Public Health Assessment

Final Release

MATLACK, INC. SITE

NEW JERSEY

EPA FACILITY ID: NJD043584101

Prepared by

NEW JERSEY DEPARTMENT OF HEALTH
Environmental and Occupational Health Surveillance Program

MAY 25, 2023

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Office of Capacity Development and Applied
Prevention Science
Atlanta, Georgia 30333

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR's Cooperative Agreement Partner pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR's Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR's Cooperative Agreement Partner addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR's Cooperative Agreement Partner which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Summary

Introduction

The Matlack site is in Woolwich Township, Gloucester County, New Jersey and operated as a truck terminal and tank-trailer cleaning facility between 1962 and 1976. The site is located within a mixed industrial, residential/rural area and contains a single-story building. Wastewater generated during tank cleaning was disposed of in an on-site unlined lagoon and underground tanks resulting in the contamination of surface water, sediment, soil, and groundwater. Potential contaminants of concerns are chlorinated volatile organic compounds, polycyclic aromatic hydrocarbons, and metals detected above health comparison values established by the Agency for Toxic Substances and Disease Registry (ATSDR). There are completed exposure pathways via incidental ingestion/dermal contact of surface water and sediment in the past, present, and future. The exposed populations are child and adult hikers accessing the off-site areas. The ATSDR and the New Jersey Department of Health (NJDOH) released a *Preliminary Health Assessment* in 1989 based on limited data, however, a complete evaluation could not be conducted since on- and off-site data were unavailable. In 1994, NJDOH and ATSDR released a *Site Review and Update* that evaluated the status of the Matlack site and identified future ATSDR activities planned for the site. The site is currently occupied by Liberty Kenworth, a truck dealership.

In May 2013, the United States Environmental Protection Agency (USEPA) listed the Matlack site to the National Priorities List (NPL). The NJDOH, in cooperation with the ATSDR, prepared the following public health assessment to evaluate the public health implications of potential exposures to contaminants found at the site.

The public comment period for this public health assessment was from August 12, 2022 to September 12, 2022. This document was shared with the current business operator, the NJDEP, the EPA, the Gloucester County Health Department, and the Gloucester County Library in Swedesboro. No comments were received.

The top priority of the NJDOH and ATSDR at this site is to ensure that the community around the site has the best information possible to safeguard its health.

Conclusions	Based on the data available for review by the NJDOH and ATSDR, there are completed exposure pathways involving surface water and sediment at Grand Sprute Run (off-site), and two potential on-site exposure pathways involving groundwater and indoor air. The NJDOH and ATSDR have reached the following conclusions in this public health assessment:
Conclusion 1	*The NJDOH and ATSDR conclude that the cumulative cancer risk from past and current exposures to site-related chlorinated volatile organic compound and polycyclic aromatic hydrocarbons from multiple pathways at the Grand Sprute Run tributary may have harmed people's health.*
Basis for Conclusion	Based on the surface water data for polycyclic aromatic hydrocarbons, the cancer risk resulting from past and current exposures from wading in the Grand Sprute Run tributary may result in an increased theoretical cancer risk for those residents who frequently used the tributary for recreational purposes, such as hiking and playing. For exposures associated with ingesting sediment and dermal contact with sediment along the Grand Sprute Run tribuary, the cancer risk from frequent exposure to volatile organic compounds in the sediment represents a low cancer risk. The contaminated area at the Grand Sprute Run is wooded and inaccessible by the area residents. As such, any interim remedial measures (such as, posting sign or fencing the area) to interrupt the pathway is not warranted.
Conclusion 2	*The NJDOH and ATSDR cannot conclude whether past exposures to site-related contaminants in the on-site production well may have harmed people's health.*
Basis for Conclusion	Based on the limited data available, site groundwater was contaminated with volatile organic compounds. In the past, the production well was the primary source of drinking water for the former Matlack employees. This is a past potential pathway because there are no data to evaluate the public health implications of the drinking water pathway. Currently, the well is out of service and the Liberty Kenworth employees are drinking bottled water.

Conclusion 3	*The NJDOH and ATSDR conclude that if the soil and groundwater contamination are not fully remediated and the land use changes (e.g., industrial to residential) in the future, the contamination may be a source of potential exposure pathways.*
Basis for Conclusion	The groundwater plume continues to discharge contaminants to the Grand Sprute Run tributary. The soil and groundwater have been delineated but not yet remediated.
Next Steps	• The NJDOH and ATSDR support the preferred remedy as described in the record of decision for the site be implemented as soon as feasible by the USEPA. The major components of the remedy include installation of two permeable reactive barriers in the groundwater; excavation and off-site disposal of contaminated soil and sediment; long-term monitoring (to assure the effectiveness of the remedy over time); and institutional controls until remedial action objectives are met. • The NJDOH and ATSDR recommend that Liberty Kenworth employees continue to use bottled water for drinking. • If land use changes, the NJDOH and ATSDR recommend an assessment of all potential pathways. Examples include an analysis of the vapor intrusion pathway and/or an analysis of incidental ingestion of on-site soil for any future planned residential homes.
For More Information	Questions about this public health assessment should be directed to: Environmental and Occupational Health Surveillance Program Consumer and Environmental Health Services New Jersey Department of Health and Senior Services P.O. Box 369 Trenton, New Jersey 08625-0369 (609) 826-4984

Statement of Issues

The Matlack Incorporated (Inc.) site is located on the south side of Route 322 in Woolwich Township, New Jersey. The site was proposed to the National Priorities List (NPL) in 1985 and then removed from the list in 1989 because it was eligible for cleanup actions under the Corrective Action program of the Resource Conservation and Recovery Act (RCRA). However, on August 9, 2011, the New Jersey Department of Environmental Protection (NJDEP) requested that the United States Environmental Protection Agency (USEPA) consider the Matlack site for NPL listing under Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). The site was listed in the NPL on May 24, 2013.

The New Jersey Department of Health (NJDOH), in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR), prepared the following public health assessment to review environmental data obtained from the site, evaluate potential human exposures to contaminants, and to determine whether the exposures are of public health concern. The primary medium contaminated at the site is the groundwater. Site investigations and planning for the site's cleanup are ongoing. The USEPA has selected a cleanup remedy for on-site contamination per the September 2017 Record of Decision (USEPA 2017a). It addresses all contaminated soil, groundwater, seep water, surface water and sediment at the site.

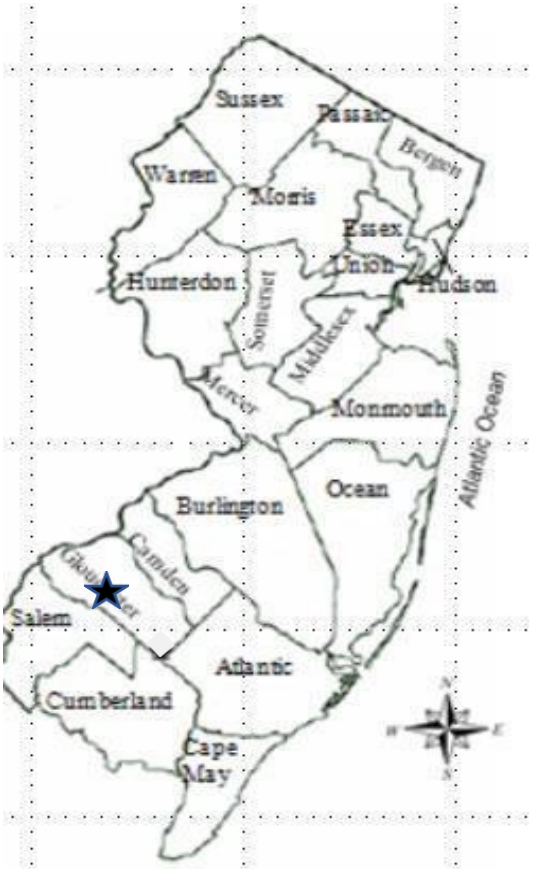

Figure 1: Location of the site

Background

Site Description and Operational History

Matlack, Inc. site is located on the south side of Route 322 in Woolwich Township, Gloucester County New Jersey (Figures 1 and 2). Currently, the 70-acre site is located within a mixed industrial, residential/rural area. The site contains a single-story structure occupied by Liberty Kenworth of South Jersey since 2018. The building and parcel used by Liberty Kenworth is located in the northeast portion of the site is fenced. The remaining portions, which are not fenced are mainly vegetated and are currently not being used/accessed. The western and southern portion of the site consists of scrub brush and fields; site is not accessible from west and south boundaries.

From the early 1900s until 1962, this site was a sand and gravel quarry. Matlack Inc. began operations in 1962 as a truck terminal and chemical tank trailer cleaning facility and continued until 2001. The site's primary waste was generated during tanker truck cleaning operations between 1962 and 1976. Truck and tanker washing activities included purging of tanks of residual material, rinsing of tanker interiors with a steam and detergent water solution along with either a mild caustic or acid solution. At least five percent of the tankers were cleaned with organic solvents that contained trichloroethylene (TCE), tetrachloroethylene (PCE), methylene chloride, toluene, acetone, methanol, and ethanol. The wastewater from cleaning operations was discharged into an unlined lagoon located southwest of the terminal building. The tanker and trailer cleaning operations generated between 5,000 and 15,000 gallons of wastewater per day. The lagoon was subsequently filled with a variety of demolition debris and other fill materials.

In 1976, Matlack began pre-treating the wastewater prior to transporting it off site for disposal. After treatment, the wastewater was stored in two 18,000-gallon open-top concrete tanks. Tanker cleaning operations discontinued in November 1997, but Matlack continued to service and store vehicles at the site until 2001. In 2001, Matlack went bankrupt and abandoned the site.

Subsequently, the NJDEP took over remediation at the site. Liberty Kenworth, a medium and heavy-duty truck sales and service center (with about 40 employees) has been operating at the site since February 2008. The operations are limited to the northeastern portion of the site and include an approximately 26,000 square feet, 14 bay service building and paved parking area.

Site Geology and Hydrogeology

The Matlack site is in the Atlantic Coastal Plain physiographic province (USEPA 2017b). The site is underlain by the Pennsauken Formation consisting of sand with some gravel and clay. This formation is approximately 30 feet thick and forms the upper unconfined aquifer which drains northwest into the Grand Sprute Run (GSR) tributary. Below the Pennsauken Formation is the Woodbury Clay and the Merchantville Formation. Together, these units vary from 54 to 62 feet thick, and separate the unconfined upper aquifer from the confined deep aquifer. Stratigraphically, the deep aquifer is a part of the undifferentiated Magothy-Raritan Formations that consist of interbedded clays and sands.

Groundwater beneath the site exists within the two separate aquifers (USEPA 2012a). The upper shallow aquifer flows on top of the Woodbury Clay and incises the Pennsauken Formation. All groundwater in the Pennsauken Formation is therefore intercepted by GSR. The shallow groundwater flow direction is across the Site north-northwest, flowing towards GSR. The water table is approximately 4 feet below the surface at the southeast comer of the site to approximately 28 feet below the surface at the northwest corner of the site. While not considered a major source of water for domestic use, a few wells within the county are located here. The

deeper aquifer[1] is separated from the upper aquifer by a clay confining unit over 50 feet thick. Groundwater in this deeper aquifer flows toward the southeast (in the direction of the Atlantic Ocean). Most of the commercial and \public water supplies within Gloucester County are contained here (USEPA 2012a).

The GSR tributary, located approximately 600 feet west of the site, is the closest surface water body. It is a shallow tributary, with depths ranging from 2 inches (0.16 feet) to nearly 3 feet. The tributary is approximately 1.25 miles, emptying into Raccoon Creek. The lower third of the tributary is primarily swamp, with little flow. GSR meanders throughout its entire length, splitting and rejoining numerous times. The flow is controlled by the amount of organic debris (fallen trees and branches) which blocks the channel. Measurements indicate a maximum velocity of 0.22 to 0.76 feet per second (USEPA 2017a). GSR is wholly contained within the 10-foot elevation contour along the entire length. The shallow groundwater in the Pennsauken Formation is intercepted by the tributary (USEPA 2017a).

Regulatory and Remedial History

In May1987, Matlack Inc. entered into an Administrative Consent Order (ACO) with the NJDEP. Between 1982 and 2001, several environmental investigations and remediation measures were implemented at the Matlack site (USEPA 2017a). Remedial measures included the installation of on and off-site groundwater monitoring wells and a groundwater remediation system consisting of twelve recovery/monitoring wells located downgradient of the former waste tanks and lagoon area. Between 1995 -2009, the groundwater remediation system operated inconsistently. There were various problems with its operation and maintenance.

Between 1992 and 2007, there were three phases of soil excavations and tank removals at the site. In 1992, two buried waste tanks (T7 and T8) located west of the terminal building and contaminated soils associated with a diesel fuel line leak area were excavated and removed. A second major soil excavation occurred in September 1999 which included excavation and removal of nine underground storage tanks (USTs). Four diesel USTs ranging in size from 15,000 to 20,000-gallons, three #2 fuel oil USTs (two 15,000-gallon and one 3,000-gallon), a 6,000-gallon motor oil UST, and a 6,000-gallon TCE UST were removed. In addition, approximately 375 feet of piping leading from the diesel tanks were excavated and removed. All excavations were backfilled with clean fill and/or uncontaminated excavated soil (USEPA 2012a).

The former unlined lagoon area and the contaminated soils were not remediated but were left in place and the lagoon was subsequently covered with a variety of demolition rubble and clean fill (USEPA 2012a). The third phase occurred in July 2006 and July 2007, when three above ground storage tanks (ASTs) ranging in size from 18,000-gallon to 50,000-gallon and two USTs (800-gallon and 3,000-gallon) associated with the former wastewater treatment system were excavated and removed. In addition, two 4,000-gallon flocculation and oil skimming USTs

[1]It should be noted that only the shallow groundwater that discharges to the stream is contaminated; the deeper aquifer is not contaminated from the site.

located very close to the treatment building were abandoned in place (emptied, cleaned, and filled with concrete).

In September 2017, EPA released the Record of Decision (ROD) to remediate contaminated soil, groundwater, seep water, surface water and sediment associated with the Matlack site (USEPA 2017a). The major components of the remedy include installation of two permeable reactive barriers (PRBs) in groundwater; excavation and off-site disposal of contaminated soil and sediment; long-term monitoring (to assure the effectiveness of the remedy over time); and institutional controls until remedial action objectives are met.

Prior NJDOH/ATSDR Involvement

In January 1989, NJDOH and ATSDR released a Preliminary Health Assessment for the site to address the public health implications of exposures to volatile organic compounds (VOCs) and polycyclic aromatics hydrocarbons (PAHs) in on-site soils and groundwater that included surrounding residential wells (ATSDR 1989). Based on the initial findings, NJDOH and ATSDR categorized the site as a potential public health concern. The assessment recommended that numerous data gaps be filled including: (1) contaminant levels in local residential wells; (2) review of remedial action taken at the contaminated residential wells; and (3) further environmental characterization and sampling of both on-site and off-site areas including sampling of soil and water.

In August 1994, NJDOH and ATSDR released a Site Review and Update which evaluated the status of the Matlack site and identified future ATSDR activities planned for the site (ATSDR 1994). Based on a review of the residential well sampling data in the area surrounding the site, the report concluded that there were no completed exposure pathways at the site, including residential exposure to groundwater via private potable wells. The NJDOH and ATSDR categorized that current and future exposures to VOCs in residential groundwater posed no apparent public health hazard. However, it was recommended that residential wells be monitored periodically (ATSDR 1994)[2].

Land Use and Demographics

There are residences directly across Route 322 and one business identified as Excel Hydraulics. A State Wildlife Management Area and the GSR, a tributary of Raccoon Creek, which flows into the Delaware River, are located to the west and northwest of the site (see Figure 2). There is open farmland on the east and south side of the site. An elementary school and daycare facility are located about 5,000 feet to the east of the site.

[2]It should be noted that the RI data collected clearly show that the private wells (on the east side of Rt 322) located upgradient of the on-site contaminated plume are not contaminated with site related contaminants and this information was not available at the time the earlier ATSDR reports were prepared.

Based on 2010 United States Census data, it is estimated that there are approximately 300 individuals living within one mile of the Matlack site. The closest residence is approximately 200 feet north of the site.

Site Visit

Staff from the NJDOH and ATSDR performed a site visit on March 8, 2016, to gather updated information about the site and surrounding area, including potential human exposure pathways to workers, trespassers, and residents. The northern portion of the property contains a single-story building occupied by Liberty Kenworth, a sales and servicer of tractor trailers. The main entrance is paved and used for storage of new and used tractor trailers and trailer beds. The groundwater recovery and treatment system are in a small building behind the main building. Beyond the treatment building towards the south end of the property is undeveloped grass and woodland. The on-site area is surrounded by 6-foot-high chain-link fence and there is no public access to the site areas during non-business hours.

The off-site areas and GSR can be accessed via an access road directly to the west of the site. This road also leads to an undeveloped woodland and eventually to the tributary. Photos from the site visit are presented in Appendix A.

Currently, the EPA is preparing the design of the remedial action and remedial activities are anticipated to start in late 2022. To help develop the remedial design, two phases of pre-design field work have been conducted thus far, and a third phase should be completed by the end of 2021 (S. Kandil, EPA Region 2, Personal Communication, Oct. 13, 2021).

Community Concerns

Past community concerns included possible private well contamination and odor complaints. These concerns were noted during the initial public health assessment in 1989 and the site review and update completed in 1994.

USEPA held a public meeting on September 14, 2017 in Woolwich, New Jersey, to present the remedial alternatives (including USEPA's preference), Proposed Plan, and answer questions. At the September 14, 2017 public meeting, the representatives of local government or the community members did not express any concern about private well contamination (USEPA 2017b).

Environmental Contamination

An evaluation of site-related environmental contamination consists of a two-tiered approach: 1) a screening analysis; and 2) a more in-depth analysis to determine public health implications of site-specific exposures (ATSDR 2005). First, maximum concentrations of

detected substances are compared to media-specific environmental guideline comparison values (CVs). If contaminant concentrations exceed their CV, it is referred to as a potential contaminant of concern (COC) and selected for further evaluation. If environmental CVs are unavailable for certain contaminants, the contaminants are retained for further evaluation. If contaminant levels are found above CVs, it does not mean that adverse health effects are likely, but that a health guideline comparison and cancer risk evaluations are necessary to assess site- specific exposures. Once exposure doses and cancer risks are estimated, they are compared with health guideline doses and allowable cancer risks to determine the likelihood of health effects.

Environmental Guideline Comparison

There are several environmental CVs available for screening environmental contaminants to identify potential COCs. These include ATSDR Environmental Media Evaluation Guides (EMEGs) and Reference Media Evaluation Guides (RMEGs). EMEGs are estimated contaminant concentrations that are not expected to result in adverse non-carcinogenic health effects. RMEGs represent the concentration in water or soil at which daily human exposure is unlikely to result in adverse non-carcinogenic effects. If the substance is a known or a probable carcinogen, ATSDR's Cancer Risk Evaluation Guides (CREGs)[3] are also considered as comparison values. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed over their lifetime (78 years).

In the absence of an ATSDR environmental CV, other comparison values may be used to evaluate contaminant levels in environmental media. These include USEPA Regional Screening Levels (RSLs)[4] used for drinking water and soil, USEPA Maximum Contaminant Levels (MCLs) used for drinking water and NJDEP soil remediation standards (NJDEP SRS) for the inhalation exposure pathway and the ingestion-dermal exposure pathway.

On-Site

Previous environmental investigation efforts have identified two primary sources of contamination: (1) A leaking diesel fuel line; and (2) the underground waste tanks. The former lagoon area was identified as the source of groundwater contamination. Specific sources of contamination and areas of concern include buried trash, the wastewater treatment system located in the waste treatment building down gradient of the former lagoon and terminal building/tank area, waste and fuel underground storage tanks (USTs) and the terminal garage bay areas located within the southern portion of the Terminal building (see Figure 3).

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[3]EPA IRIS is used to generate a cancer potency factor for ATSDR CREG.

[4]RSLs are contaminant concentrations corresponding to a fixed level of risk (i.e., a Hazard Quotient of 1, or lifetime excess cancer risk of one in one million, or 10^{-6}, which ever results in a lower contaminant concentration) in water and soil (USEPA 2011).

Soil[5]

Former Unlined Lagoon (4 to 9.5 feet): From 1962 until 1976, wastewater from the cleaning operations was disposed of in an unlined lagoon. In 1976, the unlined lagoon was filled with demolition rubble. In May 1983, the NJDEP collected soil samples and one groundwater sample for volatile organic compound (VOC) analysis. PCE and TCE were detected in the soil samples at concentrations as high as 15,835 parts per billion (ppb) and 19,850 ppb, respectively. In March 1986, samples collected from the unlined lagoon revealed PCE at 5,600 ppb and TCE at 400 ppb.

- ***Soil (0 to 2 feet):*** In March 2016, soil (0 to 2 feet depth) samples from the unlined lagoon were collected; results revealed maximum concentrations of semi-volatile organic compounds (SVOCs), polychlorinated biphenyls (PCBs), metals and pesticides exceeding their respective CVs (see Table B1, Appendix B).

- ***Soil (4 to 35 feet)***: Between 2008 and 2016, soil (4 to 35 depth) samples from the unlined lagoon and the general on-site areas were collected; results revealed maximum concentrations of SVOCs, PCBs, metals and pesticides exceeding their respective CVs (see Table B1, Appendix B).

Groundwater[5]

In May 2000 and September 2008, groundwater samples were collected; results revealed maximum concentrations of PCE at 13,600 ppb, TCE at 1,300 ppb,1,1,1-trichloroethane at 8,100 ppb, and benzene at 92 ppb in monitoring wells located approximately 220 to 250 feet southwest of the former lagoon area.

- Between 2012 and 2016, groundwater data from 22 monitoring wells were collected; results revealed maximum concentrations of VOCs, SVOCs, metals, and PCBs exceeding their respective CVs.
- Groundwater data also showed the presence of two separate contaminant plumes, a PCE, TCE, Trichloroethane (TCA) plume and a benzene, toluene, ethylbenzene, and xylene (BTEX) plume associated with past site operations within the shallow groundwater. The locations of the wells and the groundwater plumes are shown on Figure 5.

On site Production Well

A production (supply) well is located along the western side of the facility near the garage bay area and was constructed in 1976 with a proposed depth of 50 feet (see Figure 3). This well served the entire facility (40 employees). Groundwater samples collected in 1983 showed total VOCs concentrations of 1,100 ppb. No further sampling was conducted for the next 25 years.

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[5]Since there are no exposure pathways associated with on-site soil and groundwater contamination, the analytical data are not presented in this document.

Between September 2008 and May 2011, Liberty Kenworth's water system was sampled for VOCs; the maximum concentrations of 1,4-dichlorobenzene, methylene chloride, and methyl ethyl ketone detected were below ATSDR's comparison values and the EPA's MCLs (see Table B2, Appendix B). All three of these compounds were also detected in the groundwater. In 2011, during a NJDEP inspection of the facility, a Liberty Kenworth employee stated that bottled water was used for drinking.

Soil Gas

Soil gas samples (1.5 to 4 feet at 22 locations) were collected from vacant sections (former lagoon area) of the site and analyzed for total VOCs. Results indicated the highest soil gas concentration for total VOCs was 413 ppm at location SG-5, which corresponds to the approximate center of the unlined lagoon (see Figure 6).

Off-Site

GSR (Surface Water and Sediment) - In June 2005, surface water samples were collected from nine locations along the tributary. Surface water sample results revealed concentrations of PCE (up to 40 ppb) and TCE (up to 6.0 ppb) above their respective environmental guideline CVs. In 2012, additional surface water samples were collected from the tributary. Results indicated the presence of PCE, TCE, and cis-1,2-DCE above their respective environmental guideline CVs.

In April 2016, surface water samples were collected along GSR. These samples were analyzed for VOCs, SVOCs, metals and PCBs. Table B2, Appendix B shows the number of samples collected, number of non-detects, maximum/minimum concentrations and the CVs. The maximum concentrations of contaminants exceeding their respective CVs were considered as the potential COCs for the surface water (see Table B3, Appendix B).

In April 2016, sediment samples were collected along GSR, downgradient from the Site and analyzed for VOCs, SVOCs, metals and PCBs. The locations of all samples are shown on Figure 7. Table B4, Appendix B shows the number of samples collected, number of non-detects, maximum/minimum concentrations and the CVs. The maximum concentrations of contaminants exceeding their respective CVs were considered as the potential COCs for the sediment (see Table B4, Appendix B).

Biota

There are over 2,000 square feet of wetlands along the GSR tributary containing numerous ecosystems including forested upland, open field, cropland, forested wetland, open water, and tidal marsh in the vicinity of the Matlack site. Wildlife, such as rabbits, songbirds, domestic geese and deer, has been observed (USEPA 2017b). There has been no biota sampling.

Potable Wells

In 1982, four residential wells ¼ mile northwest of the site was sampled by the Gloucester County Health Department (GCHD) and analyzed for total VOCs: results indicated only one well had a VOC concentration greater than 2,000 ppb. The residence was supplied with bottled water (while the potable well was being investigated) and the well was not used. In 1991, the same well was sampled and analyzed for organic compounds; no contaminants were detected in the tap water. In 1994, ATSDR and NJDOH conducted a Site Review and Update (ATSDR 1994) and concluded the 1982 residential well sampling result could have been due to laboratory error and potential contamination of active residential wells was no longer a concern at the site. However, ATSDR and NJDOH recommended that residential wells be monitored. It should be noted that the RI data collected recently clearly show that the private wells (on the east side of Rt 322) are located upgradient of the on-site contaminated areas and this information were not available at the time the earlier ATSDR reports were prepared. In 1997, GCHD conducted an analysis (GCHD 1997) of the same potable well. The pH of the water (6.4) was found to be lower than the allowable limit (6.5 to 8.0). The concentration of manganese 0.13 mg/L) also exceeded the EPA's Secondary Drinking Water standard (i.e., 0.05 mg/L). The GCHD recommended that the owner install appropriated treatment system.

Between 2001 and 2008, potable water samples were collected from residential wells north of the Matlack site, including the high VOC contaminated well from 1982 (NJDEP 2011). The most recent (i.e., 2008) results indicated concentrations of VOCs, semi-VOCs, pesticides, PCBs and tetrahydrofuran below their applicable drinking water quality standards. Lead was the only metal detected above its drinking water Action Level (i.e., 15 ppb). The range of detected level was 6.64 ppb to 15.2 ppb. The GCHD indicated that they have followed a standard response to address such contamination as required by the New Jersey Private Well Testing Act of 2001 (PWTA[6]) (Aderholt et al., 2009) (Personal Communication to Environmental Health Coordinator, 2021). According to the NJDEP, these contaminants were evaluated and found not to be site related. Based on the location of the current ground contamination plume, groundwater flow direction and the orientation of the Grand Spruce Run, it is evident that the residential wells are located upgradient of the contaminant plume and are unlikely to be contaminated with site-related contaminants. The GCHD standard response include informing the residents about the potential adverse health effects of drinking water containing lead, suggest techniques, equipment, strategies, and funding sources available to treat the well water that exceeded lead standard.

[6]In March 2001, the New Jersey Private Well Testing Act (PWTA) was signed into law, and its regulations became effective in September 2002. The PWTA requires that the NJDEP establish, in consultation with county, regional or local health departments and health officers, a public information and education program to inform the public and appropriate professionals of 1) the PWTA itself; 2) the potential adverse health effects of consuming water from a well that does not meet the standards; 3) areas of the state at higher risk for failure due to geologic or other reasons. 4) the importance of regularly testing private well water; and 5) suggested techniques, equipment, strategies, and funding sources available to treat water that has exceeded a standard.

Summary of potential Contaminants of Concern (COC)

The contaminants detected in the environmental media are designated as the potential COCs for the Matlack site. A brief discussion of the toxicologic characteristics of these potential COCs is presented in Appendix C.

Discussion

The method for assessing whether a health hazard exists to a community is to determine whether there is a completed exposure pathway from a contaminant source to a receptor population and whether exposures to contamination are high enough to be of health concern. Site-specific exposure doses can be calculated and compared with health guidelines.

Assessment Methodology

An exposure pathway is a series of steps starting with the release of a contaminant in environmental media and ending at the interface with the human body. A completed exposure pathway consists of five elements:

1. source of contamination,
2. environmental media and transport mechanism,
3. point of exposure,
4. route of exposure, and,
5. receptor population.

Generally, the ATSDR considers three exposure categories: 1) completed exposure pathways, that is, all five elements of a pathway are present; 2) potential exposure pathways, that is, one or more of the elements may not be present, but information is insufficient to eliminate or exclude the element; and 3) eliminated exposure pathways, that is, a receptor population does not come into contact with contaminated media. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination in the past, present, and future.

When assessing an exposure risk to a potential COC, the USEPA recommends use of the 95th percentile upper confidence limit (95% UCL) of the arithmetic mean to determine the exposure point concentrations (EPC) for site-related contaminants (USEPA 2013). An EPC is an estimation of the concentration of a contaminant at the point of human exposure (USEPA 1989). For contaminants that did not have enough data (i.e., at least 8 to 10 data points) to calculate a 95% UCL in surface and sediment, the maximum contaminant concentration was used as the EPC (see Table B5 for surface water and Table B6 for sediment, Appendix B).

The site is next to a highway across from a residential neighborhood and to the east of the tributary. The exposed populations for the Matlack site include adult and child hikers at the GSR tributary area. The age range for children likely to hike would be greater than six years. The

exposure pathways for site-related contaminants are presented in Appendix B, Table B7. Appendix B.

Completed Pathways

Incidental Ingestion of off-site contaminated Sediment & Dermal contact with off-site contaminated Sediment and Surface Water at the GSR Tributary (past, present, and future): The contaminated shallow groundwater flow is intercepted by the GSR. As such surface water (see Table B3, Appendix B) and the sediment (see Table B4, Appendix B) of the GSR are contaminated with VOCs, SVOCs, and metals. The depth of water in the Run ranges from about 2 inches to nearly 3 feet in the pond areas. The downstream area where it meets the Raccoon Creek is primarily swamp with little flow. Adult and child residents access the areas for recreational purposes (i.e., hiking) and exposure is likely by incidental ingestion of sediments and dermal contact with surface water and sediment during wading.

Potential Pathways

Ingestion of groundwater from the on-site production well (past, future). In the past, the production well was the primary source of drinking water for Matlack employees. One water sample was collected in 1984 and analyzed for total VOCs, but the speciation of these VOCs was unavailable. Past exposures could not be evaluated because the specific VOC data was not available. Water samples collected in 2016 indicate the well is not currently contaminated. However, since the well is located downgradient from the contaminated areas it may get contaminated due to the movement of the contaminant plume. There is a potential pathway for future exposure since the well is downgradient from the contaminated areas.

Incidental ingestion of on-site contaminated soil (0-2 and 4-9.5 feet) (former unlined lagoon and general site property) (future). The contamination of soil has been documented and not remediated. Thus, there is a potential pathway for future exposures to soils depending on future land use. If land use changes, and the soil is exposed, this pathway needs further evaluation.

Eliminated Pathways

Incidental ingestion of on-site contaminated soils (0-2 and 4-9.5 feet) (former unlined lagoon and general site property) (past, present). Although the soils at the site are contaminated, the type of activities (i.e., truck cleaning/maintenance) in the past did not require excavation into soils. The site is also fenced (see Figure 2) and trespassing is extremely unlikely. The on-going site operation does not require excavation and as such, currently the soil exposure pathway is also considered eliminated.

Ingestion of groundwater from the on-site production well (present). The production well is being monitored quarterly and no contaminants have been detected above NJDEP drinking water standards. Residential drinking water wells are in use within a quarter mile of the Site; however, the groundwater problems identified at the site only affect the shallow groundwater

aquifer and not deeper aquifer for potable water (USEPA 2017a). As a precaution, the current occupants of the site building (Liberty Kenworth employees) have been using bottled water. This pathway is considered eliminated.

Inhalation of contaminants in indoor air (past, present, future). Past on-site cleaning operations have contaminated the groundwater with VOCs and other contaminants. Recent groundwater and soil gas sampling have identified the presence of two plumes at the site (see Figures 5 and 6). Based on the location of the groundwater plumes and groundwater flow direction, vapors from the groundwater are unlikely to impact the on-site building. A screening evaluation was also conducted to determine if the potential for vapor intrusion (VI) into indoor air from subsurface vapor sources exists (USEPA 2017a). The VI screening consisted of comparing the maximum groundwater concentration of potential COCs to both residential and commercial based Vapor Intrusion Screening Levels (VISLs). The results did not indicate any potential for a VI pathway. As such, the pathway is considered eliminated.

Ingestion of biota (past, present, future). Biota (fish) in the GSR may have ingested contaminants through surface water and soil/sediment. However, GSR is a shallow tributary with an average depth of about 2 inches. The overall water depth in the GSR makes it unsuitable for fishing. As such, the biota pathway is considered eliminated.

Public Health Implications of Completed Exposure Pathways

Once it has been determined that individuals have or are likely to have contact with site-related contaminants (i.e., a completed exposure pathway), the next step in the public health assessment process is the calculation of site-specific exposure doses. This involves looking more closely at site-specific exposure conditions, the estimation of exposure doses, and comparison to health guidelines. Health guidelines are based on data drawn from the epidemiologic and toxicological literature and often include uncertainty (or safety) factors to ensure that they are protective of human health.

Non-Cancer Health Effects

To assess non-cancer health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants that are commonly found at hazardous waste sites. An MRL is an estimate of the daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of adverse, non-cancer health effects. MRLs are developed for a route of exposure, i.e., ingestion or inhalation, over a specified time period, e.g., acute (less than 14 days); intermediate (15 to 364 days); and chronic (365 days or more). MRLs are based largely on toxicological studies in animals and on reports of human occupational (workplace) exposures. MRLs are usually extrapolated doses from observed effect levels in animal toxicological studies or occupational studies and are adjusted by a series of uncertainty (or safety) factors or using statistical models.

In toxicological literature, observed effect levels include:

- no-observed-adverse-effect level (NOAEL); and
- lowest-observed-adverse-effect level (LOAEL).

The NOAEL is the highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals. LOAEL is the lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals. In order to provide additional perspective on these health effects, the calculated exposure doses were then compared to observed effect levels (e.g., NOAEL, LOAEL). As the exposure dose increases beyond the MRL to the level of the NOAEL and/or LOAEL, the likelihood of adverse health effects increases.

To ensure that MRLs are sufficiently protective, the extrapolated values can be several hundred times lower than the observed or no-observed adverse effect levels in experimental studies. When MRLs for specific contaminants are unavailable, other health-based guidelines, such as USEPA Reference Dose (Rd.) may be used (USEPA 1989). The Rd. is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

The non-cancer adverse health effects typically are assessed by comparing the exposure dose to the reference dose (i.e., MRL or Rd.) via a ratio known as the "hazard quotient" (HQ). The HQ is defined as: Hazard Quotient = Exposure Dose/MRL or Rd. As the HQ increases above 1, the potential for harmful effects also increases. Potential COCs with a hazard quotient exceeding a value of 1 were identified as COCs and evaluated further to determine whether these contaminants pose a health threat to potentially exposed populations.

Exposure Dose Assumptions and Scenarios

ATSDR's exposure dose guidance for Surface Water (ATSDR 2018b) and Soil/Sediment (ATSDR 2018a, ATSDR 2016b), and USEPA's Exposure Factor Handbook (USEPA 2004, USEPA 2011) were used to calculate exposure doses. Exposure doses were calculated for children (6 to 21 years) and adults using the ATSDR Public Health Assessment Site Tool (PHAST). For people with typical, or average soil ingestion rates, we used a "central tendency exposure" (CTE) scenario. For people with above average ingestion rates, a "reasonable maximum exposure" (RME) scenario was used. The RME refers to people with above average exposures but still within a realistic exposure range.

Dermal Absorption of contaminants in Surface Water at the GSR tributary (past, present and future)

The site-specific exposure scenario has been hiking in the Grant Sprute Run and adjacent areas (see Photographs, Appendix C) by adults and children (greater than 6-years-old). Since the land use on both sides of the GSR (downstream of the site) is farming/industrial/commercial and there is no direct access, it is unlikely that children younger than 6 years old access the area. Dermal contact (during wading) with contaminated surface water may occur during this recreational activity. The site-specific exposure factors and exposure parameters used (by

PHAST) to calculate the dermal exposure doses are given in Table B8, Appendix B (USEPA 2004, 2011).

Based on the EPCs of cis-1,2-dichloroethene, benzene, PCE, TCE, vinyl chloride, 1,4-dioxane, benzo(a) pyrene, aluminum, arsenic and cadmium detected in the surface water and the exposure assumptions, the PHAST was used to calculate HQs for chronic exposures for adults and children (6 to 21 years old); the HQs did not exceed one (and as such the data is not presented in this document). Therefore, exposures to these contaminants in surface water are not expected to cause non-cancer adverse health effects. The chronic health guideline values for 1,1-dichloroethane, benzo(b)fluoranthene, benzo(chi)perylene, benzo(k)fluoranthene, dibenzo(a, h)anthracene, indenol(1,2,3-cd)pyrene, cobalt, copper, manganese and vanadium are unavailable.

Based on the EPCs of cis-1,2-dichloroethene, PCE, TCE, 1,4-dioxane, aluminum, cadmium, copper, cobalt and vanadium detected in the surface water and the exposure assumptions, the PHAST was used to calculate HQs for intermediate exposures for adults and children (6 to 21 years old); the HQs did not exceed one (and as such the data is not presented in this document). Therefore, exposures to these contaminants in surface water are not expected to cause non-cancer adverse health effects. The intermediate health guideline values for 1,1-dichloroethane, benzene, vinyl chloride, benzo(a) pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthere, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, arsenic and manganese are unavailable.

Based on the EPCs of cis-1,2-dichloroethene, PCE, 1,4-dioxane, arsenic and copper detected in the surface water and the exposure assumptions, the PHAST was used to calculate HQs for acute exposures for adults and children (6 to 21 years old); the HQs did not exceed one (and as such the data is not presented in this document). Therefore, exposures to these contaminants in surface water are not expected to cause non-cancer adverse health effects. The intermediate health guideline values for 1,1-dichloroethane, benzene, TCE, vinyl chloride, benzo(a) pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthere, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, aluminum, cadmium, cobalt, manganese a vanadium are unavailable.

Incidental Ingestion & Dermal Absorption of Contaminants in Sediment at the GSR

As mentioned earlier incidental ingestion and dermal contact with sediment while hiking in the Grant Sprute Run and adjacent areas (see Photographs in Appendix A) by adults and children have been the exposure scenario. The site-specific exposure parameters and exposure factors used (by PHAST) to calculate the ingestion and dermal exposure doses are given in Table B9, Appendix B.

The combined (i.e., ingestion and dermal) chronic exposure doses are calculated using PHAST. Based on the EPCs of benzene, cis-1,2-dichloroethene, vinyl chloride, benzo[a]pyrene, hexachlorobenzene, pentachlorophenol and arsenic detected in the sediment, the combined

chronic HQs (i.e., ingestion and dermal) calculated for adults and children (6-21 years old) did not exceed one (and, as such, the data is not presented in this document). Therefore, exposures to these contaminants are unlikely to cause non-cancer adverse health effects. The chronic health guideline values for benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo[k]fluoranthene, bis(2-chloroethyl) ether, indeno(1,2,3-cd)pyrene, and N-nitrosodi-N-propylamine, cobalt and manganese are unavailable. Based on the EPCs of PCE and TCE detected in the sediment the combined chronic HQs calculated for children exceeded 1 (see Table B10a and 10b, Appendix B).

The combined (i.e., ingestion and dermal) intermediate exposure doses are calculated using PHAST. Based on the EPCs of cis-1,2-dichloroethene and hexachlorobenzene detected in the sediment, the combined intermediate HQs (i.e., ingestion and dermal) calculated for adults and children (6-21 years old) did not exceed one (and, as such, the data is not presented in this document). Therefore, exposures to these contaminants are unlikely to cause non-cancer adverse health effects. The intermediate health guideline values for benzene, vinyl chloride, benzo(a)anthracene, benzo[a]pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo[k]fluoranthene, bis(2-chloroethyl) ether, indeno(1,2,3-cd)pyrene, and N-nitrosodi-N-propylamine, pentachlorophenol, arsenic, cobalt and manganese are unavailable. Based on the EPCs of PCE and TCE detected in the sediment the combined intermediate HQs calculated for children exceeded 1 (see Table B11a and 11b, Appendix B).

The combined (i.e., ingestion and dermal) acute exposure doses are calculated using PHAST. Based on the EPCs of cis-1,2-dichloroethene, TCE, hexachlorobenzene, arsenic and cobalt detected in the sediment, the combined acute HQs (i.e., ingestion and dermal) calculated for adults and children (6-21 years old) did not exceed one (and, as such, the data is not presented in this document). Therefore, exposures to these contaminants are unlikely to cause non-cancer adverse health effects. The acute health guideline values for benzene, vinyl chloride, benzo(a)anthracene, benzo[a]pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo[k]fluoranthene, bis(2-chloroethyl) ether, indeno(1,2,3-cd)pyrene, and N-nitrosodi-N-propylamine, pentachlorophenol and manganese are unavailable. Based on the EPCs of PCE detected in the sediment the combined acute HQs calculated for children exceeded 1 (see Table B12a, Appendix B).

The non-cancer adverse health effects associated with PCE and TCE are evaluated as follows:

PCE: is a manufactured chemical that is widely used for dry cleaning of fabrics and for metal-degreasing. Exposures to high concentrations of PCE can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death (ATSDR 2019). These symptoms occur almost entirely in work (or hobby) environments when people have been exposed to high concentrations. The health effects of breathing in air or drinking water with low levels of PCE are not known. Results of animal studies show that PCE can cause liver and kidney damage. PCE has been shown to cause liver tumors in mice and kidney tumors in male rats.

Based on the EPC of PCE, the combined (ingestion and dermal) chronic, intermediate and acute HQ for the RME and CTE scenarios were calculated (see Table B10a, Table 11a and Table 12a, Appendix B). A review of the HQs shows that the child age group "6 to <11 years" with the RME scenario has the highest HQs; the HQs range from 1.0 to 12 for intermediate and acute RME scenarios, respectively. It should also be noted that the chronic, intermediate, and acute MRL have the same value (i.e., 0.008 mg/kg/day) and they have been derived from the same exposure study (ATSDR 2019). The LOAEL and the uncertainty/modifying factor associated with the chronic, intermediate or acute MRL are 2.3 mg/kg/day and 300, respectively.

The acute exposure dose for the RME scenario for the age group "6 to <11 years" (i.e., 0.098 mg/kg/day) is about 23 times (2.3/0.098 = 23) lower than the LOAEL for PCE (the LOAEL is 2.3 mg/kg/day). As such, non-cancer adverse health effects from acute exposure to PCE in the sediment for the child age group "6 to <11 years" are not likely. Since the acute, intermediate, and chronic HQs for other age group children and adult are less than 13 (the HQ associated with the acute RME scenario for child age group “6 to <11 years), non-cancer adverse health effects from exposure to PCE in the sediment are also unlikely.

TCE: is used mainly as a solvent to remove grease from metal parts. Drinking large amounts of TCE may cause nausea, liver damage, unconsciousness, impaired heart function, or death. Drinking small amounts of TCE for long periods may cause liver and kidney damage, impaired immune system function, and impaired fetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with TCE for short periods may cause skin rashes. TCE is characterized as “carcinogenic to humans” by all routes of exposure. The human evidence of carcinogenicity from epidemiologic studies of TCE exposure is strong for non-Hodgkin lymphoma (NHL), but less convincing than for kidney cancer, and more limited for liver and biliary tract cancer.

It should be noted that the chronic and intermediate MRL of TCE have the same value (i.e., 0.0005 mg/kg/day) and they have been derived from three different exposure study (ATSDR 2019). The chronic oral MRL (i.e., 0.0005 mg/kg/day) uses (1) immunotoxicity in mice, (2) decreased thymus weight in female mice, and (3) fetal heart malformations in rats (ATSDR 2019) as the critical health effect. The health effect, LOAEL or Human equivalent dose (HED_{99}) UF and the resulting MRL associated with the health effect is given for each of the three health effects in Table B13, Appendix B. Since the calculated combined (ingestion and dermal) HQ exceedances are associated with younger receptors (i.e., 6 to < 11 years), decreased thymus weight has been selected as the critical health effect for this evaluation.

Based on the EPC of TCE, the combined (ingestion and dermal) chronic HQ for the RME and CTE scenarios were calculated (see Table B10b and Table 11b, Appendix B). The child age group "6 to <11 years" with the intermediate RME scenario has the highest HQ of 2.3. The intermediate exposure dose for the RME scenario for this age group (i.e., 0.0012 mg/kg/day) is about 40 times lower than the HED_{99} for TCE (i.e., 0.048 mg/kg/day). As such, non-cancer adverse health effects from exposure to TCE in the sediment for the child age group " 6 to <11 years" are not likely. Since the intermediate, and chronic HQs for other age group children and

adults are less than 2.5, non-cancer adverse health effects from exposure to TCE in the sediment of the Grant Sprute Run is unlikely for these age groups as well.

Cancer Health Effects

The site-specific lifetime excess cancer risk (LECR) indicates the cancer potential of contaminants. LECR estimates are excess cancer case estimates in an exposed population in addition to the background rate of cancer. For perspective, the lifetime risk of developing cancer in the United States is 42 per 100 individuals for males, and 38 per 100 for females. Approximately 40% of men and women will be diagnosed with cancer at some point during their lifetimes (NCI 2017, ACS 2017). Lifetime doses, used to evaluate cancerous effects, can differ depending on exposure duration and are averaged over a lifetime of exposure (i.e., 78 years). (ATSDR 2018b)

According to the United States Department of Health and Human Services (DHHS), the cancer class of contaminants detected at a site is as follows:

1 = Known human carcinogen
2 = Reasonably anticipated to be a carcinogen
3 = Not classified

Typically, health guideline comparison values developed for carcinogens are based on one excess cancer case per 1,000,000 individuals. The NJDOH considers estimated cancer risks of less or equal to one additional cancer case among one million persons exposed an unlikely increased cancer risk (expressed exponentially as 10^{-6}).

The site-specific exposure assumptions and recommended exposure factors used to calculate the LECR are the same as those used to assess non-cancer health effects. The LECRs for surface water and sediment exposures were calculated using the formula (ATSDR 2005): LECR = Cancer Exposure Dose x CSF.

Dermal Absorption of contaminants in Surface Water at the GSR tributary:

The COCs detected in the surface water of the Matlack site are given in Table B5, Appendix B. Benzene, PCE, TCE, vinyl chloride, 1,4-dioxane, PAHs, and arsenic were the carcinogens found in surface water. The LECR calculated for benzene, PCE, TCE, vinyl chloride, 1,4-dioxane, and arsenic were less than 10^{-6}; they are not retained for further investigation. The cancer risk associated with benzo[a]pyrene exceeded 10^{-6} (see Tables B14, Appendix B); benzo[a]pyrene is retained for further investigation. The DHHS has determined that some PAHs may reasonably expected to be carcinogens. As such, all PAHs detected in the surface water were included in the cancer risk assessment.

The California EPA's Office of Environmental Health Hazard Assessment (OEHHA) has developed a relative potency estimate approach for PAHs (OEHHA 2015). Using OEHHA approach, the cancer potency of carcinogenic PAHs can be estimated based on their relative

potency with reference to benzo[a]pyrene. The total benzo[a]pyrene equivalents was obtained by summing each of the individual benzo[a]pyrene equivalents. The USEPA also proposed that cancer risk for chemicals that act with a mutagenic mode of action (MOA) for carcinogenesis can be quantified using age-dependent adjustment factors (ADAFs). The ADAFs are factors by which cancer risk is multiplied to account for increased susceptibility to mutagenic compounds early in life – standard ADAFs are 10 (for ages below 2 years old), 3 (for ages 2 up to 16 years old), and 1 (for ages greater than 16) (ATSDR 2016a).

Polycyclic Aromatic Hydrocarbons (PAHs): Chronic exposure to mixtures of PAHs via inhalation and dermal contact have been found to cause cancer. In laboratory animal tests, some PAHs have caused cancer via inhalation (lung cancer), ingestion (stomach cancer), or dermal contact (skin cancer).

For each of the carcinogenic PAHs detected in the surface water, the Benzo[a]pyrene equivalents were calculated by multiplying the EPCs by the cancer potency factor (see Table B15, Appendix B). The cancer risk is calculated by multiplying Benzo[a]pyrene cancer risk by the ratio of Benzo[a]pyrene equivalent concentration to Benzo[a]pyrene concentration, or Child LECR = $1.1 \times 10^{-4} * (0.01155/0.007) = 1.81 \times 10^{-4}$ and Adult LECR = $1.5 \times 10^{-4} * (0.01155/0.007) = 2.47 \times 10^{-4}$. The LECRs for both child and adults are approximately two extra cancer cases for every 10,000 similarly exposed individuals[7]. This is considered to be an increased cancer risk.

Incidental Ingestion & Dermal Absorption of Contaminants in Sediment at the GSR tributary

The potential COCs detected in the sediment of the Matlack site are given in Table B4, Appendix B. Benzene, PCE, TCE, vinyl chloride, benzo[a]pyrene, bis(2-chloroethyl) ether, hexachlorobenzene, nitrosodi-n-propylamine, pentachlorophenol and arsenic were the carcinogens found in sediment. The combined (i.e., ingestion and dermal) LECR calculated for benzene, benzo[a]pyrene, bis(2-chloroethyl) ether, hexachlorobenzene, nitrosodi-n-propylamine, pentachlorophenol and arsenic were less than 10^{-6}; they are not retained for further investigation. Since the LECR associated with PCE, TCE and vinyl chloride exceeded 10^{-6} (see Tables B10a and B10b, Appendix B,); they are retained for further assessment.

PCE: The USEPA has recently classified PCE as "likely to be carcinogenic to humans" by all routes of exposure. Although exposure to PCE has not been directly shown to cause cancer in humans, the DHHS has determined that PCE may reasonably be anticipated to be a human carcinogen (DHHS 2016). IARC has classified PCE as a Group 2A carcinogen—probably carcinogenic to humans (limited human evidence, sufficient evidence in animals) (IARC 1995).

Using site-specific conditions, the calculated LECR (56 years for adult exposure) for PCE detected in the Matlack site for the CTE and RME scenarios were one and four extra cancer

[7]Note that this is a theoretical estimate of cancer risk that ATSDR uses as a tool for deciding whether public health actions are needed to protect health—it is not an actual estimate of cancer cases in a community.

cases for every 1,000,000 similarly exposed individuals, respectively (see Table B10a, Appendix B). This is considered to be a low cancer risk.

TCE: The USEPA characterizes TCE as carcinogenic to humans by all routes of exposure (USEPA 2012b). This conclusion is based on human epidemiology studies showing associations between human exposure to TCE and kidney cancer, non-Hodgkin's lymphoma, and liver cancer. The human studies showed increased rates of liver cancer and non-Hodgkin's lymphoma, primarily in workers who were exposed to TCE on the job. The National Toxicology Program (NTP) has determined that TCE is a "known human carcinogen". The animal studies showed increased numbers of liver, kidney, testicular, and lung tumors by two different routes of exposure (NTP 2011).

Using site-specific conditions, the calculated LECR (56 years for adult exposure) for TCE detected in the Matlack site for the CTE and RME scenarios was one and four extra cancer cases for every 1,000,000 similarly exposed individuals, respectively (see Table B10b, Appendix B). This is considered to be a low cancer risk.

Vinyl Chloride: The DHHS has determined that vinyl chloride is a known carcinogen. Studies in workers who have breathed vinyl chloride over many years showed an increased risk of liver, brain, lung cancer, and some cancers of the blood have also been observed in workers.

Using site-specific conditions, the calculated LECR (56 years for adult exposure) for vinyl chloride detected in the Matlack site for the RME scenario was one extra cancer case for every 1,000,000 similarly exposed individuals (see Table B16, Appendix B). This is considered to be a low cancer risk.

Combined Cancer Risk from Exposure to Sediment and Surface Water at the GSR tributary:

The quantitative cancer risk evaluations show that the PAHs in surface water are driving the cancer risk at two extra cancer cases for every 10,000 similarly exposed individuals while the cancer risks associated with sediment contaminants are less than four extra cancer cases for every 1,000,000 similarly exposed individuals.

Child Health Considerations

ATSDR's recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination in their environment. Children are at greater risk than adults from certain kinds of exposures to hazardous substances because they eat and breathe more than adults. They also play outdoors and often bring food into contaminated areas. Children are also smaller, resulting in higher doses of chemical exposure per body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care. An elementary school and daycare facility are located about 5,000 feet to the east of the site. The

groundwater plumes extend on-site and west of the site, which is a State Wildlife Management Area and any areas to the east of the site are not expected to be impacted by these plumes.

The NJDOH and ATSDR evaluated the potential risk for children accessing the area in the vicinity of the GSR who were exposed to VOCs, PAHs, and metals via the surface water and sediment pathways. The HQ was used to evaluate non-cancer adverse health effects; the HQs associated with dermal contact with contaminants in surface water were less than 1; as such, past exposures to these contaminants in surface water are unlikely to cause non-cancer adverse health effects in children (see Table B8, Appendix B).

For sediment ingestion and dermal contact with PCE, the combined (ingestion and dermal) acute HQ for age group “6 to <11 years” associated with the RME scenario was 13 (see Table B10, Appendix B). An analysis of non-cancer adverse health effects from acute exposure to PCE in the sediment for the child age group "6 to <11 years" was found to be not likely. Since the combined (ingestion and dermal) acute, intermediate, and chronic HQs for other age group children are less than 13, non-cancer adverse health effects from exposure to PCE in the sediment are also not likely. For sediment ingestion and dermal contact with TCE, the combined (ingestion and dermal) intermediate HQ for age group “6 to <11 years” associated with the RME scenario was 2.5 (see Table B10, Appendix B). An analysis of non-cancer adverse health effects from intermediate exposure to TCE in the sediment for the child age group "6 to <11 years" was found to be not likely. Since the combined (ingestion and dermal) intermediate and chronic HQs for other age group children are less than 2.5, non-cancer adverse health effects from exposure to TCE in the sediment are also not likely. The LECR associated with PAHs exposures in surface water for a child hiker was two extra cancer cases for every 10,000 similarly exposed individuals. This is considered to be an increased cancer risk.

Public Comment

The public comment period for this public health assessment was from August 12, 2022 to September 12, 2022. This document was shared with the current business operator, the NJDEP, the EPA, the Gloucester County Health Department, and the Gloucester County Library in Swedesboro. No comments were received.

Conclusions

The Matlack site operated as a truck terminal and tank-trailer cleaning facility between 1962 and 1976. Wastes generated during tank cleaning operations were disposed of at the site in an unlined lagoon resulting in the contamination of surface water, sediment, soil and groundwater. There are completed exposure pathways via incidental ingestion/dermal contact with surface water and sediment in the past, present and future. The exposed populations are adult and child hikers. Potential contaminants of concern are chlorinated VOCs and PAHs.

Currently, a truck sales and service center occupy the Matlack site. Based on a review of environmental data, the NJDOH and ATSDR reached the following conclusions:

The NJDOH and ATSDR conclude that the combined (ingestion and dermal) cancer risk from past and current exposures to site-related chlorinated volatile organic compounds and polycyclic aromatic hydrocarbons from multiple pathways at the Grand Sprute Run tributary may have harmed people's health. Based on the surface water data for polycyclic aromatic hydrocarbons, the cancer risk resulting from past and current exposures from wading in the Grand Sprute Run tributary may result in an increased theoretical cancer risk for those residents who frequently used the tributary for recreational purposes, such as hiking and playing. For exposures associated with ingesting sediment and dermal contact with sediment along the Grand Sprute Run tribuary, the cancer risk from frequent exposure to volatile organic compounds in the sediment represents a low cancer risk.

The NJDOH and ATSDR cannot conclude whether past exposures to site-related contaminants in the on-site production well may have harmed people's health. Based on the limited data available, site groundwater was contaminated with volatile organic compounds. In the past, the production well was the primary source of drinking water for the former Matlack employees. This is a past potential pathway because there are no data to evaluate the public health implications of the drinking water pathway. Currently, the well is out of service and the Liberty Kenworth employees are drinking bottled water.

The NJDOH and ATSDR conclude that if the soil and groundwater contamination are not fully remediated and the land use changes (e.g., industrial to residential) in the future, the contamination may be a source of potential exposure pathways. The groundwater plume continues to discharge contaminants to the Grand Sprute Run tributary. The soil and groundwater have been delineated but not yet remediated.

Recommendations

Based upon these conclusions, NJDOH and ATSDR recommend the preferred remedy as described in the record of decision for the site be implemented as soon as feasible by the USEPA. The major components of the remedy include installation of two permeable reactive barriers (PRBs) in the groundwater; excavation and off-site disposal of contaminated soil and sediment; long-term monitoring (to assess the effectiveness of the remedy over time); and institutional controls until remedial action objectives are met.

The NJDOH and ATSDR recommend that Liberty Kenworth employees continue the use of bottled water for drinking as a precautionary measure.

The contaminated area at the Grand Sprute Run is wooded and inaccessible by the area residents. As such, any interim remedial measures (such as, posting sign or fencing the area) to interrupt the pathway is not warranted. If land use changes, the NJDOH and ATSDR recommend an

assessment of all potential pathways. Examples include an analysis of the vapor intrusion pathway and/or an analysis of incidental ingestion of on-site soil for any future planned residential homes.

Public Health Action Plan

The purpose of a Public Health Action Plan is to ensure that this public health assessment not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent harmful human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the ATSDR and the NJDOH to follow-up on this plan to ensure that it is implemented. The public health action plan is as follows:

Public Health Actions Taken

1. A preliminary health assessment was completed in January 1989, which addressed the public health implications of site-related exposures to VOCs and polycyclic aromatics in on-site soils and groundwater (on-and-off-site) that included surrounding residential private wells.

2. The site was categorized as a potential public health concern because of contaminated groundwater at concentrations that may result in adverse health effects.

3. A site review and update were completed in August 1994, which evaluated the status of the Site and identified future ATSDR activities planned for the site.

Public Health Actions Underway and Planned

1. Copies of this public health assessment will be made available to concerned residents in the vicinity through township libraries and the internet.

2. The preferred remedy as indicated in the Record of Decisions (ROD) is being implemented by the USEPA.

3. NJDOH will continue to review data as it is made available. This includes new information related to investigations and remedial actions taken for areas of concern on- site as it is completed in the future. If land use changes, the NJDOH and ATSDR recommend an assessment of all potential pathways. Examples include an analysis of the vapor intrusion pathway and/or an analysis of incidental ingestion of on-site soil for any future planned residential homes.

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Report Preparation

The New Jersey Department of Health (NJDOH) prepared this Public Health Assessment for the Matlack site located in Woolwich Township, Gloucester County, New Jersey. This publication was made possible by a cooperative agreement [program #TS20-2001] with the federal Agency for Toxic Substances and Disease Registry (ATSDR). The NJDOH evaluated data of known quality using approved methods, policies, and procedures existing at the date of publication. ATSDR reviewed this document and concurs with its findings based on the information presented by the NJDOH.

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FIGURES

Figure 2: Matlack site and property boundary

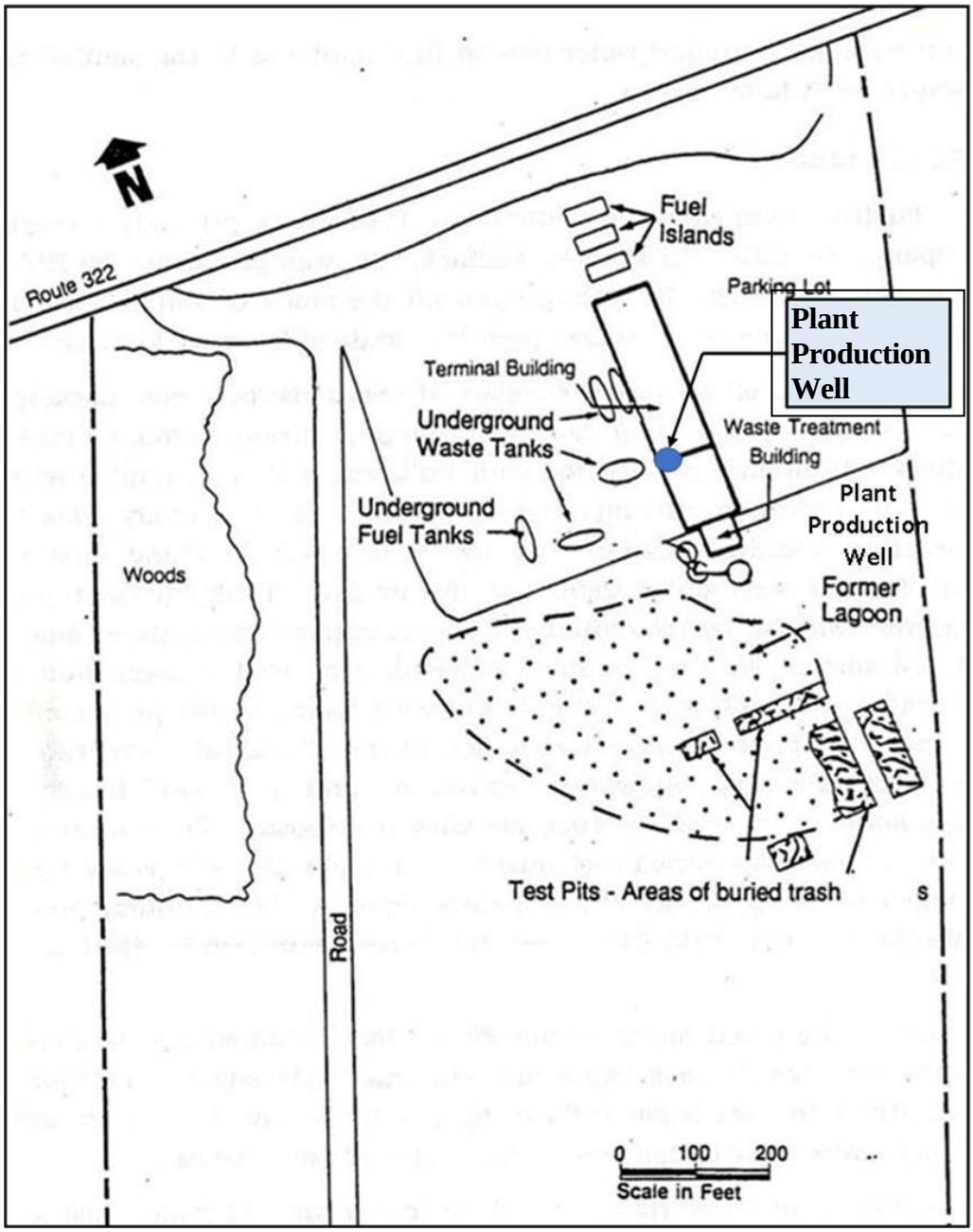

Figure 3: Matlack Historic View 1990

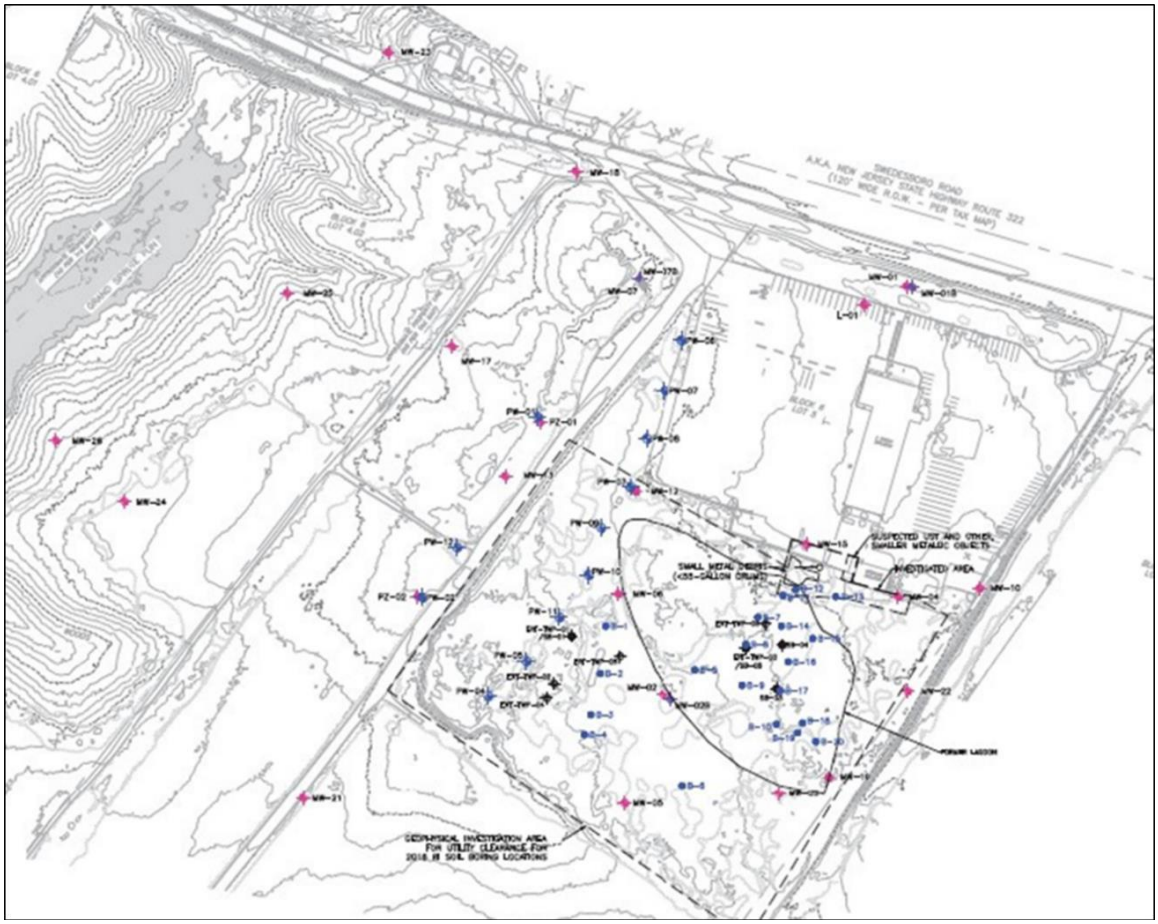

Figure 4: On and Off-site Soil Sampling Locations

Figure 5: Location of Groundwater Plumes

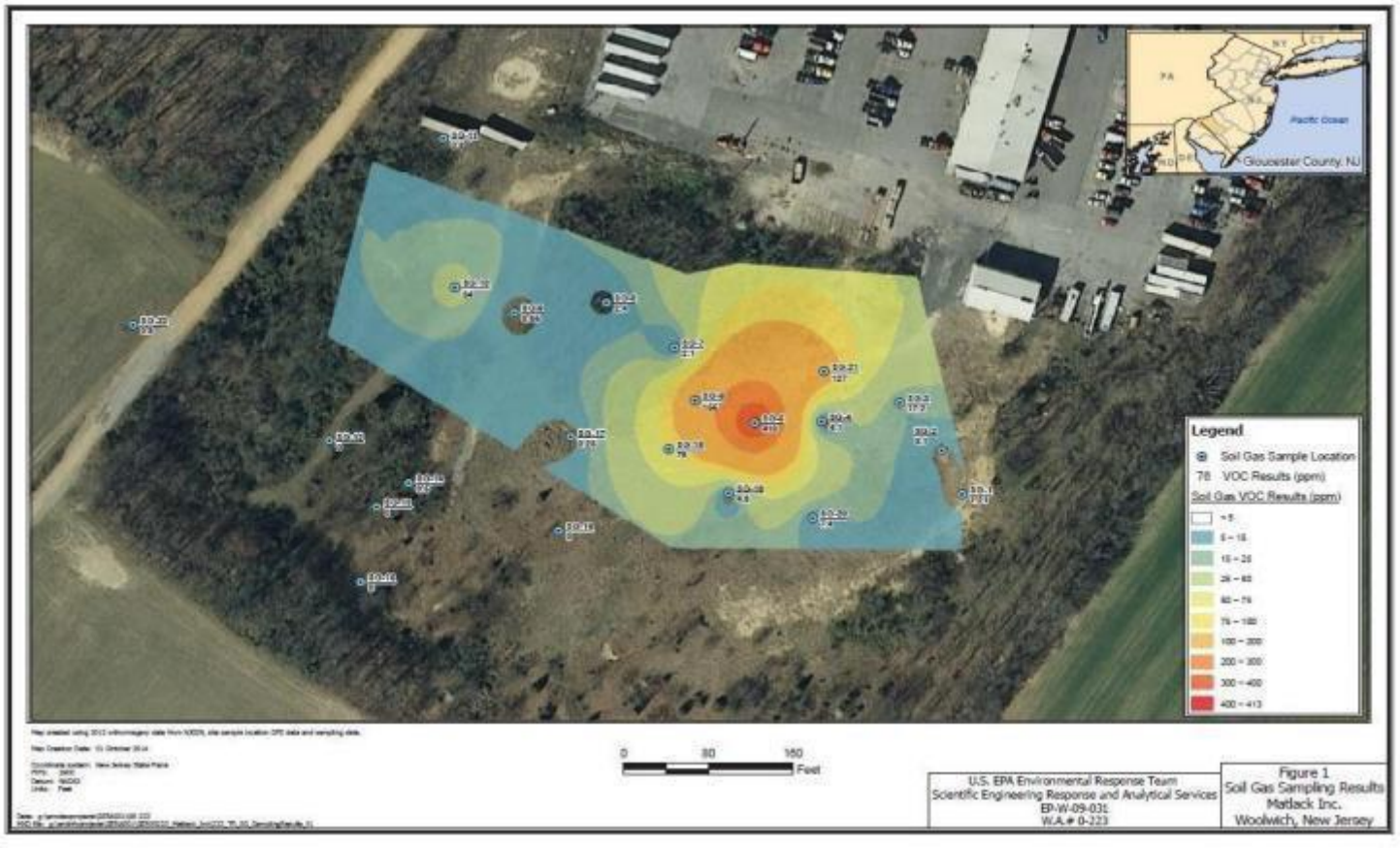

Figure 6: Soil Gas Sampling 2014

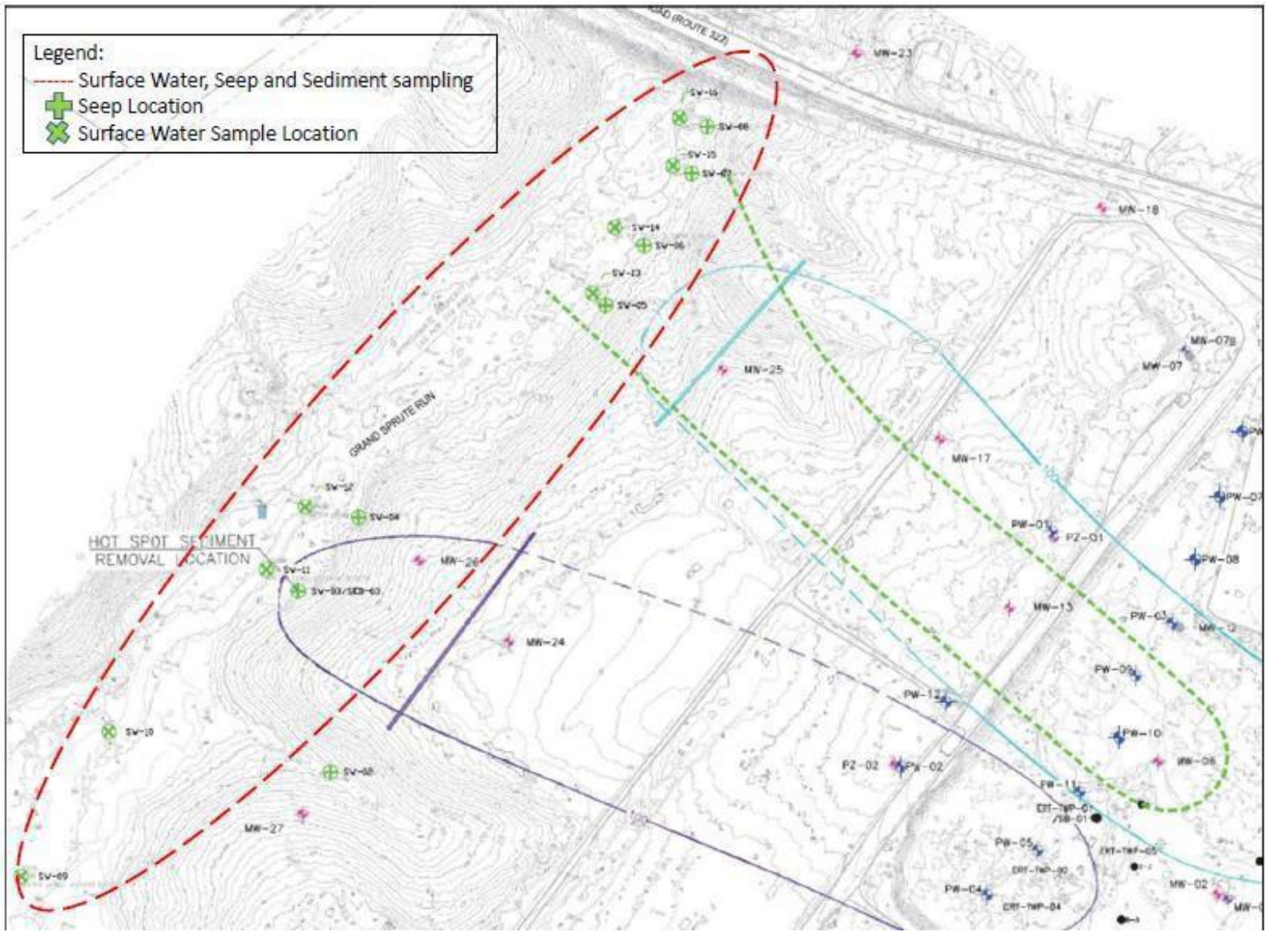

Figure 7: On and Off-site Sampling Locations

Appendix A - Photographs

[1]

[1]The photographs were taken by the NJDOH staff except the last one, which is from Google Map (readily available from internet).

Appendix A

Photographs

Main building and parking lot

Area behind site, scrub brush and field

Site view, including Route #322

Access Road between Main building and Grand Sprute Run

Soybean Field, east of Grand Sprute Run

Brush and trees outside of Grand Sprute Run

Grand Sprute Run east of Racoon Creek

Appendix B – Additional Tables

Table B1: Summary of Contaminants of Potential Concern

Media	Location	Metals	VOCs, SVOCs, PAHs
On-Site Soil (0-2')	Former Unlined Lagoon	Arsenic, Thallium	Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, bis(2-Chloroethyl) Ether, bis(2-Ethylhexyl) Phthalate, Hexachlorobenzene, N-Nitrosodi-N-Propylamine, Aroclor 1248, and Aldrin
On-Site Soil (4-35')	General Site Property (including Lagoon)	Arsenic, Thallium, Vanadium	TCE, Vinyl Chloride, bis(2-Chloroethyl) Ether, bis(2-Ethylhexyl) Phthalate, 2-Methylnaphthalene, N-Nitroso-di-N-Propylamine, Aroclor 1016, Aroclor 1242, Aroclor 1248, Aroclor 1254, Aroclor 1260
On and off-Site Ground Water	Site Property	Arsenic, Barium, Cadmium, Manganese	1,1,2,2-Tetrachloroethane, 1,1,2-Trichloroethane, 1,1-Dichloroethane, 1,2-Dibromo-3-Chloropropane, 1,2-Dibromoethane, 1,2-Dichloroethane, Benzene, Carbon Tetrachloride, cis-1,2-Dichloroethene, cis-1,3-Dichloropropene, Dibromochloromethane, Dichlorodifluoromethane, PCE, trans-1,3-Dichloropropene, TCE, Vinyl Chloride, Xylenes, 1,2,4,5-Tetachlorobenzene, 1,4-Dioxane, 2,4,6-Trichlorophenol, 4-Nitroaniline, 3,3'-Dichlorobenzidine, 4-Chloroaniline, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Biphenyl, bis(2-Chloroethyl) Ether, Chrysene, Dibenz[a,h]anthracene, Hexachlorobutadiene, Hexachlorocyclopentadiene, Hexachloroethane, Indeno[1,2,3-cd]pyrene, N-Nitrosodiphenylamine, Pentachlorophenol, Naphthalene, , Aroclor-1016, Aroclor-1221, Aroclor-1232, Aroclor-1242, Aroclor-1248, Aroclor-1254, Aroclor-1260, Aroclor-1268, Chlorobiphenyl
Off-Site Surface Water	Grand Sprute Run Tributary	Aluminum, Arsenic, Cadmium, Cobalt, Copper, Lead, Manganese, Vanadium	1,1-Dichloroethane, cis-1,2-Dichloroethene, Benzene PCE, TCE, Vinyl Chloride, 1,4-Dioxane, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene
Off-Site Sediment		Arsenic, Cobalt, Manganese	Benzene, cis-1,2-Dichloroethene, PCE, TCE, Vinyl Chloride, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, bis(2-Chloroethyl) Ether, Dibenzofuran, Dibenz(a,h)anthracene, Hexachlorobenzene, Indeno[1,2,3-cd]pyrene, N-Nitroso-di-N-Propylamine, Phenanthrene, Pentachlorophenol, 1,1,2,2-Tetrachloroethane

Table B2: On-Site Production Well Sample Results of Matlack, 2008-2011

Sample Date	Contaminant	Concentration (µg/L[a])	EPA Comparison Value[b] (µg/L)	Maximum Exceeds Comparison Value
September 2008	1,4-Dichlorobenzene	0.74	75	No
June 2009	Acetone	1.66	NA[c]	No
June 2009	2-Butanone	1.83	NA	No
June 2010	Dichloromethane	2.61	5	No

a=micrograms/Liter; b=USEPA Maximum Contaminant Level; c= Not available; d=ATSDR Environmental Media Evaluation Guide-child; e=ATSDR Reference Media Evaluation Guide-child; f=ATSDR Cancer Risk Evaluation Guide for chronic exposure.

Table B3: Off-site Surface Water Sampling Results of Matlack site, 2012 - 2016

Contaminant	No. of Samples	No. of NDs[f]	Concentration (micrograms/liter) Minimum	Concentration (micrograms/liter) Maximum	Comparison Value (micrograms/liter)	Maximum Exceeds Comparison Value
1,1,1-Trichloroethane	17	12	0.50	120	14,000 (RMEG[a])	No
1,1-Dichloroethane	17	11	0.19	10	2.7 (RSL[c])	**Yes**
1,1-Dichloroethene	17	13	0.46	14	63 (EMEG[d])	No
1,2-Dichlorobenzene	17	16	0.35	0.35	630 (RMEG)	No
1,2-Dichloroethene (cis)	25	10	0.11	61	14 (RMEG)	**Yes**
1,3-Dichlorobenzene	17	16	1.10	1.10	140 (EMEG)	No
1,4-Dichlorobenzene	17	16	1.50	1.50	490 (EMEG)	No
Acetone	17	12	5.00	25	6,300 (RMEG)	No
Benzene	17	16	1.80	1.80	0.44 (CREG)	**Yes**
Bromomethane	17	16	0.50	0.50	9.8 (EMEG)	No
Carbon Disulfide	17	16	0.28	0.28	700 (RMEG)	No
Carbon Tetrachloride	17	17	0.00	0.00	0.35 (CREG)	No
Chlorobenzene	17	15	0.26	4.70	140 (RMEG)	No
Chloroethane	17	16	0.46	0.46	21,000 (RSL)	No
Ethylbenzene	17	16	0.91	0.91	700 (RMEG)	No
Isopropylbenzene	17	16	0.97	0.97	700 (RMEG)	No
Tetrachloroethene	25	15	0.30	490	12 (CREG)	**Yes**
Trichloroethene	25	15	0.15	62	0.43 (CREG)	**Yes**
Vinyl Chloride	17	8	0.01	0.33	0.017 (CREG)	**Yes**
1,4-Dioxane	17	6	0.18	0.45	0.24 (CREG)	**Yes**
4-Chloroaniline	17	16	27	27	28 (RMEG)	**No**
Benzo[a]pyrene	17	15	5.40	7	0.012 (CREG)	**Yes**
Benzo[b]fluoranthene	17	15	5.40	7	0.034 (RSL)	**Yes**
Benzo[g,h,i]perylene	17	15	5.40	7	0.012 (CREG	**Yes**
Benzo[k]fluoranthene	17	15	5.40	7	0.34 (RSL)	**Yes**
Dibenz[a,h]anthracene	17	15	5.40	7	0.0001 (RSL)	**Yes**
Hexachlorocyclopentadiene	17	15	11	11	42 (RMEG)	No
Indeno]1,2,3-cd]pyrene	17	15	5.40	7	0.034 (RSL)	**Yes**
Aluminum[**]	17	0	22.5	32,600	7,000 (EMEG)	**Yes**
Antimony	17	16	2.2	2.2	2.8 (RMEG)	**No**
Arsenic[*]	17	1	0.31	35.8	0.016 (CREG)	**Yes**
Barium	17	0	28.2	533	1,400 (EMEG)	No

Beryllium	17	2	0.06	11.6	14 (EMEG)	No
Cadmium**	17	0	0.05	10.2	0.70 (EMEG)	**Yes**
Chromium, Total	17	2	0.22	40.3	100 (MCL)	No
Cobalt	17	1	0.36	91.2	70 (EMEG-I)	**Yes**
Copper	17	2	0.18	88.5	70 (EMEG-I)	**Yes**
Lead	17	1	0.19	471	15 (AL[h])	**Yes**
Manganese	17	0	34.7	7,470	350 (RMEG)	**Yes**
Mercury	17	14	0.07	1.2	2 (MCL)	No
Nickel	17	5	1.1	87.7	140 (RMEG)	No
Selenium	17	6	0.48	28	35 (EMEG)	No
Silver	17	13	0.01	0.84	35 (RMEG)	No
Thallium	17	16	0.71	0.71	2 (MCL)	No
Vanadium	17	4	0.33	106	70 (EMEG-I)	**Yes**
Zinc	17	1	1.8	284	2,100 (RMEG)	No

a=Not Detected; b=ATSDR Rea=Not Detected; b=ATSDR Reference Media Evaluation Guide for chronic exposure; c=USEPA Regional Screening Level for chronic exposure; d= ATSDR Environmental Media Evaluation Guide for chronic exposure; e=ATSDR Cancer Risk Evaluation Guide for chronic exposure; f= Regional Screening Levels; g=EPA Maximum Concentration Level; h= ATSDR Environmental Media Evaluation Guide for intermediate exposure; i= NJDEP Lead Action Level.*=Recommended Acute ATSDR met or exceeded; **= Recommended Intermediate ATSDR met or exceeded; ***= Recommended Acute and Intermediate ATSDR met or exceeded

Table B4: Off-site sediment sampling results of Matlack site (2005 – 2016)

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
1,1,1-Trichloroethane	17	11	0.03	5,700	11,000 (RMEG[b])	No
1,1,2,2-Tetrachloroethane	9	2	0.03	7	1.9 (CREG[c])	**Yes**
1,1,2-Trichloro-1,2,2-Trifluoroethane	9	2	0.03	7	1,7000,000 (RMEG)	No
1,1,2-Trichloroethane	9	2	0.03	7	12 (NJDEP SRS)	No
1,1-Dichloroethane	17	6	0.01	11	120 (NJDEP SRS)	No
1,1-Dichloroethene	9	7	0.03	0.03	510 (EMEG[d])	No
1,2,3-Trichlorobenzene	9	1	0.01	0.03	63 (RSL)	No
1,2,4-Trichlorobenzene	9	1	0.01	0.03	570 (RMEG)	No
1,2-Dibromo-3-Chloropropane	9	2	0.01	0.03	110 (EMEG)	No
1,2-Dibromoethane	9	7	0.03	0.03	0.19 (CREG)	No
1,2-Dichlorobenzene	17	7	0.01	3	510 (EMEG)	No
1,2-Dichloroethane	9	7	0.03	0.03	4.1 (CREG)	No
1,2-Dichloropropane	9	7	0.03	0.03	5,100 (EMEG)	No
1,3-Dichlorobenzene	17	8	0.01	3	1,100 (EMEG)	No
1,4-Dichlorobenzene	17	7	0.01	6	4,000 (EMEG)	No
2-Butanone	17	7	0.02	33	34,000 (RMEG)	No
2-Hexanone	9	6	0.02	0.06	290 (RMEG)	No
4-Methyl-2-Pentanone	9	6	0.02	0.06	6,300 (RSL)	No
Acetone	16	3	0.12	99	51,000 (RMEG)	No
Benzene	17	12	0.03	39	6.8 (CREG)	**Yes**
Bromodichloromethane	9	7	0.03	0.03	6.0 (CREG)	No
Bromoform	9	2	0.01	0.03	47 (CREG)	No
Bromomethane	9	7	0.03	0.03	80 (RMEG)	No
Carbon Tetrachloride	9	7	0.03	0.03	5.3 (CREG)	No
Chlorobenzene	17	7	0.01	37	1,100 (RMEG)	No
Chloroethane	17	14	0.03	5	14,000 (RSL)	No
Chloroform	9	7	0.03	0.03	570 (EMEG)	No

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
Chloromethane	9	7	0.03	0.03	270 (NJDEP SRS)	No
cis-1,2-Dichloroethene	23	14	0.00	4,400	110 (RMEG)	**Yes**
cis-1,3-Dichloropropene	9	7	0.03	0.03	3.7 (CREG)	No
Cyclohexane	9	7	0.03	0.03	6,500 (RSL)	No
Dibromochloromethane	9	7	0.03	0.03	4.5 (CREG)	No
Dichlorodifluoromethane	9	7	0.03	0.03	11,000 (RMEG)	No
Ethylbenzene	17	13	0.03	120	5,700 (RMEG)	No
Cumene (Isopropylbenzene)	17	12	0.00	57	5,700 (RMEG)	No
Methyl Acetate	17	12	0.03	3,800	78,000 (RSL)	No
Methyl tert-Butyl Ether	9	7	0.03	0.03	780 (NJDEP SRS)	No
Methylcyclohexane	9	7	0.01	0.03	NA[h,i]	No
Methylene Chloride	9	7	0.03	0.03	60 (CREG)	No
Nitrobenzene	9	7	0.03	0.03	110 (RMEG)	No
Styrene	9	7	0.03	0.03	11,000 (RMEG)	No
Tetrachloroethene[***]	23	9	0.01	25,000	180 (CREG)	**Yes**
Toluene	9	7	0.03	0.03	4,600 (RMEG)	No
trans-1,2-Dichloroethene	17	13	0.03	4	1,100 (RMEG)	No
trans-1,3-Dichloropropene	9	7	0.03	0.03	3.7 (CREG)	No
Trichloroethene[**]	35	11	0.01	2,100	5.6 (CREG)	**Yes**
Trichlorofluoromethane	9	7	0.03	0.03	17,000 (RMEG)	No
Vinyl Chloride	17	14	0.03	8	0.27 (CREG)	**Yes**
Xylenes	9	7	0.03	0.03	11,000 (EMEG)	No
1,2,4,5-Tetachlorobenzene	9	7	0.59	0.63	17 (RMEG)	No
1,2-Dichlorobenzene	9	0	0.07	0.26	5,100 (RMEG)	No
1,3-Dichlorobenzene	9	7	0.59	0.63	1,100 (EMEG)	No
1,4-Dichlorobenzene	9	7	0.59	0.63	4,000 (EMEG)	No
1,4-Dioxane	9	7	0.59	0.63	3.7 (CREG)	No
2,3,4,6-Tetrachlorophenol	9	7	0.59	0.63	1,700 (RMEG)	No

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
2,4,5-Trichlorophenol	9	7	0.59	0.63	5,700 (RMEG)	No
2,4,6-Trichlorophenol	9	7	1.1	1.2	34 (CREG)	No
2,4-Dichlorophenol	9	7	0.59	0.63	170 (RMEG)	No
2,4-Dimethylphenol	9	7	0.59	0.63	1,100 (RMEG)	No
2,4-Dinitrophenol	9	7	0.59	0.63	110 (RMEG)	No
2,4-Dinitrotoluene	9	7	0.59	0.63	57 (EMEG)	No
2,6-Dinitrotoluene	17	10	0.04	0.63	230 ((EMEG)	No
2-Chloronaphthalene	9	7	0.59	0.63	4,600 (RMEG)	No
2-Chlorophenol	9	0	0.07	0.26	290 (RMEG)	No
2-Methylnaphthalene	9	7	0.59	0.63	230 (RMEG)	No
2-Methylphenol	9	7	1.1	1.2	2,900 (RMEG)	No
2-Nitroaniline	9	7	0.59	0.63	780 (RSL)	No
2-Nitrophenol	9	7	1.1	1.2	35 (RSL)	No
3,3'-Dichlorobenzidine	9	7	1.1	1.2	1.2 (NJDEP SRS)	No
3-Nitroaniline	9	7	1.1	1.2	NA	No
4,6-Dinitro-2-Methylphenol	9	7	0.59	0.63	7.8 (RSL)	No
4-Bromophenyl Phenyl Ether	9	7	0.59	0.63	NA	No
4-Chloro-3-Methylphenol	9	7	1.1	1.2	7,800 (RSL)	No
4-Chloroaniline	9	7	0.59	0.63	3.5 (RSL)	No
4-Chlorophenyl Phenyl Ether	9	7	1.1	1.2	NA	No
4-Methylphenol (P-Cresol)	9	7	1.1	1.2	3,200 (RSL)	No
4-Nitroaniline	9	7	1.1	1.2	35 (RSL)	No
4-Nitrophenol	9	10	0.03	0.63	NA	No
Acenaphthene	17	7	1.1	1.2	3,400 (RMEG)	No
Acenaphthylene	17	10	0.07	0.63	3,400 (RMEG)	No
Acetophenone	17	14	0.03	1.2	5,700 (RMEG)	No
Anthracene	17	9	0.04	0.63	17,000 (RMEG)	No
Atrazine	9	7	1.1	1.2	2,000 (RMEG)	No

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
Benzaldehyde	12	9	0.07	1.2	5,700 (RMEG)	No
Benzo[a]anthracene	17	7	0.02	1.3	1.5 (RSL)	No
Benzo[a]pyrene	17	8	0.07	1.1	0.065 (CREG)	**Yes**
Benzo[b]fluoranthene	17	6	0.03	0.92	1.5 (RSL)	No
Benzo[g,h,i]perylene	17	9	0.10	0.63	0.15 (RSL)	**Yes**
Benzo[k]fluoranthene	17	8	0.07	1.4	15 (RSL)	No
Biphenyl	17	13	0.03	0.63	47 (CREG)	No
bis(2-Chloroethoxy) Methane	9	7	0.59	0.63	230 (RSL)	No
bis(2-Chloroethyl) Ether	9	7	1.1	1.2	0.34 (CREG)	**Yes**
bis(2-Chloroisopopyl) Ether	9	7	1.1	1.2	800 (RSL)	No
bis(2-Ethylhexyl) Phthalate	17	12	0.09	1.2	27 (CREG)	No
Benzyl Butyl Phthalate	9	3	0.05	6	290 (RSL)	No
Caprolactam	9	7	1.1	1.2	29,000 (RMEG)	No
Carbazole	17	10	0.05	1.2	NA	No
Chrysene	17	7	0.03	1.6	150 (RSL)	No
Dibenz(a,h)anthracene	17	10	0.07	0.63	0.15 (RSL)	**Yes**
Dibenzofuran	17	12	0.05	0.63	NA	No
Diethyl Phthalate	9	7	0.59	0.63	46,000 (RMEG)	No
Dimethyl Phthalate	9	7	0.59	0.63	5,700 (RMEG)	No
Di-N-Butyl Phthalate	17	13	0.03	0.63	5,700 (RMEG)	No
Di-N-Octyl Phthalate	17	14	0.14	1.2	23,000 (EMEG)	No
Fluoranthene	17	6	0.03	4.1	2,300 (RMEG)	No
Fluorene	17	10	0.1	0.63	2,300 (RMEG)	No
Hexachlorobenzene	9	7	0.59	0.63	0.23 (CREG)	**Yes**
Hexachlorobutadiene	9	7	0.59	0.63	4.8 (CREG)	No
Hexachlorocyclopentadiene	9	0	0.32	1.2	340 (RMEG)	No
Hexachloroethane	9	7	0.59	0.63	9.4 (CREG)	No
Indeno(1,2,3-c,d)pyrene	17	8	0.03	0.63	1.5 (RSL)	No

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
Isophorone	9	7	0.59	0.63	390 (CREG)	No
Naphthalene	16	8	0.04	0.63	1,100 (RMEG)	No
Nitrobenzene	9	7	0.59	0.63	110 (RMEG)	No
N-Nitrosodi-N-Propylamine	9	7	0.59	0.63	0.053 (CREG)	**Yes**
N-Nitrosodiphenylamine	9	7	0.59	0.63	140 (RSL)	No
Pentachlorophenol	9	7	1.1	1.2	0.94 (CREG)	**Yes**
Phenanthrene	17	8	0.09	2.5	NA	No
Phenol	9	7	1.1	1.2	17,000 (RMEG)	No
Pyrene	17	6	0.03	3.8	1,700 (RMEG)	No
Aluminum	9	0	1,180	18,100	57,000 (EMEG)	No
Antimony	9	3	1.7	3.2	23 (RMEG)	No
Arsenic	9	0	1.3	14.2	0.25 (CREG)	**Yes**
Barium	9	0	12	177	11,000 (EMEG)	No
Beryllium	9	0	0.18	0.69	110 (EMEG)	No
Cadmium	9	0	0.06	0.87	5.7 (EMEG)	No
Chromium	9	0	1.3	28.7	51 (EMEG)	No
Cobalt	9	0	0.89	57.2	23 (RSL)	**Yes**
Copper	9	0	2.1	13.3	570 (EMEG	No
Lead	9	0	13.5	71.3	400 (RSL)	No
Manganese	9	0	19.9	7,480	2,900 (RMEG)	**Yes**
Mercury	9	0	0.04	0.64	23 (NJDEP SRS)	No
Nickel	9	0	1.2	6.7	1,100 (RMEG)	No
Selenium	9	0	0.40	4.7	290 (EMEG)	No
Silver	9	0	0.01	0.07	290 (RMEG)	No
Thallium	9	3	0.85	1.6	1.6 (RSL)[xx]	No
Vanadium	9	0	2.9	15.5	570 (EMEG)	No
Zinc	9	0	2.4	59.3	17,000 (EMEG)	No
Aroclor 1016	9	6	0.07	0.12	0.19 (CREG)	No

Contaminant	No. of Samples	No. of NDs[a]	Concentration (mg/kg) Minimum	Concentration (mg/kg) Maximum	Comparison Value (mg/kg)	Maximum Exceeds Comparison Value
Aroclor 1221	9	7	0.11	0.12	0.19 (CREG)	No
Aroclor 1232	9	7	0.11	0.12	0.19 (CREG)	No
Aroclor 1242	9	7	0.11	0.12	0.19 (CREG)	No
Aroclor 1248	9	6	0.01	0.12	0.19 (CREG)	No
Aroclor 1254	9	7	0.11	0.12	0.19 (CREG)	No
Aroclor 1260	9	0	0.00	0.04	0.19 (CREG)	No
Aroclor 1262	9	7	0.11	0.12	0.19 (CREG)	No
Aroclor 1268	9	7	0.11	0.12	0.19 (CREG)	No
Chlorobiphenyl	9	6	0.07	0.12	0.19 (CREG)	No

a=Not Detected; b=ATSDR Reference Media Evaluation Guide for chronic exposure; c=ATSDR Cancer Risk Evaluation Guide for chronic exposure; d=USEPA Regional Screening Level; e=ATSDR Environmental Media Evaluation Guide for chronic exposure; f=NJDEP Residential Direct Contact Soil Cleanup Criteria; g=NJDEP Non- Residential Direct Contact Soil Cleanup Criteria; h=Not Available; i=New Jersey Department of Protection.*=Recommended Acute ATSDR met or exceeded; **= Recommended Intermediate ATSDR met or exceeded; ***= Recommended Acute and Intermediate ATSDR met or exceeded

Table B5: Exposure Point Concentration of off-site surface water contaminants

Contaminant	EPC Type	EPC*
1,1-dichloroethane	95% UCL of the mean	0.024 mg/L
1,2-dichloroethene, cis-	95% UCL of the mean	0.023 mg/L
Benzene	Maximum	0.0018 mg/L
Tetrachloroethylene	Maximum	0.43 mg/L
Trichloroethylene	Maximum	0.052 mg/L
Vinyl chloride	95% UCL of the mean	0.00019 mg/L
1,4-dioxane	95% UCL of the mean	0.00045 mg/L
Benzo(a)pyrene	Maximum	0.007 mg/L
Benzo(b)fluoranthene	Maximum	0.007 mg/L
Benzo(ghi)perylene	Maximum	0.007 mg/L
Benzo(k)fluoranthene	Maximum	0.007 mg/L
Dibenz(a,h)anthracene	Maximum	0.007 mg/L
Indeno(1,2,3-cd)pyrene	Maximum	0.007 mg/L
Aluminum	95% UCL of the mean	25 mg/L
Arsenic	95% UCL of the mean	0.013 mg/L
Cadmium	95% UCL of the mean	0.0035 mg/L
Cobalt	95% UCL of the mean	0.033 mg/L
Copper	95% UCL of the mean	0.067 mg/L
Manganese	Arithmetic mean	2.3 mg/L
Vanadium	Arithmetic mean	0.0033 mg/L

Abbreviations: EPC = exposure point concentration; mg/cm²/event = milligrams per centimeter squared per event; mg/L = milligram chemical per liter water; UCL = upper confidence limit, *Contaminant concentration converted to standard unit for calculating exposure.

Table B6: Exposure Point Concentration of off-site sediment contaminants

Contaminant	EPC Type	EPC*
Benzene	95% UCL of the mean	26 mg/kg
1,2-dichloroethene, cis-	95% UCL of the mean	1,400 mg/kg
Tetrachloroethylene	95% UCL of the mean	15,000 mg/kg
Trichloroethylene	95% UCL of the mean	630 mg/kg
Vinyl chloride	95% UCL of the mean	10 mg/kg
Benz(a)anthracene	95% UCL of the mean	0.81 mg/kg
Benzo(a)pyrene	95% UCL of the mean	0.79 mg/kg
Benzo(b)fluoranthene	95% UCL of the mean	0.65 mg/kg
Benzo(ghi)perylene	95% UCL of the mean	0.49 mg/kg
Benzo(k)fluoranthene	95% UCL of the mean	1 mg/kg
Bis(2-chloroethyl) ether	Maximum	1.2 mg/kg
Hexachlorobenzene	Maximum	0.63 mg/kg
Indeno(1,2,3-cd)pyrene	95% UCL of the mean	0.42 mg/kg
Nitrosodi-n-propylamine, n-	Maximum	0.63 mg/kg
Pentachlorophenol	Maximum	1.2 mg/kg
Arsenic	95% UCL of the mean	8 mg/kg
Cobalt	95% UCL of the mean	24 mg/kg
Manganese	95% UCL of the mean	7,100 mg/kg

Abbreviations: ABS_{GI} = gastrointestinal absorption factor; EPC = exposure point concentration; mg/kg = milligram chemical per kilogram soil; mg/kg = milligrams per kilogram; UCL = upper confidence limit

*Contaminant concentration converted to standard unit for calculating exposure.

Table B7: Major Exposure Pathways for the Matlack site

Environmental Medium	Point of Exposure	Exposure Route	Exposed Population	Past	Current	Future
Off-Site Surface Water/Sediment	Grand Sprute Run	Ingestion/Dermal	Residents Accessing off-site areas	C	C	C
Off-Site Biota	Grand Sprute Run	Ingestion	Residents Accessing off-site areas	E	E	E
Off-Site Potable Wells	Residential	Ingestion/Dermal	Residents	E	E	E
On-Site Groundwater	Production Well	Ingestion/Dermal	Employees	P	E	E
On-Site Soil (0 to 2 feet)	Lagoon	Ingestion/Dermal	Employees	E	E	P
On-Site Soil (4 to 9.5 feet)	On-Site (including Lagoon)	Ingestion/Dermal	Employees	E	E	P
On-Site Indoor Air (Vapor Intrusion)	Basement (On-site Building)	Inhalation	Employees	P	P	P

C = completed; E = eliminated; P = potential

Table B8: Site-specific exposure factors and exposure parameters used for calculating exposure dose for surface water

Site-specific Exposure Factors

Duration Category	Event Duration (hours/event)	Event Frequency (events/day)	Days per Week	Weeks per Year	Years	Exposure Group Specific $EF_{noncancer}$	Exposure Group Specific* EF_{cancer}
Acute	0.5	1	-	-	-	1	-
Intermediate	0.5	1	2	26	-	0.29	-
Chronic	0.5	1	2	26	56	0.14	= $EF_{noncancer}$ x Exposure Duration for $Cancer_{Exposure\ Group}$ (years) ÷ 78 years

Abbreviations: EF = exposure factor; NC = not calculated. *Cancer risk is averaged over a lifetime of exposure (78 years).

Site-specific Exposure Parameters

Exposure Group	Body Weight (kg)	Exposure Duration (years)	Combined Skin Surface Area (cm^2)	Notes
6 to < 11 years	31.8	5	3,824	-
11 to < 16 years	56.8	5	5,454	-
16 to < 21 years	71.6	5	6,083	-
Total Child (all age groups)	-	15	-	-
Adult	80	56	7,325	-

Abbreviations: cm^2 = centimeters square skin; kg = kilograms

Table B9: Site-specific exposure factors and exposure parameters used for calculating exposure dose for sediment

Site-specific Exposure Factors

Duration Category	Days per Week	Weeks per Year	Years	Exposure Group Specific $EF_{noncancer}$	Exposure Group Specific* EF_{cancer}
Acute	-	-	-	1	-
Intermediate	2	26	-	0.29	-
Chronic	2	26	56	0.14	= $EF_{noncancer}$ x Exposure Duration for $Cancer_{Exposure\ Group}$ (years) ÷ 78 years
Pica	2	-	-	0.29	-

Abbreviations: EF = exposure factor; NC = not calculated

Note: The dermal absorbed dose equation includes 1 event/day EF parameter. *Cancer risk is averaged over a lifetime of exposure (78 years).

Site-specific Exposure Parameters

Exposure Group	Body Weight (kg)	Exposure Duration (years)	CTE Intake Rate (mg/day)	RME Intake Rate (mg/day)	Custom Intake Rate (mg/day)	Soil-pica Intake Rate (mg/day)	Adherence Factor to Skin (mg/cm^2/event)	Combined Skin Surface Area (cm^2)	Notes
6 to < 11 years	31.8	5	60	200	-	-	0.2	1,240	-
11 to < 16 years	56.8	5	30	100	-	-	0.2	1,770	-
16 to < 21 years	71.6	5	30	100	-	-	0.2	1,950	-
Total Child (all age groups)	-	15	-	-	-	-	-	-	-
Adult	80	56	30	100	-	-	0.07	2,275	-

Abbreviations: cm^2 = centimeters square skin; CTE = central tendency exposure (typical); kg = kilograms; mg/cm^2/event = milligram chemical per centimeter square of skin per event; mg/day = milligram soil per day; RME = reasonable maximum exposure (higher)

Table B10a: Site-specific combined ingestion and dermal exposure doses for chronic exposure to tetrachloroethylene in soil at 15,000 mg/kg along with non-cancer hazard quotients and cancer risk estimates*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	CTE Cancer Risk	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration (yrs)
6 to < 11 years	0.0045	0.57	-	0.014	1.7 †	-	5
11 to < 16 years	0.0015	0.19	-	0.0042	0.52	-	5
16 to < 21 years	0.0012	0.16	-	0.0033	0.42	-	5
Total Child	-	-	9.8E-7	-	-	2.9E-6 ‡	15
Adult	0.00093	0.12	1.4E-6 ‡	0.0028	0.35	4.2E-6 ‡	56

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher); yrs = years

***The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.008 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 0.0021 $(mg/kg/day)^{-1}$.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

Table B10b: Site-specific combined ingestion and dermal exposure doses for chronic exposure to trichloroethylene in soil at 630 mg/kg along with non-cancer hazard quotients*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	CTE Cancer Risk	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration (yrs)
6 to < 11 years	0.00019	0.38	-	0.00059	1.2 †	-	5
11 to < 16 years	6.4E-05	0.13	-	0.00017	0.35	-	5
16 to < 21 years	5.2E-05	0.10	-	0.00014	0.28	-	5
Total Child	-	-	1.2E-6 ‡	-	-	3.6E-6 ‡	15
Adult	3.9E-05	0.078	1.3E-6 ‡	0.00012	0.24	3.9E-6 ‡	56

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher); yrs = years

*The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.0005 mg/kg/day and the cancer risks were calculated using the cancer slope factors of 0.022 [NHL], 0.016 [liver], 0.0093 [kidney] $(mg/kg/day)^{-1}$ and age-dependent adjustment factors.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

Table B11a: Site-specific combined ingestion and dermal exposure doses for intermediate exposure to tetrachloroethylene in soil at 15,000 mg/kg along with non-cancer hazard quotients*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	Soil-Pica Dose (mg/kg/day)	Soil-Pica Non-cancer Hazard Quotient
6 to < 11 years	0.0091	1.1 †	0.028	3.5 †	-	-
11 to < 16 years	0.0031	0.38	0.0083	1.0 †	-	-
16 to < 21 years	0.0025	0.31	0.0067	0.84	-	-
Adult	0.0019	0.23	0.0056	0.70	-	-

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher)

***The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the intermediate (two weeks to less than 1 year) minimal risk level of 0.008 mg/kg/day.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

Table B11b: Site-specific combined ingestion and dermal exposure doses for intermediate exposure to trichloroethylene in soil at 630 mg/kg along with non-cancer hazard quotients*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	Soil-Pica Dose (mg/kg/day)	Soil-Pica Non-cancer Hazard Quotient
6 to < 11 years	0.00038	0.76	0.0012	2.3 †	-	-
11 to < 16 years	0.00013	0.26	0.00035	0.70	-	-
16 to < 21 years	0.00010	0.21	0.00028	0.56	-	-
Adult	7.8E-05	0.16	0.00024	0.47	-	-

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher)

*The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the intermediate (two weeks to less than 1 year) minimal risk level of 0.0005 mg/kg/day.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

Table B12a: Site-specific combined ingestion and dermal exposure doses for acute exposure to tetrachloroethylene in soil at 15,000 mg/kg along with non-cancer hazard quotients*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	Soil-Pica Dose (mg/kg/day)	Soil-Pica Non-cancer Hazard Quotient
6 to < 11 years	0.032	4.0 †	0.098	12 †	-	-
11 to < 16 years	0.011	1.3 †	0.029	3.7 †	-	-
16 to < 21 years	0.0087	1.1 †	0.023	2.9 †	-	-
Adult	0.0065	0.82	0.020	2.5 †	-	-

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher)

*The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the acute (less than two weeks) minimal risk level of 0.008 mg/kg/day.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

Table B13: Critical health effect, LOAEL or Human Equivalent Dose (HED_{99}), UF and the resulting MRL of TCE

Health Effect	LOAEL (or HED_{99}) (mg/kg/day)	UF	MRL (mg/kg/day)
Immunotoxicity	0.37	1,000	0.00037
Thymus weight	0.048	100	0.00048
Fetal heart malformation	0.0051	10	0.0005

Table B14. Wading: Site-specific dermal exposure doses for chronic exposure to benzo(a)pyrene in surface water at 0.007 mg/L along with non-cancer hazard quotients and cancer risk estimates*

Exposure Group	Dose (mg/kg/day)	Non-cancer Hazard Quotient	Cancer Risk	Exposure Duration (yrs)
6 to < 11 years	0.00028	0.92	-	5
11 to < 16 years	0.00022	0.73	-	5
16 to < 21 years	0.00019	0.65	-	5
Total Child	-	-	1.8E-4 ‡	15
Adult	0.00021	0.70	2.6E-4 ‡	56

Source: See Table B5 and Table B8, Appendix B

Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; yrs = years

*The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the chronic (lifetime) reference dose of 0.0003 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 1.7 $(mg/kg/day)^{-1}$ and age-dependent adjustment factors, ‡Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

Table B15: Calculated Benzo(a)pyrene equivalent for the PAHs detected in the Surface Water

Potential Contaminants of Concern	EPC[a] (µg/L)	Cancer Potency Factor[b]	BaP Equivalent[c] (µg/L)	Total BaP Equivalent (µg/L)
Benzo(a)pyrene	7.0	1	7.0	11.55
Dibenz(a,h)anthracene	7.0	0.34[d]	2.38	
Benzo(b)fluoranthene	7.0	0.1	0.7	
Benzo(g,h,i)perylene	7.0	0.01	0.07	
Benzo(k)fluoranthene	7.0	0.1	0.7	
Indeno(1,2,3-cd)pyrene	7.0	0.1	0.7	

[a]Exposure Point Concentration using EPA ProUCL ® 5.1; [b]Cancer potency factor relative to benzo[a]pyrene (BaP) (OEHHA 2015); [c]BaP equivalent; [d]https://dtsc.ca.gov/wp-content/uploads/sites/31/2018/01/HERD_Poster_Benzo-a-pyrene-pdf.pdf

Table B16. Site-specific combined ingestion and dermal exposure doses for chronic exposure to vinyl chloride in soil at 10 mg/kg along with non-cancer hazard quotients*

Exposure Group	CTE Dose (mg/kg/day)	CTE Non-cancer Hazard Quotient	CTE Cancer Risk	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration (yrs)
6 to < 11 years	2.7E-06	0.00090	-	9.0E-06	0.0030	-	5
11 to < 16 years	7.6E-07	0.00025	-	2.5E-06	0.00084	-	5
16 to < 21 years	6.0E-07	0.00020	-	2.0E-06	0.00066	-	5
Total Child	-	-	3.6E-7	-	-	1.2E-6 ‡	15
Adult	5.4E-07	0.00018	2.8E-7	1.8E-06	0.00059	9.2E-7	56

Source: See Table B6 and Table B9, Appendix B

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; RME = reasonable maximum exposure (higher); yrs = years

*The calculations in this table were generated using ATSDR's PHAST v2.0.1.0. The non-cancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.003 mg/kg/day and the cancer risks were calculated using the cancer slope factors of 1.4 [from birth] 0.72 [during adulthood] $(mg/kg/day)^{-1}$.

‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

Appendix C - Toxicological Summary

The toxicological summary provided in this Appendix is based on ATSDR's ToxFAQs (https://wwwn.cdc.gov/TSP/ToxFAQs/ToxFAQsLanding.aspx). The health effects described in this section are typically known to occur at levels of exposure much higher than those that occur from environmental contamination. The chance that a health effect will occur is dependent on the amount, frequency and duration of exposure, and the individual susceptibility of exposed persons.

1,4-Dioxane is a clear liquid with a faint pleasant odor and can be released into the air, water, and soil at places where it is produced or used as a solvent. In soil, 1,4-dioxane does not stick to soil particles, is stable and does not break down, and can move from soil into groundwater. 1,4-Dioxane is a trace contaminant of some chemicals used in cosmetics, detergents, and shampoos.

Breathing low levels can cause eye and nose irritation for short periods of time. Exposure to very high levels may cause severe kidney and liver effects and possibly death. In animals, breathing vapors affects mainly the nasal cavity and the liver and kidneys. It is likely that children would show the same health effects as adults. 1,4-Dioxane may cause changes in your genes and reproductive effects in humans are unknown; however, some effects on the fetus have been seen in rats (ATSDR 2012, Giavini 1985).

In animals, tumors of the nasal cavity, liver and gall bladder tumors have been reported (ATSDR 2012; DHHS 2011; EPA IRIS 2013). 1,4-dioxane is listed as "likely to be carcinogenic to humans" by all routes of exposure (EPA IRIS 2013) and is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals" (DHHS 2011). The National Institute for Occupational Safety and Health (NIOSH) considers 1,4-dioxane a cancer-causing agent in workers (NIOSH 2010). Based on the UCL of the arithmetic mean concentration detected in surface water, the calculated LECR was three in 1,000,000,000 which is considered an unlikely increase in risk of cancer.

Aluminum is a light, silvery-white metal found in the earth's crust. It is very reactive and generally combined with other elements, most commonly with oxygen, silicon, and fluorine. Aluminum is used to make beverage cans, pots and pans, airplanes, siding and roofing, and foil. It is found in many consumer products

Aluminum occurs naturally in soil, water, and air. Mining and processing of aluminum ores or the production of aluminum metal, alloys, and compounds are its primary sources. It cannot be destroyed in the environment and settles to the ground or is washed out of the air by rain. However, very small aluminum particles can stay in the air for many days.

Workers breathing in large amounts can cause lung problems and changes that show up in chest X-rays, including decreased performance in some tests that measure functions of the nervous system. Lung effects have also been observed in animals but cannot be specifically

associated to the aluminum but may be dust related. In animals, the nervous system is a target of aluminum toxicity. Aluminum has been found in breast milk and bone disease has been reported in children caused by high levels of aluminum. We do not know if aluminum will cause birth defects in people. Birth defects have not been seen in animals.

Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide. As alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics as fire retardant. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass.

Antimony is released to the environment from natural sources and from industry. In the air, antimony is attached to very small particles that may stay in the air for many days. Most antimony particles settle in soil, where it attaches strongly to particles that contain iron, manganese, or aluminum.

Breathing high levels for a long time can irritate eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers. In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, fertility problems were observed when rats breathed very high levels of antimony for a few months.

Ingesting large doses of antimony can cause vomiting. Other effects of ingesting antimony are unknown. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it.

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Breathing high levels of inorganic arsenic can cause a sore throat or irritated lungs. Ingesting high levels of inorganic arsenic can result in death. Lower levels of arsenic can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling.

Organic arsenic compounds are used as pesticides, primarily on cotton plants. These compounds are less toxic than inorganic arsenic compounds. Exposure to high levels of some organic arsenic compounds may cause similar effects as those caused by inorganic arsenic.

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The World Health Organization (WHO), the USDHHS, and the USEPA have determined that inorganic arsenic is a human carcinogen.

Benzene is a widely used chemical formed from both natural processes and human activities. It is highly flammable liquid at room temperature. It evaporates easily into the air and has a sweet odor. Benzene is one of the top 20 chemicals used in the United States industry. Industries mix benzene with other chemicals to make plastics, resins, and nylon and synthetic fibers. Benzene is also used to make some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include volcanoes and forest fires. Benzene is also a natural part of crude oil, gasoline, and cigarette smoke.

Breathing very high levels of benzene can cause death while breathing high concentrations of benzene can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death. Long-term exposure to benzene (365 days or longer) can cause harmful effects on the bone marrow, can cause a decrease in red blood cells leading to anemia, and can cause excessive bleeding. Benzene can cause impairment of the immunity system increasing the chance of infection. Studies have shown some women exposed to high levels of benzene in air for many months had irregular menstrual periods and a reduced size of their ovaries. It is not known whether benzene exposure affects the developing fetus in pregnant women or fertility in men. Studies have shown that pregnant animals exposed to benzene in air resulted in low birth weights, delayed bone formation, and bone marrow damage.

The United States Department of Health and Human Services (DHHS) has determined that benzene is a known human carcinogen. Long-term exposure to high levels of benzene in the air can cause leukemia, cancer of the blood-forming organs.

Bis(2-chloroethyl) ether is a colorless, nonflammable liquid with a strong unpleasant odor. It dissolves easily in water, and some of it will slowly evaporate to the air. It is made in factories, and most of it is used to make pesticides. Some of it is used as a solvent, cleaner, component of paint and varnish, rust inhibitor, or as a chemical intermediate to make other chemicals. The most likely way to be exposed to methylene chloride is by breathing contaminated air or touching contaminated soil.

Bis(2-chloroethyl) ether causes irritation to the skin, eyes, throat, and lungs. In some cases, damage to the lungs can be severe enough to cause death. Breathing low concentrations will cause coughing and nose, and throat irritation. Animal studies show effects similar to those

observed in people. Some animal studies indicate that bis(2-chloroethyl) ether can affect the nervous system resulting in sluggish and slow movement, staggering, unconsciousness, and death. We do not know if bis(2-chloroethyl) ether causes reproductive effects or birth defects in people or animals.

The ability of bis(2-chloroethyl) ether to cause cancer in humans has not been established. There is some evidence that bis(2-chloroethyl) ether causes cancer in mice. The International Agency for Research on Cancer (IARC) has determined that bis(2-chloroethyl) ether is not classifiable as to its carcinogenicity in humans.

Cadmium is a natural element in the earth's crust, usually found as a mineral combined with other elements. All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Cadmium enters soil, water, and air from mining, industry, and burning coal and household wastes. The general population is exposed from breathing cigarette smoke or eating cadmium contaminated foods.

Cadmium does not break down in the environment and it can travel long distances in the air before falling to the ground or water. Some forms of cadmium dissolve in water and it binds strongly to soil particles. Fish, plants, and animals take up cadmium from the environment.

Breathing high levels of cadmium can severely damage the lungs. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea. Long-term exposure to low levels of cadmium in air, food, or water can accumulate in the kidneys and possible cause kidney disease. Other long-term effects include fragile bones. The same effects (kidney, lung, and bone damage) have been reported in children.

There is no information on birth defects in people. However, babies of animals exposed to high levels of cadmium during pregnancy had changes in behavior and learning ability. In animals, high enough exposures to cadmium before birth can reduce body weights and affect the skeleton in the developing young.

The Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds are known human carcinogens.

Cis-1,2-dichloroethene, also called 1,2-dichloroethylene, is a highly flammable, colorless liquid with a sharp, harsh odor. It is used to produce solvents and in chemical mixtures. There are two forms of 1,2-dichloroethene; one is called cis-1,2-dichloroethene and the other is called trans-1,2-di-chloroethene. Sometimes both forms are present as a mixture.

Breathing high levels of 1,2-dichloroethene can make you feel nauseous, drowsy, and tired; breathing very high levels can kill you. When animals breathed high levels of *trans*-1,2-dichloroethene for short or longer periods of time, their livers and lungs were damaged and the effects were more severe with longer exposure times. Animals that breathed very high levels of *trans*-1,2-dichloroethene had damaged hearts. Animals that ingested extremely high doses of *cis*- or *trans*-1,2-dichloroethene died. Lower doses of *cis*-1,2-dichloroethene caused effects on

the blood, such as decreased numbers of red blood cells, and also effects on the liver. The long-term (365 days or longer) human health effects after exposure to low concentrations of 1,2-dichloroethene aren't known. One animal study suggested that an exposed fetus may not grow as quickly as one that hasn't been exposed. Exposure to 1,2-dichloroethene hasn't been shown to affect fertility in people or animals.

Cobalt is a naturally occurring element found in rocks, soil, water, plants, and animals and enters the environment from natural sources including the burning of coal or oil or the production of cobalt alloys. Cobalt cannot be destroyed. It is used to produce alloys used in the manufacture of aircraft engines, magnets, grinding and cutting tools, artificial hip and knee joints. The general population is exposed to low levels of cobalt in air, water, and food.

Cobalt has both beneficial and harmful effects on health. At low levels, it is part of vitamin B12, which is essential for good health. At high levels, it may harm the lungs, heart and skin (dermatitis). Liver and kidney effects have also been observed in animals exposed to high levels of cobalt. We do not know if exposure to cobalt will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to nonradioactive cobalt.

Cancer has been shown in animals that breathed cobalt or when cobalt was placed directly into the muscle or under the skin. Based on the laboratory animal data, the International Agency for Research on Cancer (IARC) has determined that cobalt and cobalt compounds are possibly carcinogenic to humans. NTP also lists cobalt as reasonably anticipated to be a human carcinogen.

Copper is a metal that occurs naturally throughout the environment, in rocks, soil, water, and air. It is an essential element in plants and animals (including humans), which means it is necessary for us to live. Copper is used to make different kinds of products like wire, plumbing pipes, and sheet metal and combined with other metals to make brass and bronze pipes and faucets. It is released into the environment by mining, farming, and manufacturing and through wastewater releases into rivers and lakes. Copper is also released from natural sources, like volcanoes, windblown dusts, decaying vegetation, and forest fires.

You may be exposed to copper from breathing air, drinking water, eating foods, or via skin contact. Low levels of copper are essential for maintaining good health. High levels can cause harmful effects such as irritation of the nose, mouth and eyes, vomiting, diarrhea, stomach cramps, nausea, and even death. Very-high doses of copper can cause damage to your liver and kidneys and can even cause death. Studies in animals suggest that the young children may have more severe effects than adults, but we don't know if this would also be true in humans. There is a very small percentage of infants and children who are unusually sensitive to copper.

We do not know whether copper can cause cancer in humans. The EPA has determined that copper is not classifiable as to human carcinogenicity.

1,1-Dichloroethane (1,1-DCA) is a colorless, oily liquid with a sweet odor. It breaks down slowly in air and has the potential for long-range transport. 1,1-Dichloroethane does not bind strongly to soil particles unless the organic content of the soil is high. Small amounts of 1,1-DCA released to soil can evaporate into the air or move into ground water. It does not occur naturally in the environment.

1,1-DCA is used mostly as an intermediate in the manufacture of 1,1,1-trichloroethane and in a limited amount as a solvent for cleaning and degreasing. Breathing high levels of 1,1-DCA can cause anesthesia and irregular heartbeats. It is not expected to build up in the body tissues of animals. But kidney effects have been observed in cats exposed in air for long periods. However, kidney effects have not been observed in other animal species following long-term inhalation or oral exposure.

We do not know whether 1,1-DCA can produce birth defects in humans. Minor skeletal problems were observed in the fetuses of rats breathing it; decreases in body weight were also observed in the mothers.

A study in rats and mice found suggestive evidence that 1,1-DCA may cause cancer. However, the study had several flaws and the results are not conclusive. Another long-term study in mice drinking water containing 1,1-dichloroethane did not find cancer. The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC) have not evaluated the carcinogenic potential of 1,1-dichloroethane. The EPA has determined that 1,1-dichloroethane is a possible human carcinogen.

Hexachlorobenzene is a white crystalline solid that is not very soluble in water. It was widely used as a fungicide (until 1965) and was also used to make fireworks, ammunition, and synthetic rubber. Currently, there are no commercial uses of hexachlorobenzene (in the US) but, it is formed as a waste product during the manufacture of other chemicals such as trichloroethylene and tetrachloroethylene and is a contaminant in some pesticides. The most likely way to be exposed to Hexachlorobenzene is by mainly from eating low levels in contaminated food.

Breathing in large amounts of Hexachlorobenzene may cause weakness, tremors, and convulsions; skin sores; and liver and thyroid effects. It can build up in body fat (including breast tissue) and remain for long periods.

Studies in animals suggest that eating food with Hexachlorobenzene for a long time can cause cancer of the liver, kidney, and thyroid. Infants and young children seem to be more sensitive to the effects of very high levels. The USDHHS has determined that it can be reasonably anticipated to be a cancer-causing chemical, and the EPA has determined that methylene chloride is a probable cancer-causing agent in humans.

Lead is a naturally occurring metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Lead has many different uses. It is used in the

production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from gasoline, paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. People may be exposed to lead by eating food or drinking water that contains lead, spending time in areas where lead-based paints have been used and are deteriorating, and by working in a job or engaging in a hobby where lead is used. Small children are more likely to be exposed to lead by swallowing house dust or soil that contains lead, eating lead-based paint chips or chewing on objects painted with lead-based paint.

Lead can affect many organs and systems in the body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed. At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain.

Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead, for example by eating old paint chips, may develop blood anemia, severe stomachache, muscle weakness, and brain damage. A large amount of lead might get into a child's body if the child ate small pieces of old paint that contained large amounts of lead. If a child swallow’s smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, however, lead can affect a child's mental and physical growth. Exposure to lead is more dangerous for young children and fetuses. Fetuses can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead.

The USDHHS has determined that two compounds of lead (lead acetate and lead phosphate) may reasonably be anticipated to be carcinogens based on studies in animals. There is inadequate evidence to clearly determine whether lead can cause cancer in people.

Manganese is naturally occurring metal that is found in many types of rocks and cannot break down in the environment. It combines with other substances including carbon to form pesticides and is used an additive in gasolines to improve the octane rating of the gas. Manganese is an essential trace element, necessary for good health and can be found in grains, cereals, and tea.

The primary way you can be exposed to manganese is by eating food or manganese-containing nutritional supplements. Certain occupations like welding or working in a factory where steel is made may increase your chances of being exposed to high levels of manganese. Manganese is routinely contained in groundwater, drinking water, and soil at low levels.

The most common health problems in workers exposed to high levels of manganese involve the nervous system. These health effects include behavioral changes and other nervous

system effects, which include movements that may become slow and clumsy. Exposure to high levels of manganese in air can cause lung irritation and reproductive effects. Nervous system and reproductive effects have been observed in animals after high oral doses of manganese. Studies in children have suggested that extremely high levels of manganese exposure may produce undesirable effects on brain development. These changes (temporary or permanent) may not have been caused by manganese alone. Studies in animals suggest children may be more sensitive than adults.

The EPA concluded that existing scientific information cannot determine if excess manganese can cause cancer.

Mercury is a naturally occurring metal which has several forms. Metallic mercury is a shiny, silvery liquid which, when heated, can be a colorless, odorless gas. Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts," which are usually white powders or crystals. Mercury also combines with carbon to make organic mercury compounds. The most common one, methylmercury, is produced mainly by microscopic organisms in the water and soil. Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings, and batteries. Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments. People are commonly exposed to mercury by eating fish or shellfish contaminated with methylmercury, breathing vapors in air from spills, incinerators, and industries that burn mercury-containing fuels, the release of mercury from dental work, working with mercury, or practicing rituals that include mercury.

The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems. Short-term exposure to high levels of metallic mercury vapors may cause effects including lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

Young children are more sensitive to mercury than adults. Mercury in the mother's body passes to the fetus and may accumulate there. It can also pass to a nursing infant through breast milk, although the benefits of breast feeding may be greater than the possible adverse effects of mercury in breast milk.

Harmful effects due to mercury that passes from the mother to the fetus include brain damage, mental retardation, incoordination, blindness, seizures, and inability to speak. Children poisoned by mercury may develop problems with their nervous and digestive systems, and kidney damage.

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has

caused kidney tumors in male mice. The USEPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

N-Nitrosodi-N-Propylamine is a yellow liquid at room temperature that does not dissolve in water and evaporates slowly. Small amounts of it are produced as a side reaction during some manufacturing processes, as a contaminant in some commonly available weed killers (dinitroaniline-based), and during the manufacture of some rubber products. Persons may be exposed to n-Nitrosodi-npropylamine by eating foods treated with nitrite preservatives (e.g., cheeses, cured meats) and drinking certain alcoholic beverages. The general population may be exposed to it in cigarette smoke. n-Nitrosodi-n-propylamine is not likely to get into your body unless you eat certain foods, drink alcoholic beverages, or are exposed to it at a waste disposal site by breathing n-Nitrosodi-n-propylamine vapors.

Little is known about the health effects of short exposures to n-Nitrosodi-n-propylamine in experimental animals except that eating or drinking certain amounts of this chemical can cause liver disease and death. Long-term exposure of experimental animals to it in food or drinking water causes cancer of the liver, esophagus, and nasal cavities. Although human studies are not available, the animal evidence indicates that it is reasonable to expect that exposure to n-Nitrosodi-n-propylamine by eating or drinking could cause liver disease and cancer in humans. It is not known whether other effects, such as birth defects, occur in animals or could occur in humans exposed to n-Nitrosodi-n-propylamine by eating or drinking.

Liver disease and cancer due to exposure to n-Nitrosodi-n-propylamine by breathing or skin contact are, however, a possibility and a health concern.

Pentachlorophenol was widely used as a pesticide and wood preservative. Since 1984, pentachlorophenol has been restricted to certified applicators and is no longer available to the general public. It is still used industrially as a wood preservative for utility poles, railroad ties, and wharf pilings. The general populations can be exposed to very low levels of pentachlorophenol in contaminated indoor and outdoor air, food, drinking water and soil.

Exposure to high levels of pentachlorophenol can cause increases in body temperature, liver effects, damage to the immune system, reproductive effects, and developmental effects. Some of the harmful effects of pentachlorophenol are caused by the other chemicals present in technical grade pentachlorophenol.

Some studies have found an increase in cancer risk in workers exposed to high levels of technical grade pentachlorophenol for a long time, but other studies have not found this. Increases in liver, adrenal gland, and nasal tumors have been found in laboratory animals exposed to high doses of pentachlorophenol. The EPA has determined that pentachlorophenol is a probable human carcinogen and the IARC considers it possibly carcinogenic to humans.

Polycyclic Aromatic Hydrocarbons (PAHs) Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot. These include benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, benzo(g,h,i)perylene, indeno(1,2,3-cd)pyrene, phenanthrene, and naphthalene.

Some PAHs are manufactured. These pure PAHs usually exist as colorless, white, or pale yellow-green solids. PAHs are found in coal tar, crude oil, creosote, and roofing tar, but a few are used in medicines or to make dyes, plastics, and pesticides. Mice that were fed high levels of one PAH during pregnancy had difficulty reproducing and so did their offspring. These offspring also had higher rates of birth defects and lower body weights. It is not known whether these effects occur in people. Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. But these effects have not been seen in people.

The USDHHS has determined that some PAHs may reasonably be expected to be carcinogens. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

Polychlorinated biphenyls (PCBs) are mixtures of up to 209 individual chlorinated compounds (known as congeners). There are no known anthropogenic sources of PCBs. PCBs can exist as oily liquids, solids or vapor in air. Many commercial PCB mixtures are known by the trade name Aroclor. The majority of PCBs were used in dielectric fluids for use in transformers, capacitors, and other electrical equipment. Since PCBs build up in the environment and can cause harmful health effects, PCB production was stopped in the U.S. in 1977.

PCBs enter the environment during their manufacture, use, and disposal. PCBs can accumulate in fish and marine mammals, reaching levels that may be many thousands of times higher than in water. The most commonly observed health effects associated with exposures to large amounts of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage. PCB exposures in the general population are not likely to result in skin and liver effects. Most of the studies of health effects of PCBs in the general population examined children of mothers who were exposed to PCBs.

Animals administered with large PCB dose for short periods of time had mild liver damage and some died. Animals that ate smaller amounts of PCBs in food over several weeks or months developed various kinds of health effects, including anemia; acne-like skin conditions; and liver, stomach, and thyroid gland injuries. Other effects of PCBs in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects.

Few studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. The Department of Health and Human Services (DHHS) has concluded that PCBs may reasonably be anticipated to be carcinogens. The EPA and the International Agency for Research on Cancer (IARC) have determined that PCBs are probably carcinogenic to humans.

Women who were exposed to relatively high levels of PCBs in the workplace or ate large amounts of fish contaminated with PCBs had babies that weighed slightly less than babies from women who did not have these exposures. Babies born to women who ate PCB-contaminated fish also showed abnormal responses in tests of infant behavior. Some of these behaviors, such as problems with motor skills and a decrease in short-term memory, lasted for several years. Other studies suggest that the immune system was affected in children born to and nursed by mothers exposed to increased levels of PCBs. There are no reports of structural birth defects caused by exposure to PCBs or of health effects of PCBs in older children. The most likely way infants will be exposed to PCBs is from breast milk. Transplacental transfers of PCBs were also reported. In most cases, the benefits of breast-feeding outweigh any risks from exposure to PCBs in mother's milk.

Tetrachloroethylene (PCE) is a manufactured chemical that is widely used for dry cleaning of fabrics and for metal-degreasing. It is a nonflammable liquid at room temperature. It evaporates easily into the air and has a sharp, sweet odor. Most people can smell PCE when it is present in the air at a level of approximately 7,000 micrograms per cubic meter or more, although some can smell it at even lower levels. People are commonly exposed to PCE when they bring clothes from the dry cleaners.

High concentrations of PCE can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death. Irritation may result from repeated or extended skin contact with it. These symptoms occur almost entirely in work (or hobby) environments when people have been exposed to high concentrations. In industry, most workers are exposed to levels lower than those causing obvious nervous system effects, although subtler neurological effects are possible at the lower levels. The health effects of breathing in air or drinking water with low levels of PCE are not known. Results from some studies suggest that women who work in dry cleaning industries where exposures to PCE can be quite high may have more menstrual problems and spontaneous abortions than women who are not exposed. Results of animal studies, conducted with amounts much higher than those that most people are exposed to show that PCE can cause liver and kidney damage. Exposure to very high levels of PCE can be toxic to the unborn pups of pregnant rats and mice. Changes in behavior were observed in the offspring of rats that breathed high levels of the chemical while they were pregnant.

The U.S. Department of Health and Human Services (USDHHS) has determined that PCE may reasonably be anticipated to be a carcinogen. PCE has been shown to cause liver tumors in mice and kidney tumors in male rats.

Trichloroethylene (TCE) is a nonflammable, colorless liquid with a somewhat sweet odor and a sweet, burning taste. It is used mainly as a solvent to remove grease from metal parts, but it is also an ingredient in adhesives, paint removers, typewriter correction fluids, and spot removers. TCE dissolves a little in water and can remain in groundwater for a long time. It quickly evaporates from water, so it is commonly found as a vapor in the air. People can be exposed to TCE by breathing air in and around the home which has been contaminated with TCE vapors from shower water or household products, or by drinking, swimming, or showering in water that has been contaminated with TCE. Breathing small amounts of TCE may cause headaches, lung irritation, dizziness, poor coordination, and difficulty concentrating. Breathing large amounts of TCE may cause impaired heart function, unconsciousness, and death. Breathing it for long periods may cause nerve, kidney, and liver damage. Drinking large amounts of TCE may cause nausea, liver damage, unconsciousness, impaired heart function, or death. Drinking small amounts of TCE for long periods may cause liver and kidney damage, impaired immune system function, and impaired fetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with TCE for short periods may cause skin rashes.

Following U.S. EPA (2005b) Guidelines for Carcinogen Risk Assessment, TCE is characterized as "carcinogenic to humans" by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer. The kidney cancer association cannot be reasonably attributed to chance, bias, or confounding. The human evidence of carcinogenicity from epidemiologic studies of TCE exposure is strong for non-Hodgkin lymphoma (NHL), but less convincing than for kidney cancer, and more limited for liver and biliary tract cancer. In addition to the body of evidence pertaining to kidney cancer, NHL, and liver cancer, the available epidemiologic studies also provide more limited evidence of an association between TCE exposure and other types of cancer, including bladder, esophageal, prostate, cervical, breast, and childhood leukemia.

Vanadium is a naturally occurring white-to-gray metal that usually combines with other elements such as oxygen, sodium, sulfur, or chloride. Vanadium and vanadium compounds can be found in the earth's crust and in rocks, some iron ores, and crude petroleum deposits. It mainly enters the environment from natural sources and from the burning of fuel oils. Vanadium (in the form of vanadium oxide) is a component in special kinds of steel that is used for automobile parts, springs, and ball bearings. Low levels of Vanadium are found in air, water, and food; however, most people are exposed mainly from food.

Breathing high levels of Vanadium (vanadium pentoxide) in air can result in lung damage. Nausea, mild diarrhea, and stomach cramps have been reported in people who have been exposed to some vanadium compounds. In animals, ingestion of large amounts decreased red blood cells, increased blood pressure, and showed mild neurological effects. Studies in animals exposed during pregnancy have shown that vanadium can cause decreases in growth and increases in the occurrence of birth defects.

The International Agency for Research on Cancer (IARC) has classified vanadium pentoxide (a vanadium compound) as possibly carcinogenic to humans based on evidence of lung cancer in exposed mice.

Vinyl Chloride is a colorless gas. It burns easily and it is not stable at high temperatures. It has a mild, sweet odor. It is a manufactured substance that does not occur naturally. It is a biodegradation intermediate of trichloroethane, trichloroethylene, and tetrachloroethylene. Vinyl chloride is used to make polyvinyl chloride (PVC). PVC is used to make a variety of plastic products, including pipes, wire and cable coatings, and packaging materials.

Breathing high levels of vinyl chloride can cause dizziness. Breathing very high levels can cause you to pass out and breathing extremely high levels can cause death. Some people who have breathed vinyl chloride for several years have changes in the structure of their livers. People are more likely to develop these changes if they breathe high levels of vinyl chloride. Some people who work with vinyl chloride have nerve damage and develop immune reactions. The lowest levels that produce liver changes, nerve damage, and immune reaction in people are not known. Some workers exposed to very high levels of vinyl chloride have problems with the blood flow in their hands. Their fingers turn white and hurt when they go into the cold.

Animal studies have shown that long-term exposure to vinyl chloride can damage the sperm and testes. Further animals suggest that vinyl chloride might affect growth and development. It has not been proven that vinyl chloride causes birth defects in humans.

The U.S. Department of Health and Human Services has determined that vinyl chloride is a known carcinogen. Studies in workers who have breathed vinyl chloride over many years showed an increased risk of liver cancer; brain cancer, lung cancer, and some cancers of the blood have also been observed in workers. Animal studies suggest that infants and young children might be more susceptible than adults to vinyl chloride-induced cancer.