process predominates will depend on water temperature, mixing conditions and the existence of acclimated microorganisms at the site. The compound is not expected to sorb significantly to sediment or suspended organic matter in water.

Toluene theoretically may be expected to bioconcentrate because of its lipophilicity, but empirical data show that it is subject to rapid biotransformation, which greatly reduces this tendency (ATSDR, 2000). Overall, biotransfer is not expected to be significant (EPA, 1995) and biotransfer factors are not provided.

3.0 Toxicokinetics

Excretion data from humans and laboratory animals indicate that toluene is rapidly and extensively absorbed during inhalation and following oral administration (ATSDR, 2000). Data from humans suggest that pulmonary retention ranges from 50 to 83 percent of the inhaled concentration. GI absorption is practically complete in humans and animals. Administration of toluene in soil slows the rate but does not reduce the extent of GI absorption. Toluene is absorbed by the skin of humans and laboratory animals when applied as a liquid or as a water solution. Dermal uptake of toluene vapor has been demonstrated in animals. Data were not located regarding dermal uptake from soil.

Absorbed toluene is rapidly and widely distributed throughout the body (ATSDR, 2000). Highest levels are generally found in highly vascular and lipid-rich tissues, including the brain, particularly following inhalation, and the liver, particularly following ingestion. Levels in maternal tissues are generally much higher than those in fetal tissues. The nervous system is an important target for exposure to toluene, particularly following inhalation exposure.

The predominant metabolic pathway for toluene involves oxidation of the methyl group to form benzyl alcohol which is further oxidized to benzoic acid, followed by conjugation with glycine to form hippuric acid, which is recovered as the major urinary metabolite (ATSDR, 2000). A minor pathway involves hydroxylation of the benzene ring to form various cresol isomers, which undergo conjugation and excretion by the kidneys. Another minor pathway involves glutathione conjugation of benzyl alcohol to yield a mercapturic acid urinary metabolite. Quantitative differences in the relevance of these pathways have been shown for different species and different human racial groups.

The liver is the primary site of toluene biotransformation. The enzyme activity involved in toluene metabolism is low in the fetal liver, but reaches adult levels soon after birth. Low levels of detoxication activity in the fetus may increase the likelihood of developmental effects.

Toluene metabolites are rapidly excreted from the body, although excretion appears to be multi-phasic, reflecting release of toluene from adipose depots (ATSDR, 2000).

Nothing in this review of toxicokinetics suggests that laboratory animals would not serve as appropriate models for the toxicity of toluene to humans.

4.0 Dermal Exposure

EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless

empirical data demonstrate that GI absorption is significantly less than complete. As noted above, GI absorption is practically complete. Therefore, no GAF is developed for toluene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Data were not located for dermal uptake of toluene from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of toluene from soil is not evaluated. EPA (2003) provides t*, K_p, τ, B and FA values as follows:

Chemical	t*	K_p	τ	FA	B
Toluene	0.84	3.1E-2	0.35	1.0	0.1
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ= lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Acute inhalation (glue sniffing, solvent abuse, industrial accidents) or ingestion exposure to high levels of toluene induces CNS dysfunction and narcosis (ATSDR, 2000). Acute oral LD_{50} values include 2.6 to 7.5 g/kg in rats (HSDB, 2010), suggesting the acute toxicity is not high. Acute ingestion is associated with lesions in the heart, liver, lungs and kidneys (ATSDR, 2000). Prolonged oral exposure to lower levels is associated with pathological changes in the liver, kidney and brain. Changes in liver and kidney weights were observed in rats treated by gavage for 13 weeks at a dose rate of 446 mg/kg-day (NTP, 1990). Overt kidney effects were observed at higher doses (EPA, 2010). A NOAEL of 223 mg/kg-day was also observed in this NTP study.

EPA (2010) derived a verified chronic RfD for toluene, using the benchmark dose approach and the LOAEL of 446 mg/kg-day. The critical effect of absolute kidney weight was used. This approach derived a benchmark dose level of 238 mg/kg-day as the point of departure. The RfD of 8E-2 mg/kg-day is derived by dividing this value by a composite UF of 3000. This assumes a UF of 10 for each of the following: interspecies variability, intraspecies sensitivity, and subchronic to chronic extrapolation. An additional UF of 3 was added to account for deficiencies in the data base (EPA, 2010). The chronic RfD is also conservatively used for subchronic exposure.

Prolonged occupational (inhalation) exposure to toluene at concentrations near 100 ppm induces upper respiratory tract irritation and CNS dysfunction (EPA, 2010). The CNS effects are selected as the critical endpoint for a verified inhalation RfC of 5 mg/m^3. EPA (2010) considered a plethora of human studies in the derivation of this RfC. It is based on an average adjusted NOAEL from several studies of 46 mg/m^3 (EPA, 2010). An RfC of 5 mg/m^3 is derived from this value by dividing by a UF of 10 to account for inter-individual variability. This value equals an inhalation RfD of 1.4 mg/kg-day.

6.0 Carcinogenicity Evaluation

EPA (2010) verified toluene as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans) based on a lack of human data and inadequate animal testing data. Potency factors are not available for Group D compounds.

7.0 Toxicity Summary

Toxicity values for toluene 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
8E-2	8E-2	K,	4E-1/ 1.1E-1	4E-1/ 1.1E-1	CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: K = kidney, CNS = central nervous system.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 2000, ***Update Toxicological Profile for Toluene***, U.S. Public Health Service, Atlanta, Georgia, September.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.
National Toxicology Program (NTP). 1990, Toxicology and Carcinogenesis Studies of Toluene (CAS No. 108-88-3) in F344/N Rats and B5C3F1 Mice (Inhalation Studies). Public Health Service, U.S. Department of Health and Human Services; NTP TR 371. Available from: National Institute of Environmental Health Sciences, Research Triangle Park, NC.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Emergency and Remedial Response, EPA/540/R/99/005, OSWER 9285.7-02EP July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

TRICHLOROETHENE (79-01-6)

1.0 Introduction and Physical Properties

Trichloroethene is a **VOC** that occurs as a clear, colorless liquid at ambient temperatures (HSDB, 2010). It is used predominantly in the automotive, metals and textile industries for vapor degreasing of metals and solvent scouring of cotton, wool and other fabrics (ATSDR, 1997). It is also used as a chain transfer agent in the production of polyvinyl chloride and in the synthesis of pharmaceuticals, polychlorinated aliphatics, flame retardants and insecticides. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$)H_{v,b}$
131.4[a]	2.42[a]	9.85E-3[e] (25EC)	2.22[b]	7.90E-2[b] (25EC)	9.10E-6[b] (25EC)	9.08E-2[c] (25EC)	1.10E+3[b] (20-25EC)	360[d]	544[d]	7.51E+3[d]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point of pure liquid compound (EK); T_c = critical temperature (EK); $)H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]U.S. Environmental Protection Agency (EPA), 2004, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

[b]U.S. Environmental Protection Agency (EPA), 2001, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Peer Review Draft, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, March.

[c]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

[d]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

[e]U.S. Environmental Protection Agency (EPA), 2010, *EPISuite electronic database*.

2.0 Environmental Fate and Transport

Most of the trichloroethene used in the U.S. is released to the atmosphere by evaporation primarily during vapor degreasing operations (ATSDR, 1997; HSDB, 2010). Additional sources include other operations involving its manufacture and use, air stripping as part of the treatment of contaminated wastewater, and volatilization from surface water and soil. A small amount is released in wastewater from metal finishing, paint and ink formulation, electrical/electronic cleaning operations, and rubber processing industries. Spent solvents containing trichloroethene have also been disposed in landfills and discharged directly to soil.

The dominant removal mechanism for trichloroethene in the atmosphere is reaction with photochemically produced hydroxyl radicals (ATSDR, 1997). The rate of this reaction is temperature-dependent and varies with the seasons. Estimates of half-life for this reaction range from 3 to 7 days, which accounts for the relatively short residence time of the chemical in the atmosphere. Products formed include phosgene, which is a toxicant in its own right, dichloroacetyl chloride and formyl chloride. Reaction with atmospheric ozone and scavenging by rainwater are relatively minor removal mechanisms. Trichloroethene removed by precipitation is likely to revolatilize to the air.

Trichloroethene released to soil is likely to rapidly volatilize or infiltrate into the ground (HSDB, 2010). Mobility in soil is expected to be high, resulting in leaching to groundwater. The

compound is also expected to rapidly volatilize from surface water. Adsorption to sediment is insignificant.

BCF values of 4 to 39 were measured in bluegill sunfish, rainbow trout and carp (HSDB, 2010). The protocol for these studies was not described, so their relevance to field conditions is unclear. Higher concentrations were measured in algae, suggesting that biomagnification is unlikely. EPA (1995) lists trichloroethene as a chemical for which bioaccumulation is unlikely to be significant. Furthermore, the relatively low K_{ow} suggests that biotransfer and the food-chain pathways are unlikely to be significant. Therefore, biotransfer factors are not provided for trichloroethene.

3.0 Data for Evaluating Dermal Exposure

A review of toxicokinetic experiments with rats suggests that GI absorption is virtually complete (ATSDR, 1997). Fasting increases the rate of absorption. Absorption occurs more rapidly from an aqueous vehicle than from an oil vehicle. EPA (2004) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Therefore, no GAF is developed for trichloroethene and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Neat trichloroethene is readily absorbed by the skin (HSDB, 2010), but data were not located for dermal uptake of trichloroethene from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of trichloroethene from soil is not quantified. K_p, τ, FA and B values are provided by EPA (2004) for trichloroethene as follows:

Chemical	K_p	τ	FA	B
Trichloroethene	1.2E-2	0.58	1.0	0.1
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).				

4.0 Noncancer Effects Evaluation

The acute oral toxicity of trichloroethene appears to be low as indicated by LD_{50} values in rats (4.9 g/kg) and mice (2.4 g/kg) (ATSDR, 1997). Death in acutely exposed animals is accompanied by profound CNS depression. Neither the liver nor the kidney appear to be targets for acute oral exposure to trichloroethene. No RfD has been verified for trichloroethene, and no current provisional value exists. No RfC has been verified, and no current provisional RfC exists.

5.0 Carcinogenicity Evaluation

EPA (2001) formerly characterized trichloroethylene as *highly likely to produce cancer in humans* on the basis of limited evidence for increased incidences of several tumor types in exposed humans and laboratory animals, and data that suggest humans and laboratory animals share probable mechanisms of carcinogenicity. Separate SFs were derived for several individual tumor types in four human studies and for several individual tumor types in laboratory animals. Most SFs fell between 2E-2 and 4E-1 per mg/kg-day, and this range rather than a single value was formerly recommended in IRIS for both oral and inhalation exposure before

trichloroethylene was withdrawn (EPA, 2010a). An oral SF values of 5.9E-3 $(mg/kg\text{-}day)^{-1}$ and an inhalation UR of 6E-6 $(\mu g/m^3)^{-1}$ (equivalent to an inhalation SF of 2.1E-2 $[mg/kg\text{-}day]^{-1}$) are selected in this toxicological profile. Both of these values are from the California EPA Toxicity Criteria Database (CalEPA, 2010). These CalEPA values are used by EPA (2010b) for the derivation of regional screening levels.

6.0 Toxicity Summary

Toxicity values for trichloroethene 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
ND	ND	NA		ND		formerly B1	5.9E-3	formerly B1	6E-6	2.1E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per milligram per cubic meter); SFi = inhalation cancer slope factor (risk per microgram per kilogram-day); ND = not determined; NA = not applicable.
Target organ abbreviations: L = liver; K = kidney; F = unborn child; CNS = central nervous system; ES = endocrine system.
[a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.

7.0 References

Agency for Toxic Substances and Disease Registry, (ATSDR), 1997, ***Update Toxicological Profile for Trichloroethylene***, U.S. Public Health Service, Atlanta, Georgia, September, on line.

California Environmental Protection Agency (CalEPA), 2010, ***Toxicity Criteria Database***, Office of Environmental Health Hazard Assessment (OEHHA), http://www.oehha.ca.gov/risk/ChemicalDB/start.asp.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1995, "40 CFR 9, 122, 123, 131, and 132 Final Water Quality Guidance for the Great Lakes System; Final Rule," ***Federal Register***, 60(56); 15366-15425.

U.S. Environmental Protection Agency (EPA), 2001, ***Trichoroethylene Health Risk Assessment: Synthesis and Characterization***, Office of Research and Development, Washington, DC, EPA/600/P-01/002A, August.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2010a, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

U.S. Environmental Protection Agency (EPA), 2010b, ***Regional Screening Level (RSL) Summary Table***, May.

DICHLOROPROPENES

1,3-Dichloropropene

1.0 Introduction and Physical Properties

This profile addresses 1,1-dichloropropene, 1,2-dichloropropene, 1,3-dichloropropene, and 2,3-dichloropropene. All are **VOCs** and all are liquid at ambient temperatures (ATSDR, 2006; EPA, 2002; HSDB, 2010)

Various mixtures and formulations of dichloropropenes and 1,2-dichloropropane (78-87-5) are used as soil fumigants and nematocides (HSDB, 2010; Meister, 1999). It is generally applied pre-planting by injection equipment at depths of 12 to 18 inches. Compacting the treated soil, covering the treated soil with tarps, or irrigating with water are often done immediately after treatment to retard volatilization loss.

Most soil fumigant mixtures contain predominantly 1,3-dichloropropene (542-75-6), with lesser amounts of 1,2-dichloropropene (563-54-2) and traces of 1,1-dichloropropene (563-58-6) and 2,3-dichloropropene (78-88-6) as well (ATSDR, 2006; HSDB, 2010). The use of 1,3-dichloropropene as a pesticide increased after the EPA phased out the use of ethylene dibromide, and its use is likely to increase further with the planned EPA phase-out of methyl bromide. 1,3-Dichloropropene is also used in much smaller quantities in organic synthesis and as a solvent; small quantities of 2,3-dichloropropene are used in organic synthesis.

Little is known about the physical properties of 1,1-dichloropropene or 2,3-dichloropropene. 1,2-Dichloropropene occurs as a minor constituent in Telone II, one of the more common dichloropropene formulations used as a soil fumigant. There are 2 isomers of 1,3-dichloropropene (542-75-6): cis- (or Z-) 1,3-dichloropropene (10061-01-5) and trans- (or E-) 1,3-dichloropropene (10061-02-6). Many preparations and formulations of 1,3-dichloropropene contain approximately equivalent proportions of the two isomers (HSDB, 2010). Relevant physical properties of the dichloropropenes are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
1,1-Dichloropropene (563-58-6)										
110.97[a]	2.53[a,b]	2.16E-2[a,b] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	1.19E-1[a,e] (20°C)	1.18E+3[a,b] (25°C)[d]	349.65[f]	551.66[g]	7.15E+3[h]
1,2-Dichloropropene (563-54-2)										
110.97[i]	2.53[a,b]	4.91E-3[a,e] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	3.16E-2[a,e] (25°C)	2.7E+3[i,e] (25°C)	348[i]	549[g]	7.11E+3[h]
1,3-Dichloropropene (both isomers: 542-75-6)										
111[j]	2.00[i]	3.55E-3[a,b] (25°C)	1.91[a,b]	6.26E-2[k] (25°C)	1.00E-5[k] (25°C)	4.47E-2[a,e] (25°C)	2.80E+3[k] (20-25°C)	381.15[l]	587.38[l]	7.90E+3[l]

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
2,3-Dichloropropene (78-88-6)										
110.97[a]	2.42[a,b]	4.16E-3[a,e] (25°C)	1.83[a,b]	8.23E-2[c] (25°C)[d]	9.53E-6[c] (25°C)[d]	8.05E-2[i] (25°C)	2.15E+3[a,e] (25°C)	367.2[i]	551.6[i]	8.44E+3[i]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);. $H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole); ND = no data.

[a]U.S. Environmental Protection Agency (EPA), 2004a, ***EPI Suite Version 3.12*** Software for estimating physical properties, online, 30 November.
[b]Estimated.
[c]Calculated as described in Introduction to Toxicological profiles.
[d]Assumed.
[e]Measured.
[f]Agency for Toxic Substances and Disease Registry (ATSDR), 2006, ***Toxicological Profile for Dichloropropenes***, Draft for Public Comment, U.S. Department of Health and Human Services, Atlanta, GA, September, on line.
[g]Estimated by Equation 12-4 of Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[h]Estimated by Equation 13-5 of Lyman, W.J., W.F. Reehl and D.H. Rosenblatt, 1990, ***Handbook of Chemical Property Estimation Methods***, American Chemical Society, Washington, DC.
[i]Hazardous Substances Data Bank (HSDB), 2010, National Library of Medicine, on line.
[j]U.S. Environmental U.S. Environmental Protection Agency (EPA), 2004b, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.
[k]U.S. Environmental Protection Agency (EPA), 2002, ***Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites***, Office of Solid Waste and Emergency Response, OSWER 9355.4-24, December.
[l]U.S. Environmental Protection Agency (EPA), 2003, ***Users Guide for Evaluating Subsurface Vapor Intrusion into Buildings***, Office of Emergency and Remedial Response, Washington, DC, 19 June.

Considerable discrepancy was noted in the VP values for 1,2-dichloropropene. HSDB (2010) offered a value of 90.8 mm Hg (1.19E-1 atm) at 20 °C from a 1981 report, and EPA (2004a) offered a value of 24 mm Hg (3.16E-2 atm) at 25 °C from an experimental data base (not specified). The reason for the discrepancy is not apparent. The value of 24 mm Hg (3.16E-2 atm) at 25 °C from EPA (2004a) was selected as the more rigorously reviewed source.

There was also discrepancy between the log K_{ow} values for 1,3-dichloropropene. EPA (2004a) offered a value of 1.60, but the source is unclear. HSDB (2010) offered a value of 2.00, and EPA (2004a) cited experimental values of 2.03 and 2.06. The value of 2.00 from HSDB (2010) was selected as better documented and more consistent with the experimental values compiled by EPA (2004a).

Considerable discrepancy was noted in values for H for 1,3-dichloropropene. EPA (2002) offered a value for H' (unitless) of 7.26E-1, equivalent to 1.77E-2 atm-m^3/mole, from an unclear source. HSDB (2010) offered 9.76E-4 atm-m^3/mole, but the source is unclear. EPA (2004a) offered 3.55E-3 atm-m^3/mole from an experimental database. The latter value is chosen because it is surrounded with less uncertainty than the other values. Similar discrepancy was noted in values for K_{oc}. EPA (2002) offered a value for K_{oc} of 45.7 L/kg from an unclear source. HSDB

(2010) offered 290 L/kg estimated by a regression equation from the K_{ow}. EPA (2004a) offered 80.77 L/kg from an updated regression equation. The value of 80.77 L/kg (log K_{oc} = 1.91) from EPA (2002a) is selected as fraught with less uncertainty than the EPA (2002) value and estimated by a more current model than the HSDB (2010) value.

2.0 Environmental Fate and Transport

Dichloropropenes may be released in wastewater or as fugitive emissions during their production and use as chemical intermediates or soil fumigants; however releases of 1,1-, 1,2-, and 3,3-dichloropropene are probably low because no uses were located for these isomers, although they may appear at low levels in other dichloropropene mixtures and formulations. The dichloropropenes may be inadvertently formed by the chlorination of water supplies carrying organic matter (ATSDR, 2006).

Dichloropropenes released to soil are likely to be highly mobile because of their low K_{oc} values (HSDB, 2010). Although the dichloropropenes may leach to groundwater, hydrolysis and biodegradation may attenuate the leaching process (ATSDR, 2006). Also, dichloropropenes will adsorb much more readily from the vapor phase than from the dissolved phase. Volatilization from moist soil surfaces is expected to be an important removal process for the dichloropropenes. Volatilization from dry soil surfaces may also be important.

Dichloropropenes released to water are not expected to adsorb to suspended solids and sediment (HSDB, 2010). Volatilization from water surfaces is likely to be the major removal mechanism. Volatilization half-lives for a model river and model lake have been estimated at 1 and 101 hours (specific isomer[s] not specified), respectively. Hydrolysis may also be significant; a hydrolysis half-life of 11 days was estimated for the dichloropropenes at a temperature range of 15 to 25 °C. Biodegradation is also expected to be significant (ATSDR, 2006).

The dichloropropenes are expected to exist entirely in the vapor phase in the ambient atmosphere (HSDB, 2010). Vapor-phase dichloropropene is subject to reaction with photochemically-produced hydroxyl radicals; a half-life of 3 days (specific isomer[s] not specified) was estimated for this reaction. The dichloropropenes are not expected to directly photolyze in the presence of sunlight. Wet deposition may also be a significant removal mechanism (ATSDR, 2006) (1,3-dichloropropene has been detected in rainwater), but volatilization is likely to rapidly return the dichloropropenes to the atmosphere.

Their relatively low log K_{ow}, high water solubility and their potential for metabolism suggests that the dichloropropenes are unlikely to participate significantly in food-chain pathways (HSDB, 2010). 1,3-Dichloropropene was identified by the EPA (1995) as not being of concern for human health through the aquatic food chain. Therefore, biotransfer factors are not compiled or estimated.

3.0 Toxicokinetics

Data for 1,3- and 2,3-dichloropropene indicate that the dichloropropenes are readily absorbed following inhalation or oral exposure (ATSDR, 2006). Excretion data from rats indicate that 62 to 82 percent of inhaled 1,3-dichloropropene and 35 to 40 percent of inhaled 2,3-dichloropropene are absorbed. The higher absorption percentages were estimated at lower concentrations. Higher

concentrations were shown to decrease respiratory rate and saturate elimination mechanisms, which effectively reduced respiratory absorption.

Excretion data for 1,3- and 2,3-dichloropropene in rats indicate that well upwards of 90 percent of an ingested dose is absorbed from the GI tract. Excretion data from human volunteers indicate that dermal uptake occurs, but reliable quantitative data were not located.

The dichloropropenes appear to be widely distributed following absorption. After 6-hour inhalation exposure to radiolabeled 2,3-dichloropropene, tissue levels of radioactivity from highest to lowest were found in the urinary bladder, nasal turbinates, kidneys, small intestine, liver, trachea and larynx (ATSDR, 2006). Approximately 9 percent of the absorbed radioactivity was accounted for in the body tissues. Following gavage treatment with radiolabeled 1,3-dichloropropene, highest levels of radioactivity were found in the forestomach and urinary bladder; lower levels were located in a wide variety of tissues. Tissue levels fall rapidly after exposure is terminated; there appears to be no tendency for the body to accumulate the parent compound or its metabolites. It is noteworthy that the forestomach is the target organ of prolonged oral exposure, the nasal turbinates are the target of prolonged inhalation exposure, and the urinary bladder is a target organ for the carcinogenicity of 1,3-dichloropropene.

Data from rats indicate that 1,3-dichloropropene is metabolized by three primary pathways. Most important is dechlorination and conjugation with glutathione at carbon-3, followed by conversion to a mecapturic acid and acetylation to form the N-acetyl-cysteine metabolite, which is excreted (ATSDR, 2006). Excretion data from occupationally exposed humans indicate that this pathway predominates in humans as well. A minor pathway involves hydrolysis and dechlorination at carbon-3 to form 1-chloroallyl alcohol, which undergoes further oxidation and oxidative dechlorination at carbon-1 and breakage of the chain to form carbon dioxide and acetyl-CoA, which may be incorporated into body tissues. Another minor pathway involves epoxide formation followed by dechlorination and additional oxidation to form glycidaldehyde and methylglyoxal.

Data from rats indicate that 2,3-dichloropropene is subject to the same three metabolic pathways as observed for the 1,3-isomer (ATSDR, 2006). Conjugation with glutathione predominantes. The other two minor pathways (epoxide formation and hydrolysis with dechlorination) yield metabolites that have been shown to be mutagenic, which apparently accounts for the carcinogenicity of the 1,3-isomer.

Excretion of dichloropropene metabolites is primarily in the urine following inhalation exposure of rats (ATSDR, 2006). Fecal excretion is also significant, more so for 2,3-dichloropropene than for 1,3-dichloropropene. Approximately 3 percent of the absorbed amount of 2,3-dichloropropene was excreted as carbon dioxide. Similar patterns are observed after oral exposure of rats; urinary excretion predominates. cis-1,3-Dichloropropene is metabolized and excreted in the urine as mercapturic acid metabolites more efficiently than the trans-isomer. trans-1,3-Dichloropropene is metabolized to carbon dioxide more efficiently than the cis-isomer.

The limited toxicokinetic data suggest that there is more variation between the dichloropropene isomers than between species. The differences appear to be quantitative rather than qualitative, supporting the assumption that the toxicity of the isomers may be similar.

4.0 Dermal Exposure

EPA (2004b) notes that organic chemicals are generally well absorbed from the GI tract, and recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. As noted above, the dichloropropenes are extensively absorbed from the GI tract. Therefore, no GAF is developed for the dichloropropenes and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

It appears from the previous section that the dichloropropenes are absorbed through the skin, but data are not available regarding the extent of dermal uptake from soil. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004b). Therefore, dermal uptake of the dichloropropenes from soil is not quantified.

As noted above, the value selected for log K_{ow} for 1,3-dichloropropene differs slightly from that provided by EPA (2004b). Therefore, values for t*, K_p, τ, FA and B tabulated below were not taken directly from EPA (2004b). Instead, they were recalculated from MW and the accepted log K_{ow} given above using the EPA (2004b) method. Values for t*, K_p, τ, FA and B for the other isomers were also estimated from the MW and log K_{ow} values given above and the EPA (2004b) method, as follows:

Chemical	t*	K_p	τ	FA	B
1,1-Dichloropropene	1.05	1.77E-2	0.44	1	0[a]
1,2-Dichloropropene	1.05	1.77E-2	0.44	1	0[a]
1,3-Dichloropropene	1.05	7.92E-3	0.44	1	0[a]
2,3-Dichloropropene	1.05	1.50E-2	0.44	1	0[a]

t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).
[a]Sufficiently small to be rounded to zero.

5.0 Noncancer Effects Evaluation

Toxicity data were not located for 1,1-dichloropropene. Acute toxicity data for 1,2-dichloropropene include an oral LD_{50} value of 2 g/kg in rats and a dermal LD_{50} of 8750 mg/kg in rabbits (HSDB, 2010), indicating that the acute toxicity of the 1,2-isomer is relatively low. Descriptions of agonal signs or necropsy findings were not located. Data regarding prolonged exposure to 1,2-dichloropropene were not located.

Oral LD_{50} values for 1,3-dichloropropene include 94 to 713 mg/kg in rats and 215 to 640 mg/kg in mice (ATSDR, 2006; HSDB, 2010). The wide range of values within species probably reflects differences in the preparations and formulations tested. Necropsy of the decedents from one study revealed liver and kidney damage. EPA (2010) verified a chronic oral RfD for 1,3-dichloropropene of 3E-2 mg/kg-day based on chronic stomach irritation in rats treated with a microencapsulated formulation of Telone II (96 percent 1,3-dichloropropene) in the diet. In the key study, male and female rats were fed diets that provided dose rates of 0 (control), 2.5, 12.5 or 25 mg/kg-day for 24 months. Decreased body weight gain was observed in a dose-related manner that reached statistical significance in rats of both sexes in the high dose group. The incidence of hyperplasia of the epithelium of the forestomach showed a statistically significant

increase in rats in the 12.5 and 25 mg/kg-day groups. Hyperplasia of the forestomach was interpreted as a response to chronic stomach irritation, which was identified as the critical effect of prolonged ingestion exposure. The 95 percent lower confidence limit on the maximum likelihood estimate of the dose corresponding to a 10 percent response rate ($BMDL_{10}$) was estimated as 3.4 mg/kg-day. Application of an uncertainty factor of 100 (factors of 10 each for intra- and inter-species variation) to the $BMDL_{10}$ yields the verified chronic oral RfD of 3E-2 mg/kg-day. Confidence in the verified chronic oral RfD is high. Stomach irritation is considered the critical effect of prolonged oral exposure.

EPA (1997) provides a provisional subchronic oral RfD for 1,3-dichloropropene of 3E-3 mg/kg-day based on a NOEL for increased organ weights of 3 mg/kg-day in rats in a 90-day dietary study. A LOAEL was not identified. An uncertainty factor of 1000 was used. It is likely that the increase in organ weights reported by EPA (1997) may reflect increased organ-to-body weight ratios, which would be expected and consistent with the observation of decreased overall body weight gain in the chronic study described in the previous paragraph. The NOEL of 3 mg/kg-day in the 90-day study also is consistent with the effect levels observed in the chronic study described in the previous paragraph. It is not reasonable, however, that a subchronic oral RfD should be lower than a chronic oral RfD. Therefore, the verified chronic oral RfD of 3E-2 mg/kg-day is adopted as sufficiently protective for subchronic as well as chronic exposure.

Occupational exposure to high levels of 1,3-dichloropropene in air is associated with skin, eye and respiratory tract irritation (ACGIH, 1991). EPA (2010) presents a verified inhalation RfC for 1,3-dichloropropene of 2E-2 mg/m^3 based on a human equivalent adjusted 95 percent lower confidence limit on the maximum likelihood estimate of the concentration corresponding to a 10 percent response rate ($BMCL_{10}$) of 0.72 mg/m^3 in an intermittent exposure inhalation study in mice. In the key study rats and mice were exposed to technical grade 1,3-dichloropropene at concentrations of 0 (control), 5 ppm (22.7 mg/m^3), 20 ppm (90.8 mg/m^3) or 60 ppm (272 mg/m^3) 6 hours/day, 5 days/week for two years. The only treatment-related effects in rats were histopathological changes in the nasal epithelium in the high-concentration group after 24 months, but not after 6 or 12 months of exposure. Effects in mice included masses in the lungs in males in the high-dose group, and slight hypertrophy and hyperplasia of the nasal epithelium in the two lower concentration groups. Hyperplasia of the urinary bladder epithelium was also observed in female mice in the high-concentration group. Hypertrophy and hyperplasia in the nasal epithelium of mice was identified as the critical effect of inhalation exposure. An uncertainty factor of 30 was applied to the human equivalent adjusted $BMCL_{10}$ of 0.72 mg/m^3 to derive the verified chronic inhalation RfC of 2E-2 mg/m^3. The chronic inhalation RfC is equivalent to an inhalation RfD of 5.7E-3 mg/kg-day.

EPA (1997) adopted the verified chronic inhalation RfC of 2E-2 mg/m^3 (5.7E-3 mg/kg-day) as the provisional subchronic inhalation RfC. Hypertrophy and hyperplasia of the nasal epithelium is the critical effect of prolonged inhalation exposure to 1,3-dichloropropene.

Toxicity date for 2,3-dichloropropene are largely limited to an oral LD_{50} values in rats of 285 to 320 mg/kg and a dermal LD_{50} in rabbits of 1580 mg/kg (ATSDR, 2006; HSDB, 2010).

Toxicity information sufficient for RfD or RfC estimation is not available for 1,1-, 1,2- or 2,3-dichloropropene. 1,3-Dichloropropene is selected as a surrogate for 1,1-, 1,2- and 2,3-

dichloropropene because of the similarity in chemical structure. Therefore, the noncancer oral and inhalation toxicity values described for 1,3-dichloropropene are applied also to 1,1-, 1,2- and 2,3-dichloropropene.

6.0 Carcinogenicity Evaluation

In an earlier assessment, EPA (2010) verified 1,3-dichloropropene as a cancer weight-of-evidence B2 compound – probable human carcinogen – on the basis of increased incidences of various tumor types in rats and mice in three oral studies and an inhalation study. Also, 1,3-dichloropropene yields positive results in tests for mutagenic activity and is structurally similar to chemicals known to produce similar types of tumors in rodents. Consistent with more recent EPA (2005) cancer assessment guidelines, EPA (2010) characterized 1,3-dichloropropene as likely to be a human carcinogen.

EPA (2010) developed and verified oral cancer SFs of 1E-1 per mg/kg-day from the incidence of urinary bladder tumors in female mice in a two-year gavage study, 5E-2 per mg/kg-day for liver tumors in male rats in the same study, and 5E-2 per mg/kg-day for liver tumors in male rats in a two-year dietary study. EPA (2010) recommended the SF of 1E-1 per mg/kg-day because it is the most conservative estimate and there was less uncertainty regarding the actual dose delivered.

EPA (2010) developed and verified an inhalation URF of 4E-6 per $\mu g/m^3$ based on the incidence of lung tumors in male mice in a two-year inhalation study. The URF is equivalent to an inhalation SF of 1.4E-2 per mg/kg-day.

The toxicokinetic data summarized above and the EPA (2010) cancer evaluation strongly indicate that the mode of action for carcinogenicity arises from genotoxicity. EPA (2005) proposed that the oral and inhalation cancer potency factors developed for genotoxic chemicals should be modified with the age dependent adjustment factors (ADAF) of 10 for children under 2 years of age, 3 for children 2 to 16 years of age, and 1 for adults and children over 16 years of age.

7.0 Toxicity Summary

Toxicity values for 1,3-dichloropropene are summarized as follows:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure[a]		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
3E-2	3E-2	St	2E-2/ 5.7E-3	2E-2/ 5.7E-3	NE	See above	1E-1	See above	4E-6	1.4E-2

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); ND = no data; NA = not applicable.

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: St = stomach; NE = nasal epithelium. [a]sRfDo, cRfDo and SFo should be used for dermal exposure without adjustment for GI absorption.										

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2006, ***Toxicological Profile for Dichloropropenes***, Draft for Public Comment, U.S. Department of Health and Human Services, Atlanta, GA, date, on line, September.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, Cincinnati, OH, pp. 438-440.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line, accessed 21 February.

Meister, R.T., 1999, ***Farm Chemicals Handbook '99***, Volume 85, Meister Publishing Company, Willoughby, OH, p. C 134.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, Washington, DC, 9200.6-303(97-1), EPA 540/R-97-036, PB97-921199.

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.

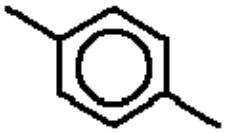
U.S. Environmental Protection Agency (EPA), 2004a, ***EPI Suite Version 3.12*** Software for estimating physical properties, online, 30 November.

U.S. Environmental Protection Agency (EPA), 2004b, ***Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final***, Office of Superfund Remediation and Technology Innovation, Washington, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2005, ***Guidelines for Carcinogen Risk Assessment***, Final, National Center for Environmental Assessment, EPA/630/P-03/001F, March.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

XYLENES (DIMETHYLBENZENE), TOTAL (1330-20-7)



p-Xylene

1.0 Introduction and Physical Properties

The xylenes, also known as dimethylbenzenes, exist as three isomers: 2-xylene (1,2-dimethylbenzene, ortho- or o-xylene) (95-47-6), 3-xylene (1,3-dimethylbenzene, meta- or m-xylene) (108-38-3) and 4-xylene (1,4-dimethylbenzene, para- or p-xylene) (106-42-3) (HSDB, 2010). The o-, m-, and p-xylene nomenclature will be used herein to be consistent with the manner in which xylene concentrations in environmental media are usually reported by the analytical laboratories.

The xylenes are **VOCs** derived from coal tar, coal gas, and petroleum distillation (ATSDR, 1995; HSDB, 2010). They occur as clear liquids in the pure state at most ambient temperatures, although 4-xylene occurs as colorless plates or prisms at temperatures below 56EF (13.33EC). Natural sources include petroleum, forest fires and volatiles from plants.

Xylenes are used in the manufacture of other organic compounds, dyes, insecticides and pharmaceuticals, and as solvents (ATSDR, 1995; HSDB, 2010). They are components of aviation gasoline, asphalt and naphtha, and automobile exhaust. Technical xylene consists of approximately 44 percent m-xylene, 20 percent o-xylene, 20 percent p-xylene, and 15 percent ethylbenzene. Relevant physical properties are compiled below:

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
o-Xylene (95-47-6)										
106.16[a]	3.12[a]	5.20E-3[b] (25°C)	2.56[b]	8.70E-2[b] (25°C)	1.00E-5[b] (25°C)	8.70E-3[a] (25°C)	1.78E+2[b] (20-25°C)	417.60[c]	630.30[c]	8.66E+3[c]
m-Xylene (108-38-3)										
106.2[d]	3.20[d]	7.34E-3[b] (25°C)	2.61[b]	7.00E-2[b] (25°C)	7.80E-6[b] (25°C)	1.09E-2[a] (25°C)	1.16E+2[b] (20-25°C)	412.27[c]	617.05[c]	8.52E+3[c]
p-Xylene (106-42-3)										
106.16[a]	3.15[a]	7.66E-3[b] (25°C)	2.59[b]	7.69E-2[b] (25°C)	8.44E-6[b] (25°C)	1.16E-2[a] (25°C)	1.85E+2[b] (20-25°C)	411.52[c]	616.20[c]	8.53E+3[c]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).

[a]Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

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

[c]U.S. Environmental Protection Agency (EPA), 2000, ***User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings, Revised***, Office of Emergency and Remedial Response, Washington, DC, December.

MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
[d]U.S. Environmental Protection Agency (EPA), 2004a, ***Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part E - Supplemental Guidance for Dermal Risk Assessment)***, Final, Office of Superfund Remediation and Technology Innovation, Washington, D.C., EPA/540/R-99/005, July.										

It is recommended that the physical properties for m-xylene should be applied to mixed xylene isomers or data where the specific isomer is not identified, because m-xylene is the major component of the technical mixture and is likely to predominate following xylene releases.

2.0 Environmental Fate and Transport

Naturally occurring sources of xylenes are petroleum, forest fires and volatiles from plants (ATSDR, 1995; HSDB, 2010). The production and use of xylenes in petroleum products and as chemical solvents and intermediates may result in their release to the environment as fugitives and through various waste streams. Off-gassing from landfills and automobile exhaust are other significant sources. Spills, leaking underground storage tanks and associated plumbing, and leaching from landfills may release xylenes to soil and groundwater.

Xylenes will exist solely in the vapor phase in the ambient atmosphere, where they are subject to degradation by reaction with photochemically-produced hydroxyl radicals as the only significant removal process (ATSDR, 1995; HSDB, 2010). The atmospheric lifetime of xylenes is about 1 to 2 days. Products of photooxidation include a wide variety of ring hydroxylation and ring cleavage products.

Volatilization is expected to be the major removal mechanism for xylenes released to surface soil, but, depending on the size and rate of release, the majority of the release may infiltrate to subsurface soil (ATSDR, 1995). Photooxidation may degrade a significant part of the small amount that remains at the surface. Biodegradation is probably the only significant subsurface removal mechanism, but it is expected to be slow. A wide range of experimental K_{oc} values have been reported in soil samples with differing pH and organic carbon content (HSDB, 2010), suggesting a wide range in the extent to which the xylenes adsorb to soil particles. Generally, however, xylene is fairly mobile and may readily leach to groundwater.

Volatilization is probably the most significant removal mechanism for xylenes in surface water (ATSDR, 1995). Biodegradation may occur in surface water, groundwater and landfill leachate, although quite slowly. Oxidative reactions are expected to be insignificant. Xylenes are expected to adsorb somewhat to suspended solids and sediment in water (HSDB, 2010).

Xylenes are not expected to participate significantly in food-chain pathways (ATSDR, 1995; HSDB, 2010); therefore, biotransfer factors are not provided.

3.0 Toxicokinetics

Xylenes are rapidly and extensively absorbed during inhalation and following oral exposure (ATSDR, 1995). In humans, approximately 50 to 73 percent of the inhaled concentration is retained. In laboratory animals, approximately 90 percent of an ingested dose is absorbed. Liquid xylene is absorbed through the skin of humans, but dermal uptake of xylene vapors approximates only 1 percent of inhalation uptake. A study in rats showed that dermal uptake of xylene adsorbed to soil is much slower than dermal uptake of the neat liquid.

Absorbed xylene is largely bound to serum protein in the blood and is distributed rapidly and widely throughout the body, but particularly to highly vascular tissues and those high in lipid content (e.g., blood, brain, liver) (ATSDR, 1995). Levels in fetal tissues approximate 2 percent of those measured in maternal brain.

The predominant metabolic pathway in humans and animals for all xylene isomers is oxidation of one of the methyl groups to the corresponding alcohol, with further oxidation to the corresponding methylbenzoic acid (ATSDR, 1995). The methylbenzoic acids conjugate with glycine to form the corresponding methylhippuric acids, which are the major urinary metabolites. Much lesser quantities of the alcohols and o-methylbenzoic acid are also excreted in the urine. Methylbenzaldehyde, proposed as the ultimate toxicant because it inactivates enzymes involved in electron transport, has been identified in laboratory animals but not in humans. Metabolism in laboratory animals differs from that in humans largely in the nature of the conjugates that are formed. Ring hydroxylation to form phenolic compounds represents a minor metabolic pathway is both humans and animals.

The xylenes are rapidly excreted from the body, largely in the form of urinary metabolites (ATSDR, 1955) as described above. Approximately 5 percent of an oral dose is eliminated in the breath as unchanged compound. Residues are slightly more persistent in adiposa and muscle tissue than other tissues of the body, but no organ or tissue accumulates xylene residues.

4.0 Dermal Exposure

EPA (2003) recommends that oral toxicity values should not be adjusted when used for dermal exposure unless empirical data demonstrate that GI absorption is significantly less than complete. Xylenes are nearly completely absorbed from the GI tract; therefore, no GAF is developed for the xylenes and the oral toxicity values described below should be used for dermal exposure without adjustment for GI absorption.

Xylene in soil is absorbed by the skin, albeit very slowly, but quantitative data were not located. VOCs tend to volatilize from soil when applied to the skin, reducing dermal uptake to toxicologically insignificant levels (EPA, 2004). Therefore, dermal uptake of xylenes from soil is not evaluated. Values for t*, K_p, τ, FA and B are provided by EPA (2003) for m-xylene as follows:

Chemical	t*	K_p	τ	FA	B
m-Xylene	1.01	5.3E-2	0.42	1.0	0.2
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); τ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

The dermal uptake parameters presented by EPA (2004) for m-xylene are applied to all the xylene isomers as well as to total xylenes.

5.0 Noncancer Effects Evaluation

Oral LD_{50} values for xylenes include 3.5 to 8.6 g/kg in rats and 1.6 to 5.6 g/kg in mice (HSDB, 2010), suggesting that the acute toxicity is low, but that mice may be slightly more sensitive than rats to the acute effects of ingested xylenes. Prolonged oral exposure of animals to xylenes is

associated with reduced body weight and survival without histopathological alterations in the internal organs (EPA, 2010). EPA (2010) confirmed a previously verified chronic oral RfD for total xylenes of 2E-1 mg/kg-day based on a NOAEL of 250 mg/kg, equivalent to a continuous dose rate of 179 mg/kg-day, for decreased body weight and survival in male rats in chronic gavage studies with mixed xylenes. The LOAEL was 500 mg/kg, equivalent to 357 mg/kg-day. Mice were also included in the study. The LOAEL in mice was 1000 mg/kg, equivalent to 714 mg/kg-day, associated with hyperactivity, which was consistently observed at 5 to 30 minutes after treatment. The mice did not exhibit the effects on body weight or survival that was observed with the rats, establishing the rat as the more sensitive species to prolonged oral exposure. An uncertainty factor of 1000 was applied to the rat NOAEL to derive the verified chronic oral RfD. Confidence in the chronic oral RfD is medium.

EPA (1997) presented provisional chronic oral RfDs for o-xylene and m-xylene of 2E+0 mg/kg-day based on an earlier IRIS evaluation of the same rat and mouse gavage study. Given the EPA (2010) re-evaluation of this study and the lowering of the chronic oral RfD by an order of magnitude, it seems prudent to adopt the verified chronic oral RfD of 2E-1 mg/kg-day for use with mixed xylenes and all xylene isomers. The critical effects of prolonged oral exposure to xylenes are reduced body weight and reduced survival. The verified chronic oral RfD is adopted as sufficiently protective for subchronic oral exposure to mixed xylenes and all xylene isomers.

Occupational exposure to xylenes induces CNS effects and GI disturbances (ACGIH, 1991). Other effects attributed to occupational exposure to xylene (blood dyscrasias, and heart, liver and kidney damage) may arise from concurrent exposure to other chemicals. EPA (2010) verified a chronic inhalation RfC of 1E-1 mg/m^3 based on CNS effects. In the key study, male rats were intermittently exposed to m-xylene at airborne concentrations of 0, 50 or 100 ppm for three months. Endpoints evaluated included spontaneous motor activity and motor coordination as measured by performance on a rotorod. Body and organ weights and limited blood chemistry and hematological parameters were also evaluated, but minor alterations in these endpoints were not attributed to exposure to m-xylene. The study identified 50 ppm as the NOAEL and 100 ppm as the LOAEL for impaired rotorod performance. The NOAEL is equivalent to an adjusted human equivalent concentration of 39 mg/m^3. Application of an uncertainty factor of 300 to the NOAEL yields the verified chronic inhalation RfC of 1E-1 mg/m^3, which is equivalent to a chronic inhalation RfD of 2.9E-2 mg/kg-day. The uncertainty factor consists of a factor of 10 to provide greater protection for more sensitive individuals, and factors of 3 each to extrapolate from animals to humans, to expand from subchronic to chronic exposure, and to account for deficiencies in the data base. The CNS is considered to be the target organ for prolonged inhalation exposure to xylenes. Confidence in the chronic inhalation RfC is medium.

No EPA-derived subchronic inhalation RfC is available for exposure to xylene. However, a preliminary subchronic inhalation RfC can be derived by applying an uncertainty factor of 100 to the NOAEL of 39 mg/m^3 in the subchronic rotorod study in rats described above. The uncertainty factor of 100 reflects the chronic uncertainty factor of 300 without the factor of 3 to expand from subchronic to chronic exposure. The preliminary subchronic inhalation RfC so derived is 4E-1 mg/m^3, which is equivalent to a subchronic inhalation RfD of 1.1E-1 mg/kg-day.

6.0 Carcinogenicity Evaluation

Xylene was previously characterized as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans). Using a more descriptive characterization, EPA (2010) concluded that the cancer *data are inadequate for an assessment of the carcinogenic potential of xylenes*. Adequate human data on the carcinogenicity of the xylenes are not available, and the available animal data are inconclusive as to the ability of xylenes to cause a carcinogenic response. Evaluations of the genotoxic effects of xylenes have consistently given negative results. Potency factors are not available.

7.0 Toxicity Summary

Toxicity values for the xylenes are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2E-1	2E-1	BW, Su	4E-1/ 1.1E-1	1E-1/ 2.9E-2	CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: BW = reduced body weight; Su = reduced survival; CNS = central nervous system.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 1995, ***Update Toxicological Profile for Xylenes***, U.S. Department of Health and Human Services, Atlanta, Georgia, August, on line.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, ***Documentation of the Threshold Limit Values and Biological Exposure Indices***, Sixth Edition, Cincinnati, OH, pp. 1732-1740.

Hazardous Substance Data Bank (HSDB), 2010, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

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.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

Miscellaneous

CYANIDE ANION (57-12-5), FREE CYANIDE, HYDROGEN CYANIDE (74-90-8)

Cyanide Anion

1.0 Introduction and Physical Properties

The term cyanide is properly used to mean the cyanide anion illustrated above. The term is also used herein to represent any form of cyanide, particularly if the form was not specified in the literature cited. The term free cyanide generally means the cyanide anion, hydrogen cyanide and some cyanide salts (e.g., sodium cyanide, potassium cyanide) that readily dissociate and release cyanide in water solutions within the biological pH range (ATSDR, 2006). Chlorine-amenable cyanide includes free cyanide as well as most organic complexes and most simple metal complexes. The chlorine-amenable fraction excludes iron cyanides, which resist degradation with release of cyanide. Total cyanide includes all of the above.

Concentrations of cyanide reported by various analytical procedures do not necessarily reflect the amount that is bioavailable, and data are not available with which to quantify bioavailability (ATSDR, 2006). Free cyanide and simple water soluble complexes (part of the chlorine-amenable fraction) are expected to be highly bioavailable. More complicated organic complexes and many metal complexes are expected to be somewhat less bioavailable, but the nature of the specific compound and pH of the matrix will greatly influence bioavailability. A reasonably conservative approach (adopted herein) is to evaluate total cyanide in the RA. A more reasonable approach, including only chlorine-amenable cyanide in the RA, might be justified if there were large differences between concentrations of chlorine-amenable and total cyanide. Large differences (upwards of an order of magnitude) might be expected in mining situations where metal complexes are expected to predominate.

Hydrogen cyanide is used in the synthesis of a wide range of organic chemicals (ATSDR, 2006; HSDB, 2010). Cyanide salts are used in metal treatment, ore extraction, as anticaking agents in road salt, and in a wide variety of other uses. Hydrogen cyanide has been used formerly to fumigate grain to control insects and rodents, and to exterminate rodents in their burrows. The cyanide anion is an **inorganic** substance. Hydrogen cyanide is a liquid, or gas above 26.5 EC (HSDB, 2010), and behaves very much like a **VOC** in the environment. Relevant physical properties of inorganic cyanide and hydrogen cyanide are compiled below:

Cyanide Anion (57-12-5)							
MW	log K_{ow}	H	K_d	D_a	D_w	VP	S
26.02[a]	NA	NA	NA	NA	NA	NA	Note 1[b]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; K_d = soil/water partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; NA = not applicable.
Note 1: Several simple complexes or salts (e.g., hydrogen cyanide, sodium cyanide, potassium cyanide) are highly soluble in water, while other salts and organic complexes are sparingly soluble or practically insoluble in water.
[a]Howard, P.H. and M. Neal, 1992, ***Dictionary of Chemical Names and Synonyms***, Lewis Publishers, Ann Arbor.
[b]Agency for Toxic Substances and Disease Registry (ATSDR), 1997, ***Update Toxicological Profile for Cyanide***, U.S. Department of Health and Human Services, Atlanta, GA, on line, September.

Hydrogen Cyanide (74-90-8)										
MW	log K_{ow}	H	log K_{oc}	D_a	D_w	VP	S	T_b	T_c	$\Delta H_{v,b}$
27.03[a]	-0.25[a]	1.33E-4[a] (25°C)[b]	-0.044[c]	2.11E-1[c] (25°C)[b]	2.44E-5[c] (25°C)[b]	9.76E-1[a] (25°C)	1E+6[a] (25°C)	298.8[a]	456.7[a]	6.02E+3[a]

MW = molecular weight (g/mole); log K_{ow} = base 10 logarithm of the octanol/water partition coefficient (unitless); H = Henry's Law constant (atm-m^3/mole) at the reference temperature; log K_{oc} = base 10 logarithm of the soil/organic carbon partition coefficient (L/kg); D_a = diffusivity in air (cm^2/second); D_w = diffusivity in water (cm^2/second); VP = vapor pressure (atm) at the reference temperature; S = solubility in water (mg/L) at the reference temperature; T_b = normal boiling point (at 1 atm) of pure liquid compound (°K); T_c = critical temperature (°K);.$H_{v,b}$ = enthalpy of vaporization at the boiling point (cal/mole).
[a]Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line, accessed 22 April.
[b]Assumed.
[c]Calculated as described in Introduction to Toxicity Profiles.

2.0 Environmental Fate and Transport

Cyanide is a naturally occurring substance (ATSDR, 2008; HSDB, 2010). Hydrogen cyanide may be produced naturally by microorganisms as well as from the degradation of cyanogenic glycosides, which are produced naturally by many plants. Several species of blue-green algae liberate hydrogen cyanide in the process of nitrate metabolism. Laetrile and amygdalin, which occur in substantial concentrations in several fruit pits (e.g., cherries, peaches, apricots, apples, pears) liberate cyanide following ingestion.

Major releases to the environment occur from metal treatment and ore extraction (primarily to water) and in automobile exhaust (primarily to the atmosphere) (ATSDR,2008). The production of hydrogen cyanide and its use as a starting material in the manufacture of acrylates, cyanide salts, herbicides and dyes as well as its former use as a fumigant may result in its release to the environment through various waste streams (HSDB, 2010). It also may be liberated from blast furnaces, gas works, coke ovens and oil shale retorting operations. Most hydrogen cyanide is emitted primarily to the atmosphere or waste water. Various cyanide salts may be released during their use in ore extraction and other metallurgic operations. Materials containing cyanide compounds disposed on land may cause elevated levels of cyanide in underlying strata and in groundwater. Cigarette smoke contains low levels of hydrogen cyanide.

Hydrogen cyanide released to the atmosphere is expected to remain solely in the vapor phase and to react slowly with photochemically generated hydroxyl radicals (HSDB, 2010). A half-life of approximately 300 to 550 days has been estimated for this reaction. Hydrogen cyanide is expected to be resistant to direct photolysis. The relatively slow rate of degradation suggests that hydrogen cyanide has the potential for transport over long distances before being removed by physical or chemical processes. Since hydrogen cyanide is miscible in water, wet deposition may be the most important removal process. Metal cyanide particles released to the air in dust are expected to be removed by both wet and dry deposition.

Cyanide may occur in the form of hydrogen cyanide, metal salts, or immobile metallocyanide complexes in soil (ATSDR, 2008; HSDB, 2010). It is expected that volatilization of hydrogen cyanide would be an important mechanism for cyanide removal from dry soil surfaces and from moist soil surfaces with pH less than 9.2. Hydrogen cyanide and many cyanide salts are very water soluble and are expected to be highly mobile in soil, readily leaching to deeper levels and to groundwater. Concentrations sufficiently low so as not to be toxic to microorganisms may undergo biodegradation in subsurface soil. Microbial degradation may yield formamide, or complete

mineralization may yield carbon dioxide and ammonia. Degradation of low concentrations in soil may substantially reduce the potential for leaching to groundwater.

Hydrogen cyanide is not expected to adsorb to suspended solids and sediment in water (HSDB, 2010). Volatilization from water surfaces is expected to be the most important removal mechanism. Biodegradation may be an important removal mechanism for low concentrations that are not toxic to microorganisms as explained for soil.

Hydrogen cyanide is volatile and labile at low concentrations. Its low log K_{ow} suggests that bioconcentration is unlikely to be significant. Some metal complexes have been shown to biotransfer, but the toxicity of these complexes is much less than that of hydrogen cyanide because of their very low solubility (ATSDR, 2008). There is no evidence of biomagnification in the food chain (ATSDR, 2008), and EPA (1995) has determined that cyanide is not a threat to human health through fish consumption. Biotransfer factors are not estimated for cyanide for these reasons.

3.0 Toxicokinetics

Inhalation exposure of humans to hydrogen cyanide results in very rapid absorption and high systemic retention (approximately 58 percent retention) (ATSDR,2008). Absorption is similarly rapid following oral exposure. Excretion data from orally treated animals suggest a minimum absorption factor of approximately 50 percent, but the extent of absorption is probably much higher. Dermal absorption of hydrogen cyanide by humans is inferred from toxicity data and supported by data from experimental animals. It is likely that some cyanide salts may also be absorbed but the extent has not been quantified.

Cyanide is rapidly and widely distributed throughout the body following absorption (ATSDR,2008).

The primary detoxication pathway involves the action of the enzyme rhodanese to combine cyanide with sulfane sulfur to yield thiocyanate, which is rapidly excreted through the urine (ATSDR, 2008). Rhodanese activity is located in most tissues of the body; the relative activity largely dependent on species. Highest levels are not generally found in the liver, which is often the primary site of metabolism for environmental contaminants. Minor detoxication pathways include pulmonary excretion of hydrogen cyanide, conversion of cyanide to carbon dioxide or other constituents that enter the single-carbon pool, and incorporation into cyanocobalamin (vitamin B_{12}). Elimination occurs rapidly, resulting in a half-life for cyanide of approximately 6 hours in humans. The incorporation of carbon into the single-carbon pool may account for the low estimate of extent of GI absorption from excretion data.

Hydrogen cyanide is the toxic moiety, complexing with ferric iron in various metalloenzymes, inhibiting mitochondrial cytochrome c oxidase and resulting in histotoxic anoxia (ATSDR,2008). Several changes follow that exacerbate the original biochemical lesion.

4.0 Dermal Exposure

GI absorption of cyanide almost certainly exceeds 50 percent as noted in Section 3.0. EPA (2004) suggests that oral toxicity values should not be adjusted when used for dermal exposure unless GI absorption is less than 50 percent. Therefore, the oral toxicity values described below are adopted without modification for use for dermal exposure

EPA (1991) stated that, "performing an oral-to-dermal extrapolation would not be appropriate because of the uncertainty that the oral RfD for cyanide would provide protection for the effects caused by dermal exposure." The available data, however, suggest that the effects following oral and dermal exposure are similar (ATSDR, 2008). Furthermore, nothing in the toxicokinetic data suggest that detoxication would occur more efficiently following oral exposure so that the oral RfD may not be sufficiently protective for dermal exposure. Therefore, it is judged that the oral RfD is an appropriate value with which to evaluate the toxicity of dermal exposure to cyanide.

Two scenarios are relevant to dermal exposure to cyanide. The first scenario is exposure to cyanide in soil. Cyanide present in soil as hydrogen cyanide would be likely to volatilize so that dermal exposure is expected to be insignificant, as noted by EPA (2004) for other VOCs. Therefore, dermal exposure to hydrogen cyanide in soil is not quantified. It is possible that cyanide may exist as various salts or complexes in soil. Although these compounds may be dermally absorbed, the extent has not been quantified, so no ABS is available with which to quantify dermal uptake. Therefore, dermal uptake of cyanide from soil is not quantified, as recommended by EPA (2004).

The second scenario is exposure to cyanide in water, which may occur as hydrogen cyanide or soluble salts. Cyanide in water will be assumed to be present entirely as hydrogen cyanide to simplify the evaluation. Values for t*, K_p, ϑ, B and FA estimated by the EPA (2004) methodology follow:

Chemical	t*	K_p	τ	FA	B
Hydrogen Cyanide	0.36	7.65E-4	0.15	1	0.0
t* = time for dermal uptake to reach steady state (hours); K_p = permeability coefficient (cm/hour); □ = lag time for chemical to cross stratum corneum (hours); FA = fraction absorbed (unitless); B = ratio of the permeability coefficient for passage across the stratum corneum relative to the permeability coefficient for passage across the viable epidermis (unitless).					

5.0 Noncancer Effects Evaluation

Oral LD_{50} values in laboratory animals range from 8 to 22 mg/kg, depending on the particular cyanide compound and laboratory species (ATSDR, 2008; HSDB, 2010), indicating that the acute oral toxicity of cyanide is high. Prolonged oral exposure is associated with weight loss, thyroid effects and nervous system effects, particularly myelin degeneration (EPA, 2010). A verified chronic oral RfD of 2E-2 mg/kg-day for free cyanide and hydrogen cyanide was derived from a NOAEL of 10.8 mg/kg-day in a 2-year study in which rats were fed diets fumigated with hydrogen cyanide. The LOAEL of 30 mg/kg-day in another rat feeding study is associated with the effects described above. An uncertainty factor of 500 was applied. Confidence in the oral RfD is medium. Loss of body weight, thyroid effects and nervous system effects are considered the critical effects for prolonged oral exposure to cyanide. EPA (1997) adopted the verified chronic oral RfD as sufficiently protective for subchronic oral exposure.

EPA (2010) verified a chronic inhalation RfC of 3E-3 mg/m^3 for hydrogen cyanide based on nervous symptoms and thyroid effects in occupationally exposed humans. The key study involved 36 non-smoking male workers employed in the electroplating sections of three factories in Egypt exposed for 5 to 10 years (1 man exposed for 15 years). Twenty non-smoking unexposed male volunteers served as controls. Breathing-zone cyanide concentrations ranged from 4.63 to 13.69 mg/m^3, with a mean of 7.07 to 11.45 mg/m^3. Symptoms reported more frequently in the exposed workers included (in decreasing order of frequency) headache, weakness, and changes in the senses of taste and smell.

Lacrimation, abdominal colic, and precordial (lower stomach) pain, salivation, and nervous instability occurred less frequently. Twenty of the exposed workers had thyroid enlargement to a mild or moderate degree, and thyroid function (iodine uptake) was altered. The lowest mean concentration recorded in the three factories, 7.07 mg/m^3, corresponding to an exposure-adjusted concentration of 2.5 mg/m^3, was designated a LOAEL. Application of an uncertainty factor of 1000 (factors of 10 each to estimate a NOAEL from a LOAEL and to provide additional protection for unusually sensitive individuals, and factors of 3 each for deficiencies in the database [lack of chronic and multigenerational reproduction studies] and for less than chronic duration) yields the verified chronic inhalation RfC of 3E-3 mg/m^3. The chronic RfC is equivalent to a chronic inhalation RfD of 8.6E-4 mg/kg-day. The thyroid and CNS are the targets for prolonged inhalation exposure. Confidence in the verified RfC is low.

A subchronic inhalation RfC of 8E-3 mg/m^3 equivalent to a subchronic inhalation RfD of 2.3E-3 mg/kg-day is derived by applying an uncertainty factor of 300 to the LOAEL of 2.5 mg/m^3 described above. The uncertainty factor of 300 reflects the chronic uncertainty factor of 1000 excluding the factor of 3 to expand from subchronic to chronic exposure.

The chronic and subchronic inhalation RfC and RfD values can be applied to any airborne form of cyanide.

6.0 Carcinogenicity Evaluation

EPA (2010) classified cyanide in EPA cancer weight-of-evidence Group D – not classifiable as to carcinogenicity to humans – because of a lack of data in humans or laboratory animals. Cancer potency factors are not derived for Group D chemicals.

7.0 Toxicity Summary

Toxicity values for cyanide/hydrogen cyanide are summarized below:

Noncancer Effects						Carcinogenicity				
Oral Exposure[a]			Inhalation Exposure			Oral Exposure		Inhalation Exposure		
sRfDo	cRfDo	TO	sRfC/ sRfDi	cRfC/ cRfDi	TO	WOE	SFo	WOE	URFi	SFi
2E-2	2E-2	BW, T, NS	8E-3/ 2.3E-3	3E-3/ 8.6E-4	T, CNS	D	NA	D	NA	NA

sRfDo = subchronic oral reference dose (milligrams per kilogram-day); cRfDo = chronic oral reference dose (milligrams per kilogram-day); TO = target organ(s) or critical effect(s); sRfC = subchronic inhalation reference concentration (milligrams per cubic meter); sRfDi = subchronic inhalation reference dose (milligrams per kilogram-day); cRfC = chronic inhalation reference concentration (milligrams per cubic meter); cRfDi = chronic inhalation reference dose (milligrams per kilogram-day); WOE = cancer weight-of-evidence evaluation; SFo = oral cancer slope factor (risk per milligram per kilogram-day); URFi = inhalation unit risk factor (risk per microgram per cubic meter); SFi = inhalation cancer slope factor (risk per milligram per kilogram-day); NA = not applicable.
Target organ or critical effect abbreviations: BW = reduced body weight; T = thyroid, NS = nervous system; CNS = central nervous system.
[a]sRfDo and cRfDo should be used for dermal exposure without adjustment for GI absorption.

8.0 References

Agency for Toxic Substances and Disease Registry (ATSDR), 2006, ***Toxicological Profile for Cyanide***, U.S. Department of Health and Human Services, Atlanta, GA, June.

Hazardous Substance Data Bank (HSDB), 2004, National Library of Medicine, on line.

U.S. Environmental Protection Agency (EPA), 1991, ***Risk Assessment Issue Paper for: Dermal Absorption of Cyanide (CASRN 57-12-5)***, National Center for Environmental Assessment, Cincinnati, OH, 3 June.

U.S. Environmental Protection Agency (EPA), 1995, "Final Water Quality Guidance for the Great Lakes System; Final Rule," (40 CFR 9, 122, 123, 131, and 132), ***Federal Register***, 60(56): 15365-15425.

U.S. Environmental Protection Agency (EPA), 1997, ***Health Effects Assessment Summary Tables, FY 1997 Update***, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, PB97-921199, July.

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, DC, EPA/540/R/99/005, OSWER 9285.7-02EP, NTIS No. PB99-963312, July.

U.S. Environmental Protection Agency (EPA), 2010, ***Integrated Risk Information System (IRIS)***, National Center for Environmental Assessment, Cincinnati, OH, on line.

APPENDIX D

RISK CHARACTERIZATION SPREADSHEETS

Table D-1

Groundskeeper, Current Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Inorganics										
Chromium	2.24E+01	7.83E-06	2.19E-05	NA	1.04E-03	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1221	1.03E-01	3.60E-08	1.01E-07	7.20E-08	1.44E-03	2.88E-09	3.33E-08	9.31E-08	6.65E-08	1.33E-03
Semivolatile Organic Compounds										
Benzo(a)anthracene	2.92E-01	1.02E-07	2.86E-07	7.45E-08	NA	7.59E-09	8.76E-08	2.45E-07	6.39E-08	NA
Benzo(a)pyrene	3.41E-01	1.19E-07	3.34E-07	8.70E-07	NA	8.87E-09	1.02E-07	2.86E-07	7.46E-07	NA
Benzo(b)fluoranthene	7.42E-01	2.59E-07	7.26E-07	1.89E-07	NA	1.93E-08	2.22E-07	6.23E-07	1.62E-07	NA
Dinitrotoluene, 2,4-	1.44E+00	5.03E-07	1.41E-06	3.42E-07	7.04E-04	2.88E-08	3.32E-07	9.29E-07	2.26E-07	4.65E-04
Total ILCR or HI				**1.55E-06**	**3.19E-03**				**1.26E-06**	**1.79E-03**

Table D-1

Groundskeeper, Current Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Inorganics							
Chromium	2.24E-06	1.57E-07	4.38E-07	6.58E-06	2.16E-03	**6.58E-06**	**3.20E-03**
Polychlorinated biphenyls (PCB)							
Aroclor 1221	1.03E-08	7.20E-10	2.02E-09	1.44E-09	NA	**1.40E-07**	**2.77E-03**
Semivolatile Organic Compounds							
Benzo(a)anthracene	2.92E-08	2.04E-09	5.71E-09	6.33E-10	NA	**1.39E-07**	**NA**
Benzo(a)pyrene	3.41E-08	2.38E-09	6.67E-09	7.39E-09	NA	**1.62E-06**	**NA**
Benzo(b)fluoranthene	7.42E-08	5.19E-09	1.45E-08	1.61E-09	NA	**3.53E-07**	**NA**
Dinitrotoluene, 2,4-	1.44E-07	1.01E-08	2.82E-08	NA	NA	**5.68E-07**	**1.17E-03**
Total ILCR or HI				**6.59E-06**	**2.16E-03**	**9.40E-06**	**7.14E-03**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-2

Groundskeeper, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Inorganics										
Aluminum	9.11E+03	3.18E-03	8.91E-03	NA	8.91E-03	NA	NA	NA	NA	NA
Chromium	1.77E+01	6.17E-06	1.73E-05	NA	8.23E-04	NA	NA	NA	NA	NA
Vanadium	2.65E+01	9.27E-06	2.60E-05	NA	5.19E-03	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1221	3.96E-02	1.38E-08	3.87E-08	2.77E-08	5.54E-04	1.11E-09	1.28E-08	3.58E-08	2.56E-08	5.11E-04
Nitroaromatics										
Dinitrotoluene, 2,6-	9.75E-01	3.41E-07	9.54E-07	2.32E-07	9.54E-04	1.95E-08	2.25E-07	6.30E-07	1.53E-07	6.30E-04
Nitrotoluene, 2-	7.66E+00	2.68E-06	7.49E-06	5.89E-07	8.32E-03	NA	NA	NA	NA	NA
Nitrotoluene, 3-	1.52E+00	5.31E-07	1.49E-06	NA	7.44E-05	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.98E-01	6.92E-08	1.94E-07	5.05E-08	NA	5.15E-09	5.94E-08	1.66E-07	4.33E-08	NA
Benzo(a)pyrene	2.02E-01	7.06E-08	1.98E-07	5.15E-07	NA	5.25E-09	6.06E-08	1.70E-07	4.42E-07	NA
Benzo(b)fluoranthene	3.12E-01	1.09E-07	3.05E-07	7.96E-08	NA	8.11E-09	9.35E-08	2.62E-07	6.83E-08	NA
Dinitrotoluene, 2,4-	1.52E+00	5.30E-07	1.48E-06	3.60E-07	7.42E-04	3.03E-08	3.50E-07	9.79E-07	2.38E-07	4.90E-04
Total ILCR or HI				**1.85E-06**	**2.56E-02**				**9.70E-07**	**1.63E-03**

Table D-2

Groundskeeper, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Inorganics							
Aluminum	9.11E-04	6.37E-05	1.78E-04	NA	1.27E-01	**NA**	**1.36E-01**
Chromium	1.77E-06	1.23E-07	3.46E-07	5.18E-06	1.70E-03	**5.18E-06**	**2.53E-03**
Vanadium	2.65E-06	1.85E-07	5.19E-07	NA	NA	**NA**	**5.19E-03**
Polychlorinated biphenyls (PCB)							
Aroclor 1221	3.96E-09	2.77E-10	7.75E-10	5.54E-10	NA	**5.38E-08**	**1.07E-03**
Nitroaromatics							
Dinitrotoluene, 2,6-	9.75E-08	6.81E-09	1.91E-08	NA	NA	**3.85E-07**	**1.58E-03**
Nitrotoluene, 2-	7.66E-07	5.35E-08	1.50E-07	NA	NA	**5.89E-07**	**8.32E-03**
Nitrotoluene, 3-	1.52E-07	1.06E-08	2.97E-08	NA	NA	**NA**	**7.44E-05**
Semivolatile Organic Compounds							
Benzo(a)anthracene	1.98E-08	1.38E-09	3.87E-09	4.29E-10	NA	**9.43E-08**	**NA**
Benzo(a)pyrene	2.02E-08	1.41E-09	3.95E-09	4.38E-09	NA	**9.62E-07**	**NA**
Benzo(b)fluoranthene	3.12E-08	2.18E-09	6.11E-09	6.76E-10	NA	**1.49E-07**	**NA**
Dinitrotoluene, 2,4-	1.52E-07	1.06E-08	2.97E-08	NA	NA	**5.98E-07**	**1.23E-03**
Total ILCR or HI				**5.19E-06**	**1.29E-01**	**8.01E-06**	**1.56E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-3

Groundskeeper, Future Scenario, Exposure to Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
Chemicals of Potential Concern	EPC mg/L	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	1.31E-01	4.58E-04	1.28E-03	6.87E-04	4.27E+00	1.31E-07	1.51E-06	4.23E-06	2.27E-06	1.41E-02	**6.89E-04**	**4.29E+00**
Iron	2.02E+00	7.04E-03	1.97E-02	NA	2.82E-02	2.02E-06	2.32E-05	6.51E-05	NA	NA	**NA**	**2.82E-02**
Manganese	1.07E+00	3.74E-03	1.05E-02	NA	2.23E-01	1.07E-06	1.23E-05	3.45E-05	NA	1.82E-02	**NA**	**2.41E-01**
Thallium	6.40E-03	2.24E-05	6.26E-05	NA	9.63E-01	6.40E-09	7.38E-08	2.07E-07	NA	3.18E-03	**NA**	**9.67E-01**
Semivolatile Organic Compounds												
Methylnaphthalene, 2-	2.56E-01	8.95E-04	2.50E-03	NA	6.26E-01	5.13E-05	5.91E-04	1.66E-03	NA	4.14E-01	**NA**	**1.04E+00**
Naphthalene	4.72E-02	1.65E-04	4.62E-04	NA	2.31E-02	4.59E-06	5.29E-05	1.48E-04	NA	7.41E-03	**NA**	**3.05E-02**
Volatile Organic Compounds												
Benzene	1.68E+00	5.87E-03	1.64E-02	3.23E-04	4.11E+00	3.75E-05	4.33E-04	1.21E-03	2.38E-05	3.03E-01	**3.47E-04**	**4.41E+00**
Chloroform	5.63E-02	1.97E-04	5.51E-04	NA	5.51E-02	7.48E-07	8.63E-06	2.42E-05	NA	2.42E-03	**NA**	**5.75E-02**
Chloromethane	3.34E-02	1.17E-04	3.27E-04	NA	NA	1.36E-07	1.57E-06	4.40E-06	NA	NA	**NA**	**NA**
Ethylbenzene	1.69E-01	5.91E-04	1.65E-03	NA	1.65E-02	1.48E-05	1.71E-04	4.79E-04	NA	4.79E-03	**NA**	**2.13E-02**
Methylene chloride	6.04E-02	2.11E-04	5.91E-04	1.58E-06	9.84E-03	3.30E-07	3.81E-06	1.07E-05	2.86E-08	1.78E-04	**1.61E-06**	**1.00E-02**
Tetrachloroethane, 1,1,2,2-	3.70E-03	1.29E-05	3.62E-05	2.59E-06	9.05E-03	6.80E-08	7.85E-07	2.20E-06	1.57E-07	5.49E-04	**2.74E-06**	**9.60E-03**
Toluene	6.92E-01	2.42E-03	6.77E-03	NA	8.46E-02	3.51E-05	4.05E-04	1.13E-03	NA	1.42E-02	**NA**	**9.88E-02**
Xylenes, total	1.13E+00	3.95E-03	1.11E-02	NA	5.53E-02	1.07E-04	1.24E-03	3.46E-03	NA	1.73E-02	**NA**	**7.26E-02**
General Chemistry												
Cyanide, total	1.10E-01	3.84E-04	1.08E-03	NA	5.38E-02	1.10E-07	1.27E-06	3.55E-06	NA	1.78E-04	**NA**	**5.40E-02**
Total ILCR or HI				**1.01E-03**	**1.05E+01**				**2.62E-05**	**7.99E-01**	**1.04E-03**	**1.13E+01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm2 - milligram per square meter
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-4

Groundskeeper, Future Scenario, Exposure to Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
	EPC	Dose	Dose			Absorbed	Dose	Dose			Total	Total
Chemicals of Potential Concern	mg/L	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ	ILCR	HI
Inorganics												
Chromium, Total	3.30E-03	1.15E-05	3.23E-05	NA	1.54E-03	3.30E-09	3.81E-08	1.07E-07	NA	3.95E-04	**NA**	**1.93E-03**
Cobalt	6.07E-03	2.12E-05	5.94E-05	NA	1.98E-01	2.43E-09	2.80E-08	7.84E-08	NA	2.61E-04	**NA**	**1.98E-01**
Iron	1.48E+00	5.16E-03	1.45E-02	NA	2.06E-02	1.48E-06	1.70E-05	4.77E-05	NA	NA	**NA**	**2.06E-02**
Manganese	3.29E-01	1.15E-03	3.22E-03	NA	6.84E-02	3.29E-07	3.79E-06	1.06E-05	NA	5.59E-03	**NA**	**7.40E-02**
Nitroaromatics												
Dinitrotoluene, 2,4-	4.67E-03	1.63E-05	4.57E-05	1.11E-05	2.29E-02	4.24E-08	4.89E-07	1.37E-06	3.32E-07	6.84E-04	**1.14E-05**	**2.36E-02**
Dinitrotoluene, 2,6-	3.99E-03	1.39E-05	3.90E-05	9.48E-06	3.90E-02	2.45E-08	2.83E-07	7.91E-07	1.92E-07	7.91E-04	**9.67E-06**	**3.98E-02**
Nitrotoluene, 2-	3.77E-02	1.32E-04	3.69E-04	2.90E-05	4.10E-01	7.31E-07	8.43E-06	2.36E-05	1.86E-06	2.62E-02	**3.09E-05**	**4.36E-01**
Nitrotoluene, 3-	4.59E-03	1.61E-05	4.50E-05	NA	2.25E-03	1.12E-07	1.29E-06	3.62E-06	NA	1.81E-04	**NA**	**2.43E-03**
Nitrotoluene, 4-	4.40E-03	1.54E-05	4.30E-05	2.46E-07	1.08E-02	9.48E-08	1.09E-06	3.06E-06	1.75E-08	7.65E-04	**2.63E-07**	**1.15E-02**
Volatile Organic Compounds												
Carbon tetrachloride	2.38E-03	8.30E-06	2.32E-05	5.81E-07	5.81E-03	9.28E-08	1.07E-06	3.00E-06	7.49E-08	7.49E-04	**6.56E-07**	**6.56E-03**
Dibromochloromethane	2.00E-04	6.99E-07	1.96E-06	5.87E-08	9.78E-05	2.22E-09	2.56E-08	7.16E-08	2.15E-09	3.58E-06	**6.09E-08**	**1.01E-04**
Dichloroethane, 1,1-	4.44E-03	1.55E-05	4.34E-05	NA	2.17E-04	5.06E-08	5.84E-07	1.63E-06	NA	8.17E-06	**NA**	**2.25E-04**
Trichloroethene	4.84E-03	1.69E-05	4.74E-05	9.99E-08	9.48E-05	1.22E-07	1.41E-06	3.95E-06	8.33E-09	7.90E-06	**1.08E-07**	**1.03E-04**
General Chemistry (mg/L)												
Nitrate-Nitrite												
Sulfate												
Total ILCR or HI				**5.06E-05**	**7.80E-01**				**2.48E-06**	**3.57E-02**	**5.31E-05**	**8.15E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

Table D-5

Indoor Worker, Future Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion					
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics							
Chromium	2.24E+01	3.91E-06	1.10E-05	NA	5.22E-04	**NA**	**5.22E-04**
Polychlorinated biphenyls (PCB)							
Aroclor 1221	1.03E-01	1.80E-08	5.04E-08	3.60E-08	7.20E-04	**3.60E-08**	**7.20E-04**
Semivolatile Organic Compounds							
Benzo(a)anthracene	2.92E-01	5.10E-08	1.43E-07	3.72E-08	NA	**3.72E-08**	**NA**
Benzo(a)pyrene	3.41E-01	5.96E-08	1.67E-07	4.35E-07	NA	**4.35E-07**	**NA**
Benzo(b)fluoranthene	7.42E-01	1.30E-07	3.63E-07	9.46E-08	NA	**9.46E-08**	**NA**
Dinitrotoluene, 2,4-	1.44E+00	2.51E-07	7.04E-07	1.71E-07	3.52E-04	**1.71E-07**	**3.52E-04**
Total ILCR or HI						**7.74E-07**	**1.59E-03**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-6

Indoor Worker, Future Scenario, Exposure to Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
	EPC	Dose	Dose			Absorbed	Dose	Dose			Total	Total
Chemicals of Potential Concern	mg/L	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ	ILCR	HI
Inorganics												
Arsenic	1.31E-01	4.58E-04	1.28E-03	6.87E-04	4.27E+00	1.31E-07	1.51E-06	4.23E-06	2.27E-06	1.41E-02	**6.89E-04**	**4.29E+00**
Iron	2.02E+00	7.04E-03	1.97E-02	NA	2.82E-02	2.02E-06	2.32E-05	6.51E-05	NA	NA	**NA**	**2.82E-02**
Manganese	1.07E+00	3.74E-03	1.05E-02	NA	2.23E-01	1.07E-06	1.23E-05	3.45E-05	NA	1.82E-02	**NA**	**2.41E-01**
Thallium	6.40E-03	2.24E-05	6.26E-05	NA	9.63E-01	6.40E-09	7.38E-08	2.07E-07	NA	3.18E-03	**NA**	**9.67E-01**
Semivolatile Organic Compounds												
Methylnaphthalene, 2-	2.56E-01	8.95E-04	2.50E-03	NA	6.26E-01	5.13E-05	5.91E-04	1.66E-03	NA	4.14E-01	**NA**	**1.04E+00**
Naphthalene	4.72E-02	1.65E-04	4.62E-04	NA	2.31E-02	4.59E-06	5.29E-05	1.48E-04	NA	7.41E-03	**NA**	**3.05E-02**
Volatile Organic Compounds												
Benzene	1.68E+00	5.87E-03	1.64E-02	3.23E-04	4.11E+00	3.75E-05	4.33E-04	1.21E-03	2.38E-05	3.03E-01	**3.47E-04**	**4.41E+00**
Chloroform	5.63E-02	1.97E-04	5.51E-04	NA	5.51E-02	7.48E-07	8.63E-06	2.42E-05	NA	2.42E-03	**NA**	**5.75E-02**
Chloromethane	3.34E-02	1.17E-04	3.27E-04	NA	NA	1.36E-07	1.57E-06	4.40E-06	NA	NA	**NA**	**NA**
Ethylbenzene	1.69E-01	5.91E-04	1.65E-03	NA	1.65E-02	1.48E-05	1.71E-04	4.79E-04	NA	4.79E-03	**NA**	**2.13E-02**
Methylene chloride	6.04E-02	2.11E-04	5.91E-04	1.58E-06	9.84E-03	3.30E-07	3.81E-06	1.07E-05	2.86E-08	1.78E-04	**1.61E-06**	**1.00E-02**
Tetrachloroethane, 1,1,2,2-	3.70E-03	1.29E-05	3.62E-05	2.59E-06	9.05E-03	6.80E-08	7.85E-07	2.20E-06	1.57E-07	5.49E-04	**2.74E-06**	**9.60E-03**
Toluene	6.92E-01	2.42E-03	6.77E-03	NA	8.46E-02	3.51E-05	4.05E-04	1.13E-03	NA	1.42E-02	**NA**	**9.88E-02**
Xylenes, total	1.13E+00	3.95E-03	1.11E-02	NA	5.53E-02	1.07E-04	1.24E-03	3.46E-03	NA	1.73E-02	**NA**	**7.26E-02**
General Chemistry												
Cyanide, total	1.10E-01	3.84E-04	1.08E-03	NA	5.38E-02	1.10E-07	1.27E-06	3.55E-06	NA	1.78E-04	**NA**	**5.40E-02**
Total ILCR or HI				**1.01E-03**	**1.05E+01**				**2.62E-05**	**7.99E-01**	**1.04E-03**	**1.13E+01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

Table D-7

Indoor Worker, Future Scenario, Exposure to Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics												
Chromium, Total	3.30E-03	1.15E-05	3.23E-05	NA	1.54E-03	3.30E-09	3.81E-08	1.07E-07	NA	3.95E-04	**NA**	**1.93E-03**
Cobalt	6.07E-03	2.12E-05	5.94E-05	NA	1.98E-01	2.43E-09	2.80E-08	7.84E-08	NA	2.61E-04	**NA**	**1.98E-01**
Iron	1.48E+00	5.16E-03	1.45E-02	NA	2.06E-02	1.48E-06	1.70E-05	4.77E-05	NA	NA	**NA**	**2.06E-02**
Manganese	3.29E-01	1.15E-03	3.22E-03	NA	6.84E-02	3.29E-07	3.79E-06	1.06E-05	NA	5.59E-03	**NA**	**7.40E-02**
Nitroaromatics												
Dinitrotoluene, 2,4-	4.67E-03	1.63E-05	4.57E-05	1.11E-05	2.29E-02	4.24E-08	4.89E-07	1.37E-06	3.32E-07	6.84E-04	**1.14E-05**	**2.36E-02**
Dinitrotoluene, 2,6-	3.99E-03	1.39E-05	3.90E-05	9.48E-06	3.90E-02	2.45E-08	2.83E-07	7.91E-07	1.92E-07	7.91E-04	**9.67E-06**	**3.98E-02**
Nitrotoluene, 2-	3.77E-02	1.32E-04	3.69E-04	2.90E-05	4.10E-01	7.31E-07	8.43E-06	2.36E-05	1.86E-06	2.62E-02	**3.09E-05**	**4.36E-01**
Nitrotoluene, 3-	4.59E-03	1.61E-05	4.50E-05	NA	2.25E-03	1.12E-07	1.29E-06	3.62E-06	NA	1.81E-04	**NA**	**2.43E-03**
Nitrotoluene, 4-	4.40E-03	1.54E-05	4.30E-05	2.46E-07	1.08E-02	9.48E-08	1.09E-06	3.06E-06	1.75E-08	7.65E-04	**2.63E-07**	**1.15E-02**
Volatile Organic Compounds												
Carbon tetrachloride	2.38E-03	8.30E-06	2.32E-05	5.81E-07	5.81E-03	9.28E-08	1.07E-06	3.00E-06	7.49E-08	7.49E-04	**6.56E-07**	**6.56E-03**
Dibromochloromethane	2.00E-04	6.99E-07	1.96E-06	5.87E-08	9.78E-05	2.22E-09	2.56E-08	7.16E-08	2.15E-09	3.58E-06	**6.09E-08**	**1.01E-04**
Dichloroethane, 1,1-	4.44E-03	1.55E-05	4.34E-05	NA	2.17E-04	5.06E-08	5.84E-07	1.63E-06	NA	8.17E-06	**NA**	**2.25E-04**
Trichloroethene	4.84E-03	1.69E-05	4.74E-05	9.99E-08	9.48E-05	1.22E-07	1.41E-06	3.95E-06	8.33E-09	7.90E-06	**1.08E-07**	**1.03E-04**
Total ILCR or HI				**5.06E-05**	**7.80E-01**				**2.48E-06**	**3.57E-02**	**5.31E-05**	**8.15E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

Table D-8

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

(Page 1 of 2)

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Inorganics										
Aluminum	9.11E+03	2.10E-04	2.93E-02	NA	2.93E-02	NA	NA	NA	NA	NA
Chromium	1.77E+01	4.07E-07	5.69E-05	NA	2.71E-03	NA	NA	NA	NA	NA
Vanadium	2.65E+01	6.12E-07	8.54E-05	NA	1.71E-02	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1221	3.96E-02	9.13E-10	1.28E-07	1.83E-09	1.82E-03	1.66E-09	3.84E-10	5.36E-08	7.67E-10	7.65E-04
Nitroaromatics										
Dinitrotoluene, 2,6-	9.75E-01	2.25E-08	3.14E-06	1.53E-08	3.14E-03	2.93E-08	6.75E-09	9.42E-07	4.59E-09	9.42E-04
Nitrotoluene, 2-	7.66E+00	1.77E-07	2.47E-05	3.88E-08	2.74E-02	NA	NA	NA	NA	NA
Nitrotoluene, 3-	1.52E+00	3.51E-08	4.89E-06	NA	2.45E-04	NA	NA	NA	NA	NA
Semivolatile Organic Compounds										
Benzo(a)anthracene	1.98E-01	4.57E-09	6.38E-07	3.33E-09	NA	7.72E-09	1.78E-09	2.49E-07	1.30E-09	NA
Benzo(a)pyrene	2.02E-01	4.66E-09	6.50E-07	3.40E-08	NA	7.88E-09	1.82E-09	2.54E-07	1.33E-08	NA
Benzo(b)fluoranthene	3.12E-01	7.20E-09	1.00E-06	5.25E-09	NA	1.22E-08	2.81E-09	3.92E-07	2.05E-09	NA
Dinitrotoluene, 2,4-	1.52E+00	3.50E-08	4.88E-06	2.38E-08	2.44E-03	4.55E-08	1.05E-08	1.46E-06	7.13E-09	7.32E-04
Total ILCR or HI				**1.22E-07**	**8.42E-02**				**2.91E-08**	**2.44E-03**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index

mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-8

Construction Worker, Current and Future Scenarios, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Inorganics							
Aluminum	3.19E-03	4.46E-06	6.22E-04	NA	4.44E-01	**NA**	**4.74E-01**
Chromium	6.18E-06	8.64E-09	1.21E-06	3.63E-07	5.94E-03	**3.63E-07**	**8.65E-03**
Vanadium	9.29E-06	1.30E-08	1.81E-06	NA	NA	**NA**	**1.71E-02**
Polychlorinated biphenyls (PCB)							
Aroclor 1221	1.39E-08	1.94E-11	2.70E-09	3.87E-11	NA	**2.63E-09**	**2.59E-03**
Nitroaromatics							
Dinitrotoluene, 2,6-	3.41E-07	4.77E-10	6.66E-08	NA	NA	**1.99E-08**	**4.08E-03**
Nitrotoluene, 2-	2.68E-06	3.75E-09	5.23E-07	NA	NA	**3.88E-08**	**2.74E-02**
Nitrotoluene, 3-	5.32E-07	7.44E-10	1.04E-07	NA	NA	**NA**	**2.45E-04**
Semivolatile Organic Compounds							
Benzo(a)anthracene	6.93E-08	9.69E-11	1.35E-08	3.00E-11	NA	**4.66E-09**	**NA**
Benzo(a)pyrene	7.07E-08	9.88E-11	1.38E-08	3.06E-10	NA	**4.76E-08**	**NA**
Benzo(b)fluoranthene	1.09E-07	1.53E-10	2.13E-08	4.73E-11	NA	**7.35E-09**	**NA**
Dinitrotoluene, 2,4-	5.31E-07	7.42E-10	1.04E-07	NA	NA	**3.09E-08**	**3.17E-03**
Total ILCR or HI				**3.63E-07**	**4.50E-01**	**5.15E-07**	**5.37E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index

Table D-9

Construction Worker, Current and Future Scenarios, Exposure to Sediment
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact					All Pathways	
		Cancer	Noncancer			Dose	Cancer	Noncancer				
	EPC	Dose	Dose			Absorbed	Dose	Dose			**Total**	**Total**
Chemicals of Potential Concern	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ	**ILCR**	**HI**
Inorganics												
Arsenic	1.52E+01	3.51E-07	4.91E-05	5.27E-07	1.64E-01	1.37E-07	3.16E-08	4.42E-06	4.75E-08	1.47E-02	**5.75E-07**	**1.78E-01**
Chromium, total	1.62E+01	3.72E-07	5.20E-05	NA	2.48E-03	NA	NA	NA	NA	NA	**NA**	**2.48E-03**
Cobalt	2.28E+01	5.25E-07	7.34E-05	NA	2.45E-01	NA	NA	NA	NA	NA	**NA**	**2.45E-01**
Manganese	3.98E+03	9.18E-05	1.28E-02	NA	2.73E-01	NA	NA	NA	NA	NA	**NA**	**2.73E-01**
Thallium	1.90E+00	4.38E-08	6.12E-06	NA	9.41E-02	NA	NA	NA	NA	NA	**NA**	**9.41E-02**
Total ILCR or HI				**5.27E-07**	**7.77E-01**				**4.75E-08**	**1.47E-02**	**5.75E-07**	**7.92E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-10

Construction Worker, Current and Future Scenarios, Exposure to Surface Water
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Dermal Contact				
		Dose	Cancer	Noncancer		
Chemicals of Potential Concern	EPC mg/L	Absorbed mg/cm^2-day	Dose mg/kg-day	Dose mg/kg-day	**Total ILCR**	**Total HI**
Inorganics						
Arsenic	6.69E-03	2.68E-08	6.17E-09	8.61E-07	9.26E-09	2.87E-03
Chromium	3.16E-02	1.27E-07	2.92E-08	4.08E-06	NA	1.51E-02
Cobalt	1.63E-02	2.61E-08	6.02E-09	8.40E-07	NA	2.80E-03
Iron	2.28E+01	9.12E-05	2.10E-05	2.94E-03	NA	NA
Manganese	1.71E+00	6.85E-06	1.58E-06	2.21E-04	NA	1.16E-01
Total ILCR or HI					**9.26E-09**	**1.37E-01**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-11

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact				
	EPC	Adult Dose	Child Dose	Resident	Adult Dose Absorbed	Adult Dose	Child Dose Absorbed	Child Dose	Resident
Chemicals of Potential Concern	mg/kg	mg/kg-day	mg/kg-day	ILCR	mg/cm^2-day	mg/kg-day	mg/cm^2-day	mg/kg-day	ILCR
Inorganics									
Aluminum	9.11E+03	3.85E-03	8.98E-03	NA	NA	NA	NA	NA	NA
Chromium	1.77E+01	7.46E-06	1.74E-05	NA	NA	NA	NA	NA	NA
Vanadium	2.65E+01	1.12E-05	2.62E-05	NA	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)									
Aroclor 1221	3.96E-02	1.67E-08	3.91E-08	1.12E-07	3.88E-10	1.04E-08	1.11E-09	1.70E-08	5.48E-08
Nitroaromatics									
Dinitrotoluene, 2,6-	9.75E-01	4.12E-07	9.62E-07	9.34E-07	6.83E-09	1.83E-07	1.95E-08	2.99E-07	3.28E-07
Nitrotoluene, 2-	7.66E+00	3.24E-06	7.55E-06	2.37E-06	NA	NA	NA	NA	NA
Nitrotoluene, 3-	1.52E+00	6.43E-07	1.50E-06	NA	NA	NA	NA	NA	NA
Semivolatile Organic Compounds									
Benzo(a)anthracene	1.98E-01	8.37E-08	1.95E-07	2.04E-07	1.80E-09	4.82E-08	5.15E-09	7.90E-08	9.29E-08
Benzo(a)pyrene	2.02E-01	8.54E-08	1.99E-07	2.08E-06	1.84E-09	4.92E-08	5.25E-09	8.06E-08	9.47E-07
Benzo(b)fluoranthene	3.12E-01	1.32E-07	3.08E-07	3.21E-07	2.84E-09	7.60E-08	8.11E-09	1.24E-07	1.46E-07
Dinitrotoluene, 2,4-	1.52E+00	6.41E-07	1.50E-06	1.45E-06	1.06E-08	2.84E-07	3.03E-08	4.65E-07	5.10E-07
Total ILCR				**7.47E-06**					**2.08E-06**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram

mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-11

On-Site Resident, Cancer Risk, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Adult Dose mg/kg-day	Inhalation Child Dose mg/kg-day	Inhalation Resident ILCR	**All Pathways Total ILCR**
Inorganics					
Aluminum	1.79E-05	1.69E-06	9.83E-07	NA	**NA**
Chromium	3.48E-08	3.27E-09	1.91E-09	2.17E-07	**2.17E-07**
Vanadium	5.23E-08	4.91E-09	2.86E-09	NA	**NA**
Polychlorinated biphenyls (PCB)					
Aroclor 1221	7.80E-11	7.33E-12	4.27E-12	2.32E-11	**1.66E-07**
Nitroaromatics					
Dinitrotoluene, 2,6-	1.92E-09	1.80E-10	1.05E-10	NA	**1.26E-06**
Nitrotoluene, 2-	1.51E-08	1.42E-09	8.26E-10	NA	**2.37E-06**
Nitrotoluene, 3-	2.99E-09	2.81E-10	1.64E-10	NA	**NA**
Semivolatile Organic Compounds					
Benzo(a)anthracene	3.90E-10	3.66E-11	2.14E-11	1.80E-11	**2.97E-07**
Benzo(a)pyrene	3.98E-10	3.74E-11	2.18E-11	1.83E-10	**3.03E-06**
Benzo(b)fluoranthene	6.15E-10	5.77E-11	3.37E-11	2.83E-11	**4.67E-07**
Dinitrotoluene, 2,4-	2.99E-09	2.80E-10	1.64E-10	NA	**1.96E-06**
Total ILCR				**2.18E-07**	**9.77E-06**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram

Table D-12

On-Site Child Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Concentration	Inhalation	
	EPC	Child Dose	Child Resident	Dose Absorbed	Child Dose	Child Resident	in Air C_a	Child Dose	Child Resident
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ
Inorganics									
Aluminum	9.11E+03	1.05E-01	1.05E-01	NA	NA	NA	1.79E-05	1.15E-05	8.19E-03
Chromium	1.77E+01	2.03E-04	9.68E-03	NA	NA	NA	3.48E-08	2.22E-08	1.10E-04
Vanadium	2.65E+01	3.05E-04	6.11E-02	NA	NA	NA	5.23E-08	3.34E-08	NA
Polychlorinated biphenyls (PCB)									
Aroclor 1221	3.96E-02	4.56E-07	6.51E-03	1.11E-09	1.98E-07	2.84E-03	7.80E-11	4.99E-11	NA
Nitroaromatics									
Dinitrotoluene, 2,6-	9.75E-01	1.12E-05	1.12E-02	1.95E-08	3.49E-06	3.49E-03	1.92E-09	1.23E-09	NA
Nitrotoluene, 2-	7.66E+00	8.81E-05	9.79E-02	NA	NA	NA	1.51E-08	9.64E-09	NA
Nitrotoluene, 3-	1.52E+00	1.75E-05	8.75E-04	NA	NA	NA	2.99E-09	1.91E-09	NA
Semivolatile Organic Compounds									
Benzo(a)anthracene	1.98E-01	2.28E-06	NA	5.15E-09	9.21E-07	NA	3.90E-10	2.49E-10	NA
Benzo(a)pyrene	2.02E-01	2.32E-06	NA	5.25E-09	9.40E-07	NA	3.98E-10	2.54E-10	NA
Benzo(b)fluoranthene	3.12E-01	3.59E-06	NA	8.11E-09	1.45E-06	NA	6.15E-10	3.93E-10	NA
Dinitrotoluene, 2,4-	1.52E+00	1.74E-05	8.72E-03	3.03E-08	5.43E-06	2.71E-03	2.99E-09	1.91E-09	NA
Total HI			**3.01E-01**			**9.04E-03**			**8.30E-03**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram

mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-12

On-Site Child Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	All Pathways Total HI
Inorganics	
Aluminum	**1.13E-01**
Chromium	**9.79E-03**
Vanadium	**6.11E-02**
Polychlorinated biphenyls (PCB)	
Aroclor 1221	**9.34E-03**
Nitroaromatics	
Dinitrotoluene, 2,6-	**1.47E-02**
Nitrotoluene, 2-	**9.79E-02**
Nitrotoluene, 3-	**8.75E-04**
Semivolatile Organic Compounds	
Benzo(a)anthracene	**NA**
Benzo(a)pyrene	**NA**
Benzo(b)fluoranthene	**NA**
Dinitrotoluene, 2,4-	**1.14E-02**
Total HI	**3.18E-01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram

Table D-13

On-Site Adult Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Concentration	Inhalation	
	EPC	Adult Dose	Adult Resident	Dose Absorbed	Adult Dose	Adult Resident	in Air C_a	Adult Dose	Adult Resident
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ
Inorganics									
Aluminum	9.11E+03	1.12E-02	1.12E-02	NA	NA	NA	1.79E-05	4.92E-06	3.51E-03
Chromium	1.77E+01	2.18E-05	1.04E-03	NA	NA	NA	3.48E-08	9.53E-09	4.69E-05
Vanadium	2.65E+01	3.27E-05	6.54E-03	NA	NA	NA	5.23E-08	1.43E-08	NA
Polychlorinated biphenyls (PCB)									
Aroclor 1221	3.96E-02	4.88E-08	6.97E-04	3.88E-10	3.03E-08	4.33E-04	7.80E-11	2.14E-11	NA
Nitroaromatics									
Dinitrotoluene, 2,6-	9.75E-01	1.20E-06	1.20E-03	6.83E-09	5.33E-07	5.33E-04	1.92E-09	5.26E-10	NA
Nitrotoluene, 2-	7.66E+00	9.44E-06	1.05E-02	NA	NA	NA	1.51E-08	4.13E-09	NA
Nitrotoluene, 3-	1.52E+00	1.87E-06	9.37E-05	NA	NA	NA	2.99E-09	8.20E-10	NA
Semivolatile Organic Compounds									
Benzo(a)anthracene	1.98E-01	2.44E-07	NA	1.80E-09	1.41E-07	NA	3.90E-10	1.07E-10	NA
Benzo(a)pyrene	2.02E-01	2.49E-07	NA	1.84E-09	1.44E-07	NA	3.98E-10	1.09E-10	NA
Benzo(b)fluoranthene	3.12E-01	3.85E-07	NA	2.84E-09	2.22E-07	NA	6.15E-10	1.68E-10	NA
Dinitrotoluene, 2,4-	1.52E+00	1.87E-06	9.35E-04	1.06E-08	8.29E-07	4.14E-04	2.99E-09	8.18E-10	NA
Total HI			**3.22E-02**			**1.38E-03**			**3.56E-03**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-13

On-Site Adult Resident, Noncancer Evaluation, Future Scenario, Exposure to Total Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	All Pathways Total HI
Inorganics	
Aluminum	**1.47E-02**
Chromium	**1.08E-03**
Vanadium	**6.54E-03**
Polychlorinated biphenyls (PCB)	
Aroclor 1221	**1.13E-03**
Nitroaromatics	
Dinitrotoluene, 2,6-	**1.73E-03**
Nitrotoluene, 2-	**1.05E-02**
Nitrotoluene, 3-	**9.37E-05**
Semivolatile Organic Compounds	
Benzo(a)anthracene	**NA**
Benzo(a)pyrene	**NA**
Benzo(b)fluoranthene	**NA**
Dinitrotoluene, 2,4-	**1.35E-03**
Total HI	**3.72E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram

Table D-14

On-Site Resident, Future Scenario, Cancer Risk from Exposure to Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					Inhalation of VOCs				All
		Adult	Child		Dose	Adult	Dose	Child		Concentration	Adult	Child		Pathways
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	ILCR	in Air mg/m^3	Dose mg/kg-day	Dose mg/kg-day	ILCR	Total ILCR
Inorganics														
Arsenic	1.31E-01	1.23E-03	7.18E-04	2.92E-03	2.62E-08	2.46E-06	4.36E-08	1.58E-06	6.06E-06	NA	NA	NA	NA	**2.93E-03**
Iron	2.02E+00	1.89E-02	1.10E-02	NA	4.03E-07	3.79E-05	6.71E-07	2.43E-05	NA	NA	NA	NA	NA	**NA**
Manganese	1.07E+00	1.01E-02	5.86E-03	NA	2.14E-07	2.01E-05	3.56E-07	1.29E-05	NA	NA	NA	NA	NA	**NA**
Thallium	6.40E-03	6.01E-05	3.51E-05	NA	1.28E-09	1.20E-07	2.13E-09	7.71E-08	NA	NA	NA	NA	NA	**NA**
Semivolatile Organic Compounds														
Methylnaphthalene, 2-	2.56E-01	2.40E-03	1.40E-03	NA	1.15E-05	1.08E-03	1.48E-05	5.35E-04	NA	1.28E-01	1.20E-02	7.00E-03	NA	**NA**
Naphthalene	4.72E-02	4.44E-04	2.59E-04	NA	1.03E-06	9.64E-05	1.32E-06	4.79E-05	NA	2.36E-02	2.22E-03	1.29E-03	4.21E-04	**4.21E-04**
Volatile Organic Compounds														
Benzene	1.68E+00	1.58E-02	9.21E-03	1.37E-03	8.39E-06	7.88E-04	1.08E-05	3.91E-04	6.49E-05	8.40E-01	7.89E-02	4.60E-02	3.37E-03	**4.81E-03**
Chloroform	5.63E-02	5.29E-04	3.08E-04	NA	1.67E-07	1.57E-05	2.16E-07	7.81E-06	NA	2.82E-02	2.64E-03	1.54E-03	NA	**NA**
Chloromethane	3.34E-02	3.14E-04	1.83E-04	NA	3.04E-08	2.86E-06	3.93E-08	1.42E-06	NA	1.67E-02	1.57E-03	9.13E-04	NA	**NA**
Ethylbenzene	1.69E-01	1.59E-03	9.26E-04	NA	3.32E-06	3.12E-04	4.28E-06	1.55E-04	NA	8.45E-02	7.93E-03	4.62E-03	NA	**NA**
Methylene chloride	6.04E-02	5.67E-04	3.31E-04	6.73E-06	7.38E-08	6.94E-06	9.53E-08	3.45E-06	7.79E-08	3.02E-02	2.83E-03	1.65E-03	7.17E-06	**1.40E-05**
Tetrachloroethane, 1,1,2,2-	3.70E-03	3.48E-05	2.03E-05	1.10E-05	1.52E-08	1.43E-06	1.96E-08	7.10E-07	4.28E-07	1.85E-03	1.74E-04	1.01E-04	5.50E-05	**6.64E-05**
Toluene	6.92E-01	6.50E-03	3.79E-03	NA	7.84E-06	7.37E-04	1.01E-05	3.66E-04	NA	3.46E-01	3.25E-02	1.89E-02	NA	**NA**
Xylenes, total	1.13E+00	1.06E-02	6.19E-03	NA	2.40E-05	2.25E-03	3.10E-05	1.12E-03	NA	5.65E-01	5.31E-02	3.09E-02	NA	**NA**
General Chemistry														
Cyanide, total	1.10E-01	1.03E-03	6.03E-04	NA	2.20E-08	2.07E-06	3.66E-08	1.32E-06	NA	NA	NA	NA	NA	**NA**
Total ILCR				**4.31E-03**					**7.14E-05**				**3.85E-03**	**8.24E-03**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter
NA - Not applicable.

Table D-15

Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			**All**
		Child		Dose	Child		Concentration	Child		**Pathways**
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	HQ	in Air mg/m^3	Dose mg/kg-day	HQ	**Total HI**
Inorganics										
Arsenic	1.31E-01	8.37E-03	2.79E+01	4.36E-08	1.84E-05	6.14E-02	NA	NA	NA	**2.80E+01**
Iron	2.02E+00	1.29E-01	1.84E-01	6.71E-07	2.83E-04	NA	NA	NA	NA	**1.84E-01**
Manganese	1.07E+00	6.84E-02	1.46E+00	3.56E-07	1.50E-04	7.91E-02	NA	NA	NA	**1.53E+00**
Thallium	6.40E-03	4.09E-04	6.29E+00	2.13E-09	8.99E-07	1.38E-02	NA	NA	NA	**6.31E+00**
Semivolatile Organic Compounds										
Methylnaphthalene, 2-	2.56E-01	1.64E-02	4.09E+00	1.48E-05	6.24E-03	1.56E+00	1.28E-01	8.17E-02	NA	**5.65E+00**
Naphthalene	4.72E-02	3.02E-03	1.51E-01	1.32E-06	5.59E-04	2.79E-02	2.36E-02	1.51E-02	1.75E+01	**1.77E+01**
Volatile Organic Compounds										
Benzene	1.68E+00	1.07E-01	2.68E+01	1.08E-05	4.57E-03	1.14E+00	8.40E-01	5.36E-01	6.23E+01	**9.03E+01**
Chloroform	5.63E-02	3.60E-03	3.60E-01	2.16E-07	9.11E-05	9.11E-03	2.82E-02	1.80E-02	1.28E+00	**1.65E+00**
Chloromethane	3.34E-02	2.13E-03	NA	3.93E-08	1.66E-05	NA	1.67E-02	1.07E-02	4.10E-01	**4.10E-01**
Ethylbenzene	1.69E-01	1.08E-02	1.08E-01	4.28E-06	1.81E-03	1.81E-02	8.45E-02	5.39E-02	1.86E-01	**3.12E-01**
Methylene chloride	6.04E-02	3.86E-03	6.43E-02	9.53E-08	4.02E-05	6.70E-04	3.02E-02	1.93E-02	6.42E-02	**1.29E-01**
Tetrachloroethane, 1,1,2,2-	3.70E-03	2.37E-04	5.91E-02	1.96E-08	8.28E-06	2.07E-03	1.85E-03	1.18E-03	NA	**6.12E-02**
Toluene	6.92E-01	4.42E-02	5.53E-01	1.01E-05	4.27E-03	5.34E-02	3.46E-01	2.21E-01	1.58E-01	**7.64E-01**
Xylenes, total	1.13E+00	7.22E-02	3.61E-01	3.10E-05	1.31E-02	6.53E-02	5.65E-01	3.61E-01	1.24E+01	**1.29E+01**
General Chemistry										
Cyanide, total	1.10E-01	7.03E-03	3.52E-01	3.66E-08	1.55E-05	7.73E-04	NA	NA	NA	**3.52E-01**
Total HI			**6.88E+01**			**3.03E+00**			**9.44E+01**	**1.66E+02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter

mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter
NA - Not applicable.

Table D-16

Adult On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Bedrock Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			All
		Noncancer		Dose	Adult		Concentration	Adult		Pathways
	EPC	Dose		Absorbed	Dose		in Air	Dose		Total
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	HI
Inorganics										
Arsenic	1.31E-01	3.59E-03	1.20E+01	2.62E-08	7.18E-06	2.39E-02	NA	NA	NA	**1.20E+01**
Iron	2.02E+00	5.52E-02	7.89E-02	4.03E-07	1.10E-04	NA	NA	NA	NA	**7.89E-02**
Manganese	1.07E+00	2.93E-02	6.24E-01	2.14E-07	5.86E-05	3.09E-02	NA	NA	NA	**6.55E-01**
Thallium	6.40E-03	1.75E-04	2.70E+00	1.28E-09	3.51E-07	5.40E-03	NA	NA	NA	**2.70E+00**
Semivolatile Organic Compounds										
Methylnaphthalene, 2-	2.56E-01	7.01E-03	1.75E+00	1.15E-05	3.14E-03	7.85E-01	1.28E-01	3.51E-02	NA	**2.54E+00**
Naphthalene	4.72E-02	1.29E-03	6.47E-02	1.03E-06	2.81E-04	1.41E-02	2.36E-02	6.47E-03	7.52E+00	**7.60E+00**
Volatile Organic Compounds										
Benzene	1.68E+00	4.60E-02	1.15E+01	8.39E-06	2.30E-03	5.74E-01	8.40E-01	2.30E-01	2.67E+01	**3.88E+01**
Chloroform	5.63E-02	1.54E-03	1.54E-01	1.67E-07	4.58E-05	4.58E-03	2.82E-02	7.71E-03	5.51E-01	**7.09E-01**
Chloromethane	3.34E-02	9.15E-04	NA	3.04E-08	8.34E-06	NA	1.67E-02	4.57E-03	1.76E-01	**1.76E-01**
Ethylbenzene	1.69E-01	4.63E-03	4.63E-02	3.32E-06	9.09E-04	9.09E-03	8.45E-02	2.31E-02	7.98E-02	**1.35E-01**
Methylene chloride	6.04E-02	1.65E-03	2.76E-02	7.38E-08	2.02E-05	3.37E-04	3.02E-02	8.26E-03	2.75E-02	**5.54E-02**
Tetrachloroethane, 1,1,2,2-	3.70E-03	1.01E-04	2.53E-02	1.52E-08	4.17E-06	1.04E-03	1.85E-03	5.07E-04	NA	**2.64E-02**
Toluene	6.92E-01	1.90E-02	2.37E-01	7.84E-06	2.15E-03	2.69E-02	3.46E-01	9.48E-02	6.77E-02	**3.32E-01**
Xylenes, total	1.13E+00	3.10E-02	1.55E-01	2.40E-05	6.57E-03	3.29E-02	5.65E-01	1.55E-01	5.34E+00	**5.52E+00**
General Chemistry										
Cyanide, total	1.10E-01	3.01E-03	1.51E-01	2.20E-08	6.03E-06	3.01E-04	5.50E-02	6.28E-04	NA	**1.51E-01**
Total HI			**2.95E+01**			**1.51E+00**			**4.05E+01**	**7.15E+01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter
NA - Not applicable.

Table D-17

On-Site Resident Cancer Risk, Future Scenario, Exposure to Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					Inhalation of VOCs				**All**
		Adult	Child		Dose	Adult	Dose	Child		Concentration	Adult	Child		**Pathways**
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	Dose mg/kg-day	ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	ILCR	in Air mg/m^3	Dose mg/kg-day	Dose mg/kg-day	ILCR	**Total ILCR**
Inorganics														
Chromium, Total	3.30E-03	3.10E-05	1.81E-05	NA	6.60E-10	6.20E-08	1.10E-09	3.97E-08	NA	NA	NA	NA	NA	**NA**
Cobalt	6.07E-03	5.70E-05	3.32E-05	NA	4.85E-10	4.56E-08	8.08E-10	2.92E-08	NA	NA	NA	NA	NA	**NA**
Iron	1.48E+00	1.39E-02	8.09E-03	NA	2.95E-07	2.77E-05	4.92E-07	1.78E-05	NA	NA	NA	NA	NA	**NA**
Manganese	3.29E-01	3.09E-03	1.80E-03	NA	6.57E-08	6.18E-06	1.09E-07	3.96E-06	NA	NA	NA	NA	NA	**NA**
Nitroaromatics														
Dinitrotoluene, 2,4-	4.67E-03	4.39E-05	2.56E-05	4.73E-05	1.90E-08	1.78E-06	2.45E-08	8.84E-07	1.81E-06	2.34E-03	2.19E-04	1.28E-04	NA	**4.91E-05**
Dinitrotoluene, 2,6-	3.99E-03	3.75E-05	2.19E-05	4.03E-05	1.10E-08	1.03E-06	1.41E-08	5.11E-07	1.05E-06	1.99E-03	1.87E-04	1.09E-04	NA	**4.14E-05**
Nitrotoluene, 2-	3.77E-02	3.54E-04	2.07E-04	1.23E-04	1.64E-07	1.54E-05	2.11E-07	7.63E-06	5.06E-06	1.89E-02	1.77E-03	1.03E-03	NA	**1.28E-04**
Nitrotoluene, 3-	4.59E-03	4.32E-05	2.52E-05	NA	5.01E-08	4.70E-06	6.46E-08	2.34E-06	NA	2.30E-03	2.16E-04	1.26E-04	NA	**NA**
Nitrotoluene, 4-	4.40E-03	4.13E-05	2.41E-05	1.05E-06	4.24E-08	3.98E-06	5.47E-08	1.98E-06	9.54E-08	2.20E-03	2.06E-04	1.20E-04	NA	**1.14E-06**
Volatile Organic Compounds														
Carbon tetrachloride	2.38E-03	2.23E-05	1.30E-05	2.47E-06	2.08E-08	1.95E-06	2.68E-08	9.68E-07	2.04E-07	1.19E-03	1.12E-04	6.50E-05	3.71E-06	**6.39E-06**
Dibromochloromethane	2.00E-04	1.88E-06	1.10E-06	2.50E-07	9.91E-10	9.31E-08	1.28E-09	4.63E-08	1.17E-08	1.00E-04	9.39E-06	5.47E-06	NA	**2.62E-07**
Dichloroethane, 1,1-	4.44E-03	4.17E-05	2.43E-05	NA	1.13E-08	1.06E-06	1.46E-08	5.28E-07	NA	2.22E-03	2.08E-04	1.21E-04	NA	**NA**
Trichloroethene	4.84E-03	4.55E-05	2.65E-05	4.25E-07	2.74E-08	2.57E-06	3.53E-08	1.28E-06	2.27E-08	2.42E-03	2.27E-04	1.32E-04	2.52E-06	**2.97E-06**
Total ILCR				**2.15E-04**					**8.25E-06**				**6.23E-06**	**2.30E-04**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-18

Child On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			**All**
		Child		Dose	Child		Concentration	Child		**Pathways**
	EPC	Dose		Absorbed	Dose		in Air	Dose		**Total**
Chemicals of Potential Concern	mg/L	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	mg/m^3	mg/kg-day	HQ	**HI**
Inorganics										
Chromium, Total	3.30E-03	2.11E-04	1.00E-02	1.10E-09	4.64E-07	1.72E-03	NA	NA	NA	**1.18E-02**
Cobalt	6.07E-03	3.88E-04	1.29E+00	8.08E-10	3.41E-07	1.14E-03	NA	NA	NA	**1.29E+00**
Iron	1.48E+00	9.44E-02	1.35E-01	4.92E-07	2.08E-04	NA	NA	NA	NA	**1.35E-01**
Manganese	3.29E-01	2.10E-02	4.47E-01	1.09E-07	4.62E-05	2.43E-02	NA	NA	NA	**4.71E-01**
Nitroaromatics										
Dinitrotoluene, 2,4-	4.67E-03	2.99E-04	1.49E-01	2.45E-08	1.03E-05	5.16E-03	2.34E-03	1.49E-03	NA	**1.55E-01**
Dinitrotoluene, 2,6-	3.99E-03	2.55E-04	2.55E-01	1.41E-08	5.96E-06	5.96E-03	1.99E-03	1.27E-03	NA	**2.61E-01**
Nitrotoluene, 2-	3.77E-02	2.41E-03	2.68E+00	2.11E-07	8.90E-05	9.89E-02	1.89E-02	1.20E-02	NA	**2.78E+00**
Nitrotoluene, 3-	4.59E-03	2.94E-04	1.47E-02	6.46E-08	2.73E-05	1.36E-03	2.30E-03	1.47E-03	NA	**1.60E-02**
Nitrotoluene, 4-	4.40E-03	2.81E-04	7.03E-02	5.47E-08	2.31E-05	5.77E-03	2.20E-03	1.40E-03	NA	**7.60E-02**
Volatile Organic Compounds										
Carbon tetrachloride	2.38E-03	1.52E-04	3.80E-02	2.68E-08	1.13E-05	2.82E-03	1.19E-03	7.58E-04	2.61E-02	**6.69E-02**
Dibromochloromethane	2.00E-04	1.28E-05	6.39E-04	1.28E-09	5.40E-07	2.70E-05	1.00E-04	6.38E-05	NA	**6.66E-04**
Dichloroethane, 1,1-	4.44E-03	2.84E-04	1.42E-03	1.46E-08	6.16E-06	3.08E-05	2.22E-03	1.42E-03	NA	**1.45E-03**
Trichloroethene	4.84E-03	3.10E-04	6.19E-04	3.53E-08	1.49E-05	2.98E-05	2.42E-03	1.55E-03	NA	**6.49E-04**
Total HI			**5.09E+00**			**1.47E-01**			**2.61E-02**	**5.27E+00**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-19

Adult On-Site Resident, Future Scenario, Noncancer Hazard from Exposure to Overburden Groundwater
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			Inhalation of VOCs			**All**
		Noncancer		Dose	Adult		Concentration	Adult		**Pathways**
Chemicals of Potential Concern	EPC mg/L	Dose mg/kg-day	HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	HQ	in Air mg/m^3	Dose mg/kg-day	HQ	**Total HI**
Inorganics										
Chromium, Total	3.30E-03	9.04E-05	4.31E-03	6.60E-10	1.81E-07	6.70E-04	NA	NA	NA	**4.97E-03**
Cobalt	6.07E-03	1.66E-04	5.54E-01	4.85E-10	1.33E-07	4.43E-04	NA	NA	NA	**5.55E-01**
Iron	1.48E+00	4.05E-02	5.78E-02	2.95E-07	8.09E-05	NA	NA	NA	NA	**5.78E-02**
Manganese	3.29E-01	9.01E-03	1.92E-01	6.57E-08	1.80E-05	9.48E-03	NA	NA	NA	**2.01E-01**
Nitroaromatics										
Dinitrotoluene, 2,4-	4.67E-03	1.28E-04	6.40E-02	1.90E-08	5.19E-06	2.60E-03	2.34E-03	6.40E-04	NA	**6.66E-02**
Dinitrotoluene, 2,6-	3.99E-03	1.09E-04	1.09E-01	1.10E-08	3.00E-06	3.00E-03	1.99E-03	5.46E-04	NA	**1.12E-01**
Nitrotoluene, 2-	3.77E-02	1.03E-03	1.15E+00	1.64E-07	4.48E-05	4.98E-02	1.89E-02	5.17E-03	NA	**1.20E+00**
Nitrotoluene, 3-	4.59E-03	1.26E-04	6.29E-03	5.01E-08	1.37E-05	6.86E-04	2.30E-03	6.29E-04	NA	**6.98E-03**
Nitrotoluene, 4-	4.40E-03	1.20E-04	3.01E-02	4.24E-08	1.16E-05	2.90E-03	2.20E-03	6.02E-04	NA	**3.30E-02**
Volatile Organic Compounds										
Carbon tetrachloride	2.38E-03	6.51E-05	1.63E-02	2.08E-08	5.69E-06	1.42E-03	1.19E-03	3.25E-04	1.12E-02	**2.89E-02**
Dibromochloromethane	2.00E-04	5.48E-06	2.74E-04	9.91E-10	2.72E-07	1.36E-05	1.00E-04	2.74E-05	NA	**2.88E-04**
Dichloroethane, 1,1-	4.44E-03	1.22E-04	6.08E-04	1.13E-08	3.10E-06	1.55E-05	2.22E-03	6.07E-04	NA	**6.23E-04**
Trichloroethene	4.84E-03	1.33E-04	2.65E-04	2.74E-08	7.50E-06	1.50E-05	2.42E-03	6.63E-04	NA	**2.80E-04**
Total HI			**2.18E+00**			**7.10E-02**			**1.12E-02**	**2.27E+00**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
mg/m^3- milligram per cubic meter

Table D-20

On-Site Resident, Future Scenario, Exposure to Sediment Cancer Risk
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion			Dermal Contact					All
		Adult	Child		Dose	Adult	Dose	Child		Pathways
Chemicals of Potential Concern	EPC mg/kg	Dose mg/kg-day	Dose mg/kg-day	Resident ILCR	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	Resident ILCR	**Total ILCR**
Inorganics										
Arsenic	1.52E+01	7.16E-07	1.67E-06	3.58E-06	3.20E-09	1.27E-08	9.14E-09	2.08E-08	5.04E-08	**3.63E-06**
Chromium, total	1.62E+01	7.59E-07	1.77E-06	NA	NA	NA	NA	NA	NA	**NA**
Cobalt	2.28E+01	1.07E-06	2.50E-06	NA	NA	NA	NA	NA	NA	**NA**
Manganese	3.98E+03	1.87E-04	4.36E-04	NA	NA	NA	NA	NA	NA	**NA**
Thallium	1.90E+00	8.92E-08	2.08E-07	NA	NA	NA	NA	NA	NA	**NA**
Total ILCR				**3.58E-06**					**5.04E-08**	**3.63E-06**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-21

On-Site Child Resident, Future Scenario, Exposure to Sediment Nonancer Risk
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			All
		Child		Dose	Child		Pathways
Chemicals of Potential Concern	EPC mg/kg	Dose mg/kg-day	Child HQ	Absorbed mg/cm^2-day	Dose mg/kg-day	Child HQ	**Total HI**
Inorganics							
Arsenic	1.52E+01	1.95E-05	6.49E-02	9.14E-09	2.43E-07	8.11E-04	**6.58E-02**
Chromium, total	1.62E+01	2.06E-05	9.83E-04	NA	NA	NA	**9.83E-04**
Cobalt	2.28E+01	2.91E-05	9.71E-02	NA	NA	NA	**9.71E-02**
Manganese	3.98E+03	5.09E-03	1.08E-01	NA	NA	NA	**1.08E-01**
Thallium	1.90E+00	2.43E-06	3.74E-02	NA	NA	NA	**3.74E-02**
Total HI			**3.09E-01**			**8.11E-04**	**3.09E-01**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-22

On-Site Adult Resident, Future Scenario, Exposure to Sediment Nonancer Risk
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion		Dermal Contact			All
		Adult		Dose	Adult		Pathways
	EPC	Dose	Adult	Absorbed	Dose	Adult	Total
Chemicals of Potential Concern	mg/kg	mg/kg-day	HQ	mg/cm^2-day	mg/kg-day	HQ	HI
Inorganics							
Arsenic	1.52E+01	2.09E-06	6.96E-03	3.20E-09	3.71E-08	1.24E-04	**7.08E-03**
Chromium, total	1.62E+01	2.21E-06	1.05E-04	NA	NA	NA	**1.05E-04**
Cobalt	2.28E+01	3.12E-06	1.04E-02	NA	NA	NA	**1.04E-02**
Manganese	3.98E+03	5.45E-04	1.16E-02	NA	NA	NA	**1.16E-02**
Thallium	1.90E+00	2.60E-07	4.00E-03	NA	NA	NA	**4.00E-03**
Total HI			**3.31E-02**			**1.24E-04**	**3.32E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/kg - milligram per kilogram
mg/cm^2 - milligram per centimeter squared

Table D-23

On-Site Resident Cancer Risk, Future Scenario, Exposure to Surface Water
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Dermal Contact				
		Adult Dose	Adult	Child Dose	Child	
Chemicals of Potential Concern	EPC mg/L	Absorbed mg/cm^2-day	Dose mg/kg-day	Absorbed mg/cm^2-day	Dose mg/kg-day	**Total ILCR**
Inorganics						
Arsenic	6.69E-03	2.01E-08	9.80E-08	2.01E-08	4.57E-08	2.16E-07
Chromium	3.16E-02	9.49E-08	4.64E-07	9.49E-08	2.16E-07	NA
Cobalt	1.63E-02	1.96E-08	9.55E-08	1.96E-08	4.46E-08	NA
Iron	2.28E+01	6.84E-05	3.34E-04	6.84E-05	1.56E-04	NA
Manganese	1.71E+00	5.14E-06	2.51E-05	5.14E-06	1.17E-05	NA
Total ILCR						**2.16E-07**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-24

Child On-Site Resident, Noncancer Risk, Future Scenario, Exposure to Surface Water
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Dermal Contact		
Chemicals of Potential Concern	EPC mg/L	Child Dose Absorbed mg/cm^2-day	Child Dose mg/kg-day	HQ
Inorganics				
Arsenic	6.69E-03	2.01E-08	5.34E-07	1.78E-03
Chromium	3.16E-02	9.49E-08	2.52E-06	9.35E-03
Cobalt	1.63E-02	1.96E-08	5.20E-07	1.73E-03
Iron	2.28E+01	6.84E-05	1.82E-03	NA
Manganese	1.71E+00	5.14E-06	1.37E-04	7.19E-02
Total HI				**8.48E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-25

Adult On-Site Resident, Noncancer Risk, Future Scenario, Exposure to Surface Water
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Dermal Contact		
Chemicals of Potential Concern	EPC mg/L	Adult Dose Absorbed mg/cm^2-day	Adult Dose mg/kg-day	HQ
Inorganics				
Arsenic	6.69E-03	2.01E-08	2.86E-07	9.53E-04
Chromium	3.16E-02	9.49E-08	1.35E-06	5.01E-03
Cobalt	1.63E-02	1.96E-08	2.79E-07	9.29E-04
Iron	2.28E+01	6.84E-05	9.74E-04	NA
Manganese	1.71E+00	5.14E-06	7.32E-05	3.85E-02
Total HI				**4.54E-02**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
mg/L - milligram per liter
mg/cm^2 - milligram per square meter
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-26

Adult Hunter, Future Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact				
	EPC	Cancer Dose	Noncancer Dose	Adult Hunter	Adult Hunter	Dose Absorbed	Cancer Dose	Noncancer Dose	Adult Hunter	Adult Hunter
Chemicals of Potential Concern	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	mg/cm^2-day	mg/kg-day	mg/kg-day	ILCR	HQ
Inorganics										
Chromium	2.24E+01	5.26E-07	1.23E-06	NA	5.84E-05	NA	NA	NA	NA	NA
Polychlorinated biphenyls (PCB)										
Aroclor 1221	1.03E-01	2.42E-09	5.64E-09	4.84E-09	8.06E-05	2.88E-09	2.23E-09	5.21E-09	4.47E-09	7.45E-05
Semivolatile Organic Compounds										
Benzo(a)anthracene	2.92E-01	6.86E-09	1.60E-08	5.01E-09	NA	7.59E-09	5.88E-09	1.37E-08	4.29E-09	NA
Benzo(a)pyrene	3.41E-01	8.01E-09	1.87E-08	5.85E-08	NA	8.87E-09	6.87E-09	1.60E-08	5.02E-08	NA
Benzo(b)fluoranthene	7.42E-01	1.74E-08	4.07E-08	1.27E-08	NA	1.93E-08	1.50E-08	3.49E-08	1.09E-08	NA
Dinitrotoluene, 2,4-	1.44E+00	3.38E-08	7.88E-08	2.30E-08	3.94E-05	2.88E-08	2.23E-08	5.20E-08	1.52E-08	2.60E-05
Total ILCR and HI				**1.04E-07**	**1.78E-04**				**8.50E-08**	**1.01E-04**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
NA - Not applicable.

Table D-26

Adult Hunter, Future Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

	Concentration in Forage C_p	Concentration in Venison C_v	Consumption of Venison				All Pathways	
			Cancer Dose	Noncancer Dose			Total	Total
Chemicals of Potential Concern	mg/kg	mg/kg	mg/kg-day	mg/kg-day	ILCR	HQ	ILCR	HI
Inorganics								
Chromium	NA	NA	NA	NA	NA	NA	**NA**	**5.84E-05**
Polychlorinated biphenyls (PCB)								
Aroclor 1221	9.58E-03	6.29E-08	4.80E-12	1.12E-11	9.60E-12	1.60E-07	**9.32E-09**	**1.55E-04**
Semivolatile Organic Compounds								
Benzo(a)anthracene	NA	NA	NA	NA	NA	NA	**9.30E-09**	**NA**
Benzo(a)pyrene	NA	NA	NA	NA	NA	NA	**1.09E-07**	**NA**
Benzo(b)fluoranthene	NA	NA	NA	NA	NA	NA	**2.36E-08**	**NA**
Dinitrotoluene, 2,4-	NA	NA	NA	NA	NA	NA	**3.81E-08**	**6.54E-05**
Total ILCR and HI					**9.60E-12**	**1.60E-07**	**1.89E-07**	**2.79E-04**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
mg/cm^2 - milligram per square meter
NA - Not applicable.

Table D-27

Hunter's Child, Future Scenario, Exposure to Surface Soil
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	EPC mg/kg	Concentration in Forage C_p mg/kg	Concentration in Venison C_v mg/kg	Consumption of Venison: Cancer Dose mg/kg-day	Consumption of Venison: Noncancer Dose mg/kg-day	ILCR	HQ	**Total ILCR**	**Total HI**
Inorganics									
Chromium	2.24E+01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Polychlorinated biphenyls (PCB)									
Aroclor 1221	1.03E-01	9.58E-03	6.29E-08	1.72E-12	2.01E-11	3.45E-12	2.87E-07	**3.45E-12**	**2.87E-07**
Semivolatile Organic Compounds									
Benzo(a)anthracene	2.92E-01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Benzo(a)pyrene	3.41E-01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Benzo(b)fluoranthene	7.42E-01	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Dinitrotoluene, 2,4-	1.44E+00	NA	NA	NA	NA	NA	NA	**NA**	**NA**
Total ILCR and HI						**3.45E-12**	**2.87E-07**	**3.45E-12**	**2.87E-07**

EPC - Exposure Point Concentration
HQ - Hazard Quotient; HI - Hazard Index
ILCR - Incremental Lifetime Cancer Risk
mg/kg - milligram per kilogram
NA - Not applicable.

Table D-28

Construction Worker, Current and Future Scenarios, Exposure to Maintenance Pit Material
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

		Incidental Ingestion				Dermal Contact				
Chemicals of Potential Concern	EPC mg/kg	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ	Dose Absorbed mg/cm^2-day	Cancer Dose mg/kg-day	Noncancer Dose mg/kg-day	ILCR	HQ
Semivolatile Organic Compounds										
Benzo(a)anthracene	6.51E-01	1.50E-08	2.10E-06	1.10E-08	NA	2.54E-08	5.86E-09	8.18E-07	4.27E-09	NA
Benzo(a)pyrene	4.98E-01	1.15E-08	1.60E-06	8.38E-08	NA	1.94E-08	4.48E-09	6.25E-07	3.27E-08	NA
Benzo(b)fluoranthene	1.01E+00	2.33E-08	3.25E-06	1.70E-08	NA	3.94E-08	9.08E-09	1.27E-06	6.63E-09	NA
Total ILCR or HI				**1.12E-07**	**NA**				**4.36E-08**	**NA**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index

mg/kg - milligram per kilogram
mg/m^3 - milligram per cubic meter
NA - Not applicable.

Table D-28

Construction Worker, Current and Future Scenarios, Exposure to Maintenance Pit Material
Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

Chemicals of Potential Concern	Concentration in Air C_a mg/m^3	Inhalation Cancer Dose mg/kg-day	Inhalation Noncancer Dose mg/kg-day	Inhalation ILCR	Inhalation HQ	**All Pathways Total ILCR**	**All Pathways Total HI**
Semivolatile Organic Compounds							
Benzo(a)anthracene	2.28E-07	3.18E-10	4.45E-08	9.87E-11	NA	**1.53E-08**	**NA**
Benzo(a)pyrene	1.74E-07	2.44E-10	3.40E-08	7.55E-10	NA	**1.17E-07**	**NA**
Benzo(b)fluoranthene	3.54E-07	4.94E-10	6.90E-08	1.53E-10	NA	**2.38E-08**	**NA**
Total ILCR or HI				**1.01E-09**	**NA**	**1.56E-07**	**NA**

EPC - Exposure Point Concentration
ILCR - Incremental Lifetime Cancer Risk
HQ - Hazard Quotient; HI - Hazard Index

APPENDIX E

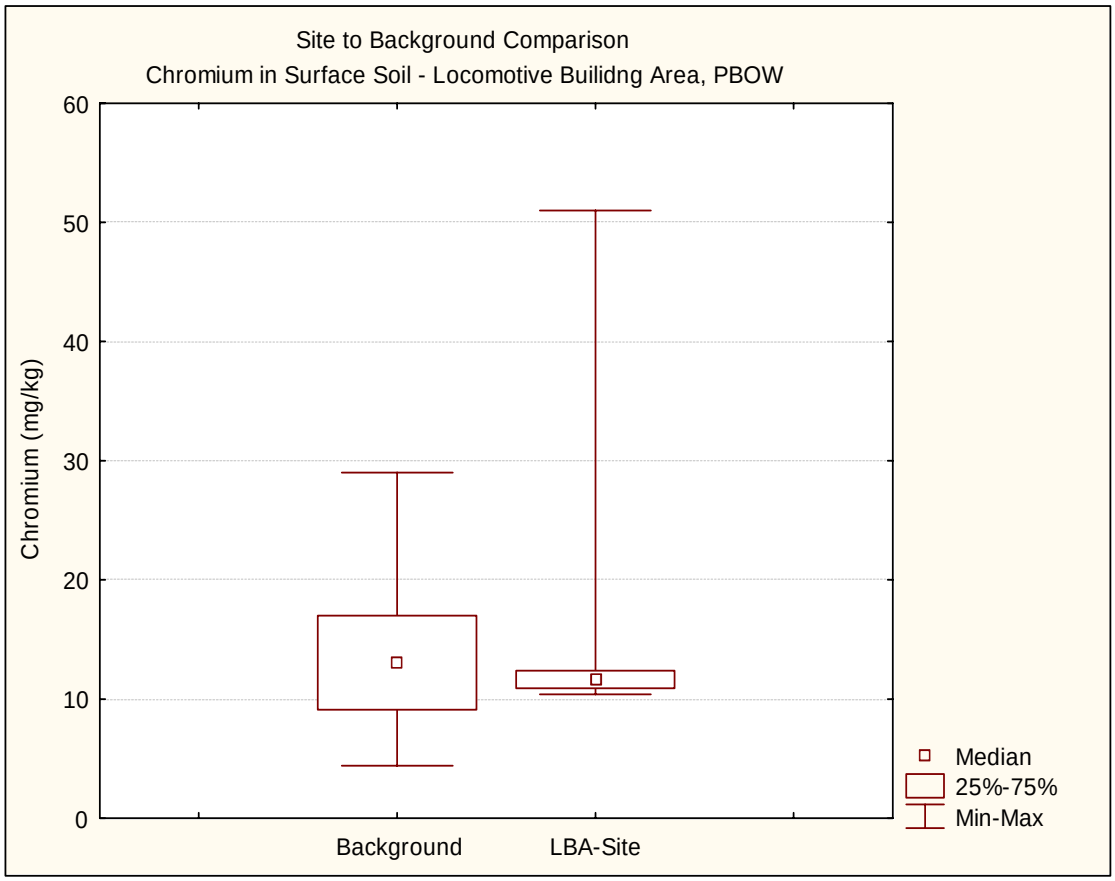
STATISTICAL TESTING FOR BACKGROUND-RELATED INORGANIC CHEMICALS OF POTENTIAL CONCERN

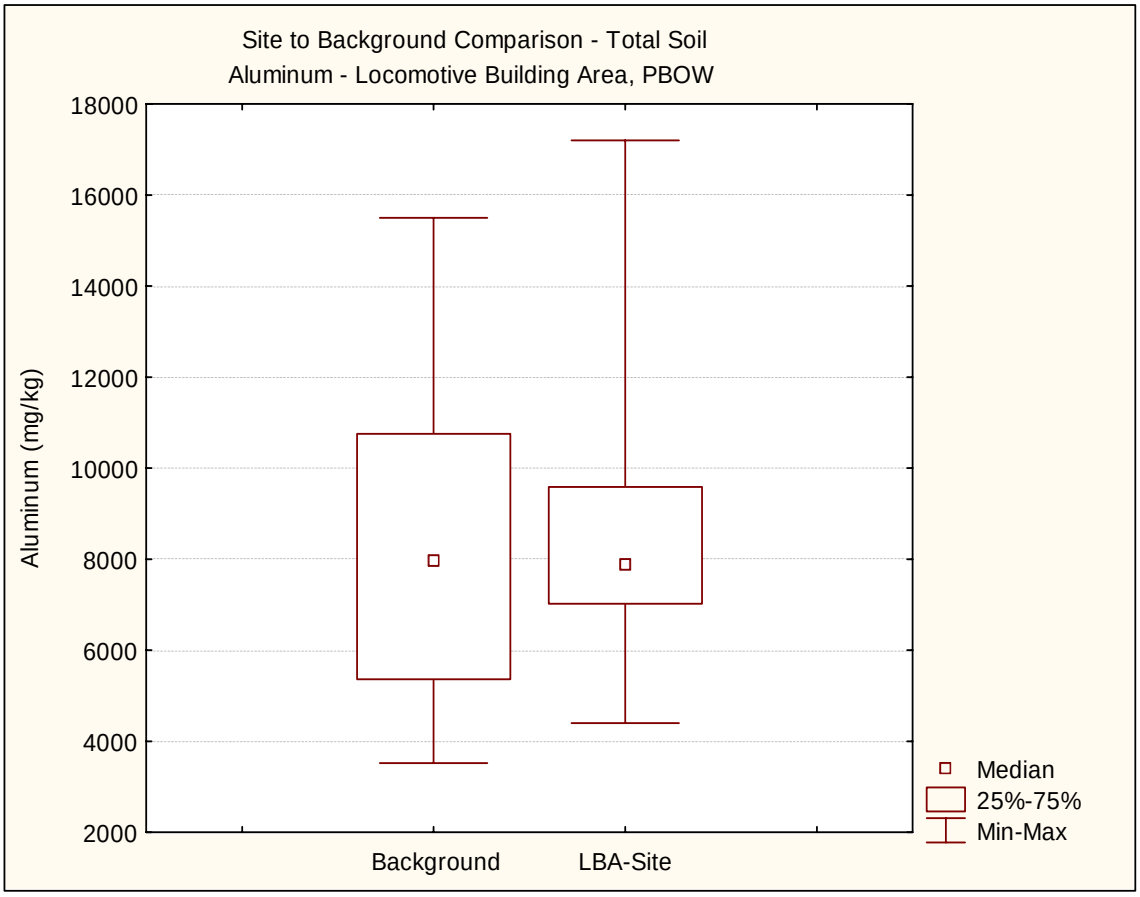
Appendix E

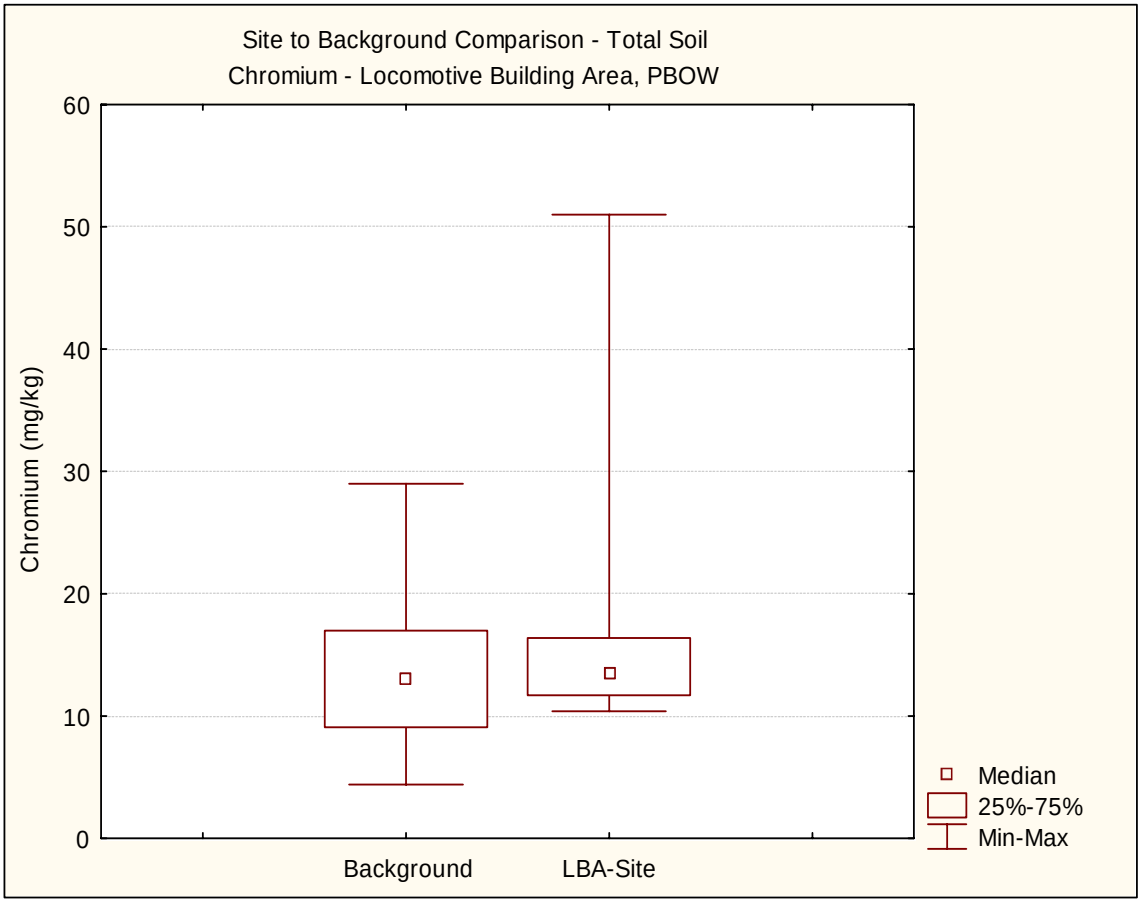
Wilcoxon Ranks Sum Tests for Locomotive Building Area
Plum Brook Ordnance Works, Sandusky, Ohio

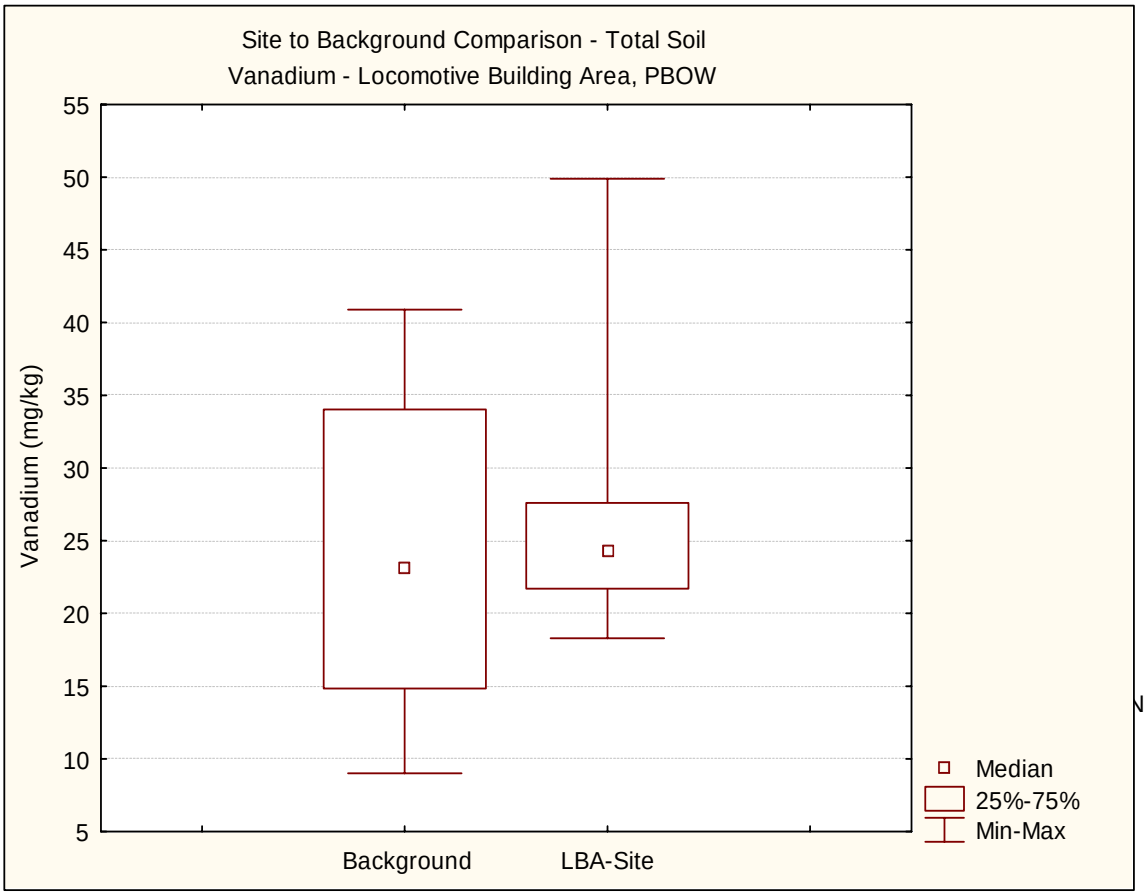
	Rank Sum	Rank Sum	U	Z	p-level	Z	p-level	Valid N	Valid N	2*1sided
Surface Soil COPCs										
Chromium	494.0000	209.0000	143.0000	0.00000	1.000000	0.00000	1.000000	26	11	1.013050
Total Soil COPCs										
Aluminum	290.5000	934.500	212.5000	-0.22087	0.825193	-0.22088	0.825185	12	37	0.827500
Chromium	731.5000	1284.500	380.5000	-1.40307	0.160597	-1.40344	0.160487	26	37	0.161698
Vanadium	286.0000	939.000	208.0000	-0.32549	0.744808	-0.32556	0.744758	12	37	0.756769
Groundwater - Overburden COPCs										
Iron	511.0000	230.0000	105.0000	-1.16024	0.245953	-1.16215	0.245176	28	10	0.257358

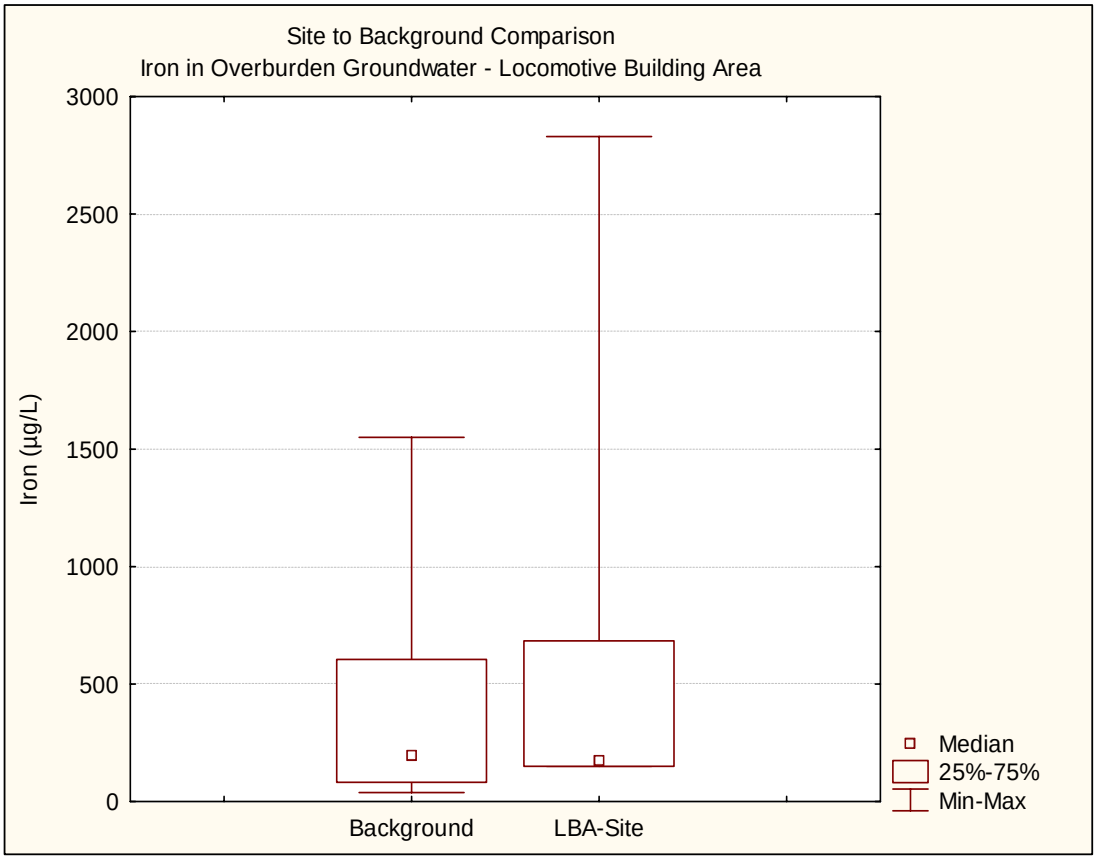
Note: None of the data sets fail the WRS test at p=0.05. Thus, none of the data sets are shown to be signicantly different from the PBOW background data sets.


Site to Background Comparison
Chromium in Surface Soil - Locomotive Builidng Area, PBOW
Chromium (mg/kg)
60
50
40
30
20
10
0
Background
LBA-Site
Median
25%-75%
Min-Max


Site to Background Comparison - Total Soil
Aluminum - Locomotive Building Area, PBOW
Aluminum (mg/kg)
18000
16000
14000
12000
10000
8000
6000
4000
2000
Background
LBA-Site
Median
25%-75%
Min-Max


Site to Background Comparison - Total Soil
Chromium - Locomotive Building Area, PBOW
Chromium (mg/kg)
60
50
40
30
20
10
0
Background
LBA-Site
Median
25%-75%
Min-Max


Site to Background Comparison - Total Soil
Vanadium - Locomotive Building Area, PBOW
Vanadium (mg/kg)
55
50
45
40
35
30
25
20
15
10
5
Background
LBA-Site
Median
25%-75%
Min-Max


Site to Background Comparison
Iron in Overburden Groundwater - Locomotive Building Area
Iron (µg/L)
3000
2500
2000
1500
1000
500
0
Background
LBA-Site
Median
25%-75%
Min-Max

RESPONSE TO COMMENTS

Response to Internal Comments
Baseline Human Health Risk Assessment (BHHRA)
for the Locomotive Building Area (Garage Maintenance Area)
Plum Brook Ordnance Works, Sandusky, Ohio,
Dated September 28, 2010

Comments by Mr. Larry Tannenbaum, Environmental Health Risk Assessment Program, U.S. Army Public Health Command, received November 29, 2010.

Comment 1: **General Comment**. We find that there is a fundamental flaw to the BHHRA effort. The Executive Summary makes it very clear that there is no completed groundwater exposure pathway. Further the reasons given for this (e.g., insufficient yield, etc.) are classic, in the sense that they are often provided in risk assessment guidance as examples of situations where the need for assessment is contra-indicated. In brief, there was no need to screen detected chemicals in groundwater or to compute risks and hazards from groundwater exposure. We are concerned about others who may see this document, and who may (wrongly) conclude that there is purpose or value in assessing groundwater exposure in the conventional manner when it is known *a priori* that the groundwater will never be used. In the document revision, please ensure that all references to groundwater exposure are removed as this medium is not of concern for humans affiliated with the LBA in any way.

Response 1: It is agreed in principle that the evaluation of incomplete exposure pathways for risk is unnecessary. However, the OEPA has maintained the position that all groundwater should be evaluated for tap water use unless the PDT decides on a on a case-by-case basis to formally eliminate quantitative evaluation of groundwater at a given site during a Team meeting. OEPA PDT members have expressed that it would consider eliminating the evaluation of groundwater at the risk assessment stage only on the basis of insufficient yield and not for reasons of naturally poor groundwater quality. OEPA PDT members have stated that at least some of the water underlying PBOW is not fit for potable use, but that the State requires that groundwater be evaluated for exposure and risk. The reason for the State's position is apparently related its groundwater policy and decision process for determination of potability. It is also noted that for PBOW, the determination of site relatedness versus background relatedness is performed at the risk assessment step. OEPA PDT members have also expressed a desire that this evaluation be performed so that a description can be provided to the public Restoration Advisory Board (RAB) illustrating the relative hypothetical risk, assuming potable use, of site-related versus background-related impacts. OEPA PDT members have also stated that risks associated with groundwater use should be evaluated so that remediation to drinking water quality (with respect to site-related contamination) may be included in the Feasibility Study. Given the generally higher hypothetical risks associated with background components, this hypothetical risk evaluation of groundwater may help to portray an eventual selected remedial alternative that does not require active groundwater remediation as more acceptable to both the State and the RAB. Also, the OEPA PDT members

have suggested that the relatively large expense of remediating groundwater may provide a potential basis for not selecting active remediation of groundwater.

It is also noted that the case-by-case decision process for determining whether groundwater should be evaluated for risk would likely take considerable time and would accordingly delay the project schedule. Therefore, to maintain the project schedules and to more transparently and consisently portray to the State and public that the background constituents generally dominate the hypothetical risks associated with groundwater, the USACE has elected to evaluate all groundwater that can be sampled from a monitoring well for risk, and then to qualitatively dismiss that pathway in the conclusions. It is noted that DOD-related nitroaromatic chemicals dominate the hypothetical risks associated with overburden groundwater, but that this pathway is eliminated from further consideration on the basis of low yield.

The BHHRA was also reviewed by Terry Walker, Risk Assessor, USACE Environmental and Munitions Center of Expertise, who had no comments as communicated October 26, 2010.