155 Cedar Lake Drive
Wentzville, Missouri 63385

GROUNDWATER HUMAN HEALTH RISK ASSESSMENT

FORMER YORK NAVAL ORDNANCE PLANT

1425 EDEN ROAD

YORK, PA 17402

21 NOVEMBER 2016

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List of Acronyms

111TCA	1,1,1-Trichloroethane
11DCE	1,1-Dichloroethene
cis-12DCE	*cis*-1,2-Dichloroethene
124TMB	1,2,4-Trimethylbenzene
135TMB	1,3,5-Trimethylbenzene
AMF	American Machine & Foundry Company
ARA	Alliance for Risk Assessment
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
bgs	Below ground surface
BSRA	Bunker and Shell Range Area
COPC	Chemical of potential concern
CTE	Central tendency exposure
EI	Environmental Indicator
EPA	U.S. Environmental Protection Agency
EPC	Exposure point concentration
EPM	Exposure pathway model
fYNOP	Former York Naval Ordnance Plant
GSC	Groundwater Sciences Corporation
GWTS	Groundwater treatment system
ID	Interim Deliverable
ILCR	Incremental lifetime cancer risk
HHRA	Human health risk assessment
HI	Hazard Index
HQ	Hazard Quotient
KM	Kaplan Meier
LNT	Linear non-threshold
LUA	Land Use Area
MCL	Maximum Contaminant Level
MSC	Medium-Specific Concentration
NETT	North End Test Track
NPBA	Northern Property Boundary Area
NRC	National Research Council
NTP	National Toxicology Program
PADEP	Pennsylvania Department of Environmental Protection
PCE	Tetrachloroethene

List of Acronyms, cont'd.

POD	Point of departure
PPRTV	Provisional Peer-Reviewed Toxicity Value
RBSL	Risk-based screening level
RME	Reasonable maximum exposure
RSL	Regional Screening Level
SPA	South Plume Area
SPBA	Southern Property Boundary Area
TCE	Trichloroethene
THQ	Target Hazard Quotient
TR	Target Risk
VDEQ	Virginia Department of Environmental Quality
VI	Vapor intrusion
VISL	Vapor Intrusion Screening Level
VOC	Volatile organic compound
WHO	World Health Organization
WPA	Western Property Area
WPL	West Parking Lot
WQCTS	Water Quality Criteria for Toxic Substances
YCIDA	York County Industrial Development Authority

Groundwater Human Health Risk Assessment

Former York Naval Ordnance Plant, York Pennsylvania

1.0 Introduction

The former York Naval Ordnance Plant (fYNOP) located in York, Pennsylvania (the Site)[1] has been used for industrial and manufacturing purposes since 1941, when it was developed by the York Safe and Lock Company, a United States Navy contractor, for the manufacture, assembly, and testing of guns and gun mounts. During World War II, the Navy took possession of the facility and operated it as the York Naval Ordnance Plant (YNOP), switching operations to manufacture and overhaul of war service weapons and equipment. In 1964, the Navy sold the YNOP to American Machine & Foundry Company (AMF), which continued similar manufacturing. In 1969, AMF merged with Harley-Davidson, and Harley-Davidson moved its motorcycle assembly operations to the AMF York facility in 1973. Harley-Davidson bought the facility from AMF in 1981, and has continued motorcycle assembly operations on a portion of the Site up to the present day.

Characterization of the fYNOP was initiated in 1984, and has proceeded under the joint U.S. Environmental Protection Agency (EPA) and Pennsylvania Department of Environmental Protection (PADEP) One Cleanup Program since 2005. The Site is zoned industrial, and has been divided into two parcels: the East Campus (172 acres) and the West Campus (58 acres). A railroad, the Heritage Rail Trail recently opened by the York County Rail Trail Authority, and an undeveloped area are located between the Site and Codorus Creek, a tributary of the Susquehanna River to the west. The Johnsons Run tributary bounds the northern portion of the Site, industrial properties bound the southern portion, and residential areas adjoin the Site to the northeast, east and southeast (Figure 1). Harley-Davidson will continue to manufacture motorcycles on the East Campus into the foreseeable future, while the West Campus was sold to the York County Industrial Development Authority (YCIDA) with activity and use limitations documented in the Buyer-Seller Agreement dated July 22, 2010, and a recorded Environmental Covenant (July 12, 2012) that runs with the land. The West Campus was sold in November 2015 to NP York 58, LLC for redevelopment. Redevelopment was initiated in the spring of 2016.

A human health risk assessment (HHRA) is being conducted at the fYNOP to examine the potential long-term exposure and health risk (both cancer and non-cancer) potentially resulting from exposure to chemicals of potential concern (COPCs) in groundwater and associated media (soil vapor and surface water in Codorus Creek) in a manner compliant with Pennsylvania Department of Environmental Protection (PADEP) Act 2 (PADEP 2002, 2015), U.S. Environmental Protection Agency (EPA) guidance (EPA 1989, 2001a, 2004, 2009, 2011a, 2014b, 2015c), and Virginia

[1] Although the term "Site" in this document refers specifically to the fYNOP property, impacted areas off-Site are also addressed, in keeping with the typical regulatory definition of the term as including all areas impacted by a facility.

Department of Environmental Quality (VDEQ) guidance (VDEQ 2014). In order to streamline the process and ensure the acceptability of the HHRA to regulatory authorities, a series of three interim deliverables (IDs) were submitted:

- ID #1: Presented the proposed exposure pathway model (EPM) to serve as a framework for the HHRA (NewFields 2014). The first draft of this document was submitted to EPA and PADEP on June 16, 2014. Revisions were made and approval from EPA was received August 13, 2014.
- ID #2: Summarized changes made to the proposed EPM presented in ID #1, developed representative data sets for each potential exposure medium, selected COPCs to be carried through the risk assessment process, and calculated reasonable maximum exposure (RME) exposure point concentrations (EPCs) for conservative exposure estimation (NewFields 2016a). Seven Land Use Areas (LUAs) were identified in accordance with current and likely future uses. Potential human exposure domains (geographic areas over which receptors may be exposed to affected media) within each LUA were identified by overlaying the LUAs on (1) the TCE/PCE and petroleum plumes, and (2) areas where groundwater is expected to be ≤ 15 feet below ground surface (bgs) (where Construction and Utility Workers could directly contact groundwater). An ecological screening assessment (ESA) for aquatic organisms in Codorus Creek was also conducted in ID #2. As no ecological COPCs were identified, no further evaluation of aquatic ecological receptors was considered warranted. ID #2 was submitted March 10, 2016, and approved by EPA on March 23, 2016.
- ID #3: Presented exposure parameter and toxicity values and calculated risk-based screening levels (RBSLs) for each COPC, exposure pathway, and receptor scenario identified in IDs #1 and #2 (NewFields 2016b). RBSLs are defined as concentrations of COPCs in relevant media that are not expected to produce any adverse health effects under defined (usually chronic) exposure conditions. RBSLs are calculated using EPA's toxicity criteria and conservative modeling and exposure assumptions based on regulatory guidance or professional judgment. As such, the suite of RBSLs developed for the Facility provide a particularly useful tool for site assessment and evaluation of remedial action alternatives. ID #3 was submitted April 18, 2016, and approved by EPA on May 9, 2016.

In the fYNOP groundwater HHRA, RBSLs are compared with both central tendency exposure (CTE) and RME EPCs to calculate ranges of individual-COPC and cumulative theoretical incremental[2] lifetime cancer risks (ILCRs) and non-cancer hazard quotients (HQs) for each receptor scenario under current and reasonably anticipated future conditions. In addition, COPCs are selected for hypothetical future (1) residential development (vapor intrusion), and (2) potable groundwater use scenarios both on- and off-Site. Maximum detected concentrations of each COPC are compared with putative cleanup goals in order to provide a screening-level estimate of the degree to which chemical concentrations exceed regulatory standards.

[2] "Incremental" denotes the theoretical chemical-induced risk over and above background cancer incidence.

2.0 Overview of the HHRA Process

The basic principles of toxicology underlie the process of quantitative human health risk assessment (HHRA) that has been developed over the past three decades by regulatory authorities such as the EPA, Agency for Toxic Substances and Disease Registry (ATSDR), and World Health Organization (WHO), as well as state agencies such as PADEP. HHRA is a systematic evaluation process designed to estimate how much contact people might have with chemicals present in environmental media, and to quantify the risk of adverse health effects that might result from that exposure (*e.g.*, EPA 1989, WHO 1999).

It is important to recognize that risk assessment is not itself a science, but rather a tool that can be useful to support remedial decision-making. Because reliable scientific data are not available for many of the elements of a risk assessment, the process relies on a number of conservative assumptions that are intended to ensure that potential exposures and risks will be consistently overestimated rather than underestimated. A tiered approach in which generic assumptions and models are replaced as necessary by more realistic assumptions and models is generally accepted as a valid means to ensure that risk assessment results provide appropriate support for effective risk management.

The HHRA process typically comprises five elements:

- **Data Review and Evaluation**. Available data are reviewed to develop data sets for use in the risk assessment, identify Site-related COPCs (defined as chemicals clearly associated with the site that are detected at a frequency greater than 5% and at concentrations higher than background levels and/or conservative COPC screening levels), and calculate EPCs representative of CTE and RME exposure conditions in each affected medium. The proposed approach for developing data sets and calculating RME EPCs was presented in ID #2 (NewFields 2016a).
- **Exposure Assessment**. The exposure assessment is based upon scenarios that define the conditions of exposure to COPCs, and considers both current and likely future site uses and is based on potentially complete exposure pathways to actual or probable human receptors (*i.e.*, the people who could come in contact with Site-related COPCs). These scenarios are summarized in the EPM that provides the framework for the HHRA. Exposure parameter values for each scenario were presented in ID #3 (NewFields 2016b).
- **Toxicity Assessment**. The toxicity assessment consists of two distinct elements: (1) hazard identification, and (2) dose-response assessment. Hazard identification describes the adverse health effects caused by a chemical, while dose-response assessment characterizes the relationship between exposure or dose and the incidence and severity of effects. EPA has developed toxicity criteria for many constituents of concern in human health risk assessment. These values are not expected to result in adverse health effects even in sensitive subpopulations exposed daily for a lifetime. Two kinds of effects are recognized: (1) non-

carcinogenic effects, and (2) carcinogenic effects. The same chemical may exert both kinds of effects. Toxicity criteria for each COPC were presented in ID #3 (NewFields 2016b).

- **Risk Characterization**. In risk characterization, exposure and toxicity data are combined to estimate the nature and magnitude of potential non-cancer hazards and theoretical incremental lifetime cancer risks to defined receptor populations. The EPA has defined the acceptable target cancer risk range one in ten thousand (0.0001 or 10^{-4}) to one in one million (0.000001 or 10^{-6}) (EPA 1991). The PADEP has identified one in one hundred thousand (0.00001, or 10^{-5}) as its target risk level for calculation of Statewide health standards (medium-specific concentrations [MSCs]) (PADEP 2002, 2015). Both agencies have adopted a non-cancer target hazard level of 1. In order to account for simultaneous exposure to multiple COPCs and routes associated with the same exposure medium, risks/hazards are summed and rounded to one significant figure (EPA 1989, 2004).
- **Uncertainty Analysis**. Like any other form of modeling, risk assessment relies on a set of assumptions and estimates, each of which has some element of uncertainty. Uncertainty analysis accounts for both variability in and lack of knowledge about measured and estimated parameters, allowing decision makers to better evaluate risk estimates in the context of the assumptions and data used in the assessment. Major sources of uncertainty in risk assessment include (1) natural variability (*e.g.*, differences in body weight in a group of people), (2) lack of knowledge about basic physical, chemical, and biological properties and processes (*e.g.*, the affinity of a constituent for soil, its solubility in water), (3) lack of accuracy in the models used to estimate key inputs (*e.g.*, dose-response models), and (4) measurement error. The uncertainties associated with dose-response relationships and weight-of-evidence carcinogenicity classification are usually greater than those of other elements. The extrapolation of rodent bioassay or occupational exposure data to much lower levels of human exposure involves a number of assumptions regarding effects thresholds, and differences among and within species, effects at different doses, and route-specific effects. The scientific validity of these assumptions is uncertain; because each of the individual extrapolations are designed to prevent underestimation of risk, in concert they result in unquantifiable but potentially very large overestimation of risk.

In this HHRA, theoretical ILCRs and non-cancer HQs are calculated by comparison of COPC-, pathway/route-, and receptor-specific risk-based screening levels (RBSLs) with EPCs. Because of the conservatism of the RBSLs, no further action will be considered in areas where potential cancer risks and non-cancer hazards associated with COPCs are below target levels. In areas where target risk and/or hazard levels are exceeded, either remedial action (including engineering and institutional controls) or further investigation may be warranted. The degree of exceedance of RBSLs in various areas can be used to prioritize response actions.

3.0 Data review and evaluation

3.1 Setting

Potential exposure media for fYNOP groundwater HHRA include groundwater, soil vapor containing volatile chemicals derived from groundwater, and surface water in a segment of Codorus Creek affected by groundwater-to-surface water flow. Comprehensive tabulations of the groundwater and surface water data used in the HHRA are provided in Appendices A and B, respectively.

3.1.1 Groundwater

Various historical manufacturing and testing processes and waste handling and disposal practices at the Site have contributed to the presence of Site-related chemicals in groundwater, particularly chlorinated solvents that were used for metal degreasing operations. The Site and related off-Site areas have been divided into seven areas of interest for investigation and remediation:

- Northern Property Boundary Area (NPBA)
- Eastern Area
- Southern Property Boundary Area (SPBA)
- South Plume Area (SPA)
- Bunker and Shell Range Area (BSRA)
- North End Test Track (NETT)
- Western Property Area (WPA)

These on- and off-Site area designations are shown on Figure 2, and specific well locations are shown on Figure 3, overlain on the TCE/PCE plumes. Also depicted on Figure 3 is a petroleum plume located in the area around MW-77 in the northeastern corner of the West Campus. The source of the plume was a leaking gasoline dispenser from a former underground storage tank that was removed in July 2010. In addition to the seven designated areas, a number of wells exist on- and off-Site outside of the designated areas. The history and groundwater monitoring results at each of the area designations is summarized in Table 1 and detailed in ID #2 (NewFields 2016a). It is also noted that there is a potential that Site-impacted groundwater might pass beneath and slightly west of Codorus Creek, as it travels through solution channels in the carbonate aquifer prior to discharging to the creek. This is discussed in detail in Section 4.1.2.1 of the Supplemental Remedial Investigation Groundwater Report (Part 2) report (GSC 2016).

3.1.2 Surface water

Codorus Creek flows to the north past the west side of the WPA (Figure 3), and is the limit of westward migration of Site-related chemicals.

The segment of Codorus Creek adjacent to the WPA is designated as Drainage Basin O (Codorus Creek from Oil Creek to the Mouth of the Susquehanna River) by PADEP (25 Pa. Code §93.9o). The interaction of Site groundwater with this segment of Codorus Creek was extensively investigated during the recent Groundwater Remedial Investigation Part 2 (GSC 2016). Bedrock within the top 200 to 250 feet bgs in the western portion of the WPA is carbonate aquifer composed of solution-prone bedrock. Under natural groundwater flow conditions (*i.e.*, in the absence of groundwater treatment system [GWTS] operation), Site-impacted groundwater flows through this karst bedrock and discharges to Codorus Creek through discrete springs and areas of diffuse groundwater discharges from the unconsolidated and fractured rock portions of the aquifer.

Detailed investigations conducted by GSC (2016) determined that the greatest impacts of Site groundwater on surface water quality occur along an approximately 0.4-mile stretch of Codorus Creek between sampling stations SW-16/SW-27 and SW-8/SW-9 (Figure 3). Accordingly, the HHRA focuses on data collected in this segment.

Johnsons Run enters the Site at the northern property line near the intersection of Eden and Paradise Roads and flows in a westerly direction generally along the northern property line. The majority of the On-Site segment of Johnsons Run appears to have been realigned or channelized in the past. Although identified as a scenario in ID #1, regular wading in Johnsons Run is thought to be unlikely. Most analytes were not detected in surface water sampling conducted by Langan in Johnson's Run in June 1998 and spring 2000; PCE and TCE were detected at estimated concentrations (below the detection limit) of 0.6 and 0.7 µg/L, respectively, in one 1998 sample, and TCE was detected in one 2000 sample at 1.4 µg/L (Langan 2002). Samples from Johnsons Run were also analyzed six times during the two shutdown periods of the groundwater extraction system in 2014 and 2015. PCE was detected in two of the six samples at estimated concentrations of 0.36 and 0.21 µg/L, and TCE was detected once at an estimated concentration of 0.25 µg/L (GSC 2016). These values are below PADEP Water Quality Criteria for Toxic Substances (WQCTS) based on human health for PCE and TCE (0.69 and 2.5 µg/L, respectively) (PADEP 2013). In addition, maximum detected concentrations of analytes in sediment samples were below available PADEP residential MSCs for direct contact with soil (PADEP 2011). Because concern regarding human health is unwarranted, wading in Johnsons Run is not evaluated in the HHRA.

3.2 Development of data sets for the HHRA

A critical element for the fYNOP groundwater HHRA is groundwater and surface water data sets of sufficient quality and quantity to reliably represent concentrations of chemicals known to be derived from the Site in potential exposure media at locations where receptors could come into contact with them. Sampling data were evaluated for their usability in the risk assessment in accordance with EPA guidance (EPA 1989, 1992). The few data flagged as "R" or "rejected" due to not meeting data usability requirements were not included in the data set. Due to the need to evaluate chemicals at low concentrations, the chemical concentrations were reported as

estimated if the analyte was identified but the concentration was less than the laboratory's quantitation limit but above the method's detection limit. The result of this method of reporting concentrations is a data set for which the detection limits may appear elevated when reported at the quantification limit.

Duplicate results were handled as follows:

- If the analyte is detected in both samples, then the average of the measured values was used to represent the compound concentration in the evaluation
- If the analyte is detected in neither sample, then the greater detection limit of the two was used to represent the compound concentration in the evaluation
- If the analyte is detected in one sample but not the other, then the detected concentration was used as the representative concentration in the evaluation

3.2.1 Groundwater data set

The chemical profile of groundwater underlying the Site has been extensively characterized in the past several decades, as described above and in numerous submittals to PADEP and EPA. Over the history of groundwater sampling at the Site, decreasing trends in concentrations of Site-related analytes have been observed in many wells (GSC 2014). On this account, and in accordance with standard EPA risk assessment guidance stating that data used for risk assessment should represent current conditions (EPA 1989, 2014a), the groundwater data set for the HHRA comprises samples collected in 2008, 2013, 2014, and 2015. In accordance with EPA and PADEP guidance (EPA 2015c), data from wells intercepting the water table are used to characterize the source strength for vapor intrusion (VI) evaluation. These data are summarized in Appendix A1. Results from all depths used to select COPCs for hypothetical future potable use of groundwater are presented in Appendix A2.

Figure 4 is a composited concentration contour map, illustrating where either TCE or PCE was detected in the "shallow" portion of the aquifer[3] (defined as any well monitoring groundwater within 75 feet of the ground surface) at an elevation of approximately 310 feet above mean sea level.[4] The contouring was interpreted by considering a number of factors, including groundwater flow direction, the three-dimensional distribution of Site-related chemicals in the aquifer, and historical chemistry trends. If the exact depth of the well screen was unknown (the condition for a few off-Site wells on the industrial properties south of the Site), then the well was assumed to monitor shallow groundwater and was included in the data set. Using this method of well or sample inclusion, several well clusters (wells monitoring different depths in the same location)

[3] It should be noted that TCE and PCE values were not added together; rather, the concentration areas were composited to illustrate the general locations of these two major Site-related chemicals.

[4] The elevation of 310 feet above mean sea level occurs approximately 100 feet below the ground surface in the NPBA and approximately 50 feet below the ground surface in all areas west of the East Campus.

were included in the data set. Data from these clusters were combined, and the highest concentration taken. Combined, these contours delineate the areas, both on- and off-Site, where groundwater quality is of potential health concern.

In order to evaluate the effect of the West Campus groundwater extraction well pump shutdown on groundwater quality, data sets representing pumping and non-pumping conditions were identified. The 2008 sampling event occurred during GWTS operation and includes the most comprehensive number of wells. However, it does not include a number of more recently installed wells. The 2013 data set provides more recent results collected while the GWTS was operating, and the 2014 data set provides results collected when the GWTS had been completely shut down for approximately two months. Results from groundwater samples collected during the 2015 SPBA VI investigation performed in order to resolve data gaps in that area (GSC 2015) were also added to the data set.

There are a few exceptions in the 2008, 2013 and 2014 data sets. In the case of 27 wells listed in Table 2, sampling was not accomplished as part of the annual sampling events, and therefore data from other sampling events were used for the HHRA. These data were combined with the appropriate sampling event, as listed in Table 2, to provide a complete database. The wells included in the list of exceptions were installed as part of separate but related investigations, and for various reasons were not sampled during the annual events.

As discussed in Section 4.2, Construction and Utility Workers could directly contact groundwater in areas where groundwater depth is 15 feet bgs or less.[5] In order to select the data set for evaluation of potential direct contact with groundwater in excavations/trenches, the area in which the depth to groundwater is less than or equal to 15 feet was determined as described in Appendix C (Figure 4). A “deep” (>15<75 feet bgs) groundwater data set was also developed in order to evaluate these receptors’ indirect exposure to vapors entering their excavations/trenches. For Commercial/Industrial Workers, shallow and deep samples were combined.

3.2.2 Surface water data set

As discussed in Section 0, the segment of Codorus Creek most likely affected by the discharge of Site groundwater is characterized by six surface water sampling locations: SW-16, SW-27, SW-13, SW-28, SW-8, and SW-9 (Figure 3). Only in-stream sampling data were used, as (1) the springs are not easily accessible for sustained or repeated exposures, and (2) analytes in spring water would be rapidly diluted in Codorus Creek. These data are summarized in Appendix B.

[5] As in the fYNOP Soils Risk Assessment (GSC 2012), it is conservatively assumed that construction excavations and utility trenches could be as deep as 15 feet. This depth is also assumed in the widely used Virginia Department of Environmental Quality guidance for evaluating exposure of workers to volatiles in a construction/utility trench (VDEQ 2014).

In order to evaluate the effect of the West Campus groundwater extraction well pump shutdown on surface water quality in Codorus Creek, data sets representing pumping and non-pumping conditions were identified. To ensure that the data sets are not skewed due to ramp up or ramp down effects from pumping, samples collected in the first few weeks following a change in pumping were considered transitional and not included in the data set. Additionally, sample results selected were during the dry season (low flow) conditions to evaluate periods characterized by the predominance of base flow (limited dilution from surface water runoff) when higher concentrations were detected. The non-pumping interval selected was between October 1, 2014 and January 27, 2015. The pumping interval selected consists of samples collected between March 15 and September 1, 2015 to represent concentrations during times when human receptors would more likely use the creek for recreational purposes. The decision to use samples collected during 2015 rather than 2013 was made to reflect the current GWTS pumping configuration.

4.0 Exposure assessment

The overall objective of the exposure assessment is to estimate the potential magnitude, frequency, duration, and routes of human exposure to site-related COPCs. The EPM proposed in ID #1 (NewFields 2014) was revised in ID #2 (NewFields 2016a) in accordance with (1) comments from EPA and PADEP, and (2) additional relevant data collection. This revised EPM (Figure 5), discussed in the following sections, provides the framework for the identification of COPCs and calculation of EPCs within defined exposure domains for each receptor scenario.

COPCs in Site groundwater may migrate and/or enter other environmental media via one or more of the following general mechanisms:

- Transport of COPCs in groundwater, and their attenuation by dispersion, diffusion, biodegradation, etc.
- Volatilization of COPCs from groundwater into soil vapor, and transport into ambient and indoor air
- Transport of COPCs in groundwater to surface water in Codorus Creek
- Bioaccumulation of COPCs in aquatic organisms

The potential for COPCs to migrate from source media to points of exposure depends on their physicochemical properties, concentration, and distribution, characteristics of the affected aquifer(s) and their interaction with surface water bodies, and climatic conditions.

4.1 Current and potential future human receptor scenarios

Current and potential future human receptor populations at the Site and off-Site areas identified in ID #1 (NewFields 2014) were:

- On-Site Maintenance Workers

- On-Site Commercial/Industrial Workers
- On-Site Trespassers
- On- and off-Site Construction Workers
- On- and off-Site Utility Workers
- Off-Site Residents
- Off-Site Fish Consumers consuming fish caught in the affected segment of Codorus Creek
- Off-Site Recreational Waders (adolescents) in Johnsons Run

Since ID #1 was prepared, a rail trail between the West Campus and Codorus Creek was opened. People traversing the rail trail adjacent to the Site may be exposed to Site-related chemical vapors emanating from groundwater into ambient air. However, this potential exposure is expected to be minor due to brief duration of contact, and dilution and dispersion in outdoor air. Therefore, this receptor scenario is not considered quantitatively in the groundwater HHRA.

Several of the receptor scenarios identified in ID #1 were eliminated from further consideration *a priori* in ID #2 (NewFields 2016a) due to the absence of complete and significant exposure pathways:

- **On-Site Maintenance Workers.** Maintenance Workers on the Site may be exposed to Site-related chemical vapors emanating from groundwater into ambient air. However, this potential exposure is expected to be minor due to dilution and dispersion in outdoor air. Therefore, this receptor scenario is not considered quantitatively in the groundwater HHRA.
- **On-Site Commercial/Industrial Workers (East Campus).** Commercial/Industrial Workers are and could in the future be present on-Site on the East Campus. Because groundwater on the Site is not currently used as a potable source, nor is it likely to be in the future, direct contact by Commercial/Industrial Workers could not occur. Harley-Davidson has an OSHA program in place that includes air/industrial hygiene monitoring, which addresses the VI pathway. Moreover, the very high air exchange rates in the industrial buildings of the East Campus effectively mitigate workers' exposures to any indoor vapors from the subsurface. Because VI is not a significant exposure pathway for current or future Commercial/Industrial workers on the East Campus, this receptor scenario is not considered quantitatively in the groundwater HHRA.
- **On-Site Trespassers.** Trespassers on the Site may be exposed to Site-related chemical vapors emanating from groundwater into ambient air. However, this potential exposure is expected to be minor due to dilution and dispersion in outdoor air. Therefore, this receptor scenario is not considered quantitatively in the groundwater HHRA.
- **Off-Site Residents.** Well surveys have documented the fact that groundwater in the vicinity of the Site is not currently used as a potable source. Although RW-4 is occasionally used for non-potable outdoor uses, VOC concentrations were either not detected or below the PADEP MSCs

in 2008, 2013, and 2014. To ensure that potable residential wells are not established in off-Site areas adjacent to the NPBA and SPBA in the future, periodic water use surveys should be conducted as part of post-remediation care plans. Therefore, potable use of groundwater by off-Site residents does not represent a current or likely future complete exposure pathway for Site-related chemicals in groundwater via direct contact (ingestion and inhalation of vapors and dermal contact during bathing).

Johnson & Ettinger (1991) model predictions indicated that indoor air concentrations of chlorinated VOCs in residential properties immediately off-Site near the NPBA and the SPBA would not exceed acceptable levels (Langan 2005, 2006). As a result, EPA issued a "Yes" determination (indicating that exposures are under control) for the Human Health Environmental Indicator (EI) form that includes the VI pathway (EPA 2005b). Additional evaluation for the NPBA was conducted in 2007, and results affirmed that there is no off-Site residential health risk via the VI pathway (Langan 2008). However, EPA subsequently determined that the methodology applied in the 2005 VI assessment was outdated and no longer sufficient to rule out a completed VI exposure pathway for off-Site residents and workers to the south (EPA 2014). Accordingly, GSC undertook a systematic assessment of the potential for VI in the SPBA, including investigations of groundwater in the saturated materials just below the water table (GSC 2015). If groundwater analytical results exceeded risk-based screening levels calculated using EPA's Vapor Intrusion Screening Level (VISL) Calculator (May 2014), then soil vapor in the vadose zone just above the capillary zone would be sampled. Because no exceedances were observed, no soil vapor samples were necessary (GSC 2015).

Two rounds of groundwater sampling in new and adjacent existing wells were analyzed for VOCs. As expected from previous investigations, concentrations of TCE and PCE within the boundary of the Harley-Davidson property were comparatively elevated, but concentrations in the newly installed shallow wells in the Canterbury Lane neighborhood adjacent to the SPBA were substantially lower. The highest concentrations of these compounds in shallow off-Site groundwater (MW-167) were below screening levels for TCE and PCE calculated using EPA's VISL calculator, indicating negligible potential for adverse health effects. Based on these results, EPA determined that VI is not expected to be a significant exposure pathway for the off-Site residential area downgradient of the SPBA, and revised the Human Exposures EI accordingly (EPA 2015d). Therefore, this receptor scenario is not considered quantitatively in the groundwater HHRA.

- **Off-Site Recreational Waders in Johnsons Run.** Although identified as a scenario in ID #1, regular wading in Johnsons Run is thought to be unlikely. Further, as discussed in Section 0, concern with regard to human health is unwarranted because concentrations of PCE and TCE in the stream under both pumping and non-pumping conditions were below PADEP WQCTS based on human health (PADEP 2013) and maximum detected concentrations of analytes in sediment samples were below available PADEP residential MSCs for direct contact with soil (PADEP 2011). For these reasons, wading in Johnsons Run is not evaluated in the HHRA.

Three receptor scenarios that were dismissed in ID #1 are considered in the groundwater HHRA:

- A recorded Environmental Covenant prohibits use of groundwater on the West Campus, and limits use of that property to certain industrial and commercial uses. Furthermore, the recorded Environmental Covenant requires Harley-Davidson's approval of any redevelopment designs. As described in ID #1, the Soils Risk Assessment for the fYNOP (GSC 2012) concluded that the potential health risk to West Campus Commercial/Industrial Workers associated with VI did not exceed acceptable levels, and in September 2005, EPA issued a "Yes" determination (indicating that exposures are under control) for the Human Health EI form that includes the VI pathway (EPA 2005b). In view of the subsequent changes in toxicity criteria and VI guidance, this conclusion was reexamined in ID #2. The soil vapor data collected by Langan (2005) (Figure 3) were compared with PADEP's draft non-residential near-source soil vapor statewide Health Standard VI screening values (PADEP 2015) as COPC screening levels. Since there were no exceedances, the scenario was proposed to be eliminated from further consideration in ID #2. However, based on reviewers' comments, it was subsequently determined that EPA's commercial scenario sub-slab soil vapor VISLs (v 346) are more appropriate COPC screening levels, and TCE was selected as a COPC based on that comparison (see Section 4.4.1). Therefore, this receptor scenario is considered quantitatively in the groundwater HHRA.
- ID #1 stated that Off-Site Commercial/Industrial Workers would not be included because the Off-Site Resident scenario is protective of their lesser potential exposures. However, because such workers may be present in non-residential areas potentially impacted by Site-related COPCs in groundwater, it was determined in ID #2 that they would be included in the groundwater HHRA.
- ID #1 stated that Codorus Creek immediately adjacent to the fYNOP does not appear to be suitable for prolonged regular direct contact recreation. However, because wading in the affected segment of Codorus Creek is possible, it was determined in ID #2 that the Recreational Wader scenario would be evaluated in the groundwater HHRA.

The current and reasonably anticipated future receptor scenarios considered in the HHRA are therefore:

- On and off-Site Commercial/Industrial Workers
- On- and off-Site Construction Workers
- On- and off-Site Utility Workers
- Off-Site Fish Consumers
- Off-Site Recreational Waders (Codorus Creek)

Future residential development on-Site is unlikely, and therefore not considered quantitatively in the HHRA. However, COPCs were selected for both VI and potable groundwater use assuming the presence of both on- and off-Site residents as hypothetical future scenarios.

Codorus Creek in the vicinity of the Site is not a current drinking water supply, and its future potable use is unlikely due to the facts that (1) the municipal wastewater treatment plant discharge is located directly across from the Site, and (2) Codorus Creek is impacted by numerous other sources along its course through the city of York. Therefore, as agreed at the parties' meeting on November 3, 2016, potable use of Codorus Creek is not considered in the HHRA.

Exposure domains based on land use are identified in the following section.

4.2 Identification of Land Use Areas and exposure domains

Land use in a given area determines what receptors may be present and what activities they may perform, which in turn determines what exposure pathways are potentially complete. Because groundwater is the source medium, and chemical plumes are present at varying depths in circumscribed areas, the Site and its adjacent areas have been divided into seven current and potential future LUAs (including Codorus Creek adjacent to the Site constituting the seventh) in order to guide the processes of data set development, COPC selection, and EPC calculation (Figure 6). The following approach was used to define the LUAs:

- The Harley-Davidson property was divided into (1) a developed area, consisting of parking lots, production buildings, and roads, along with adjacent lawn/landscaped areas and stormwater facilities (LUA #1); and (2) an undeveloped area, all of which is potentially developable (LUA #2). Construction and Utility Workers are or could be present in these areas, and Commercial/Industrial Workers could be present in LUA #2 in the event of future development. In addition, COPCs assuming hypothetical future residential development and potable use of groundwater are selected.
- The West Campus (LUA #3) consists of areas where development/construction may occur, and areas where building construction would likely be infeasible (most of the WPL, as it is located in a floodplain), but utility trenching is likely to be necessary. In the absence of deed restrictions prohibiting building in these areas, the entire West Campus was designated LUA #3. Commercial/Industrial, Construction, and Utility Workers are or could potentially be exposed to COPCs in these areas. In addition, COPCs assuming hypothetical future residential development and potable use of groundwater are selected.
- Lacking current and reasonably expected complete VI exposure pathways, residents in the off-Site areas to the north and south (LUA #4) are not considered in the groundwater HHRA. Commercial/Industrial Workers in this area are assumed to be protected by the fact that residential exposures are acceptable. However, Construction and Utility Workers could be exposed to COPCs in LUA #4. In addition, COPCs assuming hypothetical future potable use of groundwater are identified in order to inform remedy selection.
- Commercial/Industrial, Construction, and Utility Workers could potentially be exposed to Site-related chemicals in developed off-Site industrial areas (LUA #5). As noted previously herein, Site groundwater may pass beneath and slightly west of Codorus Creek prior to discharge to

the creek (GSC 2016). LUA #5 therefore extends to the north along the west side of Codorus Creek to include this area, which is currently occupied by commercial/industrial properties without groundwater supplies. The horizontal boundary of the area that may potentially be impacted by the Site groundwater was drawn along the eastern edge of the Kinzers Shale where it is close to the creek, and up to 1,000 feet west of the creek as it adjoins the Site. In addition, COPCs assuming hypothetical future residential development and potable use of groundwater are selected to inform remedy selection.Much of the area west of the WPL (LUA #6) is zoned industrial. As it is in the flood plain, the long-term presence of Commercial/Industrial Workers is unlikely. However, Construction and Utility Workers are or could potentially be exposed to COPCs in these areas, and Commercial/Industrial Workers could be present in the future if development occurs. In addition, COPCs assuming hypothetical future residential development and potable use of groundwater are selected to inform remedy selection.

- The segment of Codorus Creek impacted by Site groundwater (LUA #7) could be used by Fish Consumers and Recreational Waders.

4.3 Potentially complete exposure pathways

In order to focus attention on any pathways that may result in significant exposure to COPCs, attention is limited to those exposure pathways that are likely to be complete and potentially significant for defined receptor populations. Complete exposure pathways consist of four elements:

- A source and mechanism(s) of chemical release to the environment
- An environmental transport medium for the released chemical
- A point of potential human contact with the affected medium
- A route of entry into humans (inhalation, ingestion, or dermal contact with the affected medium)

If any of these components is missing, then the pathway is incomplete and does not contribute to receptor exposure. The rationale for selection of the potentially complete exposure pathways for on- and off-Site receptors shown in the EPM (Figure 5) is summarized in Table 3 and Table 4, respectively, and briefly discussed in the following sections.

Potential human exposure domains (geographic areas over which receptors may be exposed to affected media) within each LUA were identified by overlaying the LUAs on (1) the TCE/PCE and petroleum plumes, and (2) areas where groundwater is expected to be ≤ 15 feet bgs (where Construction and Utility Workers could directly contact groundwater) (Table 5, Figure 7). These overlays indicate portions of plumes that could serve as exposure sources for receptor scenarios in specific geographic areas, and so facilitate identification of potentially complete exposure pathways and calculation of EPCs in each LUA.

4.3.1 On-Site receptor scenarios

The rationale for identification of potentially complete exposure pathways for on-Site receptors in these areas is summarized in Table 3. On-Site Commercial/Industrial Workers in LUA #2 and #3 could be exposed to groundwater COPCs via indoor vapor inhalation. On-Site Construction and Utility Workers are or could be present in LUAs #1 – 3. When engaged in intrusive activities, these receptors could come into direct contact with groundwater in excavations and trenches in areas of the site where groundwater is within 15 feet of the ground surface (Figure 7). They could also be exposed to COPC vapors arising from groundwater both at and below the trench base for short periods of time, as the excavation and trench walls may reduce the immediate dilution and dispersion of the vapors into the outdoor air.

4.3.2 Off-Site receptor scenarios

Groundwater underlying off-Site LUAs #4 – 6 is potentially affected by Site-related COPCs, as is surface water in the adjoining segment of Codorus Creek (LUA #7). The rationale for identification of potentially complete exposure pathways for off-Site receptors in these areas is summarized in Table 4.

4.3.2.1 Commercial/Industrial Workers

Commercial/Industrial Workers are present off-Site to the south and west of Codorus Creek (LUA #5), and could in the future be present west of the WPL (LUA #6). There is no potable use of groundwater in these areas. Therefore, only potential indoor exposure via VI will be evaluated in these areas.

4.3.2.2 Construction and Utility Workers

As discussed previously for on-Site Construction and Utility Workers, exposure to COPC vapors emanating from off-Site groundwater into ambient air is expected to be minimal, and so will not be evaluated quantitatively in the groundwater HHRA. However, Off-Site Construction Workers and Utility Workers engaged in intrusive activities could come into direct contact (dermal contact and inhalation) with groundwater in excavations and trenches in areas where groundwater is within 15 feet of the ground surface (LUAs #5 and 6). In areas where depth to groundwater is greater than 15 feet, Off-Site Construction and Utility Workers in excavations and trenches may also be exposed to COPC vapors arising from groundwater for short periods of time, as the excavation and trench walls may reduce the immediate dilution and dispersion of the vapors into the outdoor air.

4.3.2.3 Fish Consumers

25 Pa. Code § Chapter 93 (relating to water quality standards) lists protected water uses for the evaluated reach of Codorus Creek (Drainage Basin O, Codorus Creek from Oil Creek to the Mouth [Page 93-165]) as warm water fishes that are indigenous to a warm water habitat and migratory

fishes that move to or from flowing water to complete their life cycle in other waters. Fishing has been observed to occur in the affected segment of Codorus Creek, potentially resulting in exposure to COPCs present in the tissues of consumed fish. Because both TCE and PCE volatilize rapidly from the water surface and have a low tendency to bioaccumulate in aquatic organisms, including fish (EPA Region 3 2006; WHO 2006; ATSDR 2014 a&b), they are unlikely to pose a risk via either direct contact or the food web.

4.3.2.4 Recreational Waders

COPCs can enter the affected segment of Codorus Creek (LUA #7) via groundwater-to-surface water flow. Potential exposure of tubers bypassing the Site would be very brief, and therefore is considered inconsequential. However, the affected segment of Codorus Creek could be used by recreational waders, potentially resulting in exposure to chemicals in surface water and sediment via incidental ingestion and dermal contact. Sediment can be an important repository for hydrophobic organic chemicals and metals. Historical data indicate that Site activities have not resulted in elevated metals concentrations in groundwater entering the affected segment of Codorus Creek. The chief chlorinated volatile chemicals related to the Site, TCE and PCE, are not expected to accumulate in surficial sediment due to their low hydrophobicity (log octanol:water partition coefficient < 4) (WHO 2006; ATSDR 2014 a&b). Codorus Creek sediment is therefore not considered to be a relevant potential human exposure medium for these compounds.

4.4 Identification of COPCs

The following selection criteria were applied to groundwater and surface water data sets to select COPCs:

- Known to be associated with former operations at the Site
- Positively detected in at least 5% of samples (EPA 1989)
- Not an essential nutrient or a common environmental element or ion (including calcium, chloride, iodine, iron, magnesium, phosphorus, potassium, and sodium) (EPA 1989)
- Maximum detected concentrations in groundwater and surface water exceed applicable conservative risk-based COPC screening level(s) for human receptors

Based on reviewers' comments, the COPC selection process described in ID #2 (NewFields 2106a) was repeated using COPC screening levels with a target cancer risk (TR) for potentially carcinogenic effects of analytes of 10^{-6} and the target hazard quotient (THQ) for non-carcinogenic effects of analytes 0.1. In addition, a groundwater COPC selection process was conducted assuming hypothetical future potable groundwater use in LUAs #1 - #6 and residential development (with attendant VI) on-Site and off-Site in LUAs #5 and #6 (LUA #4 was screened out based on residential VI in ID #2).

4.4.1 Groundwater (current and reasonably anticipated future conditions)

The samples collected in 2008 were included in the COPC selection data set as the analyte list and the coverage density are more extensive (Appendix A1). Groundwater COPCs were selected from the "shallow" (≤75 feet bgs) groundwater data set for evaluation of (1) direct contact for Construction and Utility Workers in areas where depth to groundwater is ≤ 15 feet bgs, (2) indirect contact (vapor intrusion into excavation or trench) for Construction and Utility Workers in areas where depth to groundwater is >15 to 75 feet bgs, and (3) indirect contact (vapor intrusion into buildings) for on-Site Commercial/Industrial Workers in LUAs #2 and #3 and for off-Site Commercial/Industrial Workers in LUAs #5 and #6 (Appendix D1). A comparison of Langan soil vapor data with EPA sub-slab soil vapor VISLs (TR = 10^{-6}, THQ = 0.1) (see Section 4.1) is also presented in Appendix D1. All on-Site wells were used to select volatile COPCs for the vapor inhalation exposure pathway, but only wells where depth to groundwater is ≤ 15 feet bgs were used to select COPCs for the direct contact with groundwater exposure pathway (Figure 7).

Current EPA residential tap water Regional Screening Levels (RSLs) (EPA Regions 3, 6, and 9, May 2016) (TR = 10^{-6}, THQ = 0.1) and Maximum Contaminant Levels (MCLs) (whichever is higher) were used for COPC screening for the direct contact pathway. For the vapor inhalation pathway, EPA's current (May 2016) commercial scenario VISLs (TR = 10^{-6}, THQ = 0.1) were used to screen COPCs. In accordance with PADEP guidance (PADEP 2015), the VISL spreadsheet was modified to set the groundwater temperature to 11° C, and the non-residential groundwater-to-indoor air attenuation factor to 0.00036.The selected groundwater COPCs for Construction, Utility, and Commercial/Industrial Workers by exposure pathway are summarized in Table 6. No COPCs were selected for the off-Site Commercial/Industrial Worker scenario in LUA #5 (Appendix D1).

4.4.2 Groundwater (hypothetical future residential development)

4.4.2.1 Potable use of groundwater

Groundwater COPCs were selected from data sets containing 2008 – 2015 groundwater samples from all depths for evaluation of potable use of groundwater in LUAs #1 - #6. Current EPA residential tap water RSLs (EPA Regions 3, 6, and 9, May 2016) (TR = 10^{-6}, THQ = 0.1) and MCLs (whichever is higher) were used for COPC screening. Selected COPCs are presented in Appendix D2, and summarized in Table 7.

4.4.2.2 Residential vapor intrusion

Groundwater COPCs were selected from the "shallow" (≤75 feet bgs) groundwater data set for evaluation of VI into hypothetical future residences on-Site and off-Site in LUAs #5 and #6 (VI LUA #4 was eliminated from consideration in the HHRA based on GSC's targeted groundwater investigation (GSC 2015; see Section 4.2 and ID #2 (NewFields 2016a)). EPA's current (May 2016) residential scenario VISLs (TR = 10^{-6}, THQ = 0.1) and MCLs (whichever is higher) were used to screen COPCs. In accordance with PADEP guidance (PADEP 2015), the VISL spreadsheet was

modified to set the groundwater temperature to 11° C, and the residential groundwater-to-indoor air attenuation factor to 0.0012. Selected COPCs are presented in Appendix D3, and summarized in Table 8.

4.4.3 Surface water

In order to evaluate the impact of West Campus groundwater extraction well pumping on exposures and risks to receptors contacting the affected segment of Codorus Creek, COPCs (and EPCs) were calculated based on data collected under both pumping and non-pumping conditions. As discussed in Section 0, the non-pumping interval selected was between October 1, 2014 and January 27, 2015 (full system restart). The pumping interval selected consists of samples collected between March 15 and September 1, 2015.

4.4.3.1 Fish Consumers

EPA Ambient Water Quality Criteria (AWQC) for human health based on consumption of water and fish (EPA 2015a) were used as COPC screening levels for the Fish Consumer scenario under pumping and non-pumping conditions. As shown in Appendix E, maximum concentrations of TCE and PCE exceeded these AWQC. However, as these criteria assume that Codorus Creek is the sole drinking water source for these receptors, with assumed daily water intake of 2.4 liters per day, they are considered overly conservative for a realistic fisher scenario. Therefore, a second screen was conducted comparing maximum surface water concentrations under pumping and non-pumping conditions with EPA AWQC for human health based on consumption of fish only (EPA 2015a). This more appropriate COPC screening procedure resulted in no COPCs selected for the Fish Consumer scenario. Therefore, it will not be considered further in the HHRA.

4.4.3.2 Recreational Waders

PADEP §93.8c WQCTS for human health protection (PADEP 2013) were used as COPC screening levels for the Recreational Wader scenario. For analytes lacking a WQCTS, EPA tap water RSLs (EPA Regions 3, 6, and 9, May 2016) were used for COPC screening. As shown in Appendix E, the surface water COPCs selected for the Recreational Wader scenario were TCE and PCE.

4.5 Calculation of exposure point concentrations

Site chemical data were used to calculate representative EPCs, defined as the arithmetic average of the COPC concentration that is contacted by a receptor over the exposure period. According to EPA's established policy, the arithmetic mean is considered to be the most appropriate statistic for characterizing CTE based on the fundamental assumption of random exposure within the exposure domain, and the 95% upper confidence limits (95UCL) on the arithmetic mean is used to represent RME exposure (EPA 1989, 1992, 2002b, 2013). In ID #2, only RME EPCs were presented. For the complete groundwater HHRA, CTE statistics are also considered.

EPCs were calculated using EPA's ProUCL software, v5.0.00 (EPA 2013), if the data met the criteria of having at least eight sample results, of which at least three must be detections. Non-detect values were replaced by surrogate values determined using either the Kaplan Meier (KM) or the Regression of Ordered Statistics methods. The exact method utilized was dependent on the statistical properties of the dataset. KM means were used to represent CTE. ProUCL computes UCLs using various methods based on data distributions, and then provides recommended method. The first 95UCL method was selected as the RME EPC.

4.5.1 Groundwater

As described in Section 4.2, the Site and its surrounding areas have been divided into six LUAs and Codorus Creek (Figure 6). Groundwater wells were assigned to these LUAs for purposes of EPC calculation. As discussed in Section 3.2.1, 2013 and 2014 data were generally used to calculate EPCs representative of current conditions. Due to extensive characterization in previous years, some COPCs are no longer monitored for in some wells or areas. While these COPCs are still monitored in wells where they were detected, the 2013-2014 data sets did not include the Harley-Davidson Undeveloped LUA (LUA #2). In the case of COPC 1,4-dioxane, not all samples were analyzed using sensitive analytical methods; some were reported from the general VOC analysis with elevated detection limits. Therefore, the 2008 data were used along with the current data as necessary to provide a robust data set for the EPC calculation in this area.

In accordance with EPA guidance (EPA 2014a), the RME EPC was calculated from the core of each "groundwater plume," where the core is defined as the "zone of highest concentrations of each contaminant within a delineated groundwater plume." Because all of the six LUAs have the potential for trench/excavation activities, groundwater EPCs were calculated for the core of each "groundwater plume" within the LUA. The wells sorted by LUA and exposure route for EPC determination are listed in Table 9. The TCE/PCE concentration plumes depicted in Figure 7 were used to identify the wells in the plumes' cores, defined as within the 100-ppb concentration isopleth, with the exception of the plumes in the southern portion of LUA #2, where the 50-ppb contour was used to define the VOC plume. Additionally, as the defined plume core in LUA #2 did not overlie the shallow water table portion of the LUA, wells located in that area were selected for evaluating the vapor inhalation pathway. For off-site LUAs where the core of the TCE/PCE plumes did not extend, wells were selected using the 5-ppb contour as defined on Figure 7.

Wells within the petroleum plume, located on the northern boundary between LUAs #1 and #3, are not within the TCE/PCE plume (Figure 7). Petroleum-impacted wells were therefore selected separately to calculate EPCs for petroleum compounds. The VOC EPCs were calculated using the selected wells in each of the identified plumes. No specific plume areas were identified for the non-volatile COPCs; therefore, all wells within the shallow groundwater area identified on Figure 7 were included in the EPC for direct contact.

It is noted that groundwater data for the west side of Codorus Creek (LUA #5) is limited to monitoring well MW-148A (Figure 7). If a well were installed in the future in this area, it could intercept the western edge of the Site-impacted groundwater migrating to the creek and/or pumping could draw groundwater from the east side underneath the creek to the well. The water quality in existing monitoring wells along the eastern side of the creek can therefore be regarded as providing a conservative estimate of exposure concentrations in this area.

4.5.1.1 Commercial/Industrial Workers

COPCs were selected for off-Site Commercial/Industrial Workers on-Site in LUA #3 and off-Site in LUA #6 (Table 6). EPCs (COPC concentrations in indoor air) (Appendix F) under pumping and non-pumping conditions are calculated using the following general equation:

$$C_{air} = C_{gw} \times VF_{c/iw} \quad \{1\}$$

The volatilization factor for the Commercial/Industrial Worker scenario ($VF_{c/iw}$) is calculated as:

$$VF_{c/iw}\left[\frac{L}{m^3}\right] = H'_{@T_{gw}} \times AF_{gw\_nr} \times 1{,}000 \quad \{2\}$$

where:

$H'@_{Tgw}$	=	Temperature-adjusted Henry's Law constant @ 11° C (0.2)	unitless
AF_{gw_nr}	=	default PADEP non-residential vapor attenuation factor from groundwater to indoor air (0.00036)	unitless
1,000	=	conversion factor	L/m^3

CTE and RME EPCs for Commercial/Industrial Workers are summarized in Table 10.

4.5.1.2 Construction and Utility Workers

4.5.1.2.1 Groundwater depth ≤ 15 feet

For Construction and Utility Workers, the exposure pathways that may be complete are dependent upon the depth to groundwater. As discussed in Section 4.2, where groundwater is ≤ 15 feet bgs, both Construction and Utility Workers have the potential for direct contact with the water as it seeps into the bottom of their excavation or trench. Because exposure can occur via direct contact (both inhalation and dermal contact routes), CTE and RME EPCs are calculated in terms of groundwater concentrations (Appendix F, Table 11 and Table 12, respectively).

4.5.1.2.2 Groundwater depth >15 to 75 feet

Where groundwater is deeper than 15 feet, Construction and Utility Workers may be exposed via indirect contact (VI) because excavations and trenches can impede the dispersion in outdoor air of vapors from groundwater, resulting in an enclosed-space-like exposure. There are no well-

established models available for estimating migration of volatiles from groundwater into a construction excavation or utility trench. Therefore, guidance developed by the VDEQ was used to develop vapor EPCs for both Construction and Utility Workers (VDEQ 2014). VDEQ recommends an approach based upon a combination of (1) a vadose zone model to estimate volatilization of vapors from groundwater into an excavation or trench, and (2) a box model to estimate dispersion of the vapors from the air inside the excavation or trench into the above-ground atmosphere to estimate the EPC for air in an excavation or trench.

The general equation to calculate the airborne concentration of a chemical in an excavation or trench is:

$$C_{air} = C_{gw} \times VF \qquad \{3\}$$

where:

C_{air}	=	concentration of chemical in air in the excavation or trench (µg/m^3)
C_{gw}	=	concentration of chemical in groundwater (µg/L)
VF	=	volatilization factor (see below) (L/m^3)

The generic VDEQ equation for VF where groundwater is deeper than 15 feet bgs is:

$$VF_{>15} = \frac{H \times D_{air} \times AC_{vad}^{3.33} \times A \times F \times 10^{-3} \times 10^{4} \times 3{,}600}{R \times T \times L_d \times ACH \times V \times Por_{vad}^{2}} \qquad \{4\}$$

where:

H	=	Henry's law constant	atm-m^3/mol
D_{air}	=	diffusion coefficient in air	cm^2/s
AC_{vad}	=	volumetric air content in vadose zone soil (0.25)	cm^3/cm^3
A	=	area of trench (2.22)	m^2
F	=	fraction of floor through which contaminant can enter (1)	unitless
R	=	ideal gas constant (8.2E-05)	atm-m^3/mole-°K
T	=	average system absolute temperature (298)	°K
L_d	=	distance between trench bottom and groundwater ($L_d = L_{gw}$-D_{trench})	cm
L_{gw}	=	depth to groundwater	cm
D_{trench}	=	depth of trench (8 ft or 243.84 cm)	cm
ACH	=	air changes per hour (receptor-specific)	h^{-1}
V	=	volume of trench (5.42)	m^3
Por_{vad}	=	total soil porosity in vadose zone (0.44)	cm^3/cm^3
10^{-3}	=	conversion factor	L/cm^3
10^{4}	=	conversion factor	cm^2/m^2
3,600	=	conversion factor	sec/hr

However, PADEP calculates Henry's law constants for use in deriving statewide health standard VI screening levels assuming a local groundwater temperature of 11 °C rather than the standard 25 °C (PADEP 2015). Therefore, Equation {4} is modified to calculate deep-groundwater VFs for Construction and Utility Workers ($VF_{>15\text{-}cw}$ and $VF_{>15\text{-}uw}$) at local groundwater temperature as:

$$VF_{>15\_cw} \text{ or } VF_{>15\_uw} = \frac{H'_{@T_{gw}} \times D_{air} \times AC_{vad}^{3.33} \times A \times F \times 10^{-3}\ L/cm^3 \times 10^4\ cm^2/m^2 \times 3{,}600\ sec/hr}{L_d \times \left[ACH_{cw} \text{ or } ACH_{uw}\right] \times V \times Por_{vad}^{2}} \quad \{5\}$$

where:

$H'_{@Tgw}$	=	Henry's Law constant at groundwater temperature	unitless
ACH_{cs}	=	air changes per hour for Construction Worker (360)	h^{-1}
ACH_{us}	=	air changes per hour for Utility Worker (2)	h^{-1}

All other parameters are as defined for Equation {4}. It is assumed that the ratio of width to depth for a construction excavation is greater than 1. Due to the lesser restriction of air exchange in an excavation compared to a trench, VDEQ guidance (2014) recommends an air exchange ratio of 2/hour for Utility Workers (ACH_{uw}) and 360/hour for Construction Workers (ACH_{cw}).

EPCs for groundwater vapors in excavations and trenches where groundwater is deeper than 15 feet bgs were conservatively calculated for each groundwater well to account for the variation in the terrain and water table. Groundwater vapor EPCs for Construction and Utility Workers in portions of the Site where the groundwater table is deeper than 15 feet are detailed in Appendix F and summarized in Table 13 and Table 14, respectively.

4.5.2 Surface water

The COPCs selected for the Recreational Wader scenario were PCE and TCE (Appendix E). EPCs for the direct contact with surface water by a Recreational Wader were calculated for pumping and non-pumping time intervals, as discussed in Section 4.4.3. EPCs for the pumping and non-pumping scenarios for direct contact to surface water are detailed in Appendix G and summarized in Table 15.

4.6 Uncertainties related to exposure assessment

Although the Site groundwater has been well characterized, analytical data are subject to both systematic error (bias) and random error (imprecision). The RME EPCs are conservatively calculated from the core (zone of highest concentration) of each groundwater plume, and estimated as the lower of the 95% UCL on the mean of the data set and the maximum detected value. This approach is unlikely to result in underestimation of exposure, and likely to overestimate it. That the EPCs are assumed to remain constant over the entire exposure period is likely to further overestimate exposure.

Each of the assumptions made and parameter values used to estimate the magnitude of exposure for the human exposure scenarios considered has associated uncertainty. To ensure that potential risks to human health are not underestimated, most of these assumptions and values are deliberately intended to overestimate potential exposure. Direct and/or indirect inhalation pathways are complete for the worker scenarios, but no air or contemporary soil vapor data are

available. It is therefore necessary to rely on conservative, simplistic, and unvalidated vapor transport models for exposure evaluation (Sections 4.5.1 and 6.1.2). As such, they are likely to overestimate exposure.

Taken together, the many conservative assumptions are likely to result in net overestimation of exposure to the receptor populations considered in the HHRA, to an unknown but possibly significant degree in some cases.

5.0 Toxicity assessment

The toxicity assessment combines hazard identification and dose-response assessment to characterize the relationship between the magnitude of exposure to a COPC and the nature and magnitude of adverse health effects that may result from such exposure. EPA's Integrated Risk Information System (IRIS) is the official repository of consensus and recommended non-consensus human-health toxicity criteria. Each chemical is assigned a weight-of-evidence classification that expresses its potential for human carcinogenicity. Toxicity criteria and physicochemical parameter values for each COPC are provided in Table 16.

Chemical toxicity has traditionally been divided into two categories, carcinogenic and non-carcinogenic. Regulatory toxicity criteria and health risks are calculated differently for these two types of effects because they are based on different mechanistic assumptions and expressed in different units. The two approaches are briefly discussed below.

5.1 Toxicity criteria for carcinogenic effects

Cancers are generally defined as diseases of mutation affecting cell growth and differentiation. Evidence of human carcinogenicity of a chemical is derived from two sources: (1) chronic studies with laboratory animals, and (2) human epidemiology studies where an increased incidence of cancer is associated with exposure to the chemical. EPA typically assumes that negative epidemiological data are not evidence that a chemical is not carcinogenic in humans. Specific laboratory rodent species (typically rats and mice) are generally used in cancer protocols. EPA recommends that the weight-of-evidence classification be presented for each potential carcinogen to indicate the strength of evidence that it may be a human carcinogen (EPA 1986, 1989, 2005a). However, no distinction is made among classes of carcinogens in evaluation of potential human health risks.

Determining how to quantify potential human risks associated with exposure to carcinogens has been a major focus of efforts in mechanistic and regulatory toxicology since modern human health risk assessment began in the 1970s. At that time, the molecular events of chemical carcinogenesis were poorly understood, although the correlation of genotoxicity (the ability to cause direct or indirect damage to the cellular genetic material, DNA) with carcinogenicity was well-recognized. By analogy to radiation damage, which was thought to increase cancer risk with "one hit" to DNA,

genotoxic chemicals were assumed to exhibit a linear non-threshold (LNT) dose-response: that is, it is assumed that there are no cellular defense mechanisms, and that even one molecule of a chemical carcinogen could hit a critical molecule and cause cancer to develop (*e.g.*, Albert 1994). EPA historically extended that assumption to non-genotoxic carcinogens as well (EPA 1986), although current guidance calls for consideration of mode of action both for determining the conditions under which the chemical should be considered a cancer hazard for humans (human relevance), and for determining the appropriate low-dose extrapolation approach (EPA 2005a).

Because risks at the low levels of exposure usually encountered by humans are difficult to quantify directly by either animal or epidemiological studies, mathematical models are typically used to extrapolate from high experimental to low environmental doses. The slope of the extrapolated oral dose-response curve is used to calculate the oral cancer slope factor (SF), which defines the "plausible upper bound" theoretical incremental lifetime cancer risk per unit of carcinogen (in units of risk per milligram per kilogram per day [mg/kg-day^{-1}]) (EPA 1986). For inhalation exposures, the slope of the extrapolated dose-response curve is used to calculate the inhalation unit risk (IUR), in units of risk per micrograms per cubic meter ([µg/m^3]$^{-1}$). Because the LNT assumption implies some hypothetical risk at any level of exposure, the EPA defined a hundred-fold theoretical incremental lifetime "target risk range" of one additional cancer case in the lifetimes of a hypothetical population of a million people (0.000001 or 10^{-6}) to one additional cancer case in the lifetimes of a hypothetical population of ten thousand people 0.0001 or 10^{-4}) (EPA 1991). This range defines what the Agency considers "acceptable" increases in cancer risk over background, so low that no regulatory action to protect human health is required.

5.2 Toxicity criteria for non-carcinogenic effects

Non-carcinogenic RBSLs for long-term exposure scenarios are calculated using oral reference doses (RfDs) and inhalation reference concentrations (RfCs) developed by EPA. An RfD is an estimate, "with uncertainty spanning perhaps an order of magnitude" (EPA 2011b), of the daily lifetime exposure level to humans (expressed in units of mg of chemical/kg of body weight/day – mg/kg-day), including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects (EPA 1989). RfDs are usually derived from oral exposure studies with the most sensitive species, strain and sex of experimental animal known, the assumption being that humans are as sensitive as the most sensitive organism tested. RfCs are concentrations in air (in units of mg per cubic meter – mg/m^3) that an individual may be exposed to every day for a lifetime without harm, again "with uncertainty spanning perhaps an order of magnitude" (EPA 2011b).

EPA's process for calculating RfDs and RfCs for both non-carcinogenic and carcinogenic endpoints involves two steps: (1) identification of an appropriate point of departure (POD) from an animal toxicity or human epidemiological study, and (2) extrapolation from the POD to a safe level for human exposure using uncertainty factors of 1 to 10 to account for specific sources of variability

and uncertainty (EPA 2002a, 2012a). The RfD or RfC is then calculated from the selected POD by dividing it by the product of the five UFs.

RfDs and RfCs are based on the assumption that thresholds (exposure levels below which no adverse effect is expected) exist for non-carcinogenic effects, and incorporate uncertainty factors to account for the required extrapolations from animal studies and to ensure protection of sensitive human subpopulations. That is, the RfC/RfD is expected to be below the actual threshold for adverse effect in a sensitive subgroup. As mentioned previously, current cancer risk assessment guidance calls for consideration of mode of action as an indicator of potential human relevance. In contrast, the IRIS process for identifying RfCs and RfDs does not require demonstration of a causal link between chemical exposure and particular health effects in humans.

RfDs and RfCs are compared with exposure concentrations to calculate HQs (the ratio of daily dose or air concentration to RfD or RfC) and cumulative hazard indices (HIs) (sum of HQs for individual COPCs). HQs and HIs less than or equal to 1 indicate that adverse non-cancer effects are not likely to occur, and thus can be considered to have negligible hazard. Despite the "uncertainty spanning perhaps an order of magnitude" and variability inherent in RfDs and RfCs, they have traditionally been used as "bright lines" – that is, there has historically been no estimation of the range of non-cancer hazard.

5.3 COPCs lacking toxicity criteria

All of the COPCs selected for the HHRA have EPA-approved oral and/or inhalation toxicity criteria. Route-to-route extrapolation beyond that performed by EPA was not done. In accordance with PADEP guidance, the RfD for 1,3,5-trimethylbenzene (135TMB) was used for 1,2,4-trimethylbenzene (124TMB), and the RfC for 124TMB was used for 135TMB.

5.4 Uncertainties related to toxicity assessment

The primary focus in analyzing the uncertainty of health risk estimates is usually on the values assumed for exposure parameters. These uncertainties typically range from one to two orders of magnitude. However, the uncertainties associated with dose-response relationships and weight-of-evidence carcinogenicity classification may be much greater because extrapolation of high-dose animal bioassay or occupational exposure study results to estimate human risk at much lower levels of environmental exposure involves a number of conservative assumptions regarding effects thresholds, interspecific responses, high- to low-dose extrapolation, and route-to-route extrapolation. Although the uncertainty and variability associated with each endpoint and extrapolation are generally recognized to be substantial, representation of toxicity is usually limited to single point estimates derived using uniformly conservative assumptions, with little basis for determining their accuracy and precision. In particular, the assumptions and uncertainty factors embedded in such point estimates, and the effects and impacts these have on toxicity

values and the risk/hazard estimates resulting from their use, are obscured in the risk assessment process, and therefore neglected in risk management.

In its recent review of the EPA's IRIS process, the National Research Council (NRC) Committee to Review the IRIS Process stated, "IRIS-specific guidelines for consistent, coherent, and transparent assessment and communication of uncertainty remain incompletely developed. The inconsistent treatment of uncertainties remains a source of confusion and causes difficulty in characterizing and communicating uncertainty" (NRC 2014). The Committee recommended that "[u]uncertainty analysis should be conducted systematically and coherently in IRIS assessments. To that end, EPA should develop IRIS-specific guidelines to frame uncertainty analysis and uncertainty communication. Moreover, uncertainty analysis should become an integral component of the IRIS process" (NRC 2014).

To improve documentation and presentation of dose-response information, the Committee recommended presentation of two dose-response values, a central estimate and a lower-bound (more stringent) estimate, in order to provide risk assessors with a fuller range of available information (NRC 2014). EPA was also advised to develop guidelines for uncertainty analysis and communication to support the consistent and transparent treatment of uncertainties (NRC 2014). Until such practices are implemented, both carcinogenic and non-carcinogenic toxicity assessment must be regarded as more likely to over- than underestimate health risk, to an unknown but possibly very large degree.

5.4.1 Carcinogenic effects

The linearized multi-stage model for low-dose extrapolation most often used by EPA leads to an upper-bound estimate of risk (the 95% UCL of the modeled dose-response slope). Under the assumption of dose-response linearity with no threshold, the probability that the true potency is higher than that estimated is thus only 5 percent. Recognizing the inherent tendency of this policy to overestimate risk, the EPA qualified it by stating, "[s]uch an estimate [of carcinogenic potency]…does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero. The range of risks, defined by the upper limit given by the chosen model and the lower limit which may be as low as zero, should be explicitly stated" (EPA 1986).

Even more importantly, understanding of the molecular biological changes involved in tumor development has increased tremendously in the past several decades. It is now widely understood and accepted that cancer is the end result of a prolonged multi-step process, resulting from a complex interaction of environmental factors and individual susceptibility, by which a normal cell acquires genetic alterations in key signaling pathways that allow it to transform into a tumor. These characteristics can be acquired through either genetic changes (mutations) or epigenetic (non-genotoxic) changes (transcriptional or translational changes at the DNA, RNA, or protein levels), or both (Hanahan and Weinberg, 2000, 2011; Greenman *et al.* 2007; Jarabek *et al.* 2009;

Bell 2010). Based on current understanding, many scientists have concluded that the "one-hit" LNT model of chemical carcinogenesis may not be valid even for known genotoxic chemicals TCE (*e.g.*, Jarabek *et al.* 2009; Guérard *et al.* 2015; Thomas *et al.* 2015). It is therefore highly likely that carcinogenic toxicity criteria overestimate ILCR.

Finally, it is important to understand that the target risk levels used in risk assessment are not thresholds for "significant" cancer risk. In fact, they represent an increase in risk so minute that it could never be detected given the current average background cancer incidence in the U.S. of around 40% (0.4 or 400,000 x 10^{-6}) (American Cancer Society 2016). Adding the hypothetical incremental risk of one in a million (0.000001) to this background cancer risk results in a negligible increase in lifetime cancer risk – from 0.4 to 0.400001, an increase of only 0.0002%. In contemplating these extremely small incremental cancer risks, it is also important to recall that they are not actuarial risks, but rather hypothetical calculations, and based on highly conservative upper-bound estimates of the human carcinogenic potency of chemicals – the true value of which may be zero (EPA 1986).

5.4.2 Non-carcinogenic effects of TCE

Best known for its use as a solvent for cleaning and degreasing metal parts since the early 1900s, TCE has had numerous other uses, including as an anesthetic or analgesic, a heat-transfer medium, an extraction agent for fats and oils, as an intermediate in producing chlorofluorocarbons and other chemicals, and as an ingredient in many products for industrial and consumer use (Doherty 2000; Bakke *et al.* 2007; ATSDR 2014). Use in metal degreasing has declined due to increased environmental regulations governing TCE emissions, while use as a feedstock for HFC-134a has increased (ATSDR 2014). TCE also remains a major ingredient in a variety of arts & crafts, automotive, and home maintenance products (National Library of Medicine 2015). As a result of its widespread use, TCE is one of the most commonly found chemicals at National Priority List (NPL) sites, present at 1,046 of 1,770 NPL sites (59%) and ranking 16th on the ATSDR's 2015 Substance Priority List (ATSDR 2015). It was one of the most frequently detected chemicals in untreated domestic wellhead samples between 1985 and 2002 in the U.S. Geological Survey National Water-Quality Assessment, with a relatively large percentage of samples with concentrations greater than or within one order of magnitude of the MCL (Rowe *et al.* 2007).

The fact that the toxicological criteria for both the systemic and carcinogenic effects of TCE were "under review" by EPA from the late 1980s until 2011 has long complicated assessments of potential human health risk. Given the rapid evolution in understanding of molecular mechanisms of toxic action in intervening years, the EPA's 2001 draft *Trichloroethylene Health Risk Assessment: Synthesis and Characterization* (TCE HRA) (EPA 2001b) was expected to incorporate the best available science, as articulated through several iterations of the Agency's *Guidelines for Carcinogen Risk Assessment* (EPA 1996, 1999, 2003, 2005a). Despite these efforts, the draft TCE HRA (EPA 2001b) provoked strong criticism of its fundamental assumptions and methodologies by

scientists, including many of the outside experts who had participated in the lengthy reevaluation process (Rhomberg *et al.* 2001). As a result, the TCE toxicity criteria proposed in 2001 were withdrawn in 2002.

The final *Toxicological Review of Trichloroethylene* released in early September 2011 (EPA 2011c) is also highly controversial, in part because the non-cancer toxicity criteria are so low that they are encompassed by the acceptable cancer risk range (10^{-6} to 10^{-4}). Risk-specific doses associated with the SF (0.00002 – 0.002 mg/kg-day) overlap the RfD (0.0005 mg/kg-day), and risk-specific concentrations associated with the IUR (0.24 – 24 µg/m^3) overlap the RfC (2 µg/m^3). A dose rate equal to the RfD would incur a cancer risk of 0.0005 mg/kg-d x 0.046 (mg/kg-d)$^{-1}$ = 2 x 10^{-5}, and an exposure concentration equal to the RfC would incur a cancer risk of 2 µg/m^3 x 0.0000041 (µg/m^3)$^{-1}$ = 8 x 10^{-6}. Thus, non-cancer effects will drive TCE risk assessment when (as in Pennsylvania) the target cancer risk level is greater than 10^{-6}.

This unusual circumstance highlights the importance of carefully considering the uncertainties inherent in non-cancer toxicity criteria. Despite an abundance of research on the metabolism and target organ toxicity of TCE, its mechanisms of action have not yet been clearly elucidated. It is clear that metabolism is critical for its mutagenic, carcinogenic, and other adverse health effects; indeed, most organ-specific toxicity has been attributed to specific TCE metabolites (Lash *et al.* 2014). Thus, the considerable variability in TCE metabolism among inbred mouse strains correlates with differences in susceptibility to TCE toxicity (Bradford *et al.* 2011; Yoo *et al.* 2015). Rates of TCE metabolism are higher in rodents than in humans, and markedly higher in mice than in rats, suggesting that these animals are more sensitive to some forms of TCE toxicity than are humans (Lash *et al.* 2014; Yoo *et al.* 2015).

5.4.2.1 Overview of current TCE RfC/RfD derivation

Unlike the majority of extant RfCs and RfDs, which are based on a single critical effect, the RfC and RfD for TCE are based on three primary endpoints: congenital heart defects in rats (Johnson *et al.* 2003) decreased thymus weight in female mice (Keil *et al.* 2009) and developmental immunotoxicity in mice (Peden-Adams *et al.* 2006). Supporting information was provided by a National Toxicology Program (NTP) study showing toxic nephropathy in rats (NTP 1988). All of these endpoints were observed in oral dosing studies, necessitating route-to-route extrapolation to develop the RfC. The candidate RfDs/RfCs for these endpoints represent the lower bound of a 3,000-fold range of minimum candidate values for other effect domains (EPA 2011c). Most importantly, while the immunological endpoints were derived from studies involving relatively long-term dosing and relevant to the lifetime exposures for which RfCs/RfDs are protective, congenital heart defects occurred in the offspring of rats exposed during pregnancy (22 days) (Johnson *et al.* 2003).

Variable immunotoxic effects of TCE have been reported in non-guideline studies using inbred mouse strains, including the two studies selected by EPA for development of the RfC/RfD

conducted by a single academic laboratory (Peden-Adams *et al.* 2006, Keil *et al.* 2009). Peden-Adams *et al.* (2006) assessed the immunotoxicity of TCE in B6C3F1 mice exposed via drinking water (1,400 or 14,000 µg/L) from gestation day 0 to either 3 or 8 weeks of age. EPA (2011c) identified decreased plaque-forming cell (PFC) response and increased delayed-type hypersensitivity as the main effects. Thymus mass, a sensitive indicator of immunotoxicity, was not altered at either dose at either age. Although described as evaluating "developmental immunotoxicity," the study was not designed to distinguish responses attributable to prenatal exposure alone; in fact, all responses noted could have resulted from post-natal exposure only. The absence of a positive control group precludes evaluation of the potential biological significance of the observed responses.

In summarizing the evidence for the developmental immunotoxicity of TCE, EPA (2011c) acknowledged that "[c]onsistency in response in these animal studies was difficult to ascertain due to the variations in study design (*e.g.*, animal strain used, duration of exposure, treatment levels evaluated, timing of assessments, and endpoints evaluated). Likewise, the endpoints assessed in the few epidemiological studies that evaluated immunological outcomes following developmental exposures to TCE were dissimilar from those evaluated in the animal models, and so provided no clear cross-species correlation." The responses reported by Peden-Adams *et al.* (2006) were selected because they were most sensitive immune system responses noted in animal studies. Because none of the other studies that treated mice during immune system development assessed these same endpoints, these findings have not been confirmed, let alone demonstrated to be toxicologically significant or relevant to humans. Further, because the Peden-Adams laboratory bred their own B6C3F1 mice, the comparability of their results even with duplicate experiments conducted with mice obtained from a standard laboratory animal source would be questionable.

Keil *et al.* (2009) compared the effects of TCE exposure (1,400 or 14,000 µg/L in drinking water) in female New Zealand Black/New Zealand White (NZBWF1) mice (which spontaneously develop autoimmune disease) and B6C3F1 mice (which are not genetically prone to autoimmune disease) on the development of autoantibodies, other markers of autoimmune disease, and selected parameters of immunotoxicity. Animals were exposed from 9 to 36 and 39 weeks of age, respectively. Unlike the laboratory-bred B6C3F1 mice in the Peden-Adams *et al.* study, all of these mice were purchased from a commercial supplier. Unlike Peden-Adams *et al.*'s results, the B6C3F1 mice exhibited decreased thymus weights at both dose levels (Keil *et al.* 2009). The authors interpreted their results as suggesting that TCE did not contribute to the progression of autoimmune disease in autoimmune-prone mice, but led to increased expression of markers associated with autoimmune disease in a non-genetically prone mouse strain.

A critical question is, what relevance do these unsubstantiated results in an inbred mouse strain have to humans? An expert panel convened by the National Institute of Environmental Health Sciences to examine the role of the environment in the development of autoimmune disease did not consider TCE to be known or likely to have a role in development of autoimmune diseases in

humans (Parks *et al.* 2014). The panel noted that while TCE can exacerbate systemic autoimmunity in certain animal models, "…responses are often limited and transient" (Parks *et al.* 2014). They also opined (cogently) that future studies "should be shaped by what is observed in humans, not by what is possible in mice."

Johnson *et al.* (2003) reported increased incidence of cardiac anomalies in term rat fetuses whose mothers had been exposed to 1,500 to 1,100,000 µg/L TCE in drinking water. This endpoint was "the most sensitive developmental effect by far" (EPA 2011c). Although only study of heart malformations available for conducting dose-response analysis, EPA considered it to be supported by both studies in avian species and human epidemiological data (EPA 2011c). However, a number of commenters raised serious concerns regarding both the questionable quality of this study and interpretation of the epidemiological database for cardiac defects associated with TCE exposures. EPA's unprecedented amalgamation of this serious subacute endpoint into the chronic RfC and RfD also prompted much alarm and confusion as to how to protect short- as well as long-term exposures. To address the scientific issues, EPA convened a team of Agency scientists to update the analysis of the developmental cardiac toxicity data (EPA 2012b). With respect to epidemiological data, this review determined that "…overall, the studies could not establish that the association on [*sic*] TCE exposure and cardiac defects was causal." A more recent, rigorous, and objective review of the epidemiological literature regarding the association between congenital heart defects and exposure to TCE also concluded that available studies provide no substantive or consistent evidence link with TCE exposure (Bukowski 2014).

5.4.2.2 Development of a "multiple endpoint safety range" for non-carcinogenic effects of TCE

As noted previously, while scientific considerations clearly support a range of values for non-cancer (and cancer) toxicity criteria, in practice, typically only a single toxicity value is identified. The Alliance for Risk Assessment (ARA)[6] undertook a project to develop guidance to facilitate effective risk management decisions at TCE-contaminated sites (ARA 2013). Based on this work, Dourson *et al.* (2016) proposed a process to determine a range that is reflective of the uncertainty in the TCE RfC, similar in concept to the "acceptable" cancer risk range of 10^{-6} to 10^{-4} for ILCR. This well-documented "safety range" affords risk managers flexibility in the management and/or regulatory closure of sites, like fYNOP, where the non-cancer effects of TCE drive the risk assessment.

[6] ARA is a collaboration of organizations that fosters the development of technical chemical risk assessment products and services, through a team effort of specialists and organizations dedicated to protecting public health by improving the process and efficiency of risk assessment, and to increasing the capacity for developing risk values. Coordinating with Federal and State agencies whenever possible, ARA's goal is to develop risk assessments where up-to-date assessments by major governmental agencies on the topic of interest do not exist or are not near finalization (http://allianceforrisk.org/). Its steering committee includes representatives from the U.S. Army Corps of Engineers, the Agency for Toxic Substances and Disease Registry, the National Library of Medicine, Health Canada, and several State environmental agencies.

The uncertainties inherent in each of the candidate values used to calculate EPA's RfC were evaluated both qualitatively and quantitatively. A range consisting of floor, intermediate, and ceiling values was developed for each candidate RfC. "Floor" values are the candidate RfCs identified by EPA (2011), and "ceiling" values are the PODs for each RfC – the 99th percentile estimates of the human equivalent concentrations or human equivalent doses. The overall range of these values is 2 µg/m^3 (the current EPA RfC) to 63 µg/m^3. The floor and ceiling values with the highest overall confidence (3 µg/m^3 and 30 µg/m^3) were selected as a "multiple endpoint range of safety" (Dourson *et al.* 2016). This range is "…an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime" (Dourson *et al.* 2016). By definition, values within this range are equally protective; that is, "…toxicologists cannot differentiate the "safety" of any value within a range of 3 µg/m^3 to 30 µg/m^3, nor can they differentiate among hazard quotients (HQs) developed from any value within this range" (Dourson *et al.* 2016).

A similar approach can be taken to evaluate the current TCE RfD, which is based on the same primary and supporting studies. (In fact, all of the studies used to derive the RfC were oral studies, requiring route-to-route extrapolation.) The analogous overall safety range of RfD values is 0.0005 mg/kg-d (average of candidate RfDs) to 0.4 mg/kg-d (highest 99th percentile estimate of the human equivalent dose). The multiple endpoint range of safety for the TCE RfC is 1.5 to 15 times higher than the current EPA RfC; that for the RfD is 1 to 800 times the current EPA RfD. Based on this analysis, a conservative safety range of up to ten times the current TCE RfC/RfD values is suggested for use in the fYNOP HHRA. This ten-fold range in non-cancer toxicity criteria supports a corresponding acceptable "hazard range" of 1 to 10.

6.0 Calculation of RBSLs

Chemical-, pathway-, and medium-specific RBSLs for the fYNOP are defined as concentrations of COPCs in relevant media that are not expected to produce any adverse health effects under defined exposure conditions. The fYNOP RBSLs are calculated using EPA's toxicity criteria (Section 5.0), conservative modeling assumptions, and conservative exposure assumptions. As such, they are analogous to EPA's Regional Screening Levels (RSLs) (EPA Regions 3, 6, and 9, May 2016) and PADEP medium-specific concentrations (PADEP 2011), but based on certain Site-specific parameter values in the cases of the Construction and Utility Workers and Recreational Wader. Because the Commercial/Industrial Worker is a standard scenario, no Site-specific adjustments were applied. RBSLs are calculated with a target ILCR of 0.000001 (10^{-6}) and target HQ of 0.1.

Toxicity criteria and physicochemical parameter values for each COPC are listed in Table 16, and exposure parameter values for each receptor scenario are presented in Table 17. Other parameter values are provided in the text. Equations and other parameter values were derived from EPA guidance (EPA 2004, 2009, 2011a, 2014b, 2015c; EPA Regions 3, 6, and 9, May 2016) and Virginia

Department of Environmental Quality (VDEQ) guidance for evaluating workers' exposures in construction excavations and utility trenches (VDEQ 2014).

6.1 Groundwater

Calculation of groundwater RBSLs is detailed in Appendix H.

6.1.1 Commercial/Industrial Workers

Because the Commercial/Industrial Worker is a standard receptor scenario, the current EPA industrial RSLs (EPA Regions 3, 6, and 9, May 2016) are used as RBSLs for carcinogenic effects ($RBSL_{c/iw(c)}$) and non-carcinogenic effects ($RBSL_{c/iw(nc)}$) (Table 18).

6.1.2 Construction and Utility Workers

6.1.2.1 Direct contact (groundwater ≤15 feet bgs)

Construction and Utility Workers could directly contact groundwater in excavations in areas where the depth to groundwater is ≤ 15 feet bgs via both dermal and inhalation exposure routes. For this exposure pathway, the exposure parameter values for these scenarios are considered to be the same (Table 17).

6.1.2.1.1 Dermal contact

Equations {6} through {10} and dermal model parameter values were obtained from EPA's Regional Screening Levels website (EPA Regions 3, 6, and 9, May 2016). For inorganic COPCs, the Construction or Utility Worker dermal contact RBSL ($RBSL_{inorg\_derm\_cuw}$) is calculated as:

$$RBSL_{inorg\_derm\_cuw}\left[\frac{\mu g}{L}\right] = \frac{DA_{event\_cuw} \times 1{,}000\ cm^3/L}{K_p \times ET_{cuw}} \quad \{6\}$$

Parameter		Value	Units
DA_{event_cuw}	absorbed dose per event for Construction and Utility Workers	calculated (Table 19)	$\mu g/cm^2$-event
K_p	dermal permeability coefficient of COPC in water	chemical-specific (Table 16)	cm/hour

In the case of organic chemicals for which exposure time (ET_{cuw}) is less than or equal to the chemical-specific time to reach steady state (t^*) (Table 16), the Construction or Utility Worker dermal contact RBSL ($RBSL_{org\_derm\_cuw}$) is calculated as:

$$RBSL_{org\_derm\_cuw}\left[\frac{\mu g}{L}\right] = \frac{DA_{event\_cuw} \times 1{,}000\ cm^3/L}{2 \times FA \times K_p \times \sqrt{\frac{6 \times \tau_{event} \times ET_{cuw}}{\pi}}} \quad \{7\}$$

In the case of organic chemicals for which exposure time (ET_{cuw}) is greater than t^*, the Construction or Utility Worker dermal contact RBSL ($RBSL_{cuw\_org\_derm}$) is calculated as:

$$RBSL_{org\_derm\_cuw}\left[\frac{\mu g}{L}\right] = \frac{DA_{event\_cuw} \times 1{,}000\ cm^3/L}{FA \times K_p \times \left[\frac{ET_{cuw}}{1+B} + 2 \times \tau_{event} \times \left(\frac{1+3B+B^2}{(1+B)^2}\right)\right]} \quad \{8\}$$

Parameter		Value	Units
DA_{event_cuw}	absorbed dose per event	calculated per Equations {9} and {10} (Table 19)	µg/cm^2-event
K_p	dermal permeability coefficient	chemical-specific (Table 16)	cm/hour
τ_{event}	lag time per event	chemical-specific (Table 16)	hours/event
t*	time to reach steady state	chemical-specific (Table 16)	hours
FA	fraction absorbed from water	chemical-specific (Table 16)	unitless
B	ratio of permeability coefficient through the stratum corneum to permeability coefficient across the viable epidermis	chemical-specific (Table 16)	unitless

The absorbed dose per event ($DA_{event\_cuw(c)}$) for carcinogenic effects for all categories of chemicals is calculated as:

$$DA_{event\_cuw(c)}\left[\frac{\mu g}{cm^2\text{-event}}\right] = \frac{TR \times AT_c \times BW_{cuw} \times GIABS \times 1{,}000\ \mu g/mg}{ED_{cuw} \times EF_{cuw} \times SA_{cuw} \times SF_o} \quad \{9\}$$

and $DA_{event\_cuw(nc)}$ for non-carcinogenic effects of all COPC types is calculated as:

$$DA_{event\_cuw(nc)}\left[\frac{\mu g}{cm^2\text{-event}}\right] = \frac{THQ \times AT_{nc} \times BW_{cuw} \times RfD \times GIABS \times 1{,}000\ \mu g/mg}{ED_{cuw} \times EF_{cuw} \times SA_{cuw}} \quad \{10\}$$

Parameter		Value	Units
TR	target risk	see Table 17	unitless
THQ	target hazard quotient	see Table 17	unitless
AT_c	averaging time for carcinogens	see Table 17	days
AT_{nc}	averaging time for non-carcinogens	see Table 17	days
BW_{cuw}	body weight	see Table 17	kg
SA_{cuw}	exposed body surface area	see Table 17	cm^2
EF_{cuw}	exposure frequency	see Table 17	events/year
ED_{cuw}	exposure duration	see Table 17	years
ET_{cuw}	exposure time	see Table 17	hours
GIABS	gastrointestinal absorption fraction	chemical-specific (Table 16)	unitless
SF_o	oral cancer slope factor	chemical-specific (Table 16)	(mg/kg-day)$^{-1}$
RfD	reference dose	chemical-specific (Table 16)	mg/kg-day

Values for $DA_{event\_cuw(c)}$ and $DA_{event\_cuw(nc)}$ and carcinogenic and non-carcinogenic dermal contact RBSLs are presented in Table 19.

6.1.2.1.2 Vapor inhalation

Equations {11} through {16} and vapor model parameter values were obtained from VDEQ (2014). For carcinogenic effects, the Construction or Utility Worker direct contact inhalation RBSLs ($RBSL_{dir\_inhal\_cw(c)}$ and $RBSL_{dir\_inhal\_uw(c)}$) are calculated as:

$$RBSL_{dir\_inhal\_cw(c)} \text{ or } RBSL_{dir\_inhal\_uw(c)} \left[\frac{\mu g}{L}\right] = \frac{TR \times AT_c}{EF_{cuw} \times ED_{cuw} \times ET_{cuw} / 24 \times IUR \times \left[VF_{\leq 15\_cw} \text{ or } VF_{\leq 15\_uw}\right]} \quad \{11\}$$

For non-carcinogenic effects, the Construction or Utility Worker direct contact inhalation RBSLs ($RBSL_{dir\_inhal\_cw(nc)}$ and $RBSL_{dir\_inhal\_uw(nc)}$) are calculated as:

$$RBSL_{dir\_inhal\_cw(nc)} \text{ or } RBSL_{dir\_inhal\_uw(nc)} \left[\frac{\mu g}{L}\right] = \frac{THQ \times AT_{nc} \times RfC \times 1{,}000\ \mu g/mg}{EF_{cuw} \times ED_{cuw} \times ET_{cuw} / 24 \times \left[VF_{\leq 15\_cw} \text{ or } VF_{\leq 15\_uw}\right]} \quad \{12\}$$

Parameter		Value	Units
TR	target risk	see Table 17	unitless
THQ	target hazard quotient	see Table 17	unitless
AT_c	averaging time for carcinogens	see Table 17	days
AT_{nc}	averaging time for non-carcinogens	see Table 17	days
EF_{cuw}	exposure frequency	see Table 17	events/year
ED_{cuw}	exposure duration	see Table 17	years
ET_{cuw}	exposure time	see Table 17	hours
IUR	inhalation unit risk	chemical-specific (Table 16)	$(\mu g/m^3)^{-1}$
RfC	reference concentration	chemical-specific (Table 16)	mg/m^3
$VF_{\leq 15}$	volatilization factor where depth to groundwater is ≤15' bgs	calculated per Equation {13}	L/m^3

Due to the lesser restriction of air exchange in an excavation compared to a trench, VDEQ guidance (2014) recommends that the volatilization factors for groundwater ≤15 feet bgs ($VF_{\leq 15\_cw}$ and $VF_{\leq 15\_uw}$) be calculated using an hourly air exchange rate (ACH) of 360 per hour for Construction Workers (ACH_{cw}) and 2 per hour for Utility Workers (ACH_{uw}):

$$VF_{\leq 15\_cw} \text{ or } VF_{\leq 15\_uw} \left[\frac{L}{m^3}\right] = \frac{K \times A \times F \times 10^{-3}\ L/cm^3 \times 10^4\ cm^2/m^2 \times 3{,}600\ sec/hr}{\left[ACH_{cw} \text{ or } ACH_{uw}\right] \times V} \quad \{13\}$$

where

$$K = \frac{1}{\frac{1}{k_L} + \frac{1}{H'_{@T_{gw}} \times k_G}} \quad \{14\}$$

$$k_L = \left(\frac{MW_{O_2}}{MW}\right)^{0.5} \times \frac{T_{gw}}{298} \times k_L, O_2 \quad \{15\}$$

$$k_G = \left(\frac{MW_{H_2O}}{MW}\right)^{0.335} \times \left(\frac{T_{gw}}{298}\right)^{1.005} \times k_G, H_2O \quad \{16\}$$

[Note that Equation {14} is modified from its generic form in VDEQ guidance to reflect the assumed groundwater temperature of 11° C (284° K).]

Parameter		Value	Units
K	overall mass transfer coefficient	chemical-specific (Table 16)	cm/sec
k_L	liquid-phase mass transfer coefficient	chemical-specific (Table 16)	cm/sec
H'@$_{Tgw}$	Henry's Law constant at groundwater temperature	chemical-specific (Table 16)	unitless
k_G	gas-phase mass transfer coefficient	chemical-specific (Table 16)	cm/sec
MW	molecular weight	chemical-specific (Table 16)	g/mol
A	area of the trench	2.22	m^2
F	fraction of floor through which contaminant can enter	1	unitless
ACH_{cw}	air changes per hour (construction excavation)	360	h^{-1}
ACH_{uw}	air changes per hour (utility trench)	2	h^{-1}
V	volume of trench	5.42	m^3
T_{gw}	groundwater temperature	284	°K
MW_{O2}	molecular weight of O_2	32	g/mol
k_L, O_2	liquid-phase mass transfer coefficient of oxygen at 25°C	0.002	cm/sec
MW_{H2O}	molecular weight of water	18	g/mol
k_G,H_2O	gas-phase mass transfer coefficient of water vapor at 25°C	0.833	cm/sec

Values for $VF_{\leq 15\_cw}$ and $VF_{\leq 15\_uw}$ and carcinogenic and non-carcinogenic direct contact inhalation RBSLs for Construction and Utility Workers are presented in Table 20.

6.1.2.1.3 Combined exposure routes

For volatile COPCs, combined dermal and inhalation RBSLs are calculated. For carcinogenic effects, the Construction and Utility Worker direct contact inhalation RBSLs ($RBSL_{comb\_cw(c)}$ and $RBSL_{comb\_uw(c)}$) are calculated as:

$$RBSL_{comb\_cw(c)} \text{ or } RBSL_{comb\_uw(c)}\left[\frac{\mu g}{L}\right] = \frac{1}{\frac{1}{RBSL_{org\_derm(c)}} + \frac{1}{\left[RBSL_{dir\_inhal\_cw(c)} \text{or } RBSL_{dir\_inhal\_uw(c)}\right]}} \quad \{17\}$$

For non-carcinogenic effects, the Construction or Utility Worker direct contact inhalation RBSLs ($RBSL_{comb\_cw(nc)}$ and $RBSL_{comb\_uw(nc)}$) are calculated as:

$$RBSL_{comb\_cw(nc)} \text{ or } RBSL_{comb\_uw(nc)}\left[\frac{\mu g}{L}\right] = \frac{1}{\frac{1}{RBSL_{org\_derm(c)}} + \frac{1}{\left[RBSL_{dir\_inhal\_cw(nc)} \text{or } RBSL_{dir\_inhal\_uw(nc)}\right]}} \quad \{18\}$$

Carcinogenic and non-carcinogenic combined direct contact RBSLs for the Construction and Utility Worker scenarios are presented in Table 21.

6.1.2.2 Indirect contact (groundwater >15 to 75 feet bgs)

The indirect vapor inhalation RBSLs for carcinogenic effects ($RBSL_{indir\_inhal\_cw(c)}$ and $RBSL_{indir\_inhal\_uw(c)}$) and non-carcinogenic effects ($RBSL_{indir\_inhal\_cw(nc)}$ and $RBSL_{indir\_inhal\_uw(nc)}$) are calculated based on EPA toxicity criteria and exposure assumptions from VDEQ guidance (VDEQ 2014).

For carcinogenic effects, the Construction or Utility Worker indirect contact inhalation RBSL ($RBSL_{indir\_inhal\_cuw(c)}$ is calculated as:

$$RBSL_{indir\_inhal\_cuw(c)}\left[\frac{\mu g}{m^3}\right] = \frac{TR \times AT_c}{EF_{cuw} \times ED_{cuw} \times IUR \times ET_{cuw} / 24} \quad \{19\}$$

For non-carcinogenic effects, the Construction or Utility Worker direct contact inhalation RBSLs $RBSL_{indir\_inhal\_uw(nc)}$) is calculated as:

$$RBSL_{indir\_inhal\_cuw(nc)}\left[\frac{\mu g}{m^3}\right] = \frac{THQ \times AT_{nc} \times RfC \times 1{,}000\ \mu g/mg}{EF_{cuw} \times ED_{cuw} \times ET_{cuw} / 24} \quad \{20\}$$

Parameter		Value	Units
TR	target risk	see Table 17	unitless
THQ	target hazard quotient	see Table 17	unitless
AT_c	averaging time for carcinogens	see Table 17	days
AT_{nc}	averaging time for non-carcinogens	see Table 17	days
EF_{cuw}	exposure frequency	see Table 17	events/year
ED_{cuw}	exposure duration	see Table 17	years
ET_{cuw}	exposure time	see Table 17	hours
IUR	inhalation unit risk	chemical-specific (Table 16)	$(\mu g/m^3)^{-1}$
RfC	reference concentration	chemical-specific (Table 16)	mg/m^3

Carcinogenic and non-carcinogenic indirect contact inhalation RBSLs for Construction and Utility Workers are presented in Table 22.

6.2 Surface water

Calculation of surface water RBSLs is detailed in Appendix I. Exposure of Recreational Waders to the COPCs identified for them in Codorus Creek surface water (PCE and TCE) could occur via both incidental ingestion and dermal contact. Equations {23} through {30} were obtained from EPA's Regional Screening Levels website (EPA Regions 3, 6, and 9 2015). The EPA has determined that TCE may cause kidney cancer via a mutagenic mode of action (MOA), as well as two other cancer types via non-mutagenic MOAs (EPA 2011). EPA recommends that lifetime cancer risks associated with chemicals thought to cause cancer via a mutagenic MOA should incorporate age-dependent adjustment factors (ADAFs) reflecting the assumed greater sensitivity of younger age groups. For children between the ages of 2 and 16 years, which includes the age range assumed for Recreational Waders, the recommended ADAF is 3 (EPA 2005). Since TCE requires the use of different toxicity values for cancer and mutagen equations, EPA calculated oral toxicity value

adjustment factors for carcinogenic (CAF_o) and mutagenic (MAF_o) effects (EPA Regions 3, 6, and 9 2015):

$$CAF_o\,[\text{unitless}] = \frac{\left(SF_o\ (\text{NHL}) + SF_o\ (\text{liver})\right)}{\text{Adult-based } SF_o} = \frac{0.037\ \text{kg-day/mg}}{0.046\ \text{kg-day/mg}} = 0.804 \quad \{21\}$$

$$MAF_o\,[\text{unitless}] = \frac{SF_o\ (\text{kidney})}{\text{Adult-based } SF_o} = \frac{0.0093\ \text{kg-day/mg}}{0.046\ \text{kg-day/mg}} = 0.202 \quad \{22\}$$

Parameter		Value	Units
CAF_o	oral carcinogenic adjustment factor for TCE	see Table 17	unitless
MAF_o	oral mutagenic adjustment factor for TCE	see Table 17	unitless
SF_o	adult-based oral cancer slope factor	see Table 17	$(mg/kg\text{-}day)^{-1}$
SF_o (NHL)	Oral cancer slope factor for non-Hodgkins lymphoma (NHL)	see Table 17	$(mg/kg\text{-}day)^{-1}$
SF_o (liver)	Oral cancer slope factor for liver tumor	see Table 17	$(mg/kg\text{-}day)^{-1}$
SF_o (kidney)	Oral cancer slope factor for kidney tumor	see Table 17	$(mg/kg\text{-}day)^{-1}$

Values for $DA_{event\_rec(c)}$ and $DA_{event\_rec(nc)}$ and carcinogenic and non-carcinogenic incidental ingestion, dermal contact, and combined RBSLs are presented in Table 23.

6.2.1 Incidental ingestion

For carcinogenic effects of PCE, the Recreational Wader oral RBSL ($RBSL_{rec\_oral\_PCE(c)}$) is calculated as:

$$RBSL_{rec\_oral\_PCE(c)}\left[\frac{\mu g}{L}\right] = \frac{TR \times AT_c \times BW_{rw} \times 1{,}000\ \mu g/mg}{SF_o \times EF_{rec} \times ED_{rec} \times ET_{rec} \times IR_{w\_rec}} \quad \{23\}$$

For carcinogenic and mutagenic effects of TCE, the Recreational Wader oral RBSL ($RBSL_{rec\_oral\_TCE(c)}$) is calculated as:

$$RBSL_{rec\_oral\_TCE(c)}\left[\frac{\mu g}{L}\right] = \frac{TR \times AT_c \times BW_{rec} \times 1{,}000\ \mu g/mg}{SF_o \times EF_{rec} \times ED_{rec} \times ET_{rec} \times \left[\left(CAF_o \times IR_{w\_rec}\right) + \left(MAF_o \times IR_{w\_rec} \times 3\right)\right]} \quad \{24\}$$

For non-carcinogenic effects of both compounds, the Recreational Wader oral RBSL ($RBSL_{rec\_oral\_nc}$) is calculated as:

$$RBSL_{rec\_oral\_nc}\left[\frac{\mu g}{L}\right] = \frac{THQ \times BW_{rec} \times AT_{nc} \times RfD_o \times 1{,}000\ \mu g/mg}{EF_{rec} \times ED_{rec} \times ET_{rec} \times IR_{w\_rec}} \quad \{25\}$$

Parameter		Value	Units
TR	target risk	see Table 17	unitless
THQ	target hazard quotient	see Table 17	unitless
AT_c	averaging time for carcinogens	see Table 17	days
AT_{nc}	averaging time for non-carcinogens	see Table 17	days
EF_{rec}	exposure frequency	see Table 17	events/year
ED_{rec}	exposure duration	see Table 17	years
ET_{rec}	exposure time	see Table 17	hours
IR_{w_rec}	incidental surface water ingestion rate	see Table 17	liters/hour
CAF_o	oral carcinogenic adjustment factor for TCE	see Table 17	unitless
MAF_o	oral mutagenic adjustment factor for TCE	see Table 17	unitless
SF_o	oral cancer slope factor	chemical-specific (Table 16)	$(mg/kg\text{-}day)^{-1}$
RfD	reference dose	chemical-specific (Table 16)	mg/kg-day

6.2.2 Dermal contact

Because $ET_{rec} < t^*_{rec\_PCE}$, the dermal carcinogenic RBSL for PCE ($RBSL_{rec\_dermal\_PCE(c)}$) is calculated as:

$$RBSL_{rec\_dermal\_PCE(c)}\left[\frac{\mu g}{L}\right] = \frac{DA_{event\_rec\_PCE(c)} \times 1{,}000\ cm^3/L}{2 \times FA \times K_p \times \sqrt{\frac{6 \times \tau_{event\_rec\_PCE} \times ET_{rec}}{\pi}}} \quad \{26\}$$

where

$$DA_{event\_rec\_PCE(c)}\left[\frac{\mu g}{cm^2\text{-event}}\right] = \frac{TR \times AT_{nc} \times BW_{rec} \times GIABS \times 1{,}000\ \mu g/mg}{SF_o \times EF_{rec} \times ED_{rec} \times SA_{rec}} \quad \{27\}$$

Because $ET_{rec} > t^*_{rec\_TCE}$, the dermal carcinogenic RBSL for TCE ($RBSL_{rec\_dermal\_TCE(c)}$) is calculated as:

$$RBSL_{rec\_dermal\_TCE(c)}\left[\frac{\mu g}{L}\right] = \frac{DA_{event\_rec\_TCE(c)} \times 1{,}000\ cm^3/L}{FA \times K_p \times \left[\frac{ET_{rec}}{1+B} + 2 \times \tau_{event\_rec\_TCE} \times \left(\frac{1+3B+B^2}{(1+B)^2}\right)\right]} \quad \{28\}$$

where dermal exposure is adjusted for the assumed greater sensitivity of Recreational Waders to the mutagenic effects of TCE:

$$DA_{event\_rec\_TCE(c)}\left[\frac{\mu g}{cm^2\text{-event}}\right] = \frac{TR \times AT_c \times BW_{rec} \times GIABS \times 1{,}000\ \mu g/mg}{SF_o \times EF_{rec} \times ED_{rec} \times \left[\left(CAF_o \times SA_{rec}\right) + \left(MAF_o \times SA_{rec} \times ADAF\right)\right]} \quad \{29\}$$

The non-carcinogenic dermal RBSL for PCE ($RBSL_{rec\_dermal\_PCE(nc)}$) for the Recreational Wader is also calculated using Equation {26}, and the non-carcinogenic dermal RBSL for TCE ($RBSL_{rec\_dermal\_TCE(nc)}$) for the Recreational Wader is also calculated using Equation {28}. For both compounds, the non-carcinogenic $DA_{event\_rec(nc)}$ is calculated as:

$$DA_{event\_rec(nc)}\left[\frac{\mu g}{cm^2\text{-event}}\right]=\frac{THQ \times AT_{nc} \times BW_{rec} \times RfD_o \times GIABS \times 1{,}000\ \mu g/mg}{EF_{rec} \times ED_{rec} \times SA_{rec}} \quad \{30\}$$

Parameter		Value	Units
TR	target risk	see Table 17	unitless
THQ	target hazard quotient	see Table 17	unitless
AT_c	averaging time for carcinogens	see Table 17	days
AT_{nc}	averaging time for non-carcinogens	see Table 17	days
BW_{rec}	body weight	see Table 17	kg
SA_{rec}	exposed body surface area	see Table 17	cm^2
EF_{rec}	exposure frequency	see Table 17	events/year
ED_{rec}	exposure duration	see Table 17	years
ET_{rec}	exposure time	see Table 17	hours
ADAF	age-dependent adjustment factor	see Table 17	unitless
GIABS	gastrointestinal absorption fraction	chemical-specific (Table 16)	unitless
SF_o	oral cancer slope factor	chemical-specific (Table 16)	$(mg/kg\text{-}day)^{-1}$
RfD	reference dose	chemical-specific (Table 16)	mg/kg-day
DA_{event_rec}	absorbed dose per event	chemical-specific (Table 16)	$\mu g/cm^2$-event
K_p	dermal permeability coefficient	chemical-specific (Table 16)	cm/hour
τ_{event}	lag time per event	chemical-specific (Table 16)	hours/event
FA	fraction absorbed from water	chemical-specific (Table 16)	unitless
B	ratio of permeability coefficient through the stratum corneum to permeability coefficient across the viable epidermis	chemical-specific (Table 16)	unitless

6.2.3 Combined exposure routes

The combined oral and dermal carcinogenic RBSLs ($RBSL_{rec\_comb(c)}$) for the Recreational Wader for both PCE and TCE (Table 23) are calculated as:

$$RBSL_{rec\_comb(c)}\left[\frac{\mu g}{L}\right]=\frac{1}{\frac{1}{RBSL_{rec\_oral(c)}}+\frac{1}{RBSL_{rec\_dermal(c)}}} \quad \{31\}$$

The combined oral and dermal non-carcinogenic RBSLs ($RBSL_{rec\_comb(nc)}$) for the Recreational Wader for both PCE and TCE (Table 23) are calculated as:

$$RBSL_{rec\_comb(nc)}\left[\frac{\mu g}{L}\right]=\frac{1}{\frac{1}{RBSL_{rec\_oral(nc)}}+\frac{1}{RBSL_{rec\_dermal(nc)}}} \quad \{32\}$$

7.0 Risk characterization

Risk characterization is the culmination of the HHRA process, combining the results of the toxicity and exposure assessments to provide numerical estimates of potential health risks to defined receptor populations. In the risk characterization, RBSLs for each COPC, medium, receptor, and

pathway combination are compared with EPCs in corresponding media to calculate HQs for non-carcinogenic effects and ILCRs for carcinogenic effects.

Spreadsheets showing risk calculations are presented in Appendix K.

7.1 Calculation of incremental lifetime cancer risk

Estimated theoretical ILCRs for each receptor population/pathway are calculated as:

$$\text{ILCR} = \frac{\text{CTE}_{\text{COPC/medium}} \text{ or } \text{RME}_{\text{COPC/medium}}}{\text{RBSL}_{\text{COPC/medium/pathway/scenario}}} \times \text{Target risk level} \quad \{33\}$$

Individual ILCRs are then summed to calculate a cumulative ILCR for each receptor population/pathway:

$$\text{Cumulative ILCR} = \Sigma \frac{\text{CTE}_{\text{COPC/medium}} \text{ or } \text{RME}_{\text{COPC/medium}}}{\text{RBSL}_{\text{COPC/medium/pathway/scenario}}} \times \text{Target risk level} \quad \{34\}$$

EPA and PADEP consider theoretical ILCRs of less than 10^{-4} (rounded to one significant figure) to be acceptable (EPA 1991; PADEP 2002, 2015). If the cumulative risk for a pathway is greater than 10^{-4}, then remedial action or further investigation may be considered.

7.2 Calculation of non-cancer hazard

The degree of exceedance of non-cancer thresholds (*i.e.*, the THQ of 1) is estimated by calculating the HQ (ratio of the representative COPC concentration in an exposure medium to the corresponding RBSL):

$$\text{HQ} = \frac{\text{CTE}_{\text{COPC/medium}} \text{ or } \text{RME}_{\text{COPC/medium}}}{\text{RBSL}_{\text{COPC/medium/pathway/scenario}}} \times \text{Target hazard quotient} \quad \{35\}$$

HQs for each COPC/medium/receptor scenario/pathway are summed to derive non-carcinogenic HIs for each exposure pathway in each receptor scenario:

$$\text{HI} = \Sigma \frac{\text{CTE}_{\text{COPC/medium}} \text{ or } \text{RME}_{\text{COPC/medium}}}{\text{RBSL}_{\text{COPC/medium/pathway/scenario}}} \times \text{Target hazard quotient} \quad \{36\}$$

If the HI exceeds 1 (rounded to one significant figure)[7], then constituents are grouped according to target organs or effects, and the HI recalculated. Non-cancer target organs and critical effects for COPCs are listed in Table 24. Summary of non-cancer target organs and critical effects of COPCs. Potential non-cancer hazards are considered acceptable for scenarios where target organ-specific HIs do not exceed 1.

[7] Note that rounding of HQs and HIs to one significant figure means that values between 0.95 and 1.49 are equal to 1.

7.3 Commercial/Industrial Workers

Commercial/Industrial Workers are or could be present on-Site in LUAs #2 and #3, and off-Site in LUA #6 (no COPCs were selected for this scenario in LUA #5). Their potential exposure route is indoor inhalation of COPC vapors intruding from groundwater. As shown in Table 25, total ILCR does not exceed the target risk level in LUA #2, but the TCE HQ is 4 under RME assumptions. In LUA #3, total ILCR does not exceed the target risk level under either exposure scenario, but both CTE and RME TCE HQs are elevated (Table 26). Total ILCR and HI under both CTE and RME assumptions are below respective target levels in LUA #6 (Table 27). Thus, intrusion of COPC vapors from groundwater is concluded to pose insignificant risk for Commercial/Industrial Workers off-Site.

7.4 Construction and Utility Workers

Construction and Utility Workers could be present in on-Site LUAs #1 – #3 and off-Site LUAs #5 and #6. They may be exposed to groundwater COPCs both directly (via dermal contact and inhalation in excavations/trenches where groundwater depth is less than 15 feet bgs) and indirectly (via inhalation from groundwater 15 to 75 feet bgs).

7.4.1 Direct contact with shallow groundwater

7.4.1.1 Construction Worker

ILCRs for both CTE and RME scenarios are lower than 10^{-4} in all areas, and non-cancer HIs are below 1 in LUAs #1, #2, #5, and #6 under both CTE and RME (Table 28 and Table 29). HIs exceed 1 in LUAs #1-3 and LUA #3. In LUA #1-3, the driving COPC is 124TMB. No individual rounded HQ exceeds 1, but the HQ for 124TMB is equal to 1 under RME. Because the HI exceeds 1, target organs for each COPC in shallow groundwater were identified and target organ-specific HIs calculated (Table 30). Although both benzene and 124TMB affect the blood, their critical effects differ, as does the quality of supporting data. Benzene's RfD and RfC are based on the same critical effect, decreased lymphocyte count in humans, resulting from its well-known myelotoxicity (EPA 2002c). Both criteria for benzene are accorded high confidence (Table 24). In contrast, the RfC for 124TMB, which is accorded low confidence, is based on decreased clotting time in female but not male rats exposed to 1,230,000 µg/m^3, with no evidence of myelotoxicity (Korsak 2000, EPA 2007). It is therefore considered conservative but not appropriate to sum the HQs of benzene and 124TMB. Both 124TMB and naphthalene affect body weight, so their HQs were summed. The other COPCs' target organs included liver and/or kidney, and so they were conservatively summed. As shown in Table 30, none of the resultant target-organ specific His exceed 1. Accordingly, the CTE and RME HI exceedances in LUAs #1 and 3 combined are not considered indicative of potential for adverse health effects.

In LUA #3, the driving COPCs are TCE, *cis*-1,2-dichloroethene (*cis*-12DCE), PCE, and bis-2-ethylhexylphthalate, in descending order. As shown in Table 24, these chemicals have different

target organs, so their HQs are not additive. The TCE HQ is 6 under CTE (Table 28) and 10 under RME (Table 29). The RME HQ for *cis*-12DCE is also greater than 1.

7.4.1.2 Utility Worker

Because of the assumed lower air exchange rate in utility trenches vs. construction excavations, ILCRs and HIs for the Utility Worker are proportionally higher than those calculated for Construction Workers (Table 31 and Table 32). The ILCR in LUA #1-3 was equal to 10^{-4} under CTE and 2 x 10^{-4} under RME, the only exceedance of the target cancer risk in this assessment (Table 32). The driving COPC is benzene, and the driving exposure route is inhalation.

Total HIs were greater than 1 in all LUAs under the RME scenario (Table 32), and all except LUA #5 under the CTE (Table 31). Multiple HQs are greater than 1, and the driving COPC is TCE in all areas except LUA #1-3, where petroleum-related COPCs are dominant. Driving chemicals 124TMB, benzene, and naphthalene in LUA #1-3 have different target organs, but as their individual HQs exceed 1 under both CTE and RME assumptions, these results indicate a need for reasonable precautions in planning and conducting intrusive activities in this area.

Chlorinated COPCs TCE, PCE, cis-12DCE, 11DCE, and vinyl chloride are drivers in LUA #3 under both CTE and RME, resulting in the highest HI calculated in the HHRA. The target organ for the latter two COPCs is liver, so it is appropriate to sum their HQs; the other COPCs have different target organs (Table 24). However, as in LUA #1-3, the magnitude of the calculated HQs indicates a need for reasonable care in planning and conducting intrusive activities.

TCE is the only COPC with an HQ greater than 1 in LUA #1, #2, #5 (RME only), and #6, where the HIs are slightly higher under pumping vs. non-pumping conditions.

7.4.2 Inhalation of vapors from deep groundwater

As shown in Table 33 and Table 34, neither ILCR nor HI exceed targets under CTE or RME for Construction Workers. These results indicate no concern for this exposure pathway. Similarly, neither ILCR nor HI exceed targets under CTE or RME for Utility Workers (Table 35 and Table 36). These results indicate no concern for this exposure source and pathway.

7.5 Recreational Wader

Recreational Waders could be exposed to COPCs PCE and TCE in the affected segment of Codorus Creek. Total ILCR and HI for both CTE and RME scenarios under both pumping and non-pumping conditions are well below respective target levels (Table 37). Thus, Codorus Creek surface water is concluded to pose insignificant risk to this receptor population.

7.6 Hypothetical future residential development

7.6.1 Potable use of groundwater

For each COPC identified in Table 7, the maximum detected concentration in each LUA was compared to a putative cleanup goal to provide a screening-level estimate of the degree to which conditions exceed regulatory standards. The preferred putative cleanup goal is an MCL; for COPCs lacking MCLs, current (May 2016) EPA tap water RSLs (TR = 10^{-4}, THQ = 1) were used.

As shown in Table 38, the ratio of maximum COPC concentration to putative cleanup goal (rounded to one significant figure) exceeded 1 for all COPCs in at least one LUA except 4-chloroaniline, isopropylbenzene, benzo[*a*]anthracene, benzo[*b*]fluoranthene, indeno[*1,2,3-cd*]pyrene, nickel, silver, vanadium, and zinc. Maximum rounded ratios in each LUA range from 10 in LUA #5 (due to PCE) to 7,000 in LUA #3 (due to TCE). PCE concentrations exceeded the MCL by factors of around 10 to 4,000, and TCE concentrations exceeded by factors of around 4 to 7,000. The fact that maximum concentrations of COPCs exceed regulatory standards and/or risk-based screening levels indicates that potable use of groundwater both on-Site and immediately off-Site in LUAs #5 and #6 would not be appropriate.

7.6.2 Residential vapor intrusion

For each COPC identified in Table 8, the maximum detected concentration on- and off-Site in LUAs #5 and #6 was compared to a putative cleanup goal to provide a screening-level estimate of the degree to which conditions exceed regulatory standards. The preferred putative cleanup goal is an MCL; for COPCs lacking MCLs, current (May 2016) EPA residential VISLs (TR = 10^{-4}, THQ = 1) were used. In accordance with PADEP guidance (PADEP 2015), the VISL spreadsheet was modified to set the groundwater temperature to 11° C, and the residential groundwater-to-indoor air attenuation factor to 0.0012.

As shown in Table 39, the ratio of maximum COPC concentration to putative cleanup goal (rounded to one significant figure) exceeded 1 for all COPCs except 1,1-dichloroethane, methyl *tert*-butyl ether, and naphthalene, with ratios considerably higher on-Site than off-Site. The highest ratios, exceeding 1,000, were due to benzene and TCE. The fact that maximum concentrations of COPCs exceed regulatory standards and/or risk-based screening levels indicates that residential development on-Site and immediately off-Site in LUAs #5 and #6 would not be appropriate.

7.7 Uncertainties related to risk characterization

The risk characterization process combines exposure and toxicity information to develop an estimate of the screening-level cancer risks and non-cancer hazards that may be posed by site-related COPCs to defined receptor populations. Like all modeling efforts, the risk assessment process relies on a set of assumptions and estimates with varying degrees of accuracy and validity.

Major sources of uncertainty in risk assessment include (1) adequacy of the data base, (2) natural variability in parameter values (*e.g.*, differences in body weight in a group of people), (3) lack of knowledge about basic physical, chemical, and biological properties and processes (*e.g.*, the affinity of a chemical for soil, its solubility in water), (4) assumptions in the models used to estimate key inputs (*e.g.*, dose-response models), and (5) measurement error. Underestimation of potential exposure and risk is avoided through use of upper-bound values for most parameters, including EPCs, exposure parameters, and vapor models. The greatest single source of uncertainty in risk assessment is the human relevance of effects manifested in animal studies and chemicals' dose-response relationships.

As mentioned previously (Section 5.2), HQs less than or equal to 1 (rounded to one significant figure) indicate that adverse non-cancer effects are unlikely. It is important to note, however, that HQs greater than 1 are not statistical probabilities of harm. That is, an HQ of 10 does not mean that the hazard is 10 times greater than an HQ of 1. Further, the level of concern does not increase linearly or to the same extent as HQs increase above one for different COPCs because RfDs and RfCs do not generally have equal accuracy or precision and are generally not based on the same severity of effect. Thus, an HQ of 10 for one COPC may not have the same implication (in terms of nature and magnitude of hazard) as another COPC with the same HQ (EPA 2015b).

Due to the variability in endpoints used to derive RfDs and RfCs, the acceptability of exceedances must be evaluated on a case-by-case basis, considering such factors as the confidence level of the assessment, the size of the uncertainty factors used, the slope of the dose-response curve, the magnitude of the exceedance, and the number or types of people exposed at various levels above the RfD or RfC (EPA 2015b). Based on the imprecision and uncertainties inherent in EPA's RfD/RfC for TCE, a ten-fold "multiple endpoint safety range" for the RfC (and by extension, the RfD) has been proposed as fully protective of the general population, including sensitive subgroups (Dourson *et al.* 2016; see Section 5.4.2). Substituting ten-fold higher toxicity criteria into the RBSL equations would result in ten-fold higher RBSLs. In turn, HQs calculated with these RBSLs would be ten-fold lower. Accordingly, an HQ of 10 for TCE calculated based on the current EPA RfD/RfC can be interpreted as presenting no increased risk of deleterious effects.

8.0 Summary and conclusions

The fYNOP groundwater HHRA presented in this document provides a comprehensive and conservative evaluation of potential long-term exposures and theoretical ILCR and non-cancer hazards for three current and potential future worker scenarios and a Recreational Wader who could directly and/or indirectly contact up to 23 COPCs associated with groundwater in one or more of seven on- and off-Site LUAs. As described above and detailed in the three IDs, the HHRA was designed and performed in general accordance with PADEP Act 2 (PADEP 2002, 2015) and EPA guidance (EPA 1989, 2001a, 2004, 2011a, 2014b, 2015c), and with recourse to VDEQ guidance (VDEQ 2014) for evaluation of exposures to workers performing intrusive activities.

The groundwater data set for evaluation of potential risks under current and reasonably anticipated future conditions consists of samples from wells intercepting the water table (within 75 feet of the ground surface). In order to evaluate the effect of the West Campus groundwater extraction well pump shutdown on groundwater quality, data sets representing pumping and non-pumping conditions were developed. Groundwater samples from all depths in all LUAs were used to evaluate the feasibility of hypothetical future potable use. The surface water data set includes samples from the affected segment of Codorus Creek adjacent to the Site under both extraction well pumping and non-pumping conditions.

Primary criteria for COPC selection were detection frequency and exceedance of conservative COPC screening levels calculated with a TR of 10^{-6} and THQ of 0.1. EPCs were derived from plume core data, and both CTE and RME concentrations were evaluated in order to approximate the range of COPC concentrations that receptors might encounter. PADEP's (2015) recommended groundwater temperature (11°C) and non-residential groundwater-to-indoor air attenuation factor (0.00036) were used in the vapor modeling performed. Excavation/trench vapor equations developed by VDEQ (2014) were used to calculate highly conservative EPCs for Construction and Utility Workers. COPCs for hypothetical future residential development (including potable use of groundwater) were selected by comparison with MCLs or, in their absence, relevant current EPA RSLs or VISLs. EPCs were not calculated for this screening-level evaluation; rather, maximum detected concentrations were used as worst-case exposure concentrations.

RBSLs were developed for each COPC/receptor/pathway using EPA toxicity criteria and standard default exposure parameter values for the worker scenarios and for the physical characteristics and incidental water intake rate of the Recreational Wader, assumed to be male and female children aged 6 to 11. In the absence of default exposure parameter values for the recreational scenario, these children were assumed to wade twice a week from May through September. RBSLs were compared with EPCs to calculate individual-COPC and cumulative theoretical ILCRs and non-cancer HQs for each receptor scenario. For the evaluation of hypothetical future residential development, putative cleanup goals were MCLs or, in their absence, relevant current EPA RSLs or VISLs.

Results of the fYNOP groundwater HHRA are summarized in Table 40 (ILCR) and Table 41 (HI). The "acceptable" cumulative ILCR and HI (rounded to one significant figure) are 10^{-4} and 1, respectively. However, in view of (1) recommendations that more robust descriptions of the uncertainty be provided in non-cancer risk assessment (*e.g.*, NRC 2009, 2014) and (2) the availability of a published range of RfCs for TCE based on rigorous evaluation of the uncertainty inherent in EPA's selected non-cancer endpoints (ARA 2013; Dourson *et al.* 2016) (Section 5.4.2), an HQ for TCE of up to 10 is suggested as representing both recommendations for improvement of science policy and the most reasonable interpretation of the scientific database for TCE (Section 7.6).

There were no exceedances of target ILCR or HQ for off-Site Commercial/Industrial Workers, Construction and Utility Workers exposed to vapors from groundwater deeper than 15 feet bgs, and Recreational Waders. It is therefore concluded that these receptors are not at risk from Site COPCs under the conditions assumed in the HHRA. The only exceedance of the target ILCR was for the on-Site Utility Worker in areas where groundwater is ≤ 15 feet bgs in LUA #1-3, due to the RME benzene concentration. Otherwise, potential cancer risk is not of concern at fYNOP.

Exceedances of the target HI (primarily attributable to TCE) were observed for all on-Site worker scenarios, and for Utility Workers off-Site where groundwater is within 15 feet bgs (Table 41). The highest HIs were associated with the Utility Worker scenario in areas where groundwater is ≤ 15 feet bgs, with exceedances in all areas under RME and all but LUA #5 under CTE. Although it is not known how accurate the modeled exposures to Utility Workers are, these results suggest the need for reasonable caution in planning and conducting intrusive activities in these areas. RME TCE HQs are less than 10 for the Commercial/Industrial Worker and Utility Worker in LUA #2, and for the Utility Worker in LUA #5 and LUA #6. Considering the uncertainty in the RfD/RfC for TCE, adverse health effects are not expected in these LUAs. In LUA #3, CTE HQs for TCE are less than 10 for Commercial/Industrial Workers and Construction Workers, but greater than 10 at the higher RME concentration. These results suggest that vapor intrusion could be an issue in buildings in certain parts of LUA #3. Thus, building in such areas should be preceded by appropriate soil vapor investigation, and buildings may require engineering conrols to ensure the safety of occupants.

In the evaluation of a hypothetical future residential development scenario, maximum concentrations of COPCs exceeded putative cleanup goals in all areas. These screening-level results indicate that residential development (including potable use of groundwater) on-Site and immediately off-Site in LUAs #5 and #6 would not be appropriate.

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Table 1. Summary of characteristics of groundwater in areas of interest at the Former York Naval Ordnance Plant

Characteristic	Areas of interest at fYNOP						
	NPBA	**Eastern area**	**SPBA**	**SPA**	**BSRA**	**NETT**	**WPA**
Lithology	Non-carbonate	Non-carbonate	Mostly carbonate with some non-carbonate in the western area	Mostly carbonate	Non-carbonate	Non-carbonate and carbonate	Carbonate
Shallow wells screened or open <75' bgs	CW-1A, CW-2, CW-3, CW-5, CW-6, CW-7A, MW-11, MW-12 and MW-20S	MW-2, MW-15, MW-66S, MW-91, and MW-92	MW-64 S, and MW-162 to -165 (plus well MW-108 S, designated as "Other Off-Site" in Appendix A)	MW-110, MW-167, MW-168, MW-171, MW-175, Cole D, GM-1D MW-109S, MW-151, MW-152 (23-235'), MW-166, MW-169, MW-170, MW-172, MW-173, Cole B, Cole (Flush), MW-4 (Cole), and Cole Steel)	MW-17, MW-19, MW-86S, and Springs 1 and 2	MW-70S and MW-102S	CW-9, CW-14, CW-15A, CW-16, CW-17, CW-18, MW-5, MW-6, MW-7, MW-8, MW-26, MW-27, MW-28, MW-30, MW-31S, MW-33, MW-34S, MW-35S, MW-36S, MW-37S, MW-39S, MW-45 - MW-47, MW-51S, MW-54, MW-55, MW-57, MW-77 - MW-80, MW-81S, MW-93S, MW-94, MW-95, MW-96S, MW-100S, MW-105, MW-106, MW-107, MW-116, MW-118 - MW-124, MW-126, MW-129 - MW-135, MW-144, MW-146, MW-155, MW-156, and MW-160
Depth to groundwater table	19 to 42 feet bgs, with some artesian conditions existing	39 to 90 feet bgs	30 to 40 feet bgs	12 to 37 feet bgs	9 to 30 feet bgs, plus two seasonal springs	23 to 43 feet bgs	9 to 26 feet bgs

Characteristic	Areas of interest at fYNOP						
	NPBA	**Eastern area**	**SPBA**	**SPA**	**BSRA**	**NETT**	**WPA**
Site- related VOCs detected	TCE, PCE	TCE, PCE	TCE, PCE	TCE, PCE	TCE, PCE, *cis*-12DCE	TCE, PCE, 111TCA, 11DCE	TCE, PCE, *cis*-12DCE, 111TCA, 11DCE, vinyl chloride, 1,4-dioxane, benzene, ethylbenzene, toluene, total xylenes, 1,3,5-trimethylbenzene
General trends in chemistry	Generally decreasing from 2008 and stable from 2013 to 2014	Limited recent sampling in 2008, 2013 and 2014 (VOC concentrations generally decreasing through 2008)	Generally decreasing from 2008 to 2014	TCE decreasing from 2008 - 2014; PCE stable from 2008 - 2014 north of Rt 30, and decreasing from 2008 to 2014 south of Rt 30	Limited data, but generally decreasing trends where more than 3 samples were collected, except at MW-69 (no trend)	Decreasing VOCs since 2008	Variable

Table 2. Summary of exceptions in the 2008, 2013 and 2014 data sets

Location	Event	Sample year and month	RA table column	Investigation
MW-117	1	2009 Feb	08 RI	Bldg 41
MW-117	2	2010 June	13 Res	Bldg 41
MW-118	2	2012 Aug	13 Res	Bldg 41
MW-119	1	2011 Sep	08 RI	Bldg 45/50 UST
MW-120	2	2012 Aug	13 Res	Bldg 45/50 UST
MW-121	2	2012 Aug	13 Res	Bldg 45/50 UST
MW-122	2	2012 Aug	13 Res	Bldg 45/50 UST
MW-123	2	2012 Aug	13 Res	Bldg 45/50 UST
MW-124	2	2012 Aug	13 Res	Bldg 45/50 UST
MW-125	1	2012 Aug	08 RI	Bldg 45/50 UST
MW-125	2	2013 Dec	13 Res	Bldg 45/50 UST
MW-125	3	2014 Sept	14 Res	Bldg 45/50 UST
MW-160	1	2012 Aug	08 RI	Bldg 45/50 UST
MW-160	2	2013 Dec	13 Res	Bldg 45/50 UST
MW-160	3	2014 Sept	08 RI	Bldg 45/50 UST
MW-161	3	2015 Mar or Apr	14 Res	SPBA VI
MW-162	3	2015 Mar or Apr	14 Res	SPBA VI
MW-163	3	2015 Mar or Apr	14 Res	SPBA VI
MW-164	3	2015 Mar or Apr	14 Res	SPBA VI
MW-165	3	2015 Mar or Apr	14 Res	SPBA VI
MW-166	3	2015 Mar or Apr	14 Res	SPBA VI
MW-167	3	2015 Mar or Apr	14 Res	SPBA VI
MW-168	3	2015 Mar or Apr	14 Res	SPBA VI
MW-169	3	2015 Mar or Apr	14 Res	SPBA VI
MW-170	3	2015 Mar or Apr	14 Res	SPBA VI
MW-171	3	2015 Mar or Apr	14 Res	SPBA VI
MW-172	3	2015 Mar or Apr	14 Res	SPBA VI
MW-173	3	2015 Mar or Apr	14 Res	SPBA VI
MW-174	3	2015 Mar or Apr	14 Res	SPBA VI
MW-175	3	2015 Mar or Apr	14 Res	SPBA VI
Ru-MW-5	1	2011 June	08 RI	Rutters
Ru-MW-6	1	2011 June	08 RI	Rutters

Table 3. Rationale for selection of potentially complete exposure pathways for current and potential future on-Site receptor populations considered in the groundwater HHRA

Receptor	Exposure Medium	Exposure Route	Location	Considered in HHRA?	Rationale
Commercial/ Industrial Worker	Groundwater	Ingestion Dermal contact	Indoors	No	No potable use of groundwater
			Outdoors	No	Not involved in excavation activities
	Air	Vapor inhalation	Indoors	**Yes**	Pathway potentially complete
			Outdoors	No	Work is primarily indoors
Construction Worker	Groundwater	Ingestion Dermal contact Vapor inhalation	Indoors	No	No potable use of groundwater
		Dermal contact Vapor inhalation	Outdoors (excavation)	**Yes**	Could encounter groundwater during excavation in limited areas of the site where groundwater is less than 15 ft bgs
	Air	Vapor inhalation	Indoors	No	Work is primarily outdoors
			Outdoors (excavation)	**Yes**	Could be exposed to vapors from groundwater below excavation base
Utility Worker	Groundwater	Ingestion Dermal contact Vapor inhalation	Indoors	No	No potable use of groundwater
		Dermal contact Vapor inhalation	Outdoors (trench)	**Yes**	Could encounter groundwater during excavation in limited areas of the site where groundwater is less than 15 ft bgs
	Air	Vapor inhalation	Indoors	No	Work is primarily outdoors
			Outdoors (trench)	**Yes**	Could be exposed to vapors from groundwater below trench base

Table 4. Rationale for selection of potentially complete exposure pathways for current and potential future off-Site receptor populations considered in the groundwater HHRA

Receptor	Exposure Medium	Exposure Route	Location	Considered in HHRA?	Rationale
Commercial/ Industrial Worker	Groundwater	Ingestion Dermal contact	Indoors	No	No potable use of groundwater
			Outdoors	No	Not involved in excavation activities
	Air	Vapor inhalation	Indoors	**Yes**	Pathway potentially complete
			Outdoors	No	Work is primarily indoors
Construction or Utility Worker	Groundwater	Ingestion Dermal contact Vapor inhalation	Indoors	No	No potable use of groundwater
		Dermal contact Vapor inhalation	Outdoors (excavation or trench)	**Yes**	Could encounter groundwater in excavations in areas where groundwater is less than 15 ft bgs
	Air	Vapor inhalation	Indoors	No	Work is primarily outdoors
			Outdoors (excavation or trench)	**Yes**	Could be exposed to vapors from groundwater below excavation base
Fish Consumer	Groundwater	Fish consumption	Levee area adjacent to Codorus Creek	No	Surface water data provide best indication of potential exposure
	Surface water		Codorus Creek	**Yes**	Likely to be minor due to low bioaccumulative potential of COPCs and their dilution and volatilization in surface water
Recreational Wader	Groundwater	Ingestion Dermal contact	Off-Site springs and groundwater	No	Surface water data provide best indication of potential exposure
	Surface water		Codorus Creek Johnsons Run	**Yes**	Likely to be minor due to dilution and volatilization of COPCs in surface water

Table 5. Summary of exposure media and routes for each receptor scenario by Land Use Area

Exposure medium	Receptor scenario identified in ID #2	Land Use Area[a]	Direct exposure route	Indirect exposure route
All groundwater	Commercial/Industrial Workers			
	On-Site	2, 3		Inhalation (indoor)
	Off-Site	6		
Groundwater ≤ 15' bgs	Construction and Utility Workers			
	On-Site	1 – 3	Dermal contact	
	Off-Site	4 – 6	Inhalation	
Groundwater >15 to 75' bgs	Construction and Utility Workers			Inhalation (excavation or trench)
	On-Site[b]	2, 3		
	Off-Site	4N, 4S, 5		
Surface water	Recreational Waders in affected segment of Codorus Creek	7	Incidental ingestion Dermal contact	

[a] See Figure 6
[b] Because the core of the plume in deeper groundwater is not located in LUA #1, no EPCs were calculated there (NewFields 2016a)
"N" denotes northern portion of LUA #4
"S" denotes northern portion of LUA #4

Table 6. Summary of groundwater COPCs by receptor scenario and potential exposure route

COPC	Construction and Utility Worker		Commercial/Industrial Worker	
	Direct contact	Inhalation	On-Site[a]	Off-Site[b]
1,1,1-Trichloroethane	✓			
1,1-Dichloroethane	✓		✓	
1,1-Dichloroethene	✓	✓	✓	
1,2,4-Trimethylbenzene	✓		✓	
1,3,5-Trimethylbenzene	✓		✓	
1,4-Dioxane	✓			
Benzene	✓		✓	
Chloroform		✓	✓	
cis-1,2-Dichloroethene	✓			
Ethylbenzene	✓		✓	
Isopropylbenzene	✓			
Methyl *tert*-butyl ether	✓			
Methylene chloride	✓			
Naphthalene	✓		✓	
Tetrachloroethene	✓	✓	✓	✓
Toluene	✓			
Trichloroethene	✓	✓	✓	✓
Vinyl chloride	✓	✓	✓	
bis(2-Ethylhexyl) phthalate	✓			
Pentachlorophenol	✓			
Antimony	✓			
Nickel	✓			
Vanadium	✓			

[a] LUA #3
[b] LUA #6

Table 7. Summary of groundwater COPCs and putative cleanup goals (µg/L) assuming hypothetical future potable use

COPC	On-Site			Off-Site			Putative cleanup goal	Source
	LUA #1	LUA #2	LUA #3	LUA #4	LUA #5	LUA #6		
1,1,1-Trichloroethane			✓				2.0E+02	MCL
1,1-Dichloroethane	✓		✓				2.8E+00	RSL
1,1-Dichloroethene	✓		✓				7.0E+00	MCL
1,2,4-Trimethylbenzene	✓		✓				1.5E+00	RSL
1,3,5-Trimethylbenzene	✓		✓				1.2E+01	RSL
1,4-Dioxane	✓		✓				4.6E-01	RSL
4-Chloroaniline			✓				3.7E-01	RSL
Acrylonitrile			✓				5.2E-02	RSL
Benzene	✓		✓				5.0E+00	MCL
Chloroform			✓				8.0E+01	MCL
cis-1,2-Dichloroethene	✓	✓	✓	✓			7.0E+01	MCL
Ethylbenzene	✓		✓				7.0E+02	MCL
Isopropylbenzene	✓		✓				4.5E+01	RSL
Methylene chloride	✓	✓	✓	✓			5.0E+00	MCL
Naphthalene	✓						1.70E-01	RSL
Nitrobenzene			✓				1.4E-01	RSL
Tetrachloroethene	✓	✓	✓	✓	✓	✓	5.0E+00	MCL
Toluene	✓		✓			✓	1.0E+03	MCL
Trichloroethene	✓	✓	✓	✓	✓	✓	5.0E+00	MCL
Vinyl chloride	✓		✓			✓	2.0E+00	MCL
Benzo(*a*)anthracene			✓				1.2E-02	RSL
Benzo(*b*)fluoranthene			✓				3.4E-02	RSL
bis(2-Ethylhexyl)phthalate			✓			✓	6.0E+00	MCL
Dibenzo(*a,h*)anthracene			✓				3.4E-03	RSL
Indeno(*1,2,3-cd*)pyrene			✓				3.4E-02	RSL
Pentachlorophenol			✓				1.0E+00	MCL
Arsenic	✓	✓	✓	✓			1.0E+01	MCL
Beryllium			✓	✓			4.0E+00	MCL
Chromium			✓				1.0E+02	MCL
Chromium (hexavalent)			✓	✓	✓	✓	3.5E-02	RSL
Iron				✓			1.4E+03	RSL
Lead	✓	✓	✓	✓			1.5E+01	RSL
Manganese			✓	✓		✓	4.3E+01	RSL
Nickel	✓	✓	✓	✓			3.9E+01	RSL
Silver			✓				9.4E+00	RSL
Vanadium	✓	✓	✓	✓			8.6E+00	RSL
Zinc			✓	✓			6.0E+02	RSL

MCL = Maximum Contaminant Level
RSL = EPA Regional Screening Level (May 2016, TR = 10^{-6}, THQ = 0.1)

Table 8. Summary of groundwater COPCs and putative cleanup goals (µg/L) assuming hypothetical future residential vapor intrusion

COPC	On-Site	Off-Site	Putative cleanup goal	Source
1,1,1-Trichloroethane	✓		2.0E+02	MCL
1,1-Dichloroethane	✓		1.2E+01	VISL
1,1-Dichloroethene	✓		7.0E+00	MCL
cis-1,2-Dichloroethene	✓	✓	7.0E+01	MCL
1,2,4-Trimethylbenzene	✓		6.4E+00	VISL
1,3,5-Trimethylbenzene	✓		6.4E+00	VISL*
Benzene	✓		5.0E+00	MCL
Ethylbenzene	✓		7.0E+02	MCL
Methyl tert-butyl ether	✓		6.9E+02	VISL
Naphthalene	✓		1.1E+01	VISL
Tetrachloroethene	✓	✓	5.0E+00	MCL
Toluene	✓		1.0E+03	MCL
Trichloroethene	✓	✓	5.0E+00	MCL
Vinyl chloride	✓		2.0E+00	MCL

MCL = Maximum Contaminant Level
VISL = EPA Vapor Intrusion Screening Level (May 2016, TR = 10^{-6}, THQ = 0.1)
* Value for 1,2,4 trimethylbenzene used in the absence of a VISL for 1,3,5-trimethylbenzene

Table 9. Summary of wells used to calculate EPCs by Land Use Area and groundwater VOC plume

Land Use Area	Plume ID [a]	Exposure pathway	Assumed depth to groundwater (feet) [b]	Wells within core of plume [c]
1	Harley Davidson Property (HD)	Direct contact	≤15 ft [d]	MW-19; MW-26; [MW-70D,MW-70S]; [MW-86D,MW-86S]; [MW-102D,MW-102S]; MW-103S
1 and 3	Petroleum Plume [e]	Direct contact	≤15 ft [d]	MW-77; MW-121; MW-118
	West Campus Shallow GW (WC-S)		≤15 ft [d]	MW-27; CW-15A; MW-30; MW-116; MW-117; MW-131; MW-135
	West Campus Deep GW (WC-D)	Indirect contact (VI)	16	CW-9; CW-13; MW-7; MW-37S; MW-46; MW-51S; [CW-16,MW-54]; MW-55; [MW-81D,MW-81S]; [MW-96D,MW-96S]; MW-97; MW-105; MW-106; MW-129; MW-130; MW-132; MW-134
2	HD Undeveloped Property Shallow GW (HDUD-S)	Direct contact	≤15 ft [d]	*Area not within a plume's core, so wells were selected strictly based on location:* MW-17; [MW-67D,MW-67S]
	HD Undeveloped Property Deep GW (HDUD-D)	Indirect contact (VI)	37	MW-2; MW-15; [MW-64D,MW-64S]; MW-91; MW-92; MW-161; MW-162
4	Residential Northern (RN)	Indirect contact (VI)	19	*Area not within a plume's core, so wells were selected strictly based on location along the north boundary and most are actually within the HD Undeveloped Area (LU#2):* [CW-1,CW-1A]; CW-2; CW-4; CW-5; CW-6; HERMAN (S-7); MW-9; MW-11; CW-3; MW-18S; [CW-7,CW-7A,MW-20M,MW-20S]; RW-2; RW-4 Folk
	Residential Southern (RS)		20	[MW-108D,MW-108S]; MW-109S; MW-166; MW-167; MW-168; MW-169; MW-170; MW-171; MW-172; MW-173
5	Offsite Commercial Shallow GW (OSC-S)	Direct contact	≤15 ft [d]	Cole D; GM-1D
	Offsite Commercial Deep GW (OSC-D)	Indirect contact (VI)	25	[MW-110,MW-174]
6	Western Area (WArea)	Direct contact	≤15 ft [d]	*No defined plume or core; therefore all wells within the area were used:* MW-98S; MW-99S; [MW-100I,MW-100S]; MW-101S; MW-144; MW-146; MW-155; MW-156

[a] Plume ID used in Appendix A
[b] Based on the shallowest portion of the plume's core
[c] Well clusters are identified in brackets [], maximum value from the wells in the brackets represents the well cluster for the sampling event
[d] Depth to groundwater where less than 15 feet is assumed to be at the depth of the trench
[e] Petroleum plume does not appear to commingle with the TCE/PCE or other chlorinated hydrocarbon plumes

Table 10. Summary of CTE and RME indoor air EPCs (µg/m^3) for On- and Off-Site Commercial/Industrial Workers

COPC	EPC	
	CTE	RME
On-Site (LUA #2)		
Tetrachloroethene	2.82E+01	6.95E+01
Trichloroethene	9.96E+00	3.60E+01
On-Site (LUA #3)		
1,1-Dichloroethane	6.0E-01	1.1E+00
1,1-Dichloroethene	3.6E+01	1.3E+02
1,2,4-Trimethylbenzene	3.2E+01	4.1E+01
1,3,5-Trimethylbenzene	1.6E+01	2.5E+01
Benzene	6.0E+00	1.3E+01
Chloroform	1.4E-01	2.4E-01
Ethylbenzene	5.2E+00	1.1E+01
Naphthalene	4.3E-01	1.2E+00
Tetrachloroethene	4.3E+01	9.9E+01
Trichloroethene	6.4E+01	1.4E+02
Vinyl chloride	4.8E+00	2.1E+01
Off-Site (LUA #6) – pumping		
Tetrachloroethene	1.93E+00	4.14E+00
Trichloroethene	1.84E+00	3.67E+00
Off-Site (LUA #6) – non-pumping		
Tetrachloroethene	2.19E+00	3.89E+00
Trichloroethene	1.21E+00	2.02E+00

Table 11. Summary of CTE groundwater EPCs (µg/L) for Utility and Construction Workers by Land Use Area* and plume (groundwater depth ≤ 15 feet bgs)

COPC	Land Use Area						
	1	1 & 3	2	3	5	6 - Pumping	6 - Non-Pumping
1,1,1-Trichloroethane	6.71E+00		1.35E+01	2.80E+03		1.13E+00	2.74E+00
1,1-Dichloroethane	9.27E-01			3.21E+01		6.25E-01	6.92E-01
1,1-Dichloroethene	6.75E+00		3.90E-01	5.34E+02		1.17E+00	1.28E+00
1,2,4-Trimethylbenzene		9.27E+02					
1,3,5-Trimethylbenzene		3.10E+02					
1,4-Dioxane			6.30E-01	9.59E+01			
Benzene		1.21E+03					
cis-1,2-Dichloroethene	8.63E+00			3.61E+03		1.02E+01	1.94E+01
Ethylbenzene		8.88E+02	3.45E-01				
Isopropylbenzene		6.77E+01	0.0E+00				
Methyl *tert*-butyl ether		2.05E+02	2.36E+00				
Methylene chloride		2.30E+01		8.53E+01		1.55E+00	
Naphthalene		1.95E+02	4.0E+01				
Tetrachloroethene	1.99E+01			7.78E+02	1.36E+01	1.63E+01	1.84E+01
Toluene		3.86E+03		6.50E+00	1.70E-01	3.50E-01	
Trichloroethene	1.43E+02			2.16E+03	4.14E+00	2.56E+01	1.68E+01
Vinyl chloride				6.26E+01		3.30E-01	1.08E+00
bis(2-Ethylhexyl) Phthalate			3.80E+00	9.0E+00			
Pentachlorophenol			2.40E+00	1.70E+00			
Antimony	1.11E+01			4.0E-01			
Nickel	8.67E+00			1.14E+01	2.50E+00		
Vanadium	4.28E+00			1.97E+00	1.50E+00		

Blank – COPC was not analyzed or not detected

Table 12. Summary of RME groundwater EPCs (µg/L) for Utility and Construction Workers by Land Use Area* and plume (groundwater depth ≤ 15 feet bgs)

COPC	Land Use Area						
	1	1 & 3	2	3	5	6 - Pumping	6 - Non-Pumping
1,1,1-Trichloroethane	1.40E+01		2.60E+01	6.74E+03		1.90E+00	4.89E+00
1,1-Dichloroethane	1.60E+00			8.16E+01		1.01E+00	1.11E+00
1,1-Dichloroethene	9.45E+00		3.90E-01	1.09E+03		1.76E+00	2.08E+00
1,2,4-Trimethylbenzene		1.20E+03					
1,3,5-Trimethylbenzene		4.90E+02					
1,4-Dioxane				2.0E+02			
Benzene		2.30E+03					
cis-1,2-Dichloroethene	1.10E+01		6.40E-01	1.13E+04		1.98E+01	2.99E+01
Ethylbenzene		1.90E+03					
Isopropylbenzene		8.90E+01					
Methyl *tert*-butyl ether		3.80E+02	4.60E-01				
Methylene chloride		4.10E+01		1.63E+02		2.57E+00	
Naphthalene		5.40E+02					
Tetrachloroethene	2.90E+01		4.30E+00	1.41E+03	3.10E+01	3.48E+01	3.27E+01
Toluene		8.40E+03		1.20E+01	1.70E-01	3.50E-01	
Trichloroethene	1.60E+02		4.0E+01	3.60E+03	1.50E+01	5.09E+01	2.81E+01
Vinyl chloride				3.90E+02		3.30E-01	1.50E+00
bis(2-Ethylhexyl) Phthalate	2.10E+00			9.0E+00			
Pentachlorophenol				1.70E+00			
Antimony	2.72E+01			4.70E-01			
Nickel	1.48E+01		3.80E+00	3.77E+01	2.50E+00		
Vanadium	7.01E+00		2.40E+00	2.26E+00	1.50E+00		

Blank – COPC was not analyzed or not detected

Table 13. Summary of CTE and RME groundwater vapor EPCs (µg/m^3) for Construction Workers by Land Use Area and plume (groundwater depth >15 to 75 feet bgs)

COPC	Land Use Area					
	1	2	3	4-North	4-South	5
CTE						
1,1-Dichloroethene			6.38E-03			
Chloroform			1.29E-04	1.02E-05	1.01E-05	1.62E-05
Tetrachloroethene	1.57E-04	8.67E-05	2.96E-02	4.59E-04	6.56E-05	3.73E-03
Trichloroethene	1.77E-04		4.06E-02	1.04E-03	2.95E-05	6.94E-05
Vinyl chloride	7.0E-05	8.0E-05	2.73E-03	1.96E-04		
RME						
1,1-Dichloroethene			1.39E-02			
Chloroform			2.14E-04	1.43E-05	1.51E-05	1.62E-05
Tetrachloroethene	1.57E-04	1.54E-04	7.51E-02	2.43E-03	1.10E-04	3.77E-03
Trichloroethene	2.68E-04		5.15E-02	2.27E-03	4.28E-05	7.22E-05
Vinyl chloride	7.0E-05	1.53E-04	5.44E-03	1.17E-03		

Blank – COPC was not detected

Table 14. Summary of CTE and RME groundwater vapor EPCs (µg/m^3) for Utility Workers by Land Use Area and plume (groundwater depth >15 to 75 feet bgs)

COPC	Land Use Area					
	1	2	3	4-North	4-South	5
CTE						
1,1-Dichloroethene			1.15E+00			
Chloroform			2.32E-02	1.84E-03	1.81E-03	2.91E-03
Tetrachloroethene	2.82E-02	1.56E-02	5.33E+00	8.26E-02	1.18E-02	6.72E-01
Trichloroethene	3.18E-02		7.31E+00	1.88E-01	5.31E-03	1.25E-02
Vinyl chloride	1.26E-02	1.44E-02	4.92E-01	3.52E-02		
RME						
1,1-Dichloroethene			2.51E+00			
Chloroform			3.86E-02	2.58E-03	2.71E-03	2.91E-03
Tetrachloroethene	2.82E-02	2.78E-02	1.35E+01	4.37E-01	1.98E-02	6.78E-01
Trichloroethene	4.82E-02		9.27E+00	4.09E-01	7.71E-03	1.30E-02
Vinyl chloride	1.26E-02	2.76E-02	9.79E-01	2.11E-01		

Blank – COPC was not detected or detected only once (CTE)

Table 15. Summary of CTE and RME surface water EPCs (µg/L) for Recreational Waders

COPC	Pumping		Non-Pumping	
	CTE	RME	CTE	RME
Tetrachloroethene	3.45E-01	5.82E-01	2.30E+00	4.22E+00
Trichloroethene	4.72E-01	8.40E-01	1.35E+00	2.17E+00

Table 16. Chemical-specific toxicological criteria and physicochemical parameter values

COPC	CAS	SF_O $(mg/kg\text{-}day)^{-1}$	key	IUR $(ug/m^3)^{-1}$	key	RfD (mg/kg-day)	key	RfC (mg/m^3)	key	vol	mutagen	GIABS	ABS	MW	MW Ref	H`@Tgw (11°C) (unitless)	H' Ref	HLC (atm-m^3/mole)	HLC Ref	D_{ia} (cm^2/s)
1,1,1-Trichloroethane	71-55-6					2.00E+00	I	5.00E+00	I	V		1		133.41	PHYSPROP	3.70E-01	PADEP	1.72E-02	PHYSPROP	6.48E-02
1,1,2-Trichloroethane	79-00-5	5.70E-02	I	1.60E-05	I	4.00E-03	I	2.00E-04	X	V		1		133.41	PHYSPROP	1.50E-02	PADEP	8.24E-04	PHYSPROP	6.69E-02
1,1-Dichloroethane	75-34-3	5.70E-03	C	1.60E-06	C	2.00E-01	P			V		1		98.96	PHYSPROP	1.20E-01	PADEP	5.62E-03	PHYSPROP	8.36E-02
1,1-Dichloroethene	75-35-4					5.00E-02	I	2.00E-01	I	V		1		96.944	PHYSPROP	6.30E-01	PADEP	2.61E-02	PHYSPROP	8.63E-02
1,2,4-Trimethylbenzene	95-63-6							7.00E-03	P	V		1		120.2	PHYSPROP	9.50E-02	PADEP	6.16E-03	PHYSPROP	6.07E-02
1,3,5-Trimethylbenzene	108-67-8					1.00E-02	X			V		1		120.2	PHYSPROP	1.40E-01	PADEP	8.77E-03	PHYSPROP	6.02E-02
1,4-Dioxane	123-91-1	1.00E-01	I	5.00E-06	I	3.00E-02	I	3.00E-02	I	V		1		88.107	PHYSPROP	8.60E-05	PADEP	4.80E-06	PHYSPROP	8.74E-02
Benzene	71-43-2	5.50E-02	I	7.80E-06	I	4.00E-03	I	3.00E-02	I	V		1		78.115	PHYSPROP	1.20E-01	PADEP	5.55E-03	PHYSPROP	8.95E-02
Chloroform	67-66-3	3.10E-02	C	2.30E-05	I	1.00E-02	I	9.80E-02	A	V		1		119.38	PHYSPROP	8.00E-02	PADEP	3.67E-03	PHYSPROP	7.69E-02
cis-1,2-Dichloroethene	156-59-2					2.00E-03	I			V		1		96.944	PHYSPROP	8.83E-02	PADEP	4.08E-03	PHYSPROP	8.84E-02
Ethylbenzene	100-41-4	1.10E-02	C	2.50E-06	C	1.00E-01	I	1.00E+00	I	V		1		106.17	PHYSPROP	1.40E-01	PADEP	7.88E-03	PHYSPROP	6.85E-02
Isopropylbenzene	98-82-8					1.00E-01	I	4.00E-01	I	V		1		120.2	PHYSPROP	1.60E-01	PADEP	1.15E-02	PHYSPROP	6.03E-02
Methyl tert-butyl ether	1634-04-4	1.80E-03	C	2.60E-07	C			3.00E+00	I	V		1		88.151	PHYSPROP	1.30E-02	PADEP	5.87E-04	PHYSPROP	7.53E-02
Methylene chloride	75-09-2	2.00E-03	I	1.00E-08	I	6.00E-03	I	6.00E-01	I	V	M	1		84.933	PHYSPROP	7.40E-02	PADEP	3.25E-03	PHYSPROP	9.99E-02
Naphthalene	91-20-3			3.40E-05	C	2.00E-02	I	3.00E-03	I	V		1	0.13	128.18	PHYSPROP	6.10E-03	PADEP	4.40E-04	PHYSPROP	6.05E-02
Tetrachloroethene	127-18-4	2.10E-03	I	2.60E-07	I	6.00E-03	I	4.00E-02	I	V		1		165.83	PHYSPROP	3.30E-01	PADEP	1.77E-02	PHYSPROP	5.05E-02
Toluene	108-88-3					8.00E-02	I	5.00E+00	I	V		1		92.142	PHYSPROP	1.30E-01	PADEP	6.64E-03	PHYSPROP	7.78E-02
Trichloroethene	79-01-6	4.60E-02 (a)	I	4.10E-06	I	5.00E-04	I	2.00E-03	I	V	M	1		131.39	PHYSPROP	2.00E-01	PADEP	9.85E-03	PHYSPROP	6.87E-02
Trichloroethene	79-01-6	2.16E-02 (b)	E																	
Trichloroethene	79-01-6	1.55E-02 (c)	E																	
Trichloroethene	79-01-6	9.33E-03 (d)	E																	
Vinyl chloride	75-01-4	7.20E-01	I	4.40E-06	I	3.00E-03	I	1.00E-01	I	V	M	1		62.499	PHYSPROP	7.50E-01	PADEP	2.78E-02	PHYSPROP	1.07E-01
Xylenes (Total)	1330-20-7					2.00E-01	I	1.00E-01	I	V		1		106.17	PHYSPROP	9.00E-02	PADEP	6.63E-03	PHYSPROP	6.85E-02
bis (2-Ethylhexyl) phthalate	117-81-7	1.40E-02	I	2.40E-06	C	2.00E-02	I					1	0.1	390.57	PHYSPROP			2.70E-07	EPI	1.73E-02
Pentachlorophenol	87-86-5	4.00E-01	I	5.10E-06	C	5.00E-03	I					1	0.25	266.34	PHYSPROP			2.45E-08	PHYSPROP	2.95E-02
Antimony	7440-36-0					4.00E-04	I					0.15		124.77	PHYSPROP					
Nickel	7440-02-0			2.60E-04	C	2.00E-02	I	9.00E-05	A			0.04		58.71	PHYSPROP					
Vanadium	7440-62-2					5.00E-03	S	1.00E-04	A			0.026		50.94	EPI					

(a) = adult oral slope factor; (b) = oral slope factor for non-Hodgkins lymphoma; (c) = oral slope factor for liver tumor; (d) = oral slope factor for kidney tumor

SF_O = oral slope factor; IUR = inhalation unit risk; RfD = reference dose; RfC = reference concentration; GIABS = gastrointestinal absorption fraction; ABS = dermal absorption fraction; MW = molecular weight; H' = unitless Henry's law constant; HLC = Henry's law constant; D_{ia} = diffusivity constant in air; B = ratio of the permeability coefficient of a COPC through the stratum corneum relative to its permeability coefficient across the viable epidermis; τ_{event} = lag time per event; K_p = dermal permeability coefficient in water; FA = fraction absorbed from water

Key: I = IRIS; P = PPRTV; E = EPA (2005); C = Cal EPA; M = mutagen; V = volatile; PHYSPROP = Physical Properties Database; PADEP = Pennsylvania Department of Environmental Protection (2015); EPA WATER9 = EPA wastewater treatment model; EPI = Estimation Programs Interface; RAGSE = EPA RAGS Volume 1 Part E (2004); VDEQ = Virginia Department of Environmental Quality (2014)

Table 17. Exposure parameter values

Exposure parameter		Value	Units	Source
Target cancer risk	TR	1.0E-06	unitless	
Target hazard	THQ	0.1		
Averaging time for carcinogens	AT_c	25,550	days	EPA (2014b)
Averaging time for non-carcinogens	At_{nc}	ED x 365		
Exposure duration				
Construction/Utility Worker	ED_{cuw}	1	years	VDEQ (2014)
Recreational Wader	ED_{rw}	6		Ages 11 – 16 (assumed)
Exposure frequency				
Construction/Utility Worker	EF_{cuw}	125	events/ year	VDEQ (2014)
Recreational Wader	EF_{rw}	40		2x/week from May to September (assumed)
Exposure time				
Construction/Utility Worker	ET_{cuw}	4	hours/ event	VDEQ (2014)
Recreational Wader	ET_{rw}	2		Assumed
Body weight				
Construction/Utility Worker	BW_{cuw}	80	kg	EPA (2014b)
Recreational Wader	BW_{rw}	56.8		Average male and female body weight, aged 11-16 (EPA 2011a)
Water ingestion rate (Recreational Wader)	IR_{w_rw}	0.050	L/hour	EPA Regions 3, 6, & 9 (May 2016)
Water ingestion rate for mutagens (Recreational Wader)	IR_{w_rw} x 3	0.150		
Surface area			cm^2	
Construction/Utility Worker	SA_{cuw}	3,470		VDEQ (2014)
Recreational Wader	SA_{rw}	15,900		Mean total body surface area, male and female aged 11-16 (EPA 2011a)
Surface area for mutagens (Recreational Wader)	SA_{rw} x 3	47,700		Surface area x age-dependent adjustment factor (ADAF)
Apparent thickness of stratum corneum	l_{sc}	0.01	cm	EPA (2004b)
Oral carcinogenic adjustment factor for TCE	CAF_o	0.804	unitless	EPA Regions 3, 6, & 9 (May 2016)
Oral mutagenic adjustment factor for TCE	MAF_o	0.202		

Table 18. Risk-based screening levels for the Commercial/Industrial Worker (µg/m^3)[a]

COPC	$RBSL_{c/iw}$	
	Cancer	**Non-cancer**
1,1-Dichloroethane	7.7E+00	
1,1-Dichloroethene		8.8E+01
1,2,4-Trimethylbenzene		3.1E+00
1,3,5-Trimethylbenzene[b]		3.1E+00
Benzene	1.6E+00	1.3E+01
Chloroform	5.3E-01	4.3E+01
Ethylbenzene	4.9E+00	4.4E+02
Naphthalene	3.6E-01	1.3E+00
Tetrachloroethene	4.7E+01	1.8E+01
Trichloroethene	3.0E+00	8.8E-01
Vinyl chloride	2.8E+00	4.4E+01

[a] EPA industrial air RSLs (TR = 1E-06, THQ = 0.1) (EPA Regions 3,6, & 9 May 2016)
[b] In accordance with PADEP guidance, RSL for 124TMB was used for 135TMB

Table 19. Absorbed doses per event and RBSLs for Construction and Utility Workers' dermal contact with COPCs in groundwater ≤ 15 feet bgs in an excavation/utility trench

COPC	DA_{event_cuw} (mg/cm^2-event)		$RBSL_{derm\_cuw}$ (µg/L)	
	Cancer	Non-cancer	Cancer	Non-cancer
VOCs				
1,1,1-Trichloroethane		1.35E+01		2.13E+05
1,1-Dichloroethane	8.27E-01	1.35E+00	2.62E+04	4.27E+04
1,1-Dichloroethene		3.37E-01		6.26E+03
1,2,4-Trimethylbenzene*		6.73E-02		1.84E+02
1,3,5-Trimethylbenzene		6.73E-02		2.46E+02
1,4-Dioxane	4.71E-02	2.02E-01	3.05E+04	1.31E+05
Benzene	8.57E-02	2.69E-02	1.30E+03	4.10E+02
cis-1,2-Dichloroethene		1.35E-02		2.66E+02
Ethylbenzene	4.28E-01	6.73E-01	2.01E+03	3.15E+03
Isopropylbenzene		6.73E-01		1.77E+03
Methyl *tert*-butyl ether	2.62E+00	0.0E+00	2.68E+05	
Methylene chloride	2.36E+00	4.04E-02	1.45E+05	2.49E+03
Naphthalene		1.35E-01		6.23E+02
Tetrachloroethene	2.24E+00	4.04E-02	1.22E+04	2.20E+02
Toluene		5.39E-01		3.98E+03
Trichloroethene	1.02E-01	3.37E-03	1.76E+03	5.79E+01
Vinyl chloride	6.54E-03	2.02E-02	1.78E+02	5.50E+02
SVOCs				
bis(2-Ethylhexyl) phthalate	3.37E-01	1.35E-01	1.67E+01	6.70E+00
Pentachlorophenol	1.18E-02	3.37E-02	1.03E+01	2.95E+01
Inorganics				
Antimony		4.04E-04		1.01E+02
Nickel		5.39E-03		6.73E+03
Vanadium		8.75E-04		2.19E+02

* In the absence of a reference dose for 1,2,4-trimethylbenzene, the reference dose for 1,3,5-trimethylbenzene was used as a surrogate.

Table 20. Volatilization factors and RBSLs for Construction and Utility Workers' inhalation of volatile COPCs in groundwater ≤ 15 feet bgs

COPC	Construction Worker			Utility Worker		
	$VF_{\leq 15\_cw}$ (L/m^3)	$RBSL_{dir\text{-}inhal\_cw}$ (µg/L)		$VF_{\leq 15\_uw}$ (L/m^3)	$RBSL_{dir\text{-}inhal\_uw}$ (µg/L)	
		Cancer	Non-cancer		Cancer	Non-cancer
1,1,1-Trichloroethane	3.80E-02		2.31E+05	6.84E+00		1.28E+03
1,1-Dichloroethane	4.35E-02	1.76E+04		7.83E+00	9.79E+01	
1,1-Dichloroethene	4.47E-02		7.84E+03	8.04E+00		4.36E+01
1,2,4-Trimethylbenzene	3.93E-02		3.12E+02	7.07E+00		1.73E+00
1,3,5-Trimethylbenzene*	3.96E-02		3.10E+02	7.13E+00		1.72E+00
1,4-Dioxane	1.53E-03	1.61E+05	3.44E+04	2.75E-01	8.92E+02	1.91E+02
Benzene	4.89E-02	3.22E+03	1.07E+03	8.80E+00	1.79E+01	5.97E+00
cis-1,2-Dichloroethene**	4.37E-02			7.86E+00		
Ethylbenzene	4.21E-02	1.16E+04	4.16E+04	7.58E+00	6.47E+01	2.31E+02
Isopropylbenzene	3.97E-02		1.77E+04	7.14E+00		9.81E+01
Methyl *tert*-butyl ether	3.93E-02	1.20E+05	1.34E+05	7.07E+00	6.67E+02	7.43E+02
Methylene chloride	4.63E-02	2.65E+06	2.27E+04	8.34E+00	1.47E+04	1.26E+02
Naphthalene	2.80E-02	1.29E+03	1.88E+02	5.03E+00	7.17E+00	1.04E+00
Tetrachloroethene	3.41E-02	1.39E+05	2.06E+03	6.13E+00	7.69E+02	1.14E+01
Toluene	4.51E-02		1.94E+05	8.12E+00		1.08E+03
Trichloroethene	3.81E-02	7.86E+03	9.20E+01	6.85E+00	4.36E+01	5.11E-01
Vinyl chloride	5.57E-02	5.01E+03	3.15E+03	1.0E+01	2.78E+01	1.75E+01

* In the absence of a reference concentration for 1,3,5-trimethylbenzene, the reference concentration for 1,2,4-trimethylbenzene was used as a surrogate.
** No toxicological criteria for inhalation route

Table 21. Combined dermal and inhalation RBSLs for Construction and Utility Workers' exposure to volatile COPCs (≤ 15 feet bgs) (µg/L)

COPC	Construction Worker		Utility Worker	
	$RBSL_{comb\_cw}$ (µg/L)		$RBSL_{comb\_uw}$ (µg/L)	
	Cancer	Non-cancer	Cancer	Non-cancer
1,1,1-Trichloroethane		1.11E+05		1.27E+03
1,1-Dichloroethane	1.05E+04	4.27E+04	9.76E+01	4.27E+04
1,1-Dichloroethene		3.48E+03		4.33E+01
1,2,4-Trimethylbenzene		1.16E+02		1.72E+00
1,3,5-Trimethylbenzene		1.37E+02		1.71E+00
1,4-Dioxane	2.56E+04	2.72E+04	8.67E+02	1.91E+02
Benzene	9.27E+02	2.97E+02	1.76E+01	5.89E+00
cis-1,2-Dichloroethene		2.66E+02		2.66E+02
Ethylbenzene	1.71E+03	2.93E+03	6.27E+01	2.15E+02
Isopropylbenzene		1.61E+03		9.30E+01
Methyl *tert*-butyl ether	8.29E+04	1.34E+05	6.65E+02	7.43E+02
Methylene chloride	1.38E+05	2.24E+03	1.34E+04	1.20E+02
Naphthalene	1.29E+03	1.44E+02	7.17E+00	1.04E+00
Tetrachloroethene	1.12E+04	1.98E+02	7.24E+02	1.09E+01
Toluene		3.90E+03		8.48E+02
Trichloroethene	1.44E+03	3.56E+01	4.26E+01	5.07E-01
Vinyl chloride	1.72E+02	4.68E+02	2.41E+01	1.69E+01

Table 22. RBSLs for Construction and Utility Workers' inhalation of volatile COPCs in groundwater >15 to 75 feet bgs (µg/m^3)

COPC	$RBSL_{indir\text{-}inhal\_cuw}$ (µg/m^3)	
	Cancer	Non-cancer
1,1-Dichloroethene		3.50E+02
Chloroform	5.33E+01	1.72E+02
cis-1,2-Dichloroethene		
Tetrachloroethene	4.72E+03	7.01E+01
Trichloroethene	2.99E+02	3.50E+00
Vinyl chloride	2.79E+02	1.75E+02

Table 23. Absorbed doses per event and RBSLs for Recreational Waders' direct contact with COPCs in Codorus Creek surface water (µg/L)

COPC	$DA_{event\_rec(c)}$ (µg/cm^2-event)	$DA_{event\_rec(nc)}$ (µg/cm^2-event)	Cancer RBSLs (µg/L)			Non-cancer RBSLs (µg/L)		
			Ingestion	Dermal	Combined	Ingestion	Dermal	Combined
PCE	1.81E-01	1.96E-01	2.88E+04	1.47E+03	1.40E+03	3.11E+03	1.59E+02	1.51E+02
TCE	5.86E-03	1.63E-02	9.32E+02	1.63E+02	1.39E+02	2.59E+02	4.52E+01	3.85E+01

Table 24. Summary of non-cancer target organs and critical effects of COPCs

COPC	Toxicity criterion	Source	Confidence level	Target organ	Critical effect
Benzene	RfD	IRIS	Medium	Blood	Decreased lymphocyte count
	RfC	IRIS	Medium	Blood	Decreased lymphocyte count
Bis(2-ethylhexyl)phthalate	RfD	IRIS	Medium	Liver	Increased relative liver weight
1,1-Dichloroethylene	RfD	IRIS	Medium	Liver	Liver toxicity (fatty change)
	RfC	IRIS	Medium	Liver	Toxicity (fatty change)
cis-1,2-Dichloroethylene	RfD	IRIS	Low	Kidney	Increased relative kidney weight in male rats
Ethylbenzene	RfC	IRIS	Low	Developmental	Developmental toxicity
	RfD	IRIS	Low	Liver and kidney	Liver and kidney toxicity
Isopropylbenzene	RfD	IRIS	Low	Kidney	Increased average kidney weights in female rats
	RfC	IRIS	Medium	Kidney	Increased kidney weights in female rats and adrenal weights in male and female rats
Methyl *tert*-Butyl Ether	RfC	IRIS	Medium	Liver and kidney	Increased absolute and relative liver and kidney weights and increased severity of spontaneous renal lesions (females), increased prostration (females), and swollen periocular tissue (males and females)
Methylene Chloride	RfD	IRIS	High	Liver	hepatic effects (hepatic vacuolation, liver foci)
	RfC	IRIS	Medium/high	Liver	hepatic effects (hepatic vacuolation)
Naphthalene	RfD	IRIS	Low	Body weight	Decreased mean terminal body weight in males
	RfC	IRIS	Medium	Nasal	Nasal effects: hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively
Tetrachloroethylene	RfD	IRIS	Medium	Nervous system	Neurotoxicity (color vision) (reaction time, cognitive effects)
	RfC	IRIS	Medium	Nervous system	Neurotoxicity (color vision) (reaction time, cognitive effects)
Toluene	RfD	IRIS	Medium	Kidney	Increased kidney weight
	RfC	IRIS	High	Neurological	Neurological effects in occupationally-exposed workers
Trichloroethylene	RfD	IRIS	High	Multiple	Multiple
	RfC	IRIS	High	Multiple	Multiple
1,2,4-Trimethylbenzene	RfC	PPRTV	Low	Blood	Decreased clotting time
	RfD	PPRTV	Low	Whole body; liver and kidney	Decrease in body weight gain, clinical observations, and increased serum phosphorus levels; Increased weights
1,3,5-Trimethylbenzene	RfD	PPRTV	Low	Liver	Effects
	RfC	PPRTV	Low	Respiratory, neurological, and hematological	Effects
Vinyl chloride	RfD	IRIS	Medium	Liver	Liver cell polymorphism
	RfC	IRIS	Medium	Liver	Liver cell polymorphism

Data from USDOE Risk Assessment Information System (RAIS) (accessed May 2016)
IRIS = Integrated Risk Information System; PPRTV = Provisional Peer-Reviewed Toxicity Value; RfC – reference concentration; RfD = reference dose

Table 25. On-Site Commercial/Industrial Worker (LUA #2): Potential cancer risks and non-cancer hazards associated with indoor vapor intrusion from groundwater

COPC	CTE		RME	
	ILCR	HQ	ILCR	HQ
Tetrachloroethene	6E-07	2E-01	1E-06	4E-01
Trichloroethene	3E-06	1E+00	1E-05	4E+00
Total:	**4E-06**	**1E+00**	**1E-05**	**4E+00**

Red denotes exceedance of target threshold

Table 26. On-Site Commercial/Industrial Worker (LUA #3): Potential cancer risks and non-cancer hazards associated with indoor vapor intrusion from groundwater

COPC	CTE		RME	
	ILCR	HQ	ILCR	HQ
1,1-Dichloroethane	8E-08		1E-07	
1,1-Dichloroethene		4E-02		2E-01
1,2,4-Trimethylbenzene		1E+00		1E+00
1,3,5-Trimethylbenzene		5E-01		8E-01
Benzene	4E-06	5E-02	8E-06	1E-01
Chloroform	3E-07	3E-04	5E-07	6E-04
Ethylbenzene	1E-06	1E-03	2E-06	3E-03
Naphthalene	1E-06	3E-02	3E-06	9E-02
Tetrachloroethene	9E-07	2E-01	2E-06	6E-01
Trichloroethene	2E-05	7E+00	5E-05	2E+01
Vinyl chloride	2E-06	1E-02	8E-06	5E-02
Total:	**3E-05**	**9E+00**	**7E-05**	**2E+01**

Red denotes exceedance of target threshold

Table 27. Off-Site Commercial/Industrial Worker (LUA #6): Potential cancer risks and non-cancer hazards associated with indoor vapor intrusion from groundwater under pumping and non-pumping conditions

COPC	CTE		RME	
	ILCR	HQ	ILCR	HQ
Pumping				
Tetrachloroethene	4E-08	1E-02	9E-08	2E-02
Trichloroethene	6E-07	2E-01	1E-06	4E-01
Total:	**7E-07**	**2E-01**	**1E-06**	**4E-01**
Non-pumping				
Tetrachloroethene	5E-08	1E-02	8E-08	2E-02
Trichloroethene	4E-07	1E-01	7E-07	2E-01
Total:	**4E-07**	**1E-01**	**8E-07**	**3E-01**

Table 28. Construction Worker: Potential CTE cancer risks and non-cancer hazards associated with direct contact with shallow groundwater (≤ 15 feet bgs) in excavation

COPC	Land Use Area						
	1	1&3	2	3	5	6 – pumping	6 – non-pumping
ILCR							
1,1,1-Trichloroethane							
1,1-Dichloroethane	8.8E-11			3.0E-09		5.9E-11	6.6E-11
1,1-Dichloroethene							
1,2,4-Trimethylbenzene							
1,3,5-Trimethylbenzene							
1,4-Dioxane				3.7E-09			
Benzene		1.3E-06					
cis-1,2-Dichloroethene							
Ethylbenzene		5.2E-07					
Isopropylbenzene							
Methyl *tert*-butyl ether		2.5E-09	4.2E-12				
Methylene chloride		1.7E-10		6.2E-10		1.1E-11	
Naphthalene		1.5E-07					
Tetrachloroethene	1.8E-09		2.1E-10	6.9E-08	1.2E-09	1.4E-09	1.6E-09
Toluene							
Trichloroethene	9.9E-08		2.8E-08	1.5E-06	2.9E-09	1.8E-08	1.2E-08
Vinyl chloride				3.6E-07		1.9E-09	6.3E-09
Bis(2-Ethylhexyl) Phthalate				5.4E-07			
Pentachlorophenol				1.6E-07			
Antimony							
Nickel							
Vanadium							
Total ILCR:	**1E-07**	**2E-06**	**3E-08**	**3E-06**	**4E-09**	**2E-08**	**2E-08**
HQ							
1,1,1-Trichloroethane	6.1E-06		1.2E-05	2.5E-03		1.0E-06	2.5E-06
1,1-Dichloroethane	2.2E-06			7.5E-05		1.5E-06	1.6E-06
1,1-Dichloroethene	1.9E-04		1.1E-05	1.5E-02		3.4E-05	3.7E-05
1,2,4-Trimethylbenzene		8.0E-01					
1,3,5-Trimethylbenzene		1.3E-01					
1,4-Dioxane				3.5E-04			
Benzene		4.1E-01					
cis-1,2-Dichloroethene	3.2E-03		2.4E-04	1.4E+00		3.9E-03	7.3E-03
Ethylbenzene		3.0E-02					
Isopropylbenzene		4.2E-03					
Methyl *tert*-butyl ether		1.5E-04	2.6E-07				
Methylene chloride		1.0E-03		3.8E-03		6.9E-05	
Naphthalene		1.3E-01					
Tetrachloroethene	1.0E-02		1.2E-03	3.9E-01	6.8E-03	8.2E-03	9.3E-03
Toluene		9.9E-02		1.7E-04	4.4E-06	9.0E-06	
Trichloroethene	4.0E-01		1.1E-01	6.1E+00	1.2E-02	7.2E-02	4.7E-02
Vinyl chloride				1.3E-02		7.1E-05	2.3E-04
Bis(2-Ethylhexyl) Phthalate				1.3E-01			
Pentachlorophenol				5.8E-03			
Antimony	1.1E-02			4.0E-04			
Nickel	1.3E-04		5.6E-05	1.7E-04	3.7E-05		
Vanadium	2.0E-03		1.1E-03	9.0E-04	6.9E-04		
Total HI:	**4E-01**	**2E+00**	**1E-01**	**8E+00**	**2E-02**	**8E-02**	**6E-02**

Red denotes exceedance of target threshold

Table 29. Construction Worker: Potential RME cancer risks and non-cancer hazards associated with direct contact with shallow groundwater (≤ 15 feet bgs) in excavation

COPC	Land Use Area						
	1	1&3	2	3	5	6-pump	6-nonpump
ILCR							
1,1,1-Trichloroethane							
1,1-Dichloroethane	1.5E-10			7.7E-09		9.6E-11	1.1E-10
1,1-Dichloroethene							
1,2,4-Trimethylbenzene							
1,3,5-Trimethylbenzene							
1,4-Dioxane				7.8E-09			
Benzene		2.5E-06					
cis-1,2-Dichloroethene							
Ethylbenzene		1.1E-06					
Isopropylbenzene							
Methyl *tert*-butyl ether		4.6E-09	5.5E-12				
Methylene chloride		3.0E-10		1.2E-09		1.9E-11	
Naphthalene		4.2E-07					
Tetrachloroethene	2.6E-09		3.8E-10	1.3E-07	2.8E-09	3.1E-09	2.9E-09
Toluene							
Trichloroethene	1.1E-07		2.8E-08	2.5E-06	1.0E-08	3.5E-08	1.9E-08
Vinyl chloride				2.3E-06		1.9E-09	8.7E-09
Bis(2-Ethylhexyl) Phthalate	1.3E-07			5.4E-07			
Pentachlorophenol				1.6E-07			
Antimony							
Nickel							
Vanadium							
Total ILCR:	**2E-07**	**4E-06**	**3E-08**	**6E-06**	**1E-08**	**4E-08**	**3E-08**
HQ							
1,1,1-Trichloroethane	1.3E-05		2.4E-05	6.1E-03		1.7E-06	4.4E-06
1,1-Dichloroethane	3.7E-06			1.9E-04		2.4E-06	2.6E-06
1,1-Dichloroethene	2.7E-04		1.1E-05	3.1E-02		5.1E-05	6.0E-05
1,2,4-Trimethylbenzene		1.0E+00					
1,3,5-Trimethylbenzene		2.0E-01					
1,4-Dioxane				7.3E-04			
Benzene		7.8E-01					
cis-1,2-Dichloroethene	4.1E-03		2.4E-04	4.3E+00		7.4E-03	1.1E-02
Ethylbenzene		6.5E-02					
Isopropylbenzene		5.5E-03					
Methyl *tert*-butyl ether		2.8E-04	3.4E-07				
Methylene chloride		1.8E-03		7.3E-03		1.1E-04	
Naphthalene		3.7E-01					
Tetrachloroethene	1.5E-02		2.2E-03	7.1E-01	1.6E-02	1.8E-02	1.6E-02
Toluene		2.2E-01		3.1E-04	4.4E-06	9.0E-06	
Trichloroethene	4.5E-01		1.1E-01	1.0E+01	4.2E-02	1.4E-01	7.9E-02
Vinyl chloride				8.3E-02		7.1E-05	3.2E-04
Bis(2-Ethylhexyl) Phthalate	3.1E-02			1.3E-01			
Pentachlorophenol				5.8E-03			
Antimony	2.7E-02			4.7E-04			
Nickel	2.2E-04		5.6E-05	5.6E-04	3.7E-05		
Vanadium	3.2E-03		1.1E-03	1.0E-03	6.9E-04		
Total HI:	**5E-01**	**3E+00**	**1E-01**	**2E+01**	**6E-02**	**2E-01**	**1E-01**

Red denotes exceedance of target threshold

Table 30. Calculation of target organ-specific hazard indices for the Construction Worker in LUA #1-3

COPC	Target Organ[a]	CTE HQ[b]	Target organ-specific CTE HI	RME HQ[c]	Target organ-specific RME HI
Benzene	Blood	4E-01	**4E-01**	8E-01	**8E-01**
1,2,4-Trimethylbenzene	Whole body; liver and kidney, blood	8E-01	**9E-01**	1E+00	**1E+00**
Naphthalene	Body weight, nasal	1E-01		4E-01	
Toluene	Kidney, neurological	1E-01	**3E-01**	2E-01	**5E-01**
1,3,5-Trimethylbenzene	Liver, respiratory, neurological, and hematological	1E-01		2E-01	
Ethylbenzene	Developmental, liver and kidney	3E-02		6E-02	
Isopropylbenzene	Kidney	4E-03		6E-03	
Methylene chloride	Liver	1E-03		2E-03	
Methyl *tert*-butyl ether	Liver and kidney	2E-04		3E-04	
		2E+00		**3E+00**	

Colored shading denotes same target organ
Red denotes exceedance of target threshold
[a] From Table 24
[b] From Table 28
[c] From Table 29

Table 31. Utility Worker: Potential CTE cancer risks and non-cancer hazards associated with direct contact with shallow groundwater (≤ 15 feet bgs) in trench

COPC	Land Use Area						
	1	1&3	2	3	5	6-pump	6-nonpump
ILCR							
1,1,1-Trichloroethane							
1,1-Dichloroethane	9.5E-09			3.3E-07		6.4E-09	7.1E-09
1,1-Dichloroethene							
1,2,4-Trimethylbenzene							
1,3,5-Trimethylbenzene							
1,4-Dioxane				1.1E-07			
Benzene		6.9E-05					
cis-1,2-Dichloroethene							
Ethylbenzene		1.4E-05					
Isopropylbenzene							
Methyl *tert*-butyl ether		3.1E-07	5.2E-10				
Methylene chloride		1.7E-09		6.4E-09		1.2E-10	
Naphthalene		2.7E-05					
Tetrachloroethene	2.7E-08		3.3E-09	1.1E-06	1.9E-08	2.2E-08	2.5E-08
Toluene							
Trichloroethene	3.3E-06		9.4E-07	5.1E-05	9.7E-08	6.0E-07	3.9E-07
Vinyl chloride				2.6E-06		1.4E-08	4.5E-08
Bis(2-Ethylhexyl) Phthalate				5.4E-07			
Pentachlorophenol				1.6E-07			
Antimony							
Nickel							
Vanadium							
Total ILCR:	**3E-06**	**1E-04**	**9E-07**	**6E-05**	**1E-07**	**6E-07**	**5E-07**
HQ							
1,1,1-Trichloroethane	5.3E-04		1.1E-03	2.2E-01		8.9E-05	2.2E-04
1,1-Dichloroethane	2.2E-06			7.5E-05		1.5E-06	1.6E-06
1,1-Dichloroethene	1.6E-02		9.0E-04	1.2E+00		2.7E-03	3.0E-03
1,2,4-Trimethylbenzene		5.4E+01					
1,3,5-Trimethylbenzene		1.3E-01					
1,4-Dioxane				5.0E-02			
Benzene		2.1E+01					
cis-1,2-Dichloroethene	3.2E-03		2.4E-04	1.4E+00		3.9E-03	7.3E-03
Ethylbenzene		4.1E-01					
Isopropylbenzene		7.3E-02					
Methyl *tert*-butyl ether		2.8E-02	4.6E-05				
Methylene chloride		1.9E-02		7.1E-02		1.3E-03	
Naphthalene		1.9E+01					
Tetrachloroethene	1.8E-01		2.2E-02	7.2E+00	1.2E-01	1.5E-01	1.7E-01
Toluene		4.6E-01		7.7E-04	2.0E-05	4.1E-05	
Trichloroethene	2.8E+01		7.9E+00	4.3E+02	8.2E-01	5.1E+00	3.3E+00
Vinyl chloride				3.7E-01		1.9E-03	6.4E-03
Bis(2-Ethylhexyl) Phthalate				1.3E-01			
Pentachlorophenol				5.8E-03			
Antimony	1.1E-02			4.0E-04			
Nickel	1.3E-04		5.6E-05	1.7E-04	3.7E-05		
Vanadium	2.0E-03		1.1E-03	9.0E-04	6.9E-04		
Total HI:	**3E+01**	**9E+01**	**8E+00**	**4E+02**	**9E-01**	**5E+00**	**4E+00**

Red denotes exceedance of target threshold

Table 32. Utility Worker: Potential RME cancer risks and non-cancer hazards associated with direct contact with shallow groundwater (≤ 15 feet bgs) in trench

COPC	Land Use Area						
	1	1&3	2	3	5	6-pump	6-nonpump
ILCR							
1,1,1-Trichloroethane							
1,1-Dichloroethane	1.6E-08			8.4E-07		1.0E-08	1.1E-08
1,1-Dichloroethene							
1,2,4-Trimethylbenzene							
1,3,5-Trimethylbenzene							
1,4-Dioxane				2.3E-07			
Benzene		1.3E-04					
cis-1,2-Dichloroethene							
Ethylbenzene		3.0E-05					
Isopropylbenzene							
Methyl *tert*-butyl ether		5.7E-07	6.9E-10				
Methylene chloride		3.1E-09		1.2E-08		1.9E-10	
Naphthalene		7.5E-05					
Tetrachloroethene	4.0E-08		5.9E-09	1.9E-06	4.3E-08	4.8E-08	4.5E-08
Toluene							
Trichloroethene	3.8E-06		9.4E-07	8.5E-05	3.5E-07	1.2E-06	6.6E-07
Vinyl chloride				1.6E-05		1.4E-08	6.2E-08
Bis(2-Ethylhexyl) Phthalate	1.3E-07			5.4E-07			
Pentachlorophenol				1.6E-07			
Antimony							
Nickel							
Vanadium							
Total ILCR:	**4E-06**	**2E-04**	**9E-07**	**1E-04**	**4E-07**	**1E-06**	**8E-07**
HQ							
1,1,1-Trichloroethane	1.1E-03		2.0E-03	5.3E-01		1.5E-04	3.8E-04
1,1-Dichloroethane	3.7E-06			1.9E-04		2.4E-06	2.6E-06
1,1-Dichloroethene	2.2E-02		9.0E-04	2.5E+00		4.1E-03	4.8E-03
1,2,4-Trimethylbenzene		7.0E+01					
1,3,5-Trimethylbenzene		2.0E-01					
1,4-Dioxane				1.0E-01			
Benzene		3.9E+01					
cis-1,2-Dichloroethene	4.1E-03		2.4E-04	4.3E+00		7.4E-03	1.1E-02
Ethylbenzene		8.8E-01					
Isopropylbenzene		9.6E-02					
Methyl *tert*-butyl ether		5.1E-02	6.2E-05				
Methylene chloride		3.4E-02		1.4E-01		2.1E-03	
Naphthalene		5.2E+01					
Tetrachloroethene	2.7E-01		4.0E-02	1.3E+01	2.9E-01	3.2E-01	3.0E-01
Toluene		9.9E-01		1.4E-03	2.0E-05	4.1E-05	
Trichloroethene	3.2E+01		7.9E+00	7.1E+02	3.0E+00	1.0E+01	5.5E+00
Vinyl chloride				2.3E+00		1.9E-03	8.9E-03
Bis(2-Ethylhexyl) Phthalate	1.2E-01			1.3E-01			
Pentachlorophenol				5.8E-03			
Antimony	2.7E-02			4.7E-04			
Nickel	2.2E-04		5.6E-05	5.6E-04	3.7E-05		
Vanadium	3.2E-03		1.1E-03	1.0E-03	6.9E-04		
Total HI:	**3E+01**	**2E+02**	**8E+00**	**7E+02**	**3E+00**	**1E+01**	**6E+00**

Red denotes exceedance of target threshold

Table 33. Construction Worker: Potential CTE cancer risks and non-cancer hazards associated with inhalation of volatile COPCs from deep groundwater (>15 to 75 feet bgs) in excavation

COPC	Land Use Area					
	1	2	3	4N	4S	5
ILCR						
1,1-Dichloroethene						
Chloroform			2.4E-12	1.9E-13	1.9E-13	3.0E-13
Tetrachloroethene	3.3E-14	1.8E-14	6.3E-12	9.7E-14	1.4E-14	7.9E-13
Trichloroethene	5.9E-13		1.4E-10	3.5E-12	9.9E-14	2.3E-13
Vinyl chloride	2.5E-13	2.9E-13	9.8E-12	7.0E-13		
Total ILCR:	**9E-13**	**3E-13**	**2E-10**	**4E-12**	**3E-13**	**1E-12**
HQ						
1,1-Dichloroethene			1.8E-06			
Chloroform			7.5E-08	6.0E-09	5.9E-09	9.4E-09
Tetrachloroethene	2.2E-07	1.2E-07	4.2E-05	6.5E-07	9.4E-08	5.3E-06
Trichloroethene	5.0E-06		1.2E-03	3.0E-05	8.4E-07	2.0E-06
Vinyl chloride	4.0E-08	4.6E-08	1.6E-06	1.1E-07		
Total HI:	**5E-06**	**2E-07**	**1E-03**	**3E-05**	**9E-07**	**7E-06**

Table 34. Construction Worker: Potential RME cancer risks and non-cancer hazards associated with inhalation of volatile COPCs from deep groundwater (>15 to 75 feet bgs) in excavation

COPC	Land Use Area					
	1	2	3	4N	4S	5
ILCR						
1,1-Dichloroethene						
Chloroform			4.0E-12	2.7E-13	2.8E-13	3.0E-13
Tetrachloroethene	3.3E-14	3.3E-14	1.6E-11	5.1E-13	2.3E-14	8.0E-13
Trichloroethene	9.0E-13		1.7E-10	7.6E-12	1.4E-13	2.4E-13
Vinyl chloride	2.5E-13	5.5E-13	2.0E-11	4.2E-12		
Total ILCR:	**1E-12**	**6E-13**	**2E-10**	**1E-11**	**4E-13**	**1E-12**
HQ						
1,1-Dichloroethene			4.0E-06			
Chloroform			1.2E-07	8.3E-09	8.8E-09	9.4E-09
Tetrachloroethene	2.2E-07	2.2E-07	1.1E-04	3.5E-06	1.6E-07	5.4E-06
Trichloroethene	7.6E-06		1.5E-03	6.5E-05	1.2E-06	2.1E-06
Vinyl chloride	4.0E-08	8.8E-08	3.1E-06	6.7E-07		
Total HI:	**8E-06**	**3E-07**	**2E-03**	**7E-05**	**1E-06**	**7E-06**

Table 35. Utility Worker: Potential CTE cancer risks and non-cancer hazards associated with inhalation of volatile COPCs from deep groundwater (>15 to 75 feet bgs) in trench

COPC	Land Use Area					
	1	2	3	4N	4S	5
ILCR						
1,1-Dichloroethene						
Chloroform			4.4E-10	3.5E-11	3.4E-11	5.5E-11
Tetrachloroethene	6.0E-12	3.3E-12	1.1E-09	1.8E-11	2.5E-12	1.4E-10
Trichloroethene	1.1E-10		2.4E-08	6.3E-10	1.8E-11	4.2E-11
Vinyl chloride	4.5E-11	5.2E-11	1.8E-09	1.3E-10		
Total ILCR:	**2E-10**	**5E-11**	**3E-08**	**8E-10**	**5E-11**	**2E-10**
HQ						
1,1-Dichloroethene			3.3E-04			
Chloroform			1.4E-05	1.1E-06	1.1E-06	1.7E-06
Tetrachloroethene	4.0E-05	2.2E-05	7.6E-03	1.2E-04	1.7E-05	9.6E-04
Trichloroethene	9.1E-04		2.1E-01	5.4E-03	1.5E-04	3.6E-04
Vinyl chloride	7.2E-06	8.2E-06	2.8E-04	2.0E-05		
Total HI:	**1E-03**	**3E-05**	**2E-01**	**6E-03**	**2E-04**	**1E-03**

Table 36. Utility Worker: Potential RME cancer risks and non-cancer hazards associated with inhalation of volatile COPCs from deep groundwater (>15 to 75 feet bgs) in trench

COPC	Land Use Area					
	1	2	3	4N	4S	5
ILCR						
1,1-Dichloroethene						
Chloroform			7.2E-10	4.8E-11	5.1E-11	5.5E-11
Tetrachloroethene	6.0E-12	5.9E-12	2.9E-09	9.3E-11	4.2E-12	1.4E-10
Trichloroethene	1.6E-10		3.1E-08	1.4E-09	2.6E-11	4.3E-11
Vinyl chloride	4.5E-11	9.9E-11	3.5E-09	7.6E-10		
Total ILCR:	**2E-10**	**1E-10**	**4E-08**	**2E-09**	**8E-11**	**2E-10**
HQ						
1,1-Dichloroethene			7.1E-04			
Chloroform			2.2E-05	1.5E-06	1.6E-06	1.7E-06
Tetrachloroethene	4.0E-05	4.0E-05	1.9E-02	6.2E-04	2.8E-05	9.7E-04
Trichloroethene	1.4E-03		2.6E-01	1.2E-02	2.2E-04	3.7E-04
Vinyl chloride	7.2E-06	1.6E-05	5.6E-04	1.2E-04		
Total HI:	**1E-03**	**6E-05**	**3E-01**	**1E-02**	**2E-04**	**1E-03**

Table 37. Recreational Wader: Potential cancer risks and non-cancer hazards associated with contact with Codorus Creek surface water under pumping and non-pumping conditions

COPC	CTE		RME	
	ILCR	HQ	ILCR	HQ
Pumping				
Tetrachloroethene	2E-10	2E-04	4E-10	4E-04
Trichloroethene	3E-09	1E-03	6E-09	2E-03
Total:	**4E-09**	**1E-03**	**6E-09**	**3E-03**
Non-pumping				
Tetrachloroethene	2E-09	2E-03	3E-09	3E-03
Trichloroethene	1E-08	4E-03	2E-08	6E-03
Total:	**1E-08**	**5E-03**	**2E-08**	**8E-03**

Table 38. Hypothetical future potable use of groundwater: Ratio of maximum detected concentration to putative cleanup goal

COPC	Putative cleanup goal (µg/L)	Source	Ratio of maximum detect to putative cleanup goal					
			LUA #1	LUA #2	LUA #3	LUA #4	LUA #5	LUA #6
1,1,1-Trichloroethane	2.0E+02	MCL			7E+01			
1,1-Dichloroethane	2.8E+00	RSL	7E-02		4E+00			
1,1-Dichloroethene	7.0E+00	MCL	1E+01		4E+02			
cis-1,2-Dichloroethene	7.0E+01	MCL	8E+01	1E+00	2E+02	1E+00		
1,2,4-Trimethylbenzene	1.5E+00	RSL	8E+01		7E+01			
1,3,5-Trimethylbenzene	1.2E+01	RSL	4E+00		2E+00			
1,4-Dioxane	4.6E-01	RSL	8E-01		8E+00			
4-Chloroaniline	3.7E-01	RSL			7E-01			
Acrylonitrile	5.2E-02	RSL			1E+02			
Benzene	5.0E+00	MCL	5E+02		3E+02			
Chloroform	8.0E+01	MCL			5E+00			
Ethylbenzene	7.0E+02	MCL	1E+00		3E+00			
Isopropylbenzene	4.5E+01	RSL	2E-01		2E-01			
Methylene chloride	5.0E+00	MCL	5E+00	5E+00	1E+02	2E+00		
Naphthalene	1.7E-01	RSL	3E+01					
Nitrobenzene	1.4E-01	RSL			1E+01			
Tetrachloroethene	5.0E+00	MCL	5E+02	1E+02	4E+03	9E+01	1E+01	1E+01
Toluene	1.0E+03	MCL	8E+00		7E+00			
Trichloroethene	5.0E+00	MCL	6E+02	5E+01	7E+03	3E+01	4E+00	3E+01
Vinyl chloride	2.0E+00	MCL	2E+02		6E+01			4E+00
Benzo(*a*)anthracene	1.2E-02	RSL			5E-01			
Benzo(*b*)fluoranthene	3.4E-02	RSL			9E-02			
bis(2-Ethylhexyl)phthalate	6.0E+00	MCL			2E+00			2E+00
Dibenzo(*a,h*)anthracene	3.4E-03	RSL			2E+00			
Indeno(*1,2,3-cd*)pyrene	3.4E-02	RSL			2E-01			
Pentachlorophenol	1.0E+00	MCL			4E+00			
Arsenic	1.0E+01	MCL	2E+00	2E+00	4E+00	9E+00		
Beryllium	4.0E+00	MCL			1E+00	1E+01		
Chromium	1.0E+02	MCL			1E+01			
Chromium (hexavalent)	3.5E-02	RSL			1E+03	9E+01	3E+00	9E-01
Iron, dissolved	1.4E+03	RSL				1E+00		
Lead	1.5E+01	RSL	3E+00	3E+00	7E+00	2E+01		
Manganese, dissolved	4.3E+01	RSL			2E+00	3E+00		1E-01
Nickel	3.9E+01	RSL	1E-01	1E-01	2E-01	9E-01		
Silver	9.4E+00	RSL			2E-01			
Vanadium	8.6E+00	RSL	5E-01	5E-01	9E-01	8E-01		
Zinc	6.0E+02	RSL			1E-01	2E-01		

>1<10
>10<100
>100<1000
>1000

Notes:

Blanks indicate that chemical was not selected as a COPC

Dissolved metal results were eliminated if total results were available as no filtration is assumed

MCL = Maximum Contaminant Level

RSL = EPA Regional Screening Level (May 2016, TR = 10^{-4}, THQ = 1)

Table 39. Hypothetical future residential vapor intrusion: Ratio of maximum detected concentration to putative cleanup goal

COPC	Putative cleanup goal	Source	Ratio of maximum detect to putative cleanup goal	
			On-Site	Off-Site*
1,1,1-Trichloroethane	2.0E+02	MCL	7E+01	
1,1-Dichloroethane	1.2E+01	VISL	2E-01	
1,1-Dichloroethene	7.0E+00	MCL	4E+02	
cis-1,2-Dichloroethene	7.0E+01	MCL	2E+02	1E+00
1,2,4-Trimethylbenzene	6.4E+00	VISL	2E+01	
1,3,5-Trimethylbenzene	6.4E+00	VISL**	8E+00	
Benzene	5.0E+00	MCL	2E+03	
Ethylbenzene	7.0E+02	MCL	4E+00	
Methyl *tert*-butyl ether	6.9E+02	VISL	1E-02	
Naphthalene	1.1E+01	VISL	5E-01	
Tetrachloroethene	5.0E+00	MCL	5E+02	4E+01
Toluene	1.0E+03	MCL	2E+01	
Trichloroethene	5.0E+00	MCL	1E+03	6E+01
Vinyl chloride	2.0E+00	MCL	2E+02	

>1<10
>10<100
>100<1000
>1000

Notes:
* LUAs #5 and #6
** Value for 1,2,4 trimethylbenzene used in the absence of a VISL for 1,3,5-trimethylbenzene Blanks indicate that chemical was not selected as a COPC
MCL = Maximum Contaminant Level; VISL = EPA Vapor Intrusion Screening Level (May 2016, TR = 10^{-4}, THQ = 1)

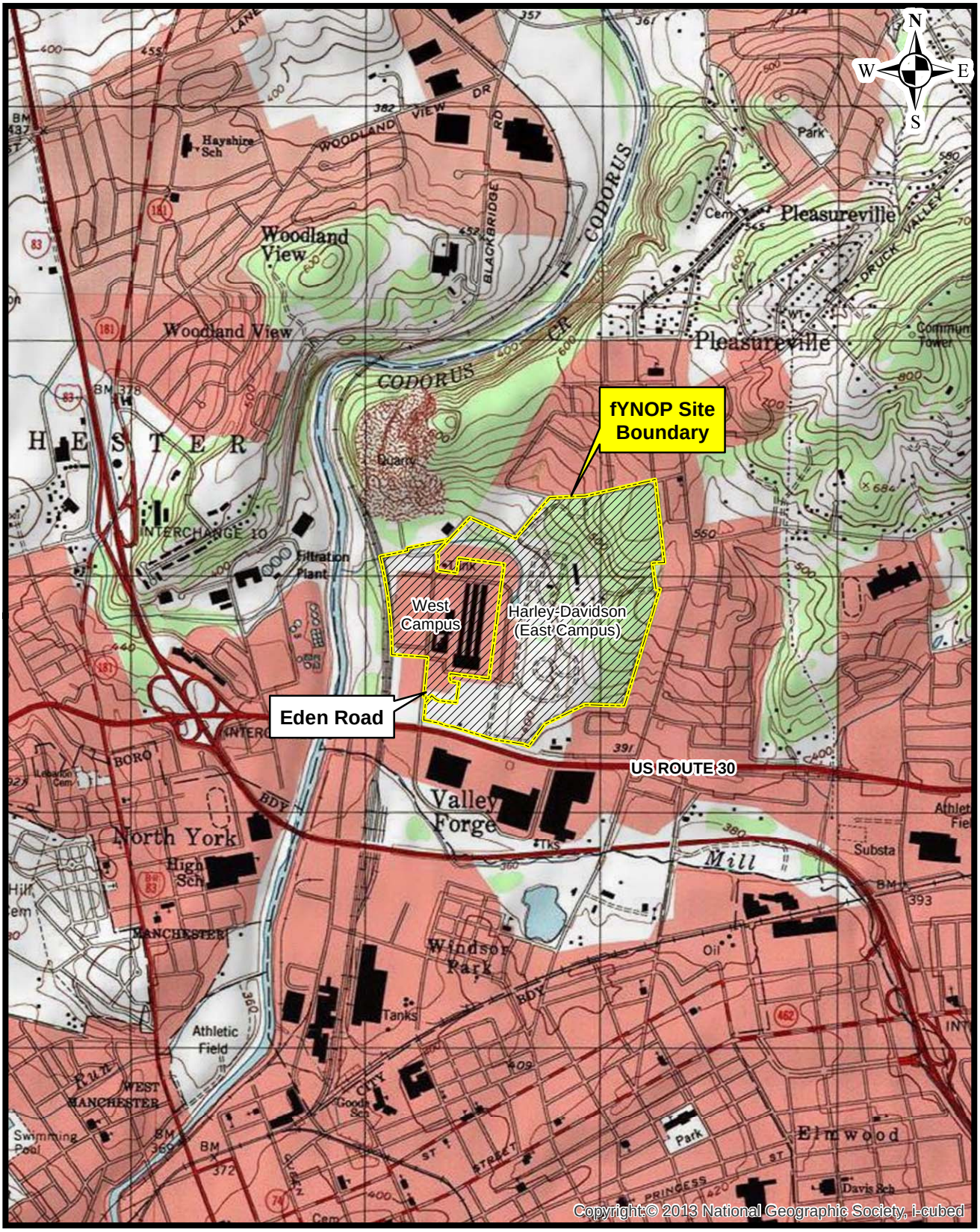
Table 40. Summary of incremental lifetime cancer risks calculated in the fYNOP Groundwater HHRA

LUA	Exposure level	Comm/Ind Worker	Groundwater ≤ 15 ft bgs			Groundwater > 15 ft bgs		Recreational Wader
			Construction Worker	Utility Worker	driver	Construction Worker	Utility Worker	
1	CTE		■	■		■	■	
	RME		■	■		■	■	
1&3	CTE		■	■				
	RME		■	■	Benzene			
2	CTE	■	■	■		■	■	
	RME	■	■	■		■	■	
3	CTE	■	■	■		■	■	
	RME	■	■	■		■	■	
4N	CTE					■	■	
	RME					■	■	
4S	CTE					■	■	
	RME					■	■	
5	CTE		■	■		■	■	
	RME		■	■		■	■	
6-pumping	CTE	■	■	■				
	RME	■	■	■				
6-nonpumping	CTE	■	■	■				
	RME	■	■	■				
7 - pumping	CTE							■
	RME							■
7 - nonpumping	CTE							■
	RME							■
■	denotes ILCR ≤ 10^{-4}							
■	denotes ILCR >10^{-4}							

Table 41. Summary of non-cancer hazard indices calculated in the fYNOP Groundwater HHRA

LUA	Exposure level	Comm/Ind Worker	driver	Groundwater ≤ 15 ft bgs				Groundwater > 15 ft bgs		Recreational Wader
				Construction Worker	driver	Utility Worker	driver	Construction Worker	Utility Worker	
1	CTE						TCE			
	RME						TCE			
1&3	CTE				124TMB		124TMB,Bz,Nap			
	RME				124TMB		124TMB,Bz,Nap			
2	CTE						TCE			
	RME		TCE				TCE			
3	CTE		TCE		c12DCE,TCE		PCE,TCE			
	RME		TCE		c12DCE,TCE		11DCE,c12DCE,PCE,TCE,VC			
4N	CTE									
	RME									
4S	CTE									
	RME									
5	CTE									
	RME						TCE			
6-pumping	CTE						TCE			
	RME						TCE			
6-nonpumping	CTE						TCE			
	RME						TCE			
7 - pumping	CTE									
	RME									
7 - nonpumping	CTE									
	RME									

denotes HI ≤ 1
denotes HI >1 ≤ 10
denotes HI >10 ≤ 100
denotes HI >100

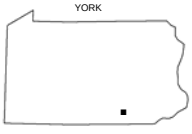


Portion of the York and York Haven PA 7.5-minute USGS Quadrangles
(2001)

Figure 1

Former York Naval Ordnance Plant
1425 Eden Road, York, PA 17402

Site Location Map



Scale (feet)
0 500 1,000 2,000

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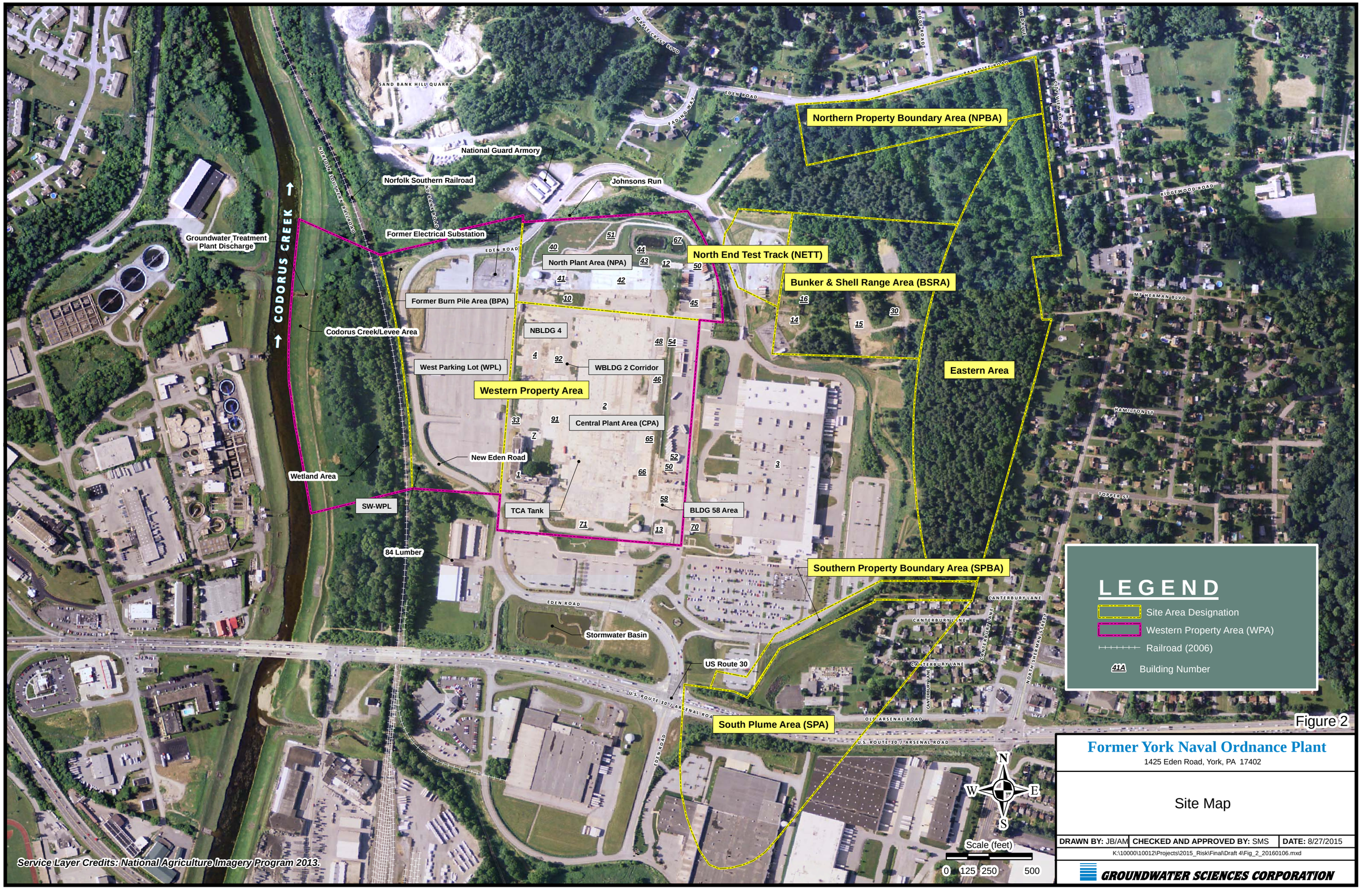

Figure 2

Former York Naval Ordnance Plant

1425 Eden Road, York, PA 17402

Site Map

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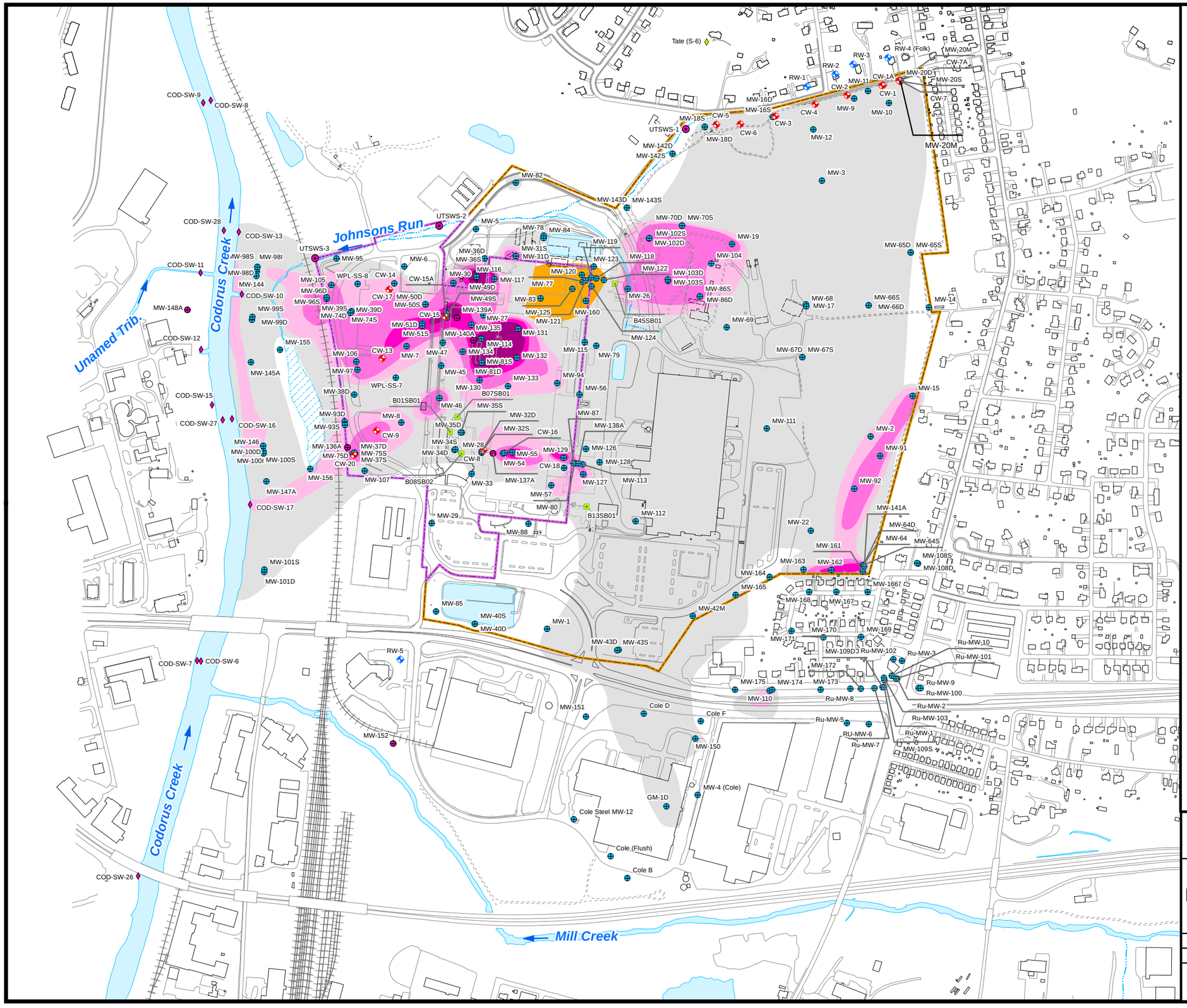

Legend

- Surface Water Sample Location
- Surface Water/Sediment Sample Location
- Soil Vapor Sample Location
- Spring
- Collection Well
- Monitoring Well
- Residential Well
- Waterloo Monitoring Well
- Existing Building
- Demolished/Slab Remains
- Demolished/Slab Removed
- West Campus Property Boundary
- Harley-Davidson Property Boundary (East Campus)
- Railroad
- Road (Paved)
- Road Curb
- Road (Unpaved)
- Walkway
- Fenceline
- Existing Stream
- Existing Water Feature
- Wetland Boundary (2006)
- TCE/PCE Concentration 5 ug/L
- TCE/PCE Concentration 50 ug/L
- TCE/PCE Concentration 100 ug/L
- TCE/PCE Concentration 500 ug/L
- TCE/PCE Concentration 1,000 ug/L
- TCE/PCE Concentration 10,000 ug/L
- Petroleum Plume

Note:
TCE/PCE concentration contours are for the shallow portion of the aquifer (defined as any well monitoring groundwater within approximately 75 feet of the ground surface).

N
W E
S

Scale (feet)
0 300 600

Figure 3

Former York Naval Ordnance Plant
1425 Eden Road, York, PA 17402

TCE/PCE and Petroleum Plumes, Monitoring Wells, Soil Vapor, and Surface Water Sampling Locations

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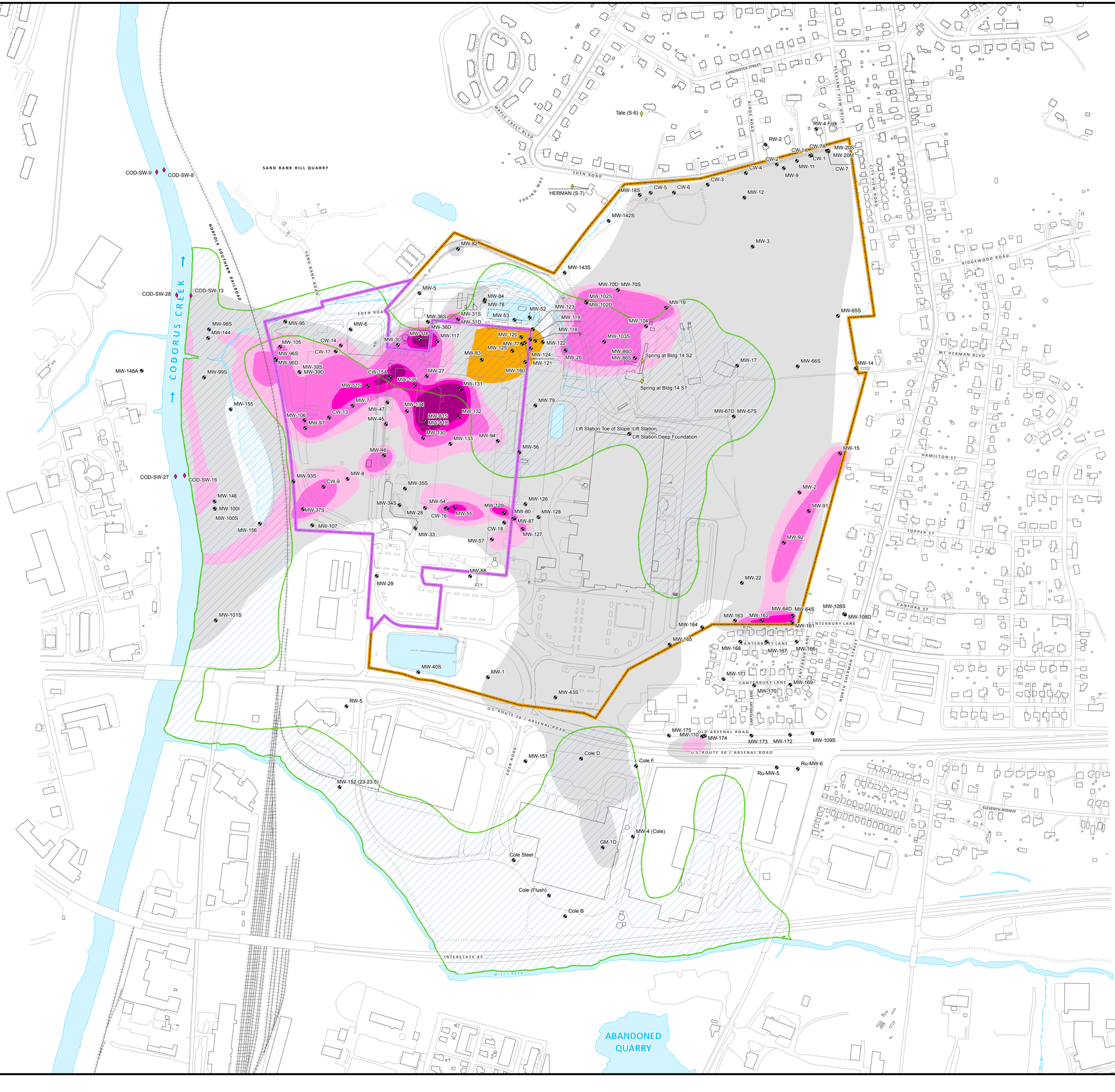

Legend

- Spring
- Well Location
- Surface Water Sample Location
- West Campus Boundary
- Harley-Davidson Property Boundary (East Campus)
- Existing Building
- Demolished/Slab Remains
- Demolished/Slab Removed
- Area Where Water Table is Within 15 Feet of Ground Surface
- Railroad
- Road (Paved)
- Road Curb
- Road (Unpaved)
- Walkway
- Fenceline
- Existing Stream
- Existing Water Feature
- Wetland Boundary (2006)
- TCE/PCE Concentration 5 ug/L
- TCE/PCE Concentration 50 ug/L
- TCE/PCE Concentration 100 ug/L
- TCE/PCE Concentration 500 ug/L
- TCE/PCE Concentration 1,000 ug/L
- TCE/PCE Concentration 10,000 ug/L
- Petroleum Plume

NOTE:
1) TCE/PCE concentration contours are for the shallow portion of the aquifer (defined as any well monitoring groundwater within approximately 75 feet of the ground surface).

Figure 4

N W E S

Scale (feet)
0 150 300 600

Former York Naval Ordnance Plant
1425 Eden Road, York, PA 17402

TCE/PCE and Petroleum Plumes, Areas Where Groundwater Depth is ≤ 15 Feet, Shallow (<75-foot) Monitoring Wells, and Surface Water Sampling Locations in the Affected Segment of Codorus Creek

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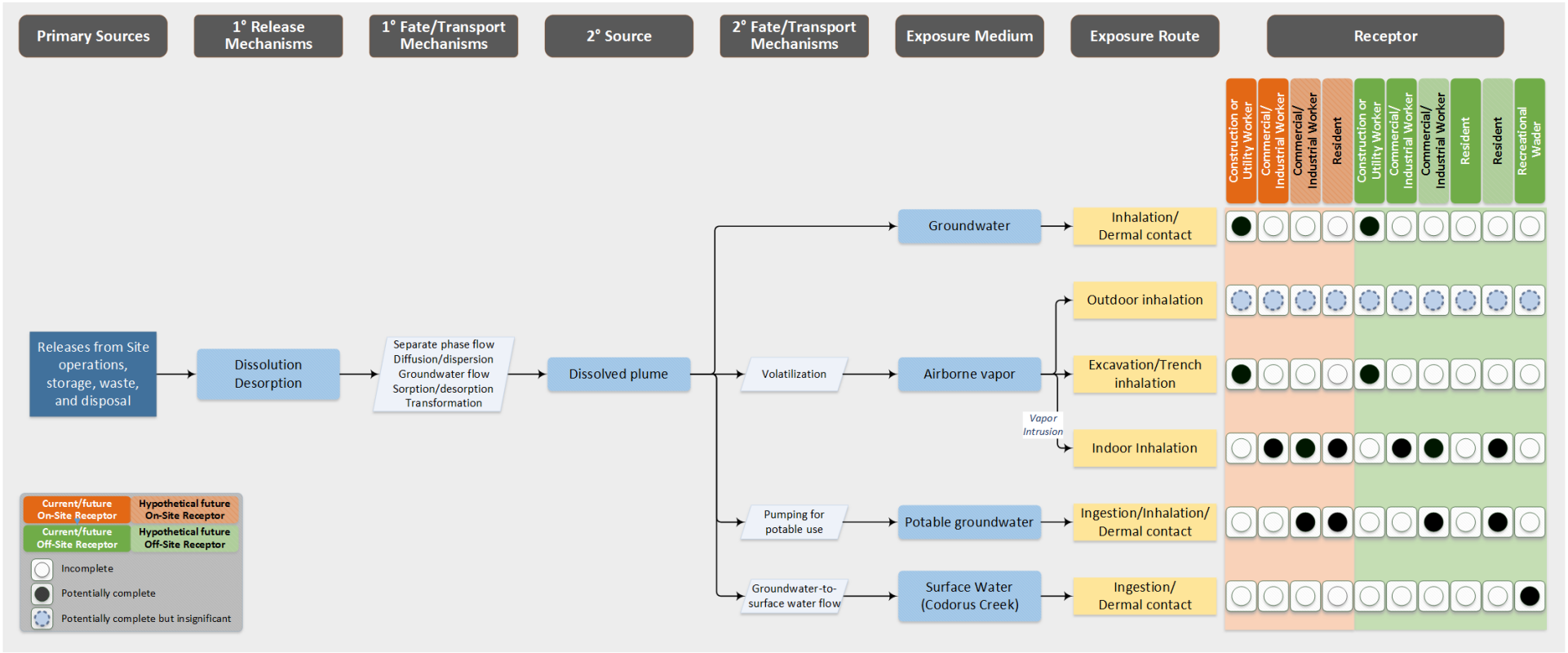
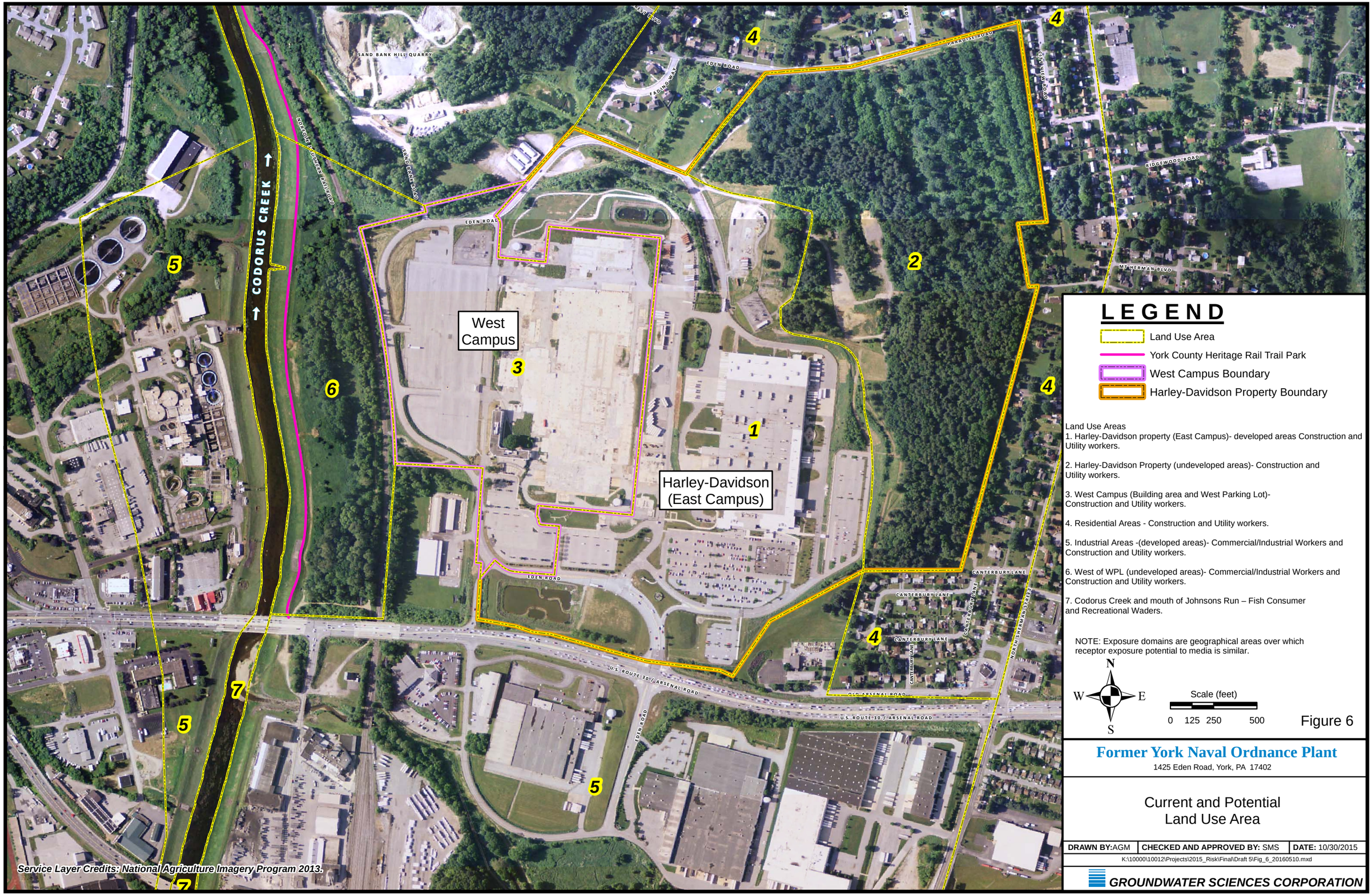


Figure 5. Exposure pathway model for the fYNOP groundwater HHRA



LEGEND

- Land Use Area
- York County Heritage Rail Trail Park
- West Campus Boundary
- Harley-Davidson Property Boundary

Land Use Areas

1. Harley-Davidson property (East Campus)- developed areas Construction and Utility workers.

2. Harley-Davidson Property (undeveloped areas)- Construction and Utility workers.

3. West Campus (Building area and West Parking Lot)- Construction and Utility workers.

4. Residential Areas - Construction and Utility workers.

5. Industrial Areas -(developed areas)- Commercial/Industrial Workers and Construction and Utility workers.

6. West of WPL (undeveloped areas)- Commercial/Industrial Workers and Construction and Utility workers.

7. Codorus Creek and mouth of Johnsons Run – Fish Consumer and Recreational Waders.

NOTE: Exposure domains are geographical areas over which receptor exposure potential to media is similar.

N W E S

Scale (feet)

0 125 250 500

Figure 6

Former York Naval Ordnance Plant

1425 Eden Road, York, PA 17402

Current and Potential Land Use Area

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Legend

- Spring
- Surface Water Sample Location
- Well Location
- Land Use Area
- Area Where Water Table is Within 15 Feet of Ground Surface
- West Campus Boundary
- Harley-Davidson Property Boundary (East Campus)
- Existing Building
- Demolished/Slab Remains
- Demolished/Slab Removed
- Railroad
- Road (Paved)
- Road Curb
- Road (Unpaved)
- Walkway
- Fenceline
- Existing Stream
- Existing Water Feature
- Wetland Boundary (2006)
- TCE/PCE Concentration 5 ug/L
- TCE/PCE Concentration 50 ug/L
- TCE/PCE Concentration 100 ug/L
- TCE/PCE Concentration 500 ug/L
- TCE/PCE Concentration 1,000 ug/L
- TCE/PCE Concentration 10,000 ug/L
- Petroleum Plume
- 7 — Landuse Area Designation
- MNW-162 — Well Selected for VOC EPC Calculation
- COD-SW-16 — Surface water Location for VOC EPC Calculation

Land Use Areas
1. Harley-Davidson property (East Campus)- developed areas Construction and Utility workers.

2. Harley-Davidson Property (undeveloped areas)- Construction and Utility workers.

3. West Campus (Building area and West Parking Lot)- Construction and Utility workers.

4. Residential Areas - Construction and Utility workers.

5. Industrial Areas -(developed areas)- Commercial/Industrial Workers and Construction and Utility workers.

6. West of WPL (undeveloped areas)- Commercial/Industrial Workers and Construction and Utility workers.

7. Codorus Creek and mouth of Johnsons Run – Fish Consumer and Recreational Waders.

NOTE:
1) Exposure domains are geographical areas over which receptor exposure potential to media is similar.

2) Yellow highlighted locations indicate they were used in the VOC EPC calculation.

3) TCE/PCE concentration contours are for the shallow portion of the aquifer (defined as any well monitoring groundwater within approximately 75 feet of the ground surface).

Figure 7

N W E S

Scale (feet)
0 150 300 600

Former York Naval Ordnance Plant
1425 Eden Road, York, PA 17402

Exposure Domains for the Groundwater HHRA

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APPENDICES A – K are on the compact disc (CD) in the front pocket of this binder.