

Public Health Assessment

Final Release

VENTRON/VELSICOL SITE

WOOD-RIDGE/CARLSTADT, BERGEN COUNTY, NEW JERSEY

EPA FACILITY ID: NJD980529879

**Prepared by
New Jersey Department of Health**

OCTOBER 4, 2016

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
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.

Use of trade names is for identification only and does not constitute endorsement by the U.S. Department of Health and Human Services. Additional copies of this report are available from:

National Technical Information Service, Springfield, Virginia
(703) 605-6000

You May Contact ATSDR Toll Free at
1-800-CDC-INFO

or

Visit our Home Page at: <http://www.atsdr.cdc.gov>

Ventron/Velsicol Site

Final Release

PUBLIC HEALTH ASSESSMENT

VENTRON/VELSICOL SITE

WOOD-RIDGE/CARLSTADT, BERGEN COUNTY, NEW JERSEY

EPA FACILITY ID: NJD980529879

Prepared by:

New Jersey Department of Health
Environmental and Occupational Health Surveillance Program
Under Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

Table of Contents

Summary	1
Statement of Issues	4
Background	4
Site Description.....	4
Site History	5
Previous Investigations/Actions.....	6
Site Visit.....	7
Demographics	7
Past ATSDR and NJDOH Activities	8
Environmental Contamination	9
Environmental Guideline Comparison	10
Site Conditions.....	11
On-site Contamination	12
Off-site Contamination	14
Contaminants of Potential Concern: Summary.....	16
Discussion	17
Assessment Methodology	17
Completed Pathways.....	17
Potential Pathways	19
Eliminated Pathways.....	19
Exposure Point Concentration	20
Public Health Implications	20
Non-Cancer Health Effects	20
Incidental ingestion of contaminated soil and sediment from on-site areas	21
Incidental ingestion of contaminated surface water from on-site areas.....	27
Inhalation of on-site contaminated ambient air	28
Incidental ingestion of contaminated soil from off-site properties	28
Cancer Health Effects	29
Incidental ingestion of contaminated soil and sediment from on-site areas	29
Incidental ingestion of contaminated surface water from on-site areas	30
Child Health Considerations	31
Health Outcome Data	31

Public Comment	32
Conclusions	32
Recommendations	33
Public Health Action Plan	33
Public Health Actions Taken by NJDOH and ATSDR	33
Public Health Actions Planned by NJDOH and ATSDR	34
References	34
Report Preparation	38

Tables

Figures

Photographs

Appendix A: Toxicologic Summaries of Contaminants of Concern

Appendix B: Sample Exposure Calculations

Appendix C: Public Comments and Responses

Summary

Introduction

On September 1, 1984 the Ventron/Velsicol site located in the boroughs of Wood-Ridge and Carlstadt, Bergen County, New Jersey was added to the National Priorities List. A mercury processing plant operated at the site from 1929 to 1974. Preliminary sampling results indicated that the soils, sediments, surface and groundwater were contaminated with organic compounds and metals including mercury. In October 2006, U.S. Environmental Protection Agency (USEPA) completed the Remedial Investigation/Feasibility Study (RI/FS) for the 38-acre site, while further investigation and delineation of off-site contamination (for example, Berry's Creek Study Area, or, BCSA¹) has been ongoing.

Through a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), the New Jersey Department of Health (NJDOH) has prepared this Public Health Assessment (PHA) of the Ventron/Velsicol site. The ATSDR and NJDOH's top priority at this site is to ensure that the community around the site has the best information possible to safeguard its health.

Conclusions

The NJDOH and ATSDR have reached three conclusions in this Public Health Assessment on the Ventron/Velsicol site:

Conclusion 1

The NJDOH and ATSDR conclude that there are no current or future site-related exposures (i.e., from ingestion of on-site soil and surface water and inhalation of ambient air) to contaminants at the developed, undeveloped (i.e., USEPA's Operable Unit 1) and off-site residential areas of the Ventron/Velsicol site that can harm people's health.

Basis for Conclusion

Contaminated soils at the on- (i.e., developed and undeveloped areas) and off-site (i.e., residential homes located to the north of the site) areas have been excavated and/or capped. The excavated areas were backfilled with clean fill. An earlier health evaluation associated with incidental ingestion of contaminated soil from off-site areas (i.e., adjacent properties, municipal repair yard and wastewater treatment plant) did not indicate any concern for harmful health effects. In addition, the site is fenced along the northern and western boundaries for access restriction. Thus, area residents are not being exposed to site-related contaminants from on-site areas.

¹The BCSA (i.e., USEPA's Operable Unit 2) also includes the on-site marsh area at the Ventron/Velsicol site.

It should be noted that the on-site marsh area is also a part of BCSA and the remedial investigation of this area is being conducted by the potential responsible parties.

Next Step The NJDOH and ATSDR recommend maintaining access restriction to ensure integrity of the remedies implemented for the on-site areas of Ventron/Velsicol site.

Conclusion 2 *Based on an earlier health consultation conducted in 1995, ATSDR concluded that past exposures associated with consumption of contaminated biota from the on-site marsh area and Berry's Creek (i.e., USEPA's Operable Unit 2) may have harmed people's health.*

Basis for Conclusion Based on the concentration of mercury detected in the biota in the past, frequent ingestion of biota from the on-site marsh area and the Berry's Creek may have resulted in exposures at levels of public health concern.

Next Steps With oversight from the USEPA, the RI of the BCSA is being conducted by the potential responsible parties.

As additional contamination data from BCSA become available, the NJDOH and ATSDR will prepare a health consultation(s) to assess the health implications of potential contamination.

Conclusion 3 *The NJDOH and ATSDR conclude that past exposures to site-related contaminants from the developed, undeveloped (i.e., USEPA's Operable Unit 1), marsh (i.e., part of USEPA's Operable Unit 2) and off-site residential properties at the Ventron/Velsicol site may have harmed people's health.*

Basis for Conclusion Based on the mercury detected in soil and ambient air, the potential for non-cancer adverse health effects associated with past exposures are possible in children and adults. Based on the lead detected in soil, the potential for lead related non-cancer adverse health effect are possible in children. Maximum lifetime excess cancer risk (associated with marsh area) was estimated to be 1 in 10,000 to the exposed population. This exposure poses a low to moderate increase in lifetime excess cancer risk, compared to the background risk of cancer from all causes.

It should be noted that there is some uncertainty in this conclusion because the soil sampling data from 0-2 feet and 0-12 inches may not represent actual surface soil conditions. This may over or underestimate the calculated exposure risk.

**For More
Information**

Questions about this Public Health Assessment should be directed to the NJDOH at (609) 826-4984.

Comments on this Public Health Assessment should be mailed to:

Environmental and Occupational Health Surveillance Program
New Jersey Department of Health
Consumer, Environmental and Occupational Health Service
P.O. Box 369
Trenton, New Jersey 08625-0369

Statement of Issues

On September 1, 1984 the Ventron/Velsicol site located in the boroughs of Wood-Ridge and Carlstadt, Bergen County, New Jersey was added to the National Priorities List. A mercury processing plant operated at the site from 1929 to 1974. Preliminary sampling results indicated that the soils, sediments, surface water and groundwater were contaminated with organic compounds and metals including mercury. Although the Agency for Toxic Substances and Disease Registry (ATSDR) prepared a preliminary Public Health Assessment (ATSDR 1989) based on limited available data at that time, a comprehensive evaluation of exposure pathway could not be conducted because a complete characterization of contamination was not available. One recommendation was to use the data from the Remedial Investigation/Feasibility Study (RI/FS) to evaluate the exposure pathways. A site review and update report (ATSDR 1993) also recommended performance of a public health assessment when the RI/FS was available for review.

In October 2006, the United States Environmental Protection Agency (USEPA) released a proposed remediation plan² for public comment after completing the RI/FS for the site. With the availability of contamination delineation data and a request (in September, 2008) from USEPA to assess the public health implications of past, current, and future exposures associated with on-site contamination, the New Jersey Department of Health (NJDOH) prepared this public health assessment report through a cooperative agreement with the ATSDR.

Further investigation and delineation of off-site contamination (Berry's Creek, Nevertouch Creek, and Diamond Shamrock/Henkel Ditches) are ongoing.

Background

Site Description

The Ventron/Velsicol site is located in the commercial/industrial section of Bergen County, New Jersey (see Figure 1) and approximately 0.6 mile to the south of Teterboro Airport (see Figure 2). The 38-acre site is located within the Hackensack Meadowlands area. The site is bordered to the west by the Diamond Shamrock/Henkel and Randolph Products properties and Park Place East, to the east by Berry's Creek, to the south by Diamond Shamrock/Henkel Ditch (south) and Nevertouch Creek, and to the north by Ethel Boulevard and a railroad track (see Figure 3). Two commercial/industrial facilities and an



Figure 1: Location of Ventron/Velsicol Site

²The Proposed Plan and the Record of decision were issued by the NJDEP with concurrence by USEPA.

empty lot on which a publicly owned treatment works (POTW) was formerly located lie immediately north of Ethel Boulevard and the railroad tracks. Approximately 15.7 of the 38 acres are within the Borough of Wood-Ridge, and the remaining 22.6 acres are within the Borough of Carlstadt. The railroad crosses Berry's Creek at the northeast corner of the site and continues south along the eastern side of Berry's Creek.

Two active warehouses, referred to as the Wolf Warehouse and the U.S. Life Warehouse, are located on the northernmost portion of the site (see Figure 3). The former mercury processing facility was located on this portion of the site (see Figure 4). This portion of the site will be referred to as the "developed" area of the site. The closest residential area is approximately 750 feet to the north. Approximately 19 acres of filled but not developed land lies generally south of the developed portion of the site. This area will be referred to as the "undeveloped" portion of the site. Together, the "developed" and "undeveloped" portions of this site comprise USEPA's Operable Unit 1 (OU1) of the Ventron/Velsicol site (see Figure 3).

The remaining 12 acres of the site, south of the undeveloped area, is generally marsh. This area will be referred to as the "marsh" portion of the site. The USEPA refers to this area as part of Operable Unit 2 (OU2) of the Ventron/Velsicol site, and it is also part of the Berry's Creek Study Area (BCSA) (see Figure 3).

Site History

Before 1927, most of the site was marshland. In 1929, F.W. Berk and Company began operating a processing plant for manufacturing mercury products near the current location of the Wolf Warehouse (NJDEP 2006). The main operations of the mercury processing plant included the manufacture of red and yellow oxides of mercury, phenyl mercuric acetate, and other organic and inorganic mercury compounds. The plant also reclaimed mercury from both in-house and customer waste products (amalgams, batteries, thermometers, impure mercury, etc.). Wastewater effluent from the mercury processing operations was initially discharged through an open ditch, and later through a pipe, into Berry's Creek. No estimates of how much mercury in the wastewater may have been released were found in the available reports. The effluent may also have contained other materials used at the mercury processing facility. The operations at this processing facility appear to be generally similar throughout its manufacturing history (Exponent 2004).

Between 1952 and 1955, the Magnesium Elektron Corporation leased a portion of the property that included a structure known as the Zirconium Building. Berk continued to operate the facility until 1960, when the corporation dissolved and the property was sold to the Wood-Ridge Chemical Corporation (WRCC), a wholly owned subsidiary of the Velsicol Chemical Corporation (Velsicol). Velsicol operated the facility until 1968, when the Ventron Corporation (Ventron), a predecessor to Morton, purchased WRCC and the approximately 7-acre parcel on which the facility was located from Velsicol. Velsicol retained ownership of the rest of the property until transferring ownership to NWI Land Management, Inc., in 1986. Ventron operated the facility until it was closed in

1974. In 1974, the parcel of land where the plant was located was sold to Robert and Rita Wolf. They demolished the plant buildings/structures in 1974; in 1975, the property was subdivided and the title of the westernmost parcel was transferred to the U.S. Life Insurance Company. Subsequently, two warehouses were constructed, one on each parcel.

The former mercury processing facility location extended to the north beyond the location of the existing warehouses, near the north property boundary (northeast of the existing Wolf Warehouse). Prior to the installation in 1961 of the railroad tracks that now border Ethel Boulevard on the north side of the site, it is possible that contaminated soils or wastes could have been placed in this area. It was reported (Exponent 2004) that subsequent to 1960, the approximately 19-acre portion of the site between the developed area and Berry's Creek was used as a dumping area for various materials including demolition material and domestic solid waste. The record of property easements confirms use of the site for municipal waste dumping by the Borough of Wood-Ridge.

Currently, three parties own the property. Jerbil, Inc. owns the U.S. Life Warehouse property, Jonathan and Roni Blonde own the Wolf Warehouse property, and the LePetomane III, Inc. Custodial Trust owns the undeveloped filled and marsh areas. The LePetomane III, Inc. Custodial Trust is the successor to NWI Land Management, Inc. following the discharge in bankruptcy of NWI's parent, Fruit of the Loom, Inc.

Previous Investigations/Actions

Beginning in the 1970s, the New Jersey Department of Environmental Protection (NJDEP) has overseen various site investigations including soil, groundwater, surface water, sediment and air quality. In 1984, the Superior Court of New Jersey issued the "Stipulation and Supplementary Order Approving Cooperative Agreement for Remedial Investigation and Feasibility Study and Amending Procedural Order Involving Remedy" in which Ventron and Velsicol agreed to investigate the site. The Stipulation was amended in 1996 to specify that Velsicol and Morton would conduct a RI/FS pursuant to an NJDEP-approved Scope of Work.

In 1990, the NJDEP performed a removal action for soil in residential areas of Wood-Ridge and Moonachie near the site. The removal actions were conducted at ten³ properties in Wood-Ridge and one property in Moonachie. The work included excavation of mercury-contaminated soil, placement of clean back-fill, revegetation, and general restoration of the properties to their original condition.

In 1996, Morton International, Inc. initiated further investigation of the site. After completing the RI/FS a proposed remediation plan was released for public comment in August 2006. The remedy for the upland portion of the site included excavation and off-site disposal of soil with greater than 620 parts per million (ppm) of mercury, capping of mercury-contaminated soil above NJDEP non-residential direct contact soil cleanup criteria, deed restrictions on properties with contamination greater than the NJDEP

³Number of residents potentially exposed (with an uncertainty factor of 2) = 4 X 10 X 2 = 80

residential soil cleanup criteria, and establishment of a clean buffer zone between capped areas and creeks or wetlands. The remedy also included a vertical hydraulic barrier system to serve as a physical barrier to groundwater flow and to encapsulate the areas of highest mercury concentrations under one of the warehouses and establishment of a Classification Exception Area and a Well Restriction Area. The remedy construction was completed in December 2010.

In May 2008, approximately 98 potentially responsible parties agreed to conduct a remedial investigation for the BCSA (i.e., USEPA's Operable Unit 2) which includes the waterways of the Barry's Creek, the tributaries and adjacent marsh areas⁴; field work began in May 2009. Data collection to characterize the site is planned through 2017.

Site Visit

On June 2, 2010, a site visit of the former Ventron/Velsicol site was conducted by representatives of the NJDOH and ATSDR. The USEPA Remediation Project Manager (RPM) briefly discussed the site history, location of the former manufacturing facility, dumping areas and the remedial activities conducted to date. The RPM also provided an update on the ongoing remediation work behind the warehouses (See Photographs 1 and 2).

The site includes buildings, pavement (in the developed portion of the site), upland vegetation, marsh vegetation, open water, debris, and localized patches of stressed vegetation. The site is fenced along the northern and western boundaries. Southern and eastern borders adjoin marsh and waterways. Human use of the site appears to be limited to transients and trespassers.

After the on-site areas, a number of off-site areas were also visited, including various accessible parts of Berry's Creek (see Photograph 3, 4 and 5) and the tide gate (see Photograph 6). The RPM also discussed the results of a survey of human activity patterns in the Berry's Creek. Direct observations by field crews and observations by cameras placed at four locations where human activity had been observed were used to conduct the survey. The results indicated that human activity is almost entirely limited to those locations where there is easy access to the waterway via relatively stable ground conditions. Fishing, bait fish capture, and crabbing appear to be the most common activities, but all at very low frequencies overall.

The potentially responsible parties have volunteered to provide and post signs warning about the fish consumption advisories.

Demographics

Using 2010 U.S. Census data, the population densities and the total populations of the three adjacent boroughs (Wood-Ridge, Carlstadt and Moonachie) are given in Figure 5.

⁴ The 12 acre "marsh" area of the site is a part of BCSA.

Past ATSDR and NJDOH Activities

In 1980, due to potential exposures to mercury by area residents through ingestion and inhalation, and at the request of the NJDEP, the NJDOH conducted a study to investigate exposure to mercury and its potential health impacts to residents in the community (NJDOH 1980). Two voluntary clinics were held where individuals filled out a questionnaire and submitted blood, urine and/or hair samples for analysis. Due to poor reproducibility, blood mercury levels were not used to assess mercury exposures. The number of individuals with urine mercury levels indicated is given below:

Number of individuals with urine mercury results

	<5 ppb	≥5 ppb	Total
Residents	106 (76.8%)	32 (23.2%)	138
Employees	96 (81.4%)	22 (18.6%)	118
Total	202 (78.9%)	54 (21.1%)	256

ppb=parts per billion

Activities including visiting the site, consuming local fish or wildlife, allowing pets to roam the site, and living in a house with basement flooding issues could not be associated with elevated urine mercury levels. The assessment did not find an immediate health hazard from mercury exposures related to the site; however, the elevated urine levels indicated low level exposures to mercury. The report also recommended construction of a fence to control or minimize exposure to contaminated soil and testing of the following individuals with the potential for extensive contact with the site:

- County Mosquito Control Commission personnel,
- children who continue to play on the site, and,
- Wood-Ridge firemen who were frequently called to extinguish brush fires of burning phragmites.

A follow up of fifteen firemen did not show any significant exposure to mercury via mercury vapor or dust blowing off the site (NJDOH 1980).

In 1989, the ATSDR conducted a preliminary Public Health Assessment of the site (ATSDR 1989). Ingestion, direct contact with groundwater, surface water, soil, sediment, inhalation of volatilized contaminants and consumption of bioaccumulated contaminants associated with recreational fishing were identified as the potential human exposure pathways. The site was considered to be of public health concern due to ongoing contaminant exposures to area residents and trespassers. The recommendation included restriction of access to the site in order to limit exposures to area residents and trespassers and characterization of the site and site contaminants.

In July 1991, the ATSDR evaluated the potential worker and customer exposure to mercury at the two warehouses located at the site (ATSDR 1991). Access to the site was partially restricted; the wooded area between the warehouses and Berry's Creek was fenced and marked with a danger sign. However, the site was accessible through a hole in

the fence. Air monitoring was conducted during the site visit using a portable instrument. Based on the information reviewed, the ATSDR concluded that the contamination at the Ventron/Velsicol site may pose a potential health threat to workers and area residents from the inhalation of mercury vapors and mercury-contaminated dusts. The report recommended indoor (i.e., warehouses) and ambient (i.e., residential areas) air monitoring.

The ATSDR conducted a site review and update of the site in 1993 (ATSDR 1993). The main community health concerns identified were the potential health effects of mercury which migrated off-site into the residential homes and municipal repair yard (at the location of a former wastewater treatment plant area). The Ventron/Velsicol site was considered to be an indeterminate public health hazard until environmental data became available for review and evaluation. The report recommended on- and off-site ambient air monitoring and a Public Health Assessment to characterize the contaminant exposure pathways.

In 1995, the ATSDR conducted a health consultation to evaluate the public health significance of (a) exposures to mercury contaminated soils in the residential homes, (b) exposures to workers at the municipal repair yard and the wastewater treatment plant, and, (c) consumption of contaminated biota from the Berry's Creek (ATSDR 1995a). The residential homes were located approximately 600 feet to the north of the site in the Boroughs of Moonachie and Wood-Ridge. The residences were constructed on the reclaimed wetlands after the mercury recovery facility began operation at the site and are believed to have been contaminated by tidal flooding from Berry's Creek, airborne transport, or use of contaminated soils for fill. An evaluation of adult and children exposure doses (calculated using worst case exposure scenario) did not indicate any potential for adverse non-cancer health effects. Contaminant exposure doses for children with soil pica⁵ tendency were also evaluated; adverse renal health effects were found to be possible. For workers (municipal repair yard and wastewater treatment plant), two potential exposure pathways were identified: the inhalation of mercury vapor and ingestion of mercury contaminated soil. Based on the calculated exposure doses, it was unlikely that adverse health effects would occur. The biota pathway was evaluated using the data collected from four edible species (i.e., carp, white perch, blue claw crab and brown bullhead catfish). The mean concentration of mercury detected in the sediment from Berry's Creek ranged from 100 to 10,000 parts per million (ppm) (ATSDR 1995a). The report concluded that while individual consumption practices may lead to significant variability in mercury ingestion, frequent ingestion of Berry's Creek biota may result in exposures at levels of public health concern. The report also recommended posting of warning signs about the hazards associated with consuming fish from Berry's Creek.

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

⁵Soil pica is a medical disorder characterized by an appetite for soil.

concentrations of detected substances are compared to media-specific environmental guideline comparison values (CVs). If concentrations exceed the environmental guideline CVs, these substances, referred to as Contaminants of Potential Concern (COPCs), are selected for further evaluation. If contaminant levels are found above environmental guideline CVs, it does not mean that adverse health effects are likely, but that a health guideline comparison is necessary to evaluate site-specific exposures. Once exposure doses are estimated, they are compared with health guideline CVs to determine the likelihood of adverse health effects.

Environmental Guideline Comparison

There are a number of CVs available for screening environmental contaminants to identify COPCs (ATSDR 2005). 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) are considered as CVs. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million persons exposed during their lifetimes (70 years).

In the absence of an ATSDR CV, CVs from other sources may be used to evaluate contaminant levels in environmental media. These include New Jersey Maximum Contaminant Levels (NJMCLs) for drinking water, USEPA MCLs for drinking water and USEPA Regional Screening Levels (RSLs). RSLs are contaminant concentrations corresponding to a fixed level of risk (i.e., a Hazard Quotient⁶ of 1, or lifetime excess cancer risk of one in one million, or 10^{-6} , whichever results in a lower contaminant concentration) in water, air, biota, and soil (USEPA 2011a). For soils and sediments, CVs also include the NJDEP Residential Direct Contact Soil Remediation Standards (RDCSRS), Ingestion-Dermal Health Based Criteria (IDHBC) and Inhalation Health Based Criteria (IHBC). Based primarily on human health impacts, these criteria also take into account natural background concentrations, analytical detection limits, and ecological effects (NJDEP 2011).

Substances exceeding applicable environmental guideline CVs were identified as COPCs and evaluated further to determine whether these contaminants pose a health threat to exposed or potentially exposed receptor populations. In instances where an environmental guideline CV or toxicologic information is unavailable, the substance may not be retained for further evaluation.

⁶The ratio of estimated site-specific exposure to a single chemical from a site over a specified period to the estimated daily exposure level at which no adverse health effects are likely to occur.

Site Conditions

Topography: The topography across most of the site is generally flat, ranging in elevation from 0 to 12.6 feet above mean sea level (msl). The 38-acre site is divided into three distinct areas: the 7-acre developed area, the 19-acre undeveloped filled area, and the 12-acre marsh area. The developed and undeveloped areas have been designated as Operable Unit 1 (OU1) and the remainder of the site has been designated as OU2.

Two warehouses occupy most of the developed area of the site. The rest of the developed area is covered by asphalt-paved surfaces, railroad tracks, and the ditch along the southwest side. The undeveloped filled area had a small basin⁷. This basin may have been a remnant of a settling basin for discharges from the plant area or the Randolph Products property (Exponent 2004). The north and west perimeters of the area are fenced; additional fencing to the east prevents site access via the tide gate. The marsh areas mostly contain phragmites. A small drainage ditch extends lengthwise through the middle of the marsh area all the way to Nevertouch Creek. At high tide, much of the marsh area is completely submerged. At low tide, the water drains, exposing sloping stream banks.

Geology: The site is located in the Newark Basin, which contains sedimentary rock consisting of primarily sandstone and shale and layered with igneous rocks. Based on previous geotechnical studies, the geologic units at the site are as follows (increasing with depth):

- Fill material, which was placed in the entire on-site area that was previously marsh. The fill thickness in the developed and undeveloped area ranges from approximately 5 to 8 feet and 3 to 14 feet, respectively.
- Meadow mat, consisting of fibrous organic peat and silt, which, if present, ranges from 0.5 to 4 feet thick.
- Fine to medium-grained sand, approximately 5 to 10 feet thick.
- Gray to red-brown silt, approximately 62 to 146 feet thick.
- Red-brown silty sand, at least 20 feet thick.
- Bedrock, consisting of reddish-brown shale, siltstone, and sandstone within the Passaic Formation.

Site Hydrogeology: Groundwater is present on the site at depths ranging from approximately 2 to 8 feet below ground surface (bgs) and generally flows to the south, toward Berry's Creek (Exponent 2004). The groundwater hydraulic gradients appear to be relatively flat over much of the site.

⁷ The basin has been eliminated as part of remediation.

Surface Water Hydrology: Surface runoff at the site is generally to the southeast, where Berry's Creek borders the site. In the developed area, the direction of surface runoff is toward the drainage ditch southwest of the existing warehouses. This ditch then flows along the West Ditch toward the Diamond Shamrock/Henkel (north) Ditch. In the undeveloped area, there are no well-defined drainage patterns. Runoff from the area flows toward the West Ditch and toward the Diamond Shamrock/ Henkel (north) Ditch. The Diamond Shamrock/Henkel (north) Ditch flows in a southeasterly direction into Berry's Creek.

On-site Contamination

Soil/Sediment: The Remedial Investigation (RI) for the site was conducted by Exponent (2004) on behalf of Morton International, Inc. The RI included collection of environmental samples from areas identified as the developed area, the undeveloped filled area, and the marsh area (see Figure 2). Samples were analyzed for Volatile Organic Compounds (VOCs), Semi-volatile Organic Compounds (SVOCs) and metals. Soil sampling results obtained during monitoring well installation and during the warehouse evaluation study were also included in the dataset.

Developed Area Soil: Up to 15 samples (0 to 2 feet depth⁸) were collected from the developed area soil (see Table 1). The number of analyses, number of non-detects, range and mean of the contaminants detected in the soil (0 to 2 feet depth) are presented in Table 1. Maximum concentrations of total mercury, aluminum, chromium, copper, thallium, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene detected in soil exceeded their respective environmental guideline CVs; they are considered as the COPCs for the developed area soil.

Undeveloped/Marsh Area Soil/Sediment: Up to 25 samples (0 to 2 feet depth) were collected from the undeveloped area soil (see Table 2). Maximum concentrations of total mercury, aluminum, antimony, arsenic, cadmium, chromium, lead, manganese, thallium, zinc, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[ghi]perylene, bis[2-ethylhexyl]phthalate, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene detected in soil exceeded their respective environmental guideline CVs; they are considered as the COPCs for the undeveloped area soil.

The contaminants in the sediment of onsite basin and the West Ditch located within the undeveloped area were also evaluated. Maximum concentrations of mercury, arsenic, chromium, lead, Aroclor 1260, benzo[a]anthracene, benzo[a]pyrene and dibenz[a,h]anthracene detected in the sediment of the onsite basin (see Table 3) and mercury, arsenic, cadmium, chromium, benzo[a]anthracene, benzo[a]pyrene and dibenz[a,h]anthracene detected in the sediment of the West Ditch (see Table 4) exceeded their respective environmental guideline CVs; they are considered as the COPCs for the onsite basin and the West Ditch.

⁸The ATSDR considers samples collected from 0-3" depth as surface soil

Sediment samples were also collected from marsh area; sediment is defined as the upper 0 to 15 centimeters (cm) of sediment. Maximum concentrations of mercury, arsenic, cadmium, chromium, lead, benzo[a]anthracene, benzo[a]pyrene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene detected in sediment exceeded their respective environmental guideline CVs (see Table 5); they are considered as the COPCs for the marsh area located within the undeveloped area.

Subsurface Soil: Subsurface soil samples were collected from depths of 1 to 20 feet below ground surface from the developed and undeveloped areas. Maximum concentrations of mercury, antimony, arsenic, cadmium, chromium, lead, manganese, thallium, vanadium, zinc, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene and dibenz[a,h]anthracene detected in the subsurface soil exceeded their respective environmental guideline CVs (see Table 6); they are considered as the COPCs for the onsite subsurface soil.

Groundwater: On-site groundwater samples were collected from monitoring wells located in the developed and undeveloped filled areas. Maximum concentrations of mercury, arsenic, cadmium, manganese, thallium, benzene and toluene detected in the groundwater exceeded their respective environmental guideline CVs (see Table 7); they are considered as the COPCs for the groundwater.

The area residential homes are provided with public water supplies (ATSDR 1993).

Surface Water: Surface water samples were collected from the onsite basin, west ditch and the marsh area/Berry's Creek. Whole (unfiltered) and filtered water samples (for dissolved metals) were analyzed. Since unfiltered water better represents the potential for exposure from surface water, only the unfiltered concentrations were evaluated for identifying COPCs. Maximum concentrations of mercury, arsenic, lead thallium and 1,1,2,2-tetrachloroethane detected in the surface water exceeded their respective environmental guideline CVs (see Table 8); they are considered as the COPCs for the surface water.

Ambient Air: In 1978, at the request of the NJDEP, an extensive on-site mercury monitoring was conducted by the USEPA's Environmental Monitoring and Surveillance Laboratory (Research Triangle Park, NC). Ambient mercury levels were measured for a period of five days at various locations (see Figure 6). Air samples were collected at 8, 12 and 24 hour intervals. The weather conditions during the sampling were for the most part hot and humid; however, the soil was quite wet due to a severe thunderstorm that occurred during sampling.

The results were reported as elemental mercury. The concentration of mercury detected ranged from 0.29 to 3.3 $\mu\text{g}/\text{m}^3$. The average concentrations at sites 2, 3 and 4 (see Figure 6) during the five-day sampling period were 0.76, 1.03 and 1.5 $\mu\text{g}/\text{m}^3$, respectively.

In July 1991, the ATSDR evaluated the potential worker and customer exposure to mercury at the two warehouses located at the site (ATSDR 1991). Air monitoring was conducted using a portable instrument (Jerome meter). Mercury was detected at a concentration of 20 $\mu\text{g}/\text{m}^3$ at a point along the railroad tracks adjacent to the fence and 40 $\mu\text{g}/\text{m}^3$ at an exhaust vent on the foundation of the furniture distribution center located at 3 Ethel Boulevard.

Maximum concentrations of air mercury levels detected in the ambient air exceeded the environmental guideline CV (0.2 $\mu\text{g}/\text{m}^3$); mercury is considered as the COPC for the on-site ambient air in the past.

The NJDEP (1991) conducted two rounds of air sampling in the vicinity of the site (Exponent 2004) in June, 1989, and October, 1990. Particulate and gaseous mercury samples were collected from six locations in the vicinity of the site (three samples were within the site boundaries) and one background location. Only one of these samples, a gaseous mercury sample located near the gate entrance to the undeveloped filled area, had a concentration above detection limits (0.72 $\mu\text{g}/\text{m}^3$); the level also exceeded the environmental guideline CV for mercury (0.2 $\mu\text{g}/\text{m}^3$).

In 1997, air samples were collected during the warehouse evaluation study (Exponent 2004). Particulate mercury concentrations ranged from 0.0001 to 0.0017 $\mu\text{g}/\text{m}^3$ (see Figure 7). The highest concentration was in the Wolf Warehouse. The lowest mercury particulate levels were in the U.S. Life Warehouse and at a location between the two warehouses. Gaseous mercury sampling was conducted concurrently with the particulate mercury sampling. Gaseous mercury concentrations ranged from 0.0014 to 0.0606 $\mu\text{g}/\text{m}^3$ (see Figure 7). The highest concentration was within the undeveloped filled area.

Since 1997, mercury levels detected in the ambient air did not exceed the environmental guideline CVs (0.2 $\mu\text{g}/\text{m}^3$); mercury is not considered a COPC for the on-site ambient air at present and in the future.

Off-site Contamination

Off-site Properties: The locations referred to as “off-site properties” consist of the following properties: the Blum Property, the Prince Packaging property, the EJB property, the Lin-Mor property, Ethel Boulevard and the railroad property. During the time when the mercury processing plant was in operation, the adjacent areas were wooded and marshland and were not developed (see Figure 4). Soil sampling results from these areas are presented in Table 9. Maximum concentrations of mercury, lead, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene detected in the soil exceeded their respective environmental guideline CVs (see Table 9); they are considered as the COPCs for the off-site area soil.

Residential Properties: Residential properties are located approximately 600 feet to the north of the site (see Figure 2). Twenty-four properties were identified for soil sampling. The maximum and mean concentration of mercury detected in the soil was 60 ppm and 10 ppm, respectively. Results (0 to 12 inches depth) indicated that nine properties were contaminated at levels exceeding the NJDEP Residential Direct Contact Soil Remediation Standard (RDCSRS); mercury is considered as the COPC for the residential properties.

Municipal Repair Yard/Wastewater Treatment Plant: Ambient air mercury concentrations were measured using a portable instrument (Jerome monitor) during a site visit (ATSDR 1993). The maximum ambient air mercury concentration detected at the border of the municipal repair yard/wastewater treatment plant was $40 \mu\text{g}/\text{m}^3$. The maximum concentration of mercury detected in the soil of the municipal repair yard was 600 ppm. Maximum concentrations of ambient air mercury levels detected in the ambient air and soil exceeded environmental guideline CVs ($0.2 \mu\text{g}/\text{m}^3$ and 14 ppm, respectively); mercury is considered as the COPC for the off-site ambient air and soil.

Contaminants of Potential Concern: Summary

The following contaminants detected in the environmental media are designated as the COPCs for the Ventron/Velsicol site:

Location/ Media	Metals	VOCs/SVOCs
On-site		
Developed Area Soil	Mercury, aluminum, chromium, copper and thallium	Benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene
Undeveloped Area Soil/Sediment	Mercury, aluminum, antimony, arsenic, cadmium, chromium, lead, manganese, thallium and zinc	Benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, bis[2-ethylhexyl]phthalate, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene
Subsurface Soil	Mercury, antimony, arsenic, cadmium, chromium, lead, manganese, thallium, vanadium and zinc	Benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene and dibenz[a,h]anthracene
Groundwater	Mercury, arsenic, cadmium, manganese and thallium	Benzene and toluene
Surface Water	Mercury, arsenic, lead and thallium	1,1,2,2-Tetrachloroethane
Ambient Air	Mercury	
Off-site		
Soil (adjacent areas)	Mercury and lead	Benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene
Soil in Residential Homes	Mercury	
Soil and Ambient Air in Municipal Repair Yard/WWTP	Mercury	

Discussion

The method for assessing whether a health hazard exists to a community is to determine whether there is a completed or potentially completed exposure pathway from a contaminant source to a receptor population and whether exposures to contamination are high enough to be of health concern (ATSDR 2005). Site-specific exposure doses are calculated and compared with health guideline CVs.

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 mechanisms;
3. point of exposure;
4. route of exposure; and
5. receptor population.

Generally, the ATSDR considers three exposure pathway 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, one or more of the elements is absent. 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.

The ATSDR generally considers dermal exposures to be a minor contributor to the overall exposure dose relative to the contribution of ingestion and inhalation exposures (ATSDR 2005). As such, dermal exposure will not be evaluated in this assessment.

Based on sampling data, results and knowledge of accessibility to contaminated area, the following exposure pathways for individuals who live (or lived) near the site were identified.

Completed Pathways

Incidental ingestion of contaminated soil and sediment from on-site (developed, undeveloped and marsh) areas (past). On-site soils (0 to 2 feet depth) and sediment were contaminated with VOCs, PAHs and metals. In the past, the site was not fenced and area residents frequently visited the site for recreation (NJDOH 1980). Residents, including

children, were exposed to contaminants while engaging in outdoor recreational activities. This scenario includes site visitors and trespassers.

The most likely individuals who visited the site (developed, undeveloped and marsh area) for recreation were adults and older children (i.e., 6 to 18 years old) who live (or lived) near the site. Younger children were not expected to visit the on-site areas given the distance of the nearest residence from the site.

Incidental ingestion of contaminated soil from off-site (the Blum Property, the Prince Packaging property, the EJB property, the Lin-Mor property, Ethel Boulevard and the railroad property) areas (past): Off-site soils (0 to 2 feet depth) were contaminated with site-related contaminants. In the past, the adjacent off-site areas were wooded and undeveloped, and area residents frequently visited these adjacent areas (NJDOH 1980). Residents, including children, were exposed to contaminants while engaging in outdoor recreational activities.

Incidental ingestion of contaminated soil from off-site (residential properties, municipal repair yard and wastewater treatment plant) areas (past): The residential properties are located approximately 600 feet to the north of the site (see Figure 2). The contaminated properties were remediated by excavating and removing all soils exhibiting mercury concentrations exceeding 14 ppm (ATSDR 1995a). A health evaluation of adult and children exposed to mercury-contaminated soil in backyard soils did not indicate the potential for any adverse health effects.

The municipal repair yard and the former wastewater treatment plant were located to the north of the site (see Figure 2). Mercury was detected in the soil of the municipal repair yard and wastewater treatment plant.⁹ An evaluation of mercury exposure doses did not indicate any non-cancer adverse health effects (ATSDR 1995a).

Incidental ingestion of surface water (past): Area residents visited the site frequently for recreational purposes. Therefore, contaminant exposures through incidental ingestion of surface water were assumed to have occurred in the past.

Inhalation of ambient air (past): Air sampling indicated that the on-site ambient air was contaminated with mercury vapor. Inhalation of ambient air was an exposure pathway for the employees of the businesses that occupied the site after 1974 and residents who visited the site.

Ingestion of contaminated biota from off-site areas (past): Contamination of Berry's Creek with mercury, PCBs and other contaminants has been historically documented since the late 1920s (ATSDR 1995a). Therefore, contaminant exposures through ingestion of biota have occurred in the past.

⁹Mercury was also detected in the ambient air of the municipal repair yard and wastewater treatment plant. An evaluation of the levels detected did not indicate the potential for non-cancer adverse health effects (ATSDR 1995).

A summary of completed exposure pathways identified for the site is presented in Table 10.

Potential Pathways

Ingestion of contaminated biota from off-site areas (present, future): Mercury, PCBs and other contaminants were detected in the water, soil and sediment of Berry's Creek and adjacent areas. Under the oversight of EPA, the remedial investigation of Berry's Creek is being conducted by the potential responsible parties.

The NJDOH, in cooperation with the ATSDR, will prepare a separate health consultation to evaluate contaminant exposures associated with the consumption of biota from Berry's Creek.

Eliminated Pathways

Incidental ingestion of contaminated on-site soil and sediment (developed, undeveloped and marsh) area (present and future): As summarized earlier in the report, the preferred remedy for the site was completed in November 2010. The remedy included excavation and off-site disposal of soil, capping of mercury-contaminated soil, construction of a vertical hydraulic barrier system, establishment of a Classification Exception Area and a Well Restriction Area, and deed restrictions. Therefore, the pathways associated with the incidental ingestion of soil and sediment from on-site areas is considered eliminated.

Ingestion of contaminated biota from on-site areas (past, present, future): Although it is possible the on-site plants may be contaminated by site-related contaminants, the exposure pathways are considered incomplete because no edible plants were noted during site visit. The on-site surface water bodies (i.e., the West Ditch and the onsite basin) were determined not to support fish or other aquatic organisms that would be consumed by people. Therefore, this pathway is considered eliminated.

Incidental ingestion of contaminated groundwater (past, present and future): The residential homes located near the site (north-east and north-west) are provided with public water supplies (ATSDR 1993). Therefore, the groundwater ingestion pathway is considered eliminated.

Groundwater at the site generally flows to the south, toward Berry's Creek (Exponent 2004). Therefore, the soil vapor intrusion pathway is also considered eliminated.

Inhalation of on- and off-site ambient air (present and future): As discussed earlier, since 1997, the ambient air mercury concentration results were below the allowable levels. As such, the present and future on- and off-site ambient air pathways are considered eliminated.

Exposure Point Concentration

The exposure point concentration (EPC), or the concentration term in the exposure equation, is derived to reflect a representative concentration at the exposure point or points over the exposure period (EPA 1989). Consistent with guidance from ATSDR (2005), the 95% upper confidence limit (UCL) was used to estimate the EPC. Where the 95% UCL¹⁰ was greater than the maximum value, the maximum concentration was applied.

Public Health Implications

Once it has been determined that individuals have or are likely to come in 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 is called a health guideline comparison, which involves looking more closely at site-specific exposure conditions, the estimation of exposure doses, and the evaluation with health guideline comparison values (CVs). Health guideline CVs are based on data drawn from epidemiological/toxicological literature and often include uncertainty or safety factors to ensure that they are amply protective of human health.

Non-Cancer Health Effects

To assess the possibility of 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 through the use of statistical models. In toxicological literature, effect levels include:

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

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

¹⁰ EPC calculations were conducted using EPA's ProUCL software (EPA 2007).

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 comparison values such as USEPA Reference Dose (RfD) may be used. The RfD 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.

Incidental ingestion of contaminated soil and sediment from on-site areas (past)

In the past, the manufacturing buildings occupied a small area near the northwest portion of the site; the remainder of the site was vacant and was not fenced; the fence on the west and north side of the site was erected in 1979 (NJDEP ca.1980). The area residents accessed the site routinely for recreational purposes. Exposures are based on incidental ingestion of contaminated soil; non-cancer exposure doses were calculated using the following formula:

$$\text{Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW}$$

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;

C = concentration of contaminant in soil (mg/kg);

IR = soil ingestion rate (kg/day);

EF = exposure factor representing the site-specific exposure scenario; and,

BW = body weight (kg)

In the past, the mercury processing plant and the waste disposal areas were not fenced; the area around the plant was mostly undeveloped and wooded (see Figure 4). It is likely that prior to the erection of the security fence in 1979, area residents visited the site for recreational purposes (e.g., fishing, crabbing). The most likely visitors/trespassers were adults and older children (i.e., 6 to 18 years old). Younger children would not be expected to trespass within the area, given the distance between the site and the nearest homes. In this assessment, visitors/trespassers were assumed to be exposed either to soil, sediment or surface water on a given visit. This assessment used 130 visits per year in the on- and off-site areas. This was derived by NJDEP assuming approximately 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001).

Developed Area: Based on the estimated exposure point concentrations (EPCs), the non-cancer exposure doses calculated for arsenic, chromium, copper and thallium for children and adults were lower than the corresponding health guideline CVs; as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects.

Based on the EPC of mercury detected in the soil (0 to 2 feet depth), prior to the remediation of the site (see Table 11), the chronic exposure doses calculated for children and adults exceeded the RfD for mercuric chloride. The health guideline CVs of benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer adverse health effects associated with exposures to mercury and PAHs are evaluated as follows:

Mercury: Mercury is a naturally occurring metal which exists in the environment in several forms (ATSDR 1999a). Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or “salts”. 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. Environmental exposure to mercury occurs from breathing contaminated air, ingesting contaminated water and food. Mercury, at high levels, may damage the brain, kidneys, and developing fetus.

Since a chronic oral MRL and RfD are unavailable for mercury, the calculated exposure dose for children and adults was compared to the health guideline CV for intermediate exposures (see Table 11). The oral MRL for mercury (0.002 mg/kg/day) for intermediate exposures is based on increased kidney weight of rats exposed to mercuric chloride once every five days for twenty-six weeks (ATSDR 1999a). An uncertainty factor of 100 and a NOAEL of 0.23 mg/kg/day were used to calculate the MRL. Since the child exposure dose (i.e., 0.0041 mg/kg/day) was about 56 times lower than the NOAEL for intermediate exposures, no intermediate adverse health effects are likely from past exposures.

In order to assess the chronic exposures, the estimated mercury exposure doses for children (0.0041 mg/kg/day) and adults (0.00051 mg/kg/day) were expressed as mercuric chloride by multiplying the exposure dose with appropriate molecular weights, i.e., $0.0041 \times 271/200 = 0.0055$ mg/kg/day and $0.00051 \times 271/200 = 0.0007$ mg/kg/day, respectively. The chronic exposure doses exceeded the RfD of 0.0003 mg/kg/day for mercuric chloride. The chronic oral RfD for mercuric chloride is based on autoimmune effects (USEPA 2011b). An uncertainty factor of 1000 and a LOAEL of 0.317 mg/kg/day were used to calculate the oral RfD (0.0003 mg/kg/day). The child and adult exposure doses (i.e., 0.0055 mg/kg/day and 0.0007 mg/kg/day) were 57 and 450 times lower than the LOAEL, respectively (see Table 12). As such, the risk of non-cancer adverse health effects for past exposures to mercury detected in soil is considered low but possible.

PAHs: PAHs are a class of over 100 different compounds that are found in and formed during incomplete combustion of coal, oil, wood, or other organic substances (ATSDR 1995b). More commonly they are found in petroleum-based products such as coal tar, asphalt, creosote, and roofing tar. In the environment, PAHs are found as complex mixtures of compounds, and many have similar toxicological effects and environmental fate. Because combustion processes produce them, PAHs are widespread

in the environment. PAHs have been found to exhibit anti-androgenic¹¹ properties in human cell cultures and are implicated in the loss of fertility in males (Kizu 2003). Non-cancer adverse health effects associated with PAH exposures have been observed in animals but generally not in humans (ATSDR 1995b). Non-cancer effects are usually seen at much higher levels than found in the environment. The main potential concern for PAH exposures is for cancer effects.

The chronic past exposure doses for children and adults were calculated based on the EPC of PAHs detected in the soil (see Table 11). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available and are shown below:

Reference Dose for Chronic Oral Exposure			
PAH	NOAEL (mg/kg/day)	RfD (mg/kg/day)	Health Effect
Acenaphthene	175	0.06	Hepatotoxicity
Anthracene	1,000	0.3	No observed effect
Fluoranthene	125	0.04	Nephropathy, increased liver weights, hematological alterations, and clinical effects
Fluorene	125	0.04	Decreased red blood count, packed cell volume and hemoglobin
Naphthalene	71	0.02	Decreased mean terminal body weight in males
Pyrene	75	0.03	Kidney effects (renal tubular pathology, decreased kidney weights)

The RfDs of these PAHs are based on the NOAEL and are much higher than the exposure doses calculated for the PAHs detected in the developed area. Based on the 95% UCL of arithmetic mean of benzo[b]fluoranthene (the PAH with the highest concentration), the calculated chronic child exposure dose (0.000003 mg/kg/day) was about 6,666 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table 11).

As such, non-cancer adverse health effects associated with past exposures from the ingestion of PAH contaminated soil at the Ventron/Velsicol site are unlikely in children and adults.

Undeveloped Area: Based on the estimated EPCs, the non-cancer exposure doses calculated for antimony, arsenic, cadmium, chromium, manganese, thallium, zinc and bis[2-ethylhexyl]phthalate for children and adults were lower than the corresponding health guideline CVs (see Table 12); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. Based on the EPC of mercury

¹¹Antiandrogenic substances block the action of androgens, the hormones responsible for male characteristics.

detected in the soil (0 – 2 feet depth) (see Table 12), the chronic exposure doses calculated for children exceeded the health-based CVs; the chronic exposure dose calculated for adults for mercury were below the health-based CVs. The mean concentration of lead detected in the soil was above the health guideline CVs. The health-based CVs of benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; the PAHs were retained for further evaluation. The non-cancer adverse health effects associated with exposures to lead, mercury and PAHs are evaluated as follows:

Lead: Accumulation of lead in the body can cause damage to the nervous system, gastrointestinal system, kidneys, and red blood cells (ATSDR 2006). Children, infants, and fetuses are the most sensitive populations. Lead may cause learning difficulties and stunted growth, or may endanger fetal development. Health effects associated with lead exposure, particularly changes in children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold (i.e., no NOAEL or LOAEL is available). It should be noted there is no known safe level of lead. The mean concentration of lead detected in the on-site areas (800 mg/kg) exceeded the NJDEP RDCSCC of 400 mg/kg.

Lead exposures associated with the intermittent recreational use of lead-contaminated on-site areas were evaluated using the USEPA's integrated exposure uptake biokinetic (IEUBK) model (USEPA 1994a). The IEUBK model estimates a plausible distribution of blood lead levels centered on the geometric mean blood lead levels from available exposure information. Blood lead levels are indicators of exposure, and are also the most widely used index of internal lead body burdens associated with potential health effects. The model also calculates the probability that children's blood lead levels will exceed a level of concern. In using the IEUBK model, the USEPA recommends that the lead concentration in site soil does not result in a 5% probability of exceeding a blood lead concentration of 10 µg/dL (USEPA 1986; CDC 1991; USEPA 1994a; 1994b). The UCL of the arithmetic mean of lead levels in soils (0 to 2 feet depth) was used as an input value to calculate expected children's blood lead levels due to incidental ingestion. The assumptions for the recreational exposure scenario for children aged 72 to 84 months are as follows:

1. Children were exposed to soil containing lead each time the former facility areas were visited.
2. Model default values were used for all other variables (USEPA 2002) including residential soil and dust.

It should be noted that on January 4, 2012, CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) recommended that CDC adopt the 97.5 percentile for children one to five years old as the reference value for designating elevated blood lead levels in children. The 97.5 percentile currently is 5 µg/dL (CDC 2012a). On June 7, 2012, the CDC released a statement indicating concurrence with the

recommendations of the ACCLPP (CDC 2012b). The EPA is evaluating the impact of the new recommended blood lead levels on the remediation standards.

The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 5 µg/dL (P₅) for children are shown in the following table:

Exposure Scenario		
Age (months)	Four Site Visits Per Week ^a	
	Blood Lead Level ^b (µg/dL)	P ₅ (%) ^c
72 - 84	5.6	60

^aWeighted soil lead concentration (800 ppm x 4/7 + 200 ppm x 3/7) = 540 ppm (USEPA 2003a);

^bGeometric mean lead levels in blood; ^cprobability of blood lead level > 5 µg/dL

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 72 to 84 months¹² were slightly above the reference value (5 µg/dL); the probabilities of blood lead levels exceeding 5 µg/dL for children ages 72 to 84 months exceeded 5 percent. Therefore, potential for adverse health effects associated with lead exposures is considered possible.

An adult blood lead model estimated a geometric mean blood lead level among adult workers and 95th percentile of blood lead level among fetuses as 2.3 µg/dL and 6.9 µg/dL, respectively (USEPA 2003b). As such, potential for adverse health effects to fetuses associated with lead exposures at the Ventron/Velsicol site was considered possible.

Mercury: As mentioned earlier, since a chronic oral MRL or RfD are unavailable for mercury, the calculated exposure dose was compared to health guideline CV for intermediate exposures (see Table 12). The child exposure dose (i.e., 0.0006 mg/kg/day) was about 3 times lower than the MRL for intermediate exposures (0.002 mg/kg/day); as such, no intermediate adverse health effects are likely (ATSDR 1999a).

In order to assess the chronic exposures, the estimated mercury exposure doses for children (0.00065 mg/kg/day) were expressed as mercuric chloride, i.e., 0.00065*271/200 = 0.00088 mg/kg/day (ATSDR 1999a). The chronic exposure doses exceeded the RfD of 0.0003 mg/kg/day. An uncertainty factor of 1,000 and a LOAEL of 0.317 mg/kg/day were used to calculate the oral RfD (0.0003 mg/kg/day). The child exposure dose (i.e., 0.00088 mg/kg/day) was about 360 times lower than the LOAEL (see Table 12). As such, the risk of non-cancer adverse health effects for exposures to mercury detected in soil is considered unlikely.

PAHs: Using the same approach described earlier, based on the 95% UCL of arithmetic mean of benzo[b]fluoranthene (the PAH with the highest concentration), the

¹²The most likely individuals who visited the site (developed, undeveloped and marsh area) for recreation were adults and older children (i.e., 6 to 18 years old) who live (or lived) near the site. Younger children were not expected to visit the on-site areas given the distance of the nearest residence from the site.

calculated chronic child exposure dose (0.00001 mg/kg/day) was 2,000 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table 12) (ATSDR 1995b). As such, non-cancer adverse health effects associated with past exposures from the ingestion of PAH contaminated soil from undeveloped area at the Ventron/Velsicol site are unlikely in children and adults.

Marsh Area: The non-cancer exposure doses calculated for arsenic and cadmium for children and adults were lower than the corresponding health guideline CVs (see Table 13); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. Based on the EPC of chromium and mercury detected in the soil (0 to 2 feet depth) (see Table 13), the chronic exposure doses calculated for children exceeded the health-based CVs; the chronic exposure doses calculated for adults were below the health-based CVs. The health guideline CVs of benzo[a]anthracene, benzo[a]pyrene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable. The non-cancer adverse health effects associated with exposures to chromium, mercury and PAHs are evaluated as follows:

Chromium: Chromium may occur in several forms; in nature, chromium (III) is much more common than the more toxic chromium (VI) (ATSDR 2005). Chromium measured in the soil was reported as total chromium. Since the form of chromium in soil is a function of source materials and environmental conditions, to be conservative, the total chromium was assumed to be in the more toxic chromium (VI) form. It should be noted, however, that this assumption may result in an overestimation of exposure dose and potential for health effects.

The chronic oral RfD for chromium (VI) (0.003 mg/kg/day) is based on water consumption in a group of male and female rats (USEPA 2011b). An uncertainty/modifying factor of 900 and a NOAEL (i.e., the dose that showed no effect in animal studies) of 2.5 mg/kg/day were used to calculate the oral RfD. Based on the EPC of chromium detected in the soil, the child exposure doses (i.e., 0.004 mg/kg/day) were 625 times lower than the NOAEL (see Table 13). Based on the fact that the RfD is based on NOAEL and all the chromium detected was assumed to be in the chromium (VI) form, the non-cancer adverse health effects for exposures by ingestion to chromium detected in soil is considered unlikely.

Mercury: As mentioned earlier, since a chronic oral MRL or RfD are unavailable for mercury, the calculated exposure dose was compared to health guideline CV for intermediate exposures (see Table 12). The child exposure dose (i.e., 0.001 mg/kg/day) was lower than the MRL for intermediate exposures (0.002 mg/kg/day); as such, no intermediate adverse health effects are likely (ATSDR 1999a).

In order to assess the chronic exposures, the estimated mercury exposure doses for children (0.001 mg/kg/day) were expressed as mercuric chloride, i.e., $0.001 \times 271/200 = 0.0013$ mg/kg/day. The chronic exposure doses exceeded the RfD of 0.0003 mg/kg/day (ATSDR 1999a). An uncertainty factor of 1,000 and a LOAEL of 0.317 mg/kg/day were used to calculate the oral RfD (0.0003 mg/kg/day). The child exposure dose (i.e.,

0.0013 mg/kg/day) was about 233 times lower than the LOAEL (see Table 13). As such, the risk of non-cancer adverse health effects for exposures to mercury detected in soil is considered unlikely.

PAHs: Using the same approach described earlier, based on the mean concentration of benzo[a]pyrene (the PAH with the highest concentration), the calculated chronic child exposure dose (0.0000024 mg/kg/day) was about 8,000 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table 13). As such, the risk of non-cancer adverse health effects associated with past exposures from the ingestion of PAH contaminated soil from marsh area at the Ventron/Velsicol site is unlikely in children and adults.

Incidental ingestion of contaminated surface water from on-site areas (past)

Residents, including children, could have been exposed to surface water contaminants during outdoor recreational activities at the site. In order to assess exposures from incidental ingestion of surface water (from on-site basin, west ditch or marsh area), an exposure dose was calculated using the following formula:

$$\text{Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW}$$

where mg/kg/day = milligrams of contaminant/kilogram of body weight/day;
 C = concentration of contaminant in water (mg/L);
 IR = ingestion rate (L/day);
 EF = exposure factor representing the site-specific exposure scenario; and,
 BW = body weight (kg).

As discussed earlier, the following exposure assumptions (USEPA 1997) were used to calculate surface water contaminant doses (see Table 14).

Incidental Ingestion Rate (mL/day)	Number of Days Exposed Per Year	No. of years Exposed
50	130 days	30

Based on the EPC of arsenic, mercury, thallium and 1,1,2,2-tetrachloroethane detected in the surface water, the chronic exposure doses calculated for adults and children were lower than the corresponding health guideline CVs (see Table 14).

Although the EPC of lead exceeded the AL,¹³ it should be noted that the AL is based on a default ingestion rate of 2 L/day. Since the incidental ingestion rate (0.05 L/day) was 40 times lower than the default ingestion rate, in order to ingest the equivalent amount of lead, the lead concentration could be as high as 600 µg/L. As such,

¹³Action Level for Lead in drinking water

past exposures associated with incidental ingestion of surface water from the on-site surface water sources are unlikely to cause non-cancer adverse health effects.

Inhalation of on-site contaminated ambient air (past)

In 1978, on-site mercury monitoring was conducted by the USEPA's Environmental Monitoring and Surveillance Laboratory (Research Triangle Park, NC) (NJDOH 1980). The concentration of mercury detected ranged from 0.29 to 3.3 $\mu\text{g}/\text{m}^3$. The average concentrations at three sites (see Figure 6) were 0.76, 1.03 and 1.5 $\mu\text{g}/\text{m}^3$, respectively.

In July 1991, the ATSDR evaluated the potential worker and customer exposure to mercury at the two warehouses located at the site using a portable instrument (Jerome meter) (ATSDR 1991). Mercury concentration was 20 $\mu\text{g}/\text{m}^3$ at a point along the railroad tracks adjacent to the fence and 40 $\mu\text{g}/\text{m}^3$ at an exhaust vent on the foundation of the furniture distribution center located at 3 Ethel Boulevard.

These levels exceeded the EPA's RfC^{14} of 0.3 $\mu\text{g}/\text{m}^3$ for chronic exposure to elemental mercury vapor. The chronic inhalation RfC for mercury (0.3 $\mu\text{g}/\text{m}^3$) is based on hand tremors, increases in memory disturbances, and slight subjective and objective evidence of autonomic dysfunction. An uncertainty factor of 30 and a LOAEL of 9 $\mu\text{g}/\text{m}^3$ were used to calculate the inhalation RfC (ATSDR 1999a). Although the detected levels were ambient levels and the exposure conditions were not chronic (24 hours/day), the levels were 2 to 3 times higher than the LOAEL and, as such, there was a potential for non-cancer adverse health effects in children and adults from inhalation of mercury vapor.

Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. 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 (ATSDR 1999a).

Incidental ingestion of contaminated soil from off-site properties (past, present, future)

Off-site properties: The off-site properties were wooded areas/marshland and were not developed (see Figure 4). The exposure assumptions used for estimating exposure doses were the same as the on-site areas (i.e., 130 visits per year). As mentioned earlier, since a chronic oral MRL or RfD are unavailable for mercury, the calculated exposure dose was compared to health guideline CV for intermediate exposures. The child exposure dose (i.e., 0.0008 $\text{mg}/\text{kg}/\text{day}$) was lower than the MRL for intermediate exposures (0.002 $\text{mg}/\text{kg}/\text{day}$); as such, no intermediate adverse health effects are likely (ATSDR 1999a).

¹⁴The receptor for this completed exposure pathway are the employees and trespassers, therefore, occupational standards are not appropriate levels to be used for comparison.

In order to assess the chronic exposures, the estimated mercury exposure doses for children (0.0008 mg/kg/day) were expressed as mercuric chloride, i.e., $0.0008 \times 271/200 = 0.00108$ mg/kg/day. The chronic exposure doses exceeded the RfD of 0.0003 mg/kg/day for mercuric chloride (ATSDR 1999a). An uncertainty factor of 1,000 and a LOAEL of 0.317 mg/kg/day were used to calculate the oral RfD (0.0003 mg/kg/day). The child exposure dose (i.e., 0.00108 mg/kg/day) was about 175 times lower than the LOAEL (see Table 15). As such, the risk of non-cancer adverse health effects for exposures to mercury detected in soil is considered unlikely.

Cancer Health Effects

The site-specific lifetime excess cancer risk (LECR) indicates the cancer potential of contaminants. LECR estimates are usually expressed in terms of excess cancer cases in an exposed population in addition to the background rate of cancer. For perspective, the lifetime risk of being diagnosed with cancer in the United States is 44 per 100 individuals for males and 38 per 100 for females; the lifetime risk of being diagnosed with any of several common types of cancer ranges between 1 in 10 and 1 in 100 (ACS 2011). Typically, health guideline CVs developed for carcinogens are based on one excess cancer case per one million individuals (10^{-6}).

The U.S. Department of Health and Human Services (USDHHS) cancer classes for the Ventron/Velsicol site contaminants are presented in Tables 16-20. The cancer classes are defined as follows:

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

Incidental ingestion of contaminated soil and sediment from on-site areas (past)

Developed Area: The cancer classes of the COPCs detected in the soil of the developed area are given in Table 16. Arsenic and PAHs were the carcinogens found in soil. Exposure doses for cancer evaluation were calculated using the following formula:

$$\text{Cancer Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW} * \frac{ED}{AT}$$

where, C = concentration of contaminant in soil (mg/kg);
 IR = soil ingestion rate (kg/day);
 EF = exposure factor representing the site-specific exposure scenario;
 ED = exposure duration¹⁵ (year);
 BW = body weight (kg); and,
 AT = averaging time (year).

¹⁵A lifetime exposure time of 30 years was used based on default residency time.

A relative potency estimate approach was developed (OEHHA 2016) to assess the cancer risks associated with PAHs (with the exception of dibenz[a,h]anthracene). Using this approach, the cancer potency of carcinogenic PAHs can be estimated based on their relative potency with reference to benzo[a]pyrene. For each of the carcinogenic PAHs, the benzo[a]pyrene equivalence was calculated by multiplying the mean concentration detected with the cancer potency factor. The total benzo[a]pyrene equivalence was then obtained by summing each of the individual benzo[a]pyrene equivalences (see Table 16).

Based on previously described exposure assumptions, LECRs were calculated by multiplying the exposure dose by the cancer slope factor. The cancer slope factor is defined as the slope of the dose-response curve obtained from animal and/or human cancer studies and is expressed as the inverse of the daily exposure dose, i.e., $(\text{mg/kg/day})^{-1}$.

LECRs associated with ingestion of contaminated soil during recreational activities in the developed area of the Ventron/Velsicol site were evaluated (see Table 16). Based on the EPCs of PAHs and arsenic concentrations detected in soil, the calculated LECRs showed a low increase in the cancer risk (1 in 100,000 individuals exposed including children) who were exposed to contaminated soil in this area.

Undeveloped Area: LECRs associated with ingestion of contaminated soil during recreational activities in the undeveloped area of the Ventron/Velsicol site were evaluated (see Table 17). Based on the EPCs of PAHs, arsenic and cadmium detected in soil, the calculated LECRs showed a low increase in the cancer risk (9 in 100,000 individuals exposed, including children) who were exposed to contaminated soil in this area.

Marsh Area: LECRs associated with ingestion of contaminated soil during recreational activities in the marsh area of the Ventron/Velsicol site were evaluated (see Table 18). Based on the EPCs of PAHs, arsenic, cadmium and chromium¹⁶ detected in soil, the calculated LECRs showed a low to moderate increase in the cancer risk (1 in 10,000 individuals exposed including children) who were exposed to contaminated soil in this area.

Incidental ingestion of contaminated surface water from on-site areas (past)

The cancer class of the COPCs detected in the surface water is given in Table 19; arsenic was the only carcinogen in the surface water pathway. Based on the EPC, the LECR is 5 in ten million to the exposed population, which is considered to be no increase in cancer risk.

Incidental ingestion of contaminated soil from off-site areas (past, present, future)

¹⁶The CSF for chromium (+6) was obtained from NJDEP. The NJDEP derived a CSF of 0.5 $(\text{mg hexavalent chromium/kg/day})^{-1}$ using chronic bioassay data of male mice from the 2008 NTP study and US EPA cancer assessment guidelines (NJDEP 2009).

The cancer class of the contaminants detected in the soil of adjacent areas is given in Table 20; PAHs were found to be the carcinogens. Based on the calculated EPC of the PAHs, the cumulative LECR is 2 in 100,000 to the exposed population. The LECR is considered a low increase in cancer risk.

Child Health Considerations

The NJDOH and ATSDR recognize 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 types of exposures to hazardous substances. Their lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most important, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

The NJDOH and ATSDR evaluated the potential risk for children resided in the area who may have been exposed to site contaminants. The incidental exposure doses to mercury detected in the on-site and adjacent properties soil exceeded the health-based CVs; the exposure doses were 57 to 360 times lower than the LOAEL. Although the mean ambient air mercury concentrations exceeded the RfC, the levels were 6 to 9 times lower than the LOAEL. Based on the fact that oral and inhalation exposures were significantly lower than the adverse effect level (i.e., the LOAEL), there was a low potential for non-cancer adverse health effects in children from exposures to mercury.

Based on the concentrations of PAHs and metals detected, a LECR between 5 in ten million and 1 in 10,000 were determined for residents (including children) due to past exposures. This exposure poses no expected to low increase in lifetime excess cancer risk.

Health Outcome Data

A review of health outcome data (e.g., adverse pregnancy outcomes, cancers, deaths) may be conducted to assess the public health impact of these completed exposure pathways. However, the size of the exposed population (i.e., about 80) is relatively small and an evaluation of available health data would be unlikely to produce statistically valid findings. Health data at the municipal level would not be relevant since the potentially exposed population is a small proportion of the three townships (total population of about 16,500).

Public Comment

The public comment period for this public health assessment was from March 4, 2015 through April 05, 2015. The comments and the responses are given in Appendix C.

Conclusions

Manufacturing operations at the former Ventron/Velsicol site and uncontrolled release of hazardous wastes have resulted in the contamination of soil, water, biota and ambient air. There were completed exposure pathways via the incidental ingestion of contaminated soil and inhalation of mercury vapor in the past. Contaminants of concern are arsenic, lead, cadmium, chromium, mercury, bis[2-ethylhexyl]phthalate and PAHs in the soil and mercury vapor in the ambient air. The exposed population included area residents and trespassers. The ATSDR and NJDOH have reached the following conclusions in this report.

The NJDOH and ATSDR conclude that there are no current or future site-related exposures (i.e., from ingestion of on-site soil and surface water and inhalation of ambient air) to contaminants at the developed, undeveloped (i.e., USEPA's Operable Unit 1) and off-site residential areas of the Ventron/Velsicol site that can harm people's health.

Contaminated soils at the on- (i.e., developed and undeveloped areas) and off-site (i.e., residential homes located to the north of the site) areas have been excavated and/or capped. The excavated areas were backfilled with clean fill. An earlier health evaluation (ATSDR 1995) associated with incidental ingestion of contaminated soil from off-site areas (i.e., adjacent properties, municipal repair yard and wastewater treatment plant) did not indicate any concern for harmful health effects. In addition, the site is fenced along the northern and western boundaries for access restriction. Thus, area residents are not being exposed to site-related contaminants.

Based on an earlier health consultation conducted in 1995, ATSDR concluded that past exposures associated with consumption of contaminated biota from the on-site marsh area and Berry's Creek (i.e., USEPA's Operable Unit 2) may have harmed people's health.

Based on the concentration of mercury detected in the biota in the past, frequent ingestion of biota may have resulted in exposures at levels of public health concern. Under the oversight of USEPA, the remedial investigation of the BCSA is being conducted by the potential responsible parties.

The NJDOH and ATSDR conclude that past exposures to site-related contaminants from the developed, undeveloped (i.e., USEPA's Operable Unit 1), marsh (i.e., part of USEPA's Operable Unit 2) and off-site residential properties at the Ventron/Velsicol site may have harmed people's health.

Based on the mercury detected in soil and ambient air, the potential for non-cancer adverse health effects associated with past exposures are possible in children and adults. Based on the lead detected in soil, the potential for lead related non-cancer adverse health effects are possible in children. Maximum cumulative lifetime excess cancer risks were estimated to be 1 in 10,000 to the exposed population. This exposure poses a low to moderate increase in lifetime excess cancer risk in comparison to the background risk of cancer from all causes. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0-2 feet and 0-12 inches depth, which may not represent actual soil conditions. This may over or underestimate the calculated exposure risk.

Recommendations

The NJDOH and ATSDR recommend maintaining site access restriction to ensure integrity of the remedies for the Ventron/Velsicol site.

The NJDOH and ATSDR recommend completing the RI/FS and the remedy for the marsh area and all the adjacent water bodies, including Berry's Creek, Nevertouch Creek, and the Diamond Shamrock/Henkel Ditches as soon as feasible.

Public Health Action Plan

The purpose of a Public Health Action Plan (PHAP) 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 adverse 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 actions to be implemented by the ATSDR and NJDOH are as follows:

Public Health Actions Taken by NJDOH and ATSDR

1. Contamination data collected from the Ventron/Velsicol site were evaluated by the NJDOH and ATSDR.
2. Representatives of the NJDOH conducted a site visit of the Ventron/Velsicol site on June 2, 2010.
3. Copies of this Public Health Assessment were provided to the public via the township library and posted to the Internet for public comment.

Public Health Actions Planned by NJDOH and ATSDR

1. Under the oversight of EPA, the remedial investigation of the Berry's Creek is being conducted by the potential responsible parties. The NJDOH, in cooperation with the ATSDR, will prepare a separate health consultation to evaluate contaminant exposures associated with the consumption of biota from Berry's Creek.

References

[ACS] American Cancer Society: Cancer Facts and Figures. 2011. Accessed on Sept. 23, 2011 at: <http://www.cancer.org/Research/CancerFactsFigures/CancerFactsFigures/cancer-facts-figures-2011>.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1989. Preliminary Public Health Assessment: Ventron/Velsicol, Wood-Ridge, New Jersey, July 1991.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1991. Health Consultation: Ventron/Velsicol NPL Site, Wood-Ridge Borough, Bergen County, New Jersey, CERCLIS No. NJD980529879, April 10, 1989.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1993. Site Review and Update: Ventron/Velsicol, Wood-Ridge Borough, Bergen County, New Jersey, CERCLIS No. NJD980529879, April 20, 1993.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1995a. Public Health Consultation: Ventron/Velsicol (aka Berry's Creek), Wood-Ridge/Carlstadt, Bergen County, New Jersey, January, 1995.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1995b. Toxicological profile for Polycyclic Aromatic Compounds (PAHs). US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999a. Toxicological profile for Mercury. US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999b. Toxicological profile for Cadmium. US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2000a. Toxicological profile for Arsenic. US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2000b. Toxicological profile for Chromium. US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005. Public Health Assessment Guidance Manual. US Department of Health and Human Services, Atlanta, Georgia.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2006. Toxicological profile for Lead. US Department of Health and Human Services, Atlanta, Georgia.

[CDC] Centers for Disease Control. 1991. Preventing lead poisoning in young children. U.S. Department of Health and Human Services, October.

[CDC] Centers for Disease Control and Prevention 2012a. Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention: Report of the Advisory Committee on Childhood Lead Poisoning Prevention, Centers for Disease Control and Prevention. U.S. Department of Health and Human Services, January.

[CDC] Centers for Disease Control and Prevention 2012b. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in "Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention." Centers for Disease Control and Prevention, June 7, 2012. Available at: http://www.cdc.gov/nceh/lead/ACCLPP/CDC_Response_Lead_Exposure_Recs.pdf.

Exponent. 2004. Operable Unit 1: Remedial Investigation Report for the Ventron/Velsicol site, Wood-Ridge/Carlstadt, New Jersey, June 2004.

Exponent. 2005. Operable Unit 1: Final Human Health Risk Assessment, Ventron/Velsicol site, Wood-Ridge/Carlstadt, New Jersey, July 2005.

Kizu R, Kazumsa O, Toriba A, et al. 2003. Antiandrogenic activities of diesel exhaust particle extracts in PC3/AR human prostate carcinoma cells. Toxicol Sci. 76: 299-309.

[NJDEP] New Jersey Department of Environmental Protection. Ca. 1980. Mercury Levels in Berry's Creek. Lipsky D, Reed RJ, and Harkov R. Office of Cancer and Toxic Substance Research, New Jersey, ca. 1980.

[NJDEP] New Jersey Department of Environmental Protection. 1991. Summary of air sampling for mercury at the Berry's Creek site. Memo dated March 23, 1991. State of New Jersey Department of Environmental Protection, Trenton, NJ.

[NJDEP] New Jersey Department of Environmental Protection. 2001. Comments regarding draft human health risk assessment dated April 2001. Letter dated September 14, 2001 from G. Zervas, NJDEP, to J. Hoek, Civil & Environmental Consultants, Inc. New Jersey Department of Environmental Protection, Trenton, NJ.

[NJDEP] New Jersey Department of Environmental Protection. 2006. Superfund Record of Decision: Ventron/Velsicol site, Wood-Ridge and Carlstadt, New Jersey, October 2006.

[NJDEP] New Jersey Department of Environmental Protection. 2009. Derivation of an Ingestion-Based Soil Remediation Criterion for Cr+6 Based on the NTP Chronic Bioassay Data for Sodium Dichromate Dihydrate. Office of Science. Trenton, NJ 2009.

[NJDEP] New Jersey Department of Environmental Protection. 2011. Residential Direct Contact Health Based Criteria and Soil Remediation Standard, 2011.

[NJDOH] New Jersey Department of Health. 1980. Mercury in Berry's Creek – Health Study. Presentation by D. Patel, M. Lakat and R. Altman at the Mercury in Berry's Creek Ecosystem Conference, Meadowlands Hilton, 3 November, 1980.

[OEHHA] Office of Environmental Health Hazard Assessment, California Environmental Protection Agency (Cal/EPA). 2016. Air Toxicology and Epidemiology: Adoption of Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. [accessed 2016 March 01]. Available from: http://www.oehha.ca.gov/air/hot_spots/hotspots2015.html.

[USEPA] United States Environmental Protection Agency 1986. Air Quality Criteria for Lead. Environmental Criteria and Assessment Office, Office of Research and Development, Research Triangle Park, N.C. EPA 600/8-83-028 a-f, June 1986.

[USEPA] U.S. Environmental Protection Agency. 1989. Risk assessment guidance for Superfund. Volume 1: Human health evaluation manual (Part A). Interim Final Report. EPA 540/1-89/002. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

[USEPA] United States Environmental Protection Agency. 1994a. Guidance Manual for the IEUBK Model for Lead in Children. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-15-1. February 1994.

[USEPA] United States Environmental Protection Agency. 1994b. Memorandum: OSWER Directive: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive #9355.4-12. August 1994.

[USEPA] U.S. Environmental Protection Agency. 1997. Exposure Factor Handbook (Volume I, II and III), EPA/600/P-95/002Fa, b and c, August 1997.

[USEPA] United States Environmental Protection Agency. 2002. User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows® Version – 32 Bit Version. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-42. May 2002.

[USEPA] United States Environmental Protection Agency. 2003a. Assessing Intermittent or Variable Exposures at Lead Sites. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-76. November 2003.

[USEPA] United States Environmental Protection Agency. 2003b. Recommendations of the Technical Review Workgroup for Lead for an Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil, EPA-540-R-03-001, January 2003.

[USEPA] U.S. Environmental Protection Agency. 2007. User Guide ProUCL® Version 4.1.00. [accessed 2011 February]. Available from:
<http://www.epa.gov/osp/hstl/tsc/software.htm>.

[USEPA] U.S. Environmental Protection Agency. 2011. Regional Screening Levels (Formerly PRGs). [accessed 2011 January]. Available from:
<http://www.epa.gov/region9/superfund/prg/>.

[USEPA] U.S. Environmental Protection Agency. 2011a. Exposure Factor Handbook (Volume I, II and III), EPA/600/R-09/052F, September 2011.

[USEPA] U.S. Environmental Protection Agency. 2011b. Integrated Risk Information System (IRIS) database. [accessed 2011 January]. Available from:
<http://www.epa.gov/iris/subst/01b41.htm>.

Report Preparation

This Public Health Assessment for the Ventron/Velsicol site, located in Wood-Ridge and Carlstadt (Bergen County) New Jersey, was prepared by the New Jersey Department of Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies and procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

Authors

Tariq Ahmed, PhD, PE
Research Scientist
New Jersey Department of Health

Alicia Curtis Stephens, M.S.
Research Scientist
New Jersey Department of Health

ATSDR Reviewers

Eva McLanahan, Ph.D., REHS/RS
Commander, U.S. Public Health Service
Technical Project Officer

ATSDR Regional Representatives:

Leah T. Graziano, R.S.
Regional Director
Office of Regional Operations, Region II

Elena Vaouli, M.P.H.
Commander, U.S. Public Health Service
Associate Regional Representative
Office of Regional Operations, Region II

Table 1: Soil (0 to 2 feet depth) Sampling Results of the Developed Area (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Metals (mg/kg)							
Mercury (total)	14	0	9.3	13,800	1,800	6.7 (RSL ^d)	Yes
Aluminum	9	0	3,370	12,000	7,000	50,000 (EMEG ^e)	No
Arsenic	11	3	ND	11	5	0.5 (CREG ^f)	Yes
Barium	11	0	26.9	818	180	10,000 (EMEG)	No
Beryllium	9	8	ND	0.68	0.25	100 (EMEG)	No
Cadmium	14	6	ND	3.4	1	5 (EMEG)	No
Chromium	11	0	6.6	131	34	50 (EMEG ^g)	Yes
Copper	14	0	12.4	7,420	630	3,100 (RSL)	Yes
Lead	14	0	17.8	390	120	400 (RSL)	No
Manganese	11	0	110	540	270	3,000 (RMEG ^h)	No
Nickel	11	2	ND	87.8	28.4	1,000 (RMEG)	No
Selenium	11	9	ND	1.1	0.4	300 (EMEG)	No
Silver	11	4	ND	9.6	2	500 (RMEG)	No
Thallium	14	11	ND	5.4	1.1	5.1 (RSL)	Yes
Vanadium	9	0	6	140	40	390 (RSL)	No
Zinc	14	0	89	2,110	500	20,000 (EMEG)	No
Volatile Organic Compounds (mg/kg)							
Benzene	11	6	ND	2.8	0.3	10 (CREG)	No
Chlorobenzene	11	10	ND	0.0012	0.005	1,000 (RMEG)	No
Toluene	11	9	ND	0.011	0.006	4,000 (RMEG)	No

Table 1: (Cont'd.)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Semivolatile Organic Compounds (mg/kg)							
Acenaphthene	9	3	ND	0.23	0.14	3,000 (RMEG)	No
Acenaphthylene	9	7	ND	0.069	0.18	NA ⁱ	
Anthracene	9	2	ND	0.46	0.17	20,000 (RMEG)	No
Benz[a]anthracene	12	2	ND	1.4	0.5	0.15 (RSL)	Yes
Benzo[a]pyrene	12	2	ND	1.1	0.4	0.1 (CREG)	Yes
Benzo[b]fluoranthene	12	1	ND	1.4	0.5	0.15 (RSL)	Yes
Benzo[ghi]perylene	9	2	ND	0.52	0.24	NA	
Benzo[k]fluoranthene	12	1	ND	0.565	0.25	1.5 (RSL)	No
bis[2-Ethylhexyl]phthalate	12	1	ND	10.8	2.8	35 (RSL)	No
Chrysene	9	1	ND	1.4	0.5	15 (RSL)	No
Dibenz[a,h]anthracene	12	6	ND	0.15	0.12	0.015 (RSL)	Yes
Fluoranthene	9	0	0.094	2.6	0.8	2,000 (RMEG)	No
Fluorene	9	2	ND	0.37	0.16	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	12	2	ND	0.47	0.23	0.15 (RSL)	Yes
2-Methylnaphthalene	9	6	ND	0.19	0.18	NA	
Naphthalene	9	7	ND	0.094	0.19	1,000 (RMEG)	No
Pyrene	9	0	ND	2.6	0.8	2,000 (RMEG)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Value; ^eATSDR Environmental Media Evaluation Guide;^fATSDR Cancer Risk Evaluation Guide; ^gATSDR EMEG based on hexavalent chromium; ^hATSDR Reference Media Evaluation Guide; ⁱNot Available

Table 2: Soil (0 to 2 feet depth) Sampling Results of the Undeveloped Area (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum		
Metals (mg/kg)						
Mercury (total)	25	0	1.2	548	6.7 (RSL ^d)	Yes
Methylmercury	9	0	0.00059	0.322	20 (EMEG ^e)	No
Aluminum	24	0	3,580	11,000	50,000 (EMEG)	No
Antimony	24	6	ND	53.7	20 (RMEG ^f)	Yes
Arsenic	25	4	ND	26.4	0.5 (CREG ^g)	Yes
Barium	25	0	33.3	608	10,000 (EMEG)	No
Beryllium	24	23	ND	0.26	100 (EMEG)	No
Cadmium	25	9	ND	21.2	5 (EMEG)	Yes
Chromium	25	0	11.3	1,150	50 (EMEG ^h)	Yes
Copper	25	0	22.8	1,010	3,100 (RSL)	No
Lead	25	0	39.3	4,320	400 (RSL)	Yes
Manganese	25	0	66.3	3,090	3,000 (RMEG)	Yes
Nickel	25	2	ND	81.7	1,000 (RMEG)	No
Selenium	25	15	ND	2	300 (EMEG)	No
Silver	25	3	ND	93.8	500 (RMEG)	No
Thallium	25	23	ND	21.9	5.1 (RSL)	Yes
Vanadium	24	0	9.9	175	390 (RSL)	No
Zinc	25	6	ND	25,400	20,000 (EMEG)	Yes
Volatile Organic Compounds (mg/kg)						
Benzene	25	24	ND	0.0036	10 (CREG)	No
Toluene	25	24	ND	0.0022	4,000 (RMEG)	No

Table 2: (Cont'd.)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Semivolatile Organic Compounds (mg/kg)							
Acenaphthene	24	16	ND	1.2	0.5	3,000 (RMEG)	No
Acenaphthylene	24	19	ND	0.099	0.5	NA	
Anthracene	24	8	ND	4.1	0.6	20,000 (RMEG)	No
Benz[a]anthracene	24	2	ND	4	0.9	0.15 (RSL)	Yes
Benzo[a]pyrene	24	2	ND	10	1	0.1 (CREG)	Yes
Benzo[b]fluoranthene	24	5	ND	13	2	0.15 (RSL)	Yes
Benzo[ghi]perylene	24	2	ND	2.2	0.7	NA	
Benzo[k]fluoranthene	24	5	ND	4.7	0.7	1.5 (RSL)	Yes
bis[2-Ethylhexyl]phthalate	24	11	ND	380	30	35 (RSL)	Yes
Chrysene	24	2	ND	12	1	15 (RSL)	No
Dibenz[a,h]anthracene	24	8	ND	0.9	0.27	0.015 (RSL)	Yes
Fluoranthene	24	2	ND	26	2	2,000 (RMEG)	No
Fluorene	24	16	ND	1.1	0.5	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	24	3	ND	2.6	0.3	0.15 (RSL)	Yes
2-Methylnaphthalene	24	21	ND	0.12	0.5	NA	
Naphthalene	24	19	ND	0.63	0.5	1,000 (RMEG)	No
Pyrene	24	1	ND	24	2	2,000 (RMEG)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Reference Media Evaluation Guide; ^gATSDR Cancer Risk Evaluation Guide; ^hATSDR EMEG based on hexavalent chromium; ⁱNot Available

Table 3: Sediment Sampling Results of the Onsite Basin (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c	
			Minimum	Maximum			
Metals (mg/kg)							
Mercury (total)	2	0	1,280	1,290	1,285	6.7 (RSL ^d)	Yes
Mercury (total) (0-2 cm)	2	0	856	1,180	1,020	6.7 (RSL)	Yes
Methylmercury	2	0	0.0982	0.126	0.112	20 (EMEG ^e)	No
Arsenic	2	0	3.6	8.8	6.2	0.5 (CREG ^f)	Yes
Cadmium	2	0	0.9	1.6	1.2	5 (EMEG)	No
Chromium	2	0	55.4	65.6	60.5	50 (EMEG ^g)	Yes
Copper	2	0	94	136	115	3,100 (RSL)	No
Lead	2	0	188	469	329	400 (RSL)	Yes
Nickel	2	0	14.2	28.1	21.2	1,000 (RMEG ^h)	No
Silver	2	0	1.1	2.4	1.8	500 (RMEG)	No
Zinc	2	0	556	844	700	20,000 (EMEG)	No
Semivolatile Organic Compounds (mg/kg)							
Aroclor® 1248	2	0	0.19	0.24	0.21	0.4 (CREG)	No
Aroclor® 1260	2	0	0.26	0.49	0.37	0.4 (CREG)	Yes
Acenaphthene	2	1	ND	0.1	0.1	3,000 (RMEG)	No
Anthracene	2	0	0.25	0.35	0.3	20,000 (RMEG)	No
Benz[a]anthracene	2	0	0.9	1.7	1.3	0.15 (RSL)	Yes
Benzo[a]pyrene	2	0	0.8	1.6	1.2	0.1 (CREG)	Yes
Benzo[ghi]perylene	2	0	0.58	1.2	0.9	NA ⁱ	No
Benzo[k]fluoranthene	2	0	0.34	0.66	0.5	1.5 (RSL)	No
Chrysene	2	0	0.9	1.6	1.2	15 (RSL)	No
Dibenz[a,h]anthracene	2	0	0.17	0.32	0.25	0.015 (RSL)	Yes

Table 3: (Cont'd.)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CV _S ^b	COPC ^c
			Minimum	Maximum	Mean		
Metals (mg/kg)							
SemiVolatile Organic Compounds (mg/kg)							
Fluoranthene	2	0	1.7	2.8	2.2	2,000 (RMEG)	No
Fluorene	2	0	0.11	0.17	0.14	2,000 (RMEG)	No
Phenanthrene	2	0	1.2	1.8	1.5	NA	No
Pyrene	2	0	1.61	2.9	2.3	2,000 (RMEG)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide; ^gATSDR EMEG based on hexavalent chromium; ^hATSDR Reference Media Evaluation Guide; ⁱNot Available

Table 4: Sediment Sampling Results of the West Ditch (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum		
Metals (mg/kg)						
Mercury (total)	3	0	19	155	64.8	6.7 (RSL ^d)
Methylmercury	3	0	0.01191	0.06654	0.03091	20 (EMEG ^e)
Arsenic	3	0	2.6	5.1	4.2	0.5 (CREG ^f)
Cadmium	3	0	2.2	9.1	5.3	5 (EMEG)
Chromium	3	0	88.4	156	131	50 (EMEG ^g)
Copper	3	0	143	194	162	3,100 (RSL)
Lead	3	0	224	274	246	400 (RSL)
Nickel	3	2	24.1	29.2	27.1	1,000 (RMEG ^h)
Silver	3	0	4.3	4.3	1.5	500 (RMEG)
Zinc	3	0	434	3,540	1,920	20,000 (EMEG)
Semivolatile Organic Compounds (mg/kg)						
Acenaphthylene	3	1	0.18	0.27	0.28	NA ⁱ
Anthracene	3	0	0.17	0.35	0.27	20,000 (RMEG)
Benz[a]anthracene	3	0	0.23	0.56	0.35	0.15 (RSL)
Benzo[a]pyrene	3	0	0.3	0.63	0.41	0.1 (CREG)
Benzo[ghi]perylene	3	0	0.27	0.55	0.37	NA
Benzo[k]fluoranthene	3	0	0.28	0.58	0.38	1.5 (RSL)
Chrysene	3	0	0.33	0.69	0.47	15 (RSL)
Dibenz[a,h]anthracene	3	0	0.091	0.27	0.18	0.015 (RSL)

Table 4: (Cont'd.)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
SemiVolatile Organic Compounds (mg/kg)							
Fluoranthene	3	0	0.51	1.1	0.8	2,000 (RMEG)	No
Naphthalene	3	2	0.1	0.1	0.1	1,000 (RMEG)	No
Phenanthrene	3	0	0.18	0.39	0.27	NA	
Pyrene	3	0	0.38	0.78	0.56	2,000 (RMEG)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide; ^gATSDR EMEG based on hexavalent chromium; ^hATSDR Reference Media Evaluation Guide; ⁱNot Available

Table 5: Sediment Sampling Results of the Marsh Area (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum		
Metals (mg/kg)						
Mercury (total)	19	0	25.1	1,090	270	6.7 (RSL ^d)
Methylmercury	19	0	0.0039	0.233	0.06	20 (EMEG ^e)
Arsenic	19	1	ND	150	40	0.5 (CREG ^f)
Cadmium	19	0	3.8	68.3	22.2	5 (EMEG)
Chromium	19	0	142	2,170	1,000	50 (EMEG ^g)
Copper	19	0	44.2	730	360	3,100 (RSL)
Lead	19	0	51.6	401	280	400 (RSL)
Nickel	19	0	18.2	274	140	1,000 (RMEG ^h)
Silver	19	2	ND	9.6	5.3	500 (RMEG)
Zinc	19	0	868	22,700	5,000	20,000 (EMEG)
Semiolatlite Organic Compounds (mg/kg)						
Acenaphthene	19	18	ND	0.11	0.6	3,000 (RMEG)
Acenaphthylene	19	18	ND	0.48	0.06	NA ⁱ
Anthracene	19	17	ND	1.1	0.6	20,000 (RMEG)
Benz[a]anthracene	19	0	0.13	5.8	0.6	0.15 (RSL)
Benzo[a]pyrene	19	0	0.15	4.9	0.6	0.1 (CREG)
Benzo[g,h,i]perylene	19	0	0.1	2.7	0.5	NA
Benzo[k]fluoranthene	19	7	ND	1.6	0.5	1.5 (RSL)
Chrysene	19	0	0.16	5.6	0.7	15 (RSL)
Dibenz[a,h]anthracene	19	12	ND	0.17	0.48	0.015 (RSL)
Fluoranthene	19	0	0.22	10	1	2,000 (RMEG)

Table 5: (Cont'd.)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Semivolatle Organic Compounds (mg/kg)							
Fluorene	19	18	ND	0.29	0.29	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	19	0	0.095	3.2	0.5	0.15 (RSL)	Yes
2-Methylnaphthalene	19	18	ND	0.085	0.06	NA	
Naphthalene	19	17	ND	0.59	0.44	1,000 (RMEG)	No
Phenanthrene	19	7	ND	4.1	0.6	NA	
Pyrene	19	0	0.24	9	1	2,000 (RMEG)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide; ^gATSDR EMEG based on hexavalent chromium; ^hATSDR Reference Media Evaluation Guide; ⁱNot Available

Table 6: Subsurface Soil Sampling Results of the On-site Areas (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum		
Metals (mg/kg)						
Mercury (total)	50	0	0.1942	34,700	1,100	Yes
Methylmercury	7	0	0.0001	0.00744	0.00294	No
Antimony	40	7	ND	41.2	6.5	Yes
Arsenic	45	1	ND	120	14	Yes
Barium	45	0	29.5	1,290	490	No
Beryllium	40	38	ND	2	0.37	No
Cadmium	45	0	0.4	36.1	6	Yes
Chromium	45	0	6.4	606	106	Yes
Copper	45	0	6.7	2,190	290	No
Lead	45	0	5	3,830	1,050	Yes
Manganese	45	0	16.5	23,300	900	Yes
Nickel	45	0	8.2	317	61	No
Selenium	45	29	ND	6.4	1.3	No
Silver	45	9	ND	84.8	4.1	No
Thallium	45	41	ND	12.9	1.4	Yes
Vanadium	40	0	7.4	980	72	Yes
Zinc	45	0	26.8	43,200	2,200	Yes
Volatile Organic Compounds (mg/kg)						
Benzene	45	38	ND	0.33	0.02	No
Chlorobenzene	45	44	ND	0.01	0.0067	No
Toluene	45	40	ND	70	2.1	No
Xylene	40	37	ND	110	3	No

Table 6: (Cont'd)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Semivolatile Organic Compounds (mg/kg)							
Benz[a]anthracene	40	10	ND	62	2.1	0.15 (RSL)	Yes
Benzo[a]pyrene	40	12	ND	52	1.8	0.1 (CREG)	Yes
Benzo[b]fluoranthene	40	10	ND	64	2.4	0.15 (RSL)	Yes
bis[2-Ethylhexyl]phthalate	40	7	ND	22	1.9	35 (RSL)	No
Dibenz[a,h]anthracene	40	19	ND	1.3	0.21	0.015 (RSL)	Yes

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Reference Media Evaluation Guide; ^gATSDR Cancer Risk Evaluation Guide; ^hATSDR EMEG based on hexavalent chromium

Table 7: On-site Groundwater Sampling Results (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum	Mean		
Metals (µg/L)							
Mercury	13	8	ND	8.2	0.9	2 (MCL ^d)	Yes
Methylmercury	13	0	0.00014	0.02	0.0044	3 (EMEG ^e)	No
Arsenic	13	10	ND	14.6	3.2	0.02 (CREG ^f)	Yes
Cadmium	13	11	ND	2.5	0.4	1 (RMEG ^g)	Yes
Lead	13	11	ND	0.9	0.6	15 (AL ^h)	No
Manganese	13	0	74.1	3,880	1,390	500 (RMEG)	Yes
Nickel	13	0	2.1	117	22	200 (RMEG)	No
Selenium	13	12	ND	2.34	1.3	50 (EMEG)	No
Thallium	13	11	ND	5.4	3.4	2.4 (RSL)	Yes
Volatile Organic Compounds (µg/L)							
Benzene	13	11	ND	140	20	0.6 (CREG)	Yes
Chlorobenzene	13	9	ND	15	5	200 (RMEG)	No
1,2-Dichloroethene	13	11	ND	45	8	200 (RMEG)	No
Toluene	13	12	ND	1,700	100	800 (RMEG)	Yes
Xylene	13	12	ND	390	30	2,000 (EMEG)	No
Semivolatile Organic Compounds (µg/L)							
bis[2-Ethylhexyl]phthalate	13	12	ND	2	20	4.8 (RSL)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Maximum Contaminant Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide; ^gATSDR Reference Media Evaluation Guide; ^hUSEPA Action Level for Lead in drinking water

Table 8: On-site Surface Water Sampling Results (Source: Exponent 2004)

Location	Analyte	No. of Analysis	No of NDs ^a	Concentration			Environmental Guideline CVs ^b	COPC ^c					
				Minimum	Maximum	Mean							
On-site Basin	Metals (µg/L)												
	Methylmercury - Filtered	2	0	0.00085	0.00098	0.00092	3 (EMEG)	No					
	Mercury (total) - Unfiltered	2	0	0.0058	0.0176	0.0117	2 (MCL)	No					
	Methylmercury - Unfiltered	2	0	0.00218	0.00233	0.00225	3 (EMEG)	No					
	Lead - Filtered	2	1	ND	4.7	4.7	15 (AL)	No					
	Lead - Unfiltered	2	0	2	4.2	3.1	15 (AL)	No					
West Ditch	Mercury (total) unfiltered	3	0	0.402	0.738	0.573	2 (MCL)	No					
	Methylmercury unfiltered	3	0	0.00114	0.00277	0.00184	3 (EMEG)	No					
	Lead - Unfiltered	3	0	4	19	9	15 (AL)	Yes					
	Mercury (total) - Filtered	15	13	ND	0.24	0.12	2 (MCL)	No					
Marsh	Methylmercury - Filtered	15	0	0.00008	0.00047	0.00027	3 (EMEG)	No					
	Mercury (total) - Unfiltered	15	1	ND	15.6	2.7	2 (MCL)	Yes					
	Methylmercury - Unfiltered	15	0	0.00058	0.00464	0.00135	3 (EMEG)	No					
	Thallium - Filtered	15	14	ND	5.3	2.7	2.4 (RSL)	Yes					
	Arsenic - Unfiltered	15	12	ND	14.2	2.9	0.02 (CREG)	Yes					
	Lead - Unfiltered	15	5	ND	119	11	15 (AL)	Yes					
	Thallium - Unfiltered	15	13	ND	9.5	3.6	2.4 (RSL)	Yes					
	Volatile Organic Compounds (µg/L)												
1,1,2,2-Tetrachloroethane								15	14	3	5	0.067 (RSL)	Yes
Semivolatile Organic Compounds (µg/L)													
bis[2-Ethylhexyl]phthalate								15	10	1	4	4.8 (RSL)	No

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Maximum Contaminant Levels; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide; ^gATSDR Reference Media Evaluation Guide; ^hUSEPA Action Level for Lead in drinking water

Table 9: Soil (0 to 2 feet depth) Sampling Results of the non-residential Off-site Areas (Source: Exponent 2004)

Analyte	No. of Analysis	No of NDs ^a	Concentration		Environmental Guideline CVs ^b	COPC ^c
			Minimum	Maximum		
Metals (mg/kg)						
Mercury	26	2	ND	554	33	Yes
Copper	22	0	13.5	202	60	No
Lead	22	0	3.61	410	150	Yes
Zinc	22	0	26.9	777	250	No
Semivolatile Organic Compounds (mg/kg)						
Benz[a]anthracene	22	1	ND	15	2	Yes
Benzo[a]pyrene	22	1	ND	18	2	Yes
Benzo[b]fluoranthene	22	1	ND	22	3	Yes
Benzo[k]fluoranthene	22	2	ND	11	1	Yes
Dibenz[a,h]anthracene	22	9	ND	2.1	0.5	Yes
Indeno[1,2,3-cd]pyrene	22	4	ND	6.8	1	Yes

^aNon-detects; ^bComparison Value; ^cContaminant of Potential Concern; ^dUSEPA Regional Screening Value; ^eATSDR Environmental Media Evaluation Guide; ^fATSDR Cancer Risk Evaluation Guide

Table 10: Summary of Exposure Pathways

Medium	Point of Exposure	Exposure Route	Exposed Population	Exposure Pathway Classification		
				Past	Present	Future
On-site						
Surface soils	Developed, Undeveloped and Marsh Areas	Ingestion	Residents, hunters, recreators	Completed	Eliminated	Eliminated
Surface water	On-site basin, West Ditch, and Marsh	Ingestion	Residents, hunters, recreators	Completed	Eliminated	Eliminated
Biota	On-site basin, West Ditch, and Marsh	Ingestion	Residents, hunters, recreators	Eliminated	Eliminated	Eliminated
Ambient Air	On-site areas	Inhalation	Residents, hunters, recreators	Completed	Eliminated	Eliminated
Off-site						
Surface Soil	Off-site areas	Ingestion, inhalation	Residents, hunters, recreators	Completed	Completed	Completed
	Residential properties	Ingestion, inhalation	Residents	Completed	Eliminated	Eliminated
	Municipal repair yard and wastewater treatment plant	Ingestion, inhalation	Workers	Completed	Completed	Completed
Biota	Berry's Creek	Ingestion	Residents, hunters, recreators	Completed	Potential	Potential

Table 11: Comparison of Surface Soil Exposure Dose associated with the developed area with the Health Guideline

Contaminants of Potential Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)			Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b			
Metals							
Arsenic	6.2	95% UCL	2.5 x10 ⁻⁵	3.15 x10 ⁻⁶	0.0003 (MRL ^d)	No	
Chromium	59	95% UCL	2.4 x10 ⁻⁴	3.0 x10 ⁻⁵	0.001 (MRL)	No	
Copper	1,700	95% UCL	6.9 x10 ⁻³	8.6 x10 ⁻⁴	0.04 (RfDo ^e)	No	
Mercury (total)	1,000 ^f	95% UCL	4.1 x10 ⁻³	5.1 x10 ⁻⁴	0.0003 (RfD)	Yes	
Thallium	1.8	95% UCL	7.36 x10 ⁻⁶	9.15 x10 ⁻⁷	0.000065 (RfD _o)	No	
Semivolatile Organic Compounds (SVOCs)							
Benz[a]anthracene	0.7	95% UCL	2.86 x10 ⁻⁶	3.56 x10 ⁻⁷	NA ^g		
Benzo[a]pyrene	0.57	95% UCL	2.33 x10 ⁻⁶	2.9 x10 ⁻⁷	NA		
Benzo[b]fluoranthene	0.72	95% UCL	2.94 x10 ⁻⁶	3.66 x10 ⁻⁷	NA		
Dibenz[a,h]anthracene	0.15	Max	6.14 x10 ⁻⁷	7.63 x10 ⁻⁸	NA		
Indeno[1,2,3-cd]pyrene	0.23	Mean	2.5 x10 ⁻⁷	9.41 x10 ⁻⁷	NA		

^aChild exposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 200 mg/day ingestion rate and 17.4 kg body weight; ^bAdult Exposure Scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate and 70 kg body weight; ^cHealth Guideline Value; ^dATSDR Minimal Risk Levels; ^eReference Dose in EPA Regional Screening Levels; ^fThe mean is calculated after excluding the sample 13,800 mg/kg in Table 1; ^gNot Available

Table 12: Comparison of Surface Soil Exposure Dose associated with the Undeveloped area with the Health Guideline

Contaminants of Potential Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Metals						
Antimony	8.2	95% UCL	3.36 x10 ⁻⁵	4.17 x10 ⁻⁶	0.0004 (Rfd ^d)	No
Arsenic	9.1	95% UCL	3.73 x10 ⁻⁵	4.63 x10 ⁻⁶	0.0003 (MRL ^e)	No
Cadmium	6.1	95% UCL	2.5 x10 ⁻⁵	3.10 x10 ⁻⁶	0.0001 (MRL)	No
Chromium	770	95% UCL	3.15 x10 ⁻³	3.92 x10 ⁻⁴	0.003 (Rfd)	No
Lead	800	Mean				Yes
Manganese	530	95% UCL	2.17 x10 ⁻³	2.70 x10 ⁻⁴	0.05 (Rfd)	No
Mercury	160	95% UCL	6.55 x10 ⁻⁴	8.14 x10 ⁻⁵	0.0003 (RfD)	Yes
Thallium	3.5	95% UCL	1.43 x10 ⁻⁵	1.78 x10 ⁻⁶	0.000065 (Rfd ^f)	No
Zinc	18,000	95% UCL	7.37 x10 ⁻²	9.16 x10 ⁻³	0.3 (MRL)	No
Semivolatile Organic Compounds (SVOCs)						
Benz[a]anthracene	2	95% UCL	8.19 x10 ⁻⁶	1.02 x10 ⁻⁶	NA ^g	
Benzo[a]pyrene	2.4	95% UCL	9.83 x10 ⁻⁶	1.22 x10 ⁻⁶	NA	
Benzo[b]fluoranthene	2.7	95% UCL	1.11 x10 ⁻⁵	1.37 x10 ⁻⁶	NA	
Benzo[k]fluoranthene	0.7	Mean	2.87 x10 ⁻⁶	3.56 x10 ⁻⁷	NA	
Dibenz[a,h]anthracene	0.27	Mean	1.11 x10 ⁻⁶	1.37 x10 ⁻⁷	NA	

Table 12: (Cont'd.)

Contaminants of Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Semivolatile Organic Compounds (SVOCs)						
Indeno[1,2,3-cd]pyrene	0.3	Mean	1.23 x10 ⁻⁶	1.52 x10 ⁻⁷	NA	
Bis[2-ethylhexyl]phthalate	54	95% UCL	2.21 x10 ⁻⁴	2.75 x10 ⁻⁵	0.02 (Rfd _o)	No

^aChild exposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 200 mg/day ingestion rate and 17.4 kg body weight; ^bAdult Exposure Scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate and 70 kg body weight; ^cHealth Guideline Value; ^dEPA Reference Dose; ^eATSDR Minimal Risk Levels; ^fReference Dose in EPA Regional Screening Levels; ^gNot Available

Table 13: Comparison of Sediment Exposure Dose associated with the Marsh area with the Health Guideline

Contaminants of Potential Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Metals						
Arsenic	40	Mean	1.64 x10 ⁻⁴	2.04 x10 ⁻⁵	0.0003 (MRL ^d)	No
Cadmium	22.2	Mean	9.09 x10 ⁻⁵	1.13 x10 ⁻⁵	0.0001	No
Chromium	1,000	Mean	4.09 x10 ⁻³	5.09 x10 ⁻⁴	0.003 (RfD ^e)	Yes
Mercury (total)	270	Mean	1.11 x10 ⁻³	1.37 x10 ⁻⁴	0.0003 (RfD ^f)	Yes
Semivolatile Organic Compounds (SVOCs)						
Benz[a]anthracene	0.6	Mean	2.46 x10 ⁻⁶	3.05 x10 ⁻⁷	NA ^f	
Benzo[a]pyrene	0.6	Mean	2.46 x10 ⁻⁶	3.05 x10 ⁻⁷	NA	
Benzo[k]fluoranthene	0.5	Mean	2.05 x10 ⁻⁶	2.54 x10 ⁻⁷	NA	
Dibenz[a,h]anthracene	0.48	Mean	1.97 x10 ⁻⁶	2.44 x10 ⁻⁷	NA	
Indeno[1,2,3-cd]pyrene	0.5	Mean	2.05 x10 ⁻⁶	2.54 x10 ⁻⁷	NA	

^aChild exposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 200 mg/day ingestion rate and 17.4 kg body weight; ^bAdult Exposure Scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate and 70 kg body weight; ^cHealth Guideline Value; ^dATSDR Minimal Risk Levels; ^eEPA Reference Dose; ^fNot Available

Table 14: Comparison of Surface Water Exposure Dose with the Health Guideline CVs

Contaminants of Potential Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Metals						
Arsenic	2.9	Mean	2.97 x10 ⁻⁶	7.38 x10 ⁻⁷	0.0003 (MRL ^d)	No
Lead	71.1	Mean				
Mercury (total)	5.45	Mean	5.58 x10 ⁻⁶	1.39 x10 ⁻⁶	0.0003	No
Thallium	3.6	Mean	3.68 x10 ⁻⁶	9.16 x10 ⁻⁷	0.000065 (RfDo ^e)	No
Volatile Organic Compounds (VOCs)						
1,1,2,2-Tetrachloroethane	1	Mean	1.02 x10 ⁻⁶	2.54 x10 ⁻⁷	0.02	No

^aChild exposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 50 mL/day ingestion rate and 17.4 kg body weight; ^bAdult Exposure Scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 50 mL/day ingestion rate and 70 kg body weight; ^cHealth Guideline Value; ^dATSDR Minimal Risk Levels; ^eReference Dose in EPA Regional Screening Levels

Table 15: Comparison of Surface Soil Exposure Dose associated with the off-site properties with the Health Guideline CVs

Contaminants of Potential Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline ^c (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Metals						
Mercury (total)	195	95%UCL	8.0 x10 ⁻⁴	9.92 x10 ⁻⁵	0.0003 (RfD)	Yes
Lead	150	Mean				No
Semivolatile Organic Compounds (SVOCs)						
Benz[a]anthracene	6.04	95%UCL	2.47 x10 ⁻⁵	3.07 x10 ⁻⁶	NA ^d	
Benzo[a]pyrene	6.5	95%UCL	2.66 x10 ⁻⁵	3.3 x10 ⁻⁶	NA	
Benzo[b]fluoranthene	8.2	95%UCL	3.35 x10 ⁻⁵	4.17 x10 ⁻⁶	NA	
Benzo[k]fluoranthene	4.4	95%UCL	1.8 x10 ⁻⁵	2.23 x10 ⁻⁶	NA	
Dibenz[a,h]anthracene	2.1	Max	8.6 x10 ⁻⁶	1.06 x10 ⁻⁶	NA	
Indeno[1,2,3-cd]pyrene	3.61	95%UCL	1.47 x10 ⁻⁵	1.83 x10 ⁻⁶	NA	

^aChild exposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 200 mg/day ingestion rate and 17.4 kg body weight; ^bAdult Exposure Scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate and 70 kg body weight; ^cHealth Guideline Value; ^dNot Available

Table 16: Calculated LECR associated with the Contaminants detected in the Developed Area

Contaminants of Potential Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose ^e (mg/kg/day)	CSF ^d (mg/kg/d) ⁻¹	LECR ^e
Polycyclic Aromatic Compounds (PAHs)								
Dibenz[a,h]anthracene	0.15	2	-	-	-	3.27 x10 ⁻⁸	4.1	1.34 x10 ⁻⁷
Benz[a]anthracene	0.7	2	0.1	0.07	0.735	1.6 x10 ⁻⁷	7.3	1.17 x10 ⁻⁶
Benzo[a]pyrene	0.57	2	1	0.57				
Benzo[b]fluoranthene	0.72	2	0.1	0.072				
Indeno[1,2,3-cd]pyrene	0.23	2	0.1	0.023				
Metals								
Arsenic	6.2	1				1.35 x10 ⁻⁶	1.5	2.02 x10 ⁻⁶
Chromium	59	1				1.28 x10 ⁻⁵	0.5 ^f	6.43 x10 ⁻⁶
Copper	1,700	NA ^g						
Mercury (total)	1,000	NA						
Thallium	1.8	NA						
							Sum =	9.6 x10 ⁻⁶

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cExposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dCancer Slope Factor; ^eLifetime Excess Cancer Risk; ^f(NJDEP 2009); ^gNot Available

Table 17: Calculated LECR associated with the Contaminants detected in the Undeveloped Area

Contaminants of Potential Concern	Max. Conc. (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose ^c (mg/kg/day)	CSF ^d (mg/kg/d) ⁻¹	LECR ^e
Polycyclic Aromatic Compounds (PAHs)								
Benz[a]anthracene	2	2	0.1	0.2	2.94	6.41 x10 ⁻⁷	7.3	4.68 x10 ⁻⁶
Benzo[a]pyrene	2.4	2	1	2.4				
Benzo[b]fluoranthene	2.7	2	0.1	0.27				
Benzo[k]fluoranthene	0.7	2	0.1	0.07				
Bis[2-ethylhexyl]phthalate	54					1.18 x10 ⁻⁵	0.014	1.65 x10 ⁻⁷
Metals								
Antimony	8.2	NA ^f						
Arsenic	9.1	1				1.98 x10 ⁻⁶	1.5	2.98 x10 ⁻⁶
Cadmium	6.1	1				1.33 x10 ⁻⁶	0.0018	2.39 x10 ⁻⁹
Chromium	770	NA				1.68 x10 ⁻⁴	0.5 ^g	8.4 x10 ⁻⁵
Lead	4,320							
Manganese	530	NA						
Mercury (total)	160	NA						
Thallium	3.5	NA						
Zinc	18,000	NA						
							Sum =	9.1 x10 ⁻⁵

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cExposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dCancer Slope Factor; ^eLifetime Excess Cancer Risk; ^fNot Available; ^g(NJDEP 2009)

Table 18: Calculated LECR associated with the Contaminants detected in the Marsh Area

Contaminants of Potential Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose ^c (mg/kg/day)	CSF ^d (mg/kg/d) ⁻¹	LECR ^e
Polycyclic Aromatic Compounds (PAHs)								
Dibenz[a,h]anthracene	0.48	2	-	-	-	1.05 x10 ⁻⁷	4.1	4.3 x10 ⁻⁷
Benz[a]anthracene	0.6	2	0.1	0.06	0.76	1.66 x10 ⁻⁷	7.3	1.21 x10 ⁻⁶
Benzo[a]pyrene	0.6	2	1	0.6				
Benzo[b]fluoranthene	0.5	2	0.1	0.05				
Indeno[1,2,3-cd]pyrene	0.5	2	0.1	0.05				
Metals								
Arsenic	40	1				8.72 x10 ⁻⁶	1.5	1.31 x10 ⁻⁵
Cadmium	22.2	1				4.84 x10 ⁻⁶	0.0018	8.71 x10 ⁻⁹
Chromium	1,000	1				2.18 x10 ⁻⁴	0.5 ^f	1.09 x10 ⁻⁴
Lead	280	NA ^g						
Mercury (total)	270	NA						
							Sum =	1.24 x10 ⁻⁴

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cExposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dCancer Slope Factor; ^eLifetime Excess

Cancer Risk; ^f(NJDEP 2009); ^gNot Available

Table 19: Calculated LECR based on Concentration of Contaminants detected in Surface Water

Contaminants of Potential Concern	Concentration (µg/L)	DHHS ^a Cancer Class	Exposure Dose ^b (mg/kg/day)	CSFi ^c (mg/kg/day) ⁻¹	LECR ^d
Metals					
Arsenic	2.9	1	3.16 x10 ⁻⁷	1.5	4.74 x10 ⁻⁷
Lead	71.1				
Mercury (total)	5.45	3	1.39 x10 ⁻⁶		
Thallium	3.6	NA ^e	9.16 x10 ⁻⁷		
Volatile Organic Compounds					
1,1,2,2-Tetrachloroethane	1	NA	2.54 x10 ⁻⁷		
				Sum =	4.74 x10 ⁻⁷

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified

^bExposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 50 mL/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^cCancer Slope Factor; ^dLifetime Excess Cancer Risk; ^eNot Available

Table 20: Calculated LECR associated with the Contaminants detected in the Off-site Areas

Contaminants of Potential Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose ^c (mg/kg/day)	CSF ^d (mg/kg/d) ⁻¹	LECR ^e
Polycyclic Aromatic Compounds (PAHs)								
Dibenz[a,h]anthracene	4.4	2	-	-	-	4.58 x10 ⁻⁷	4.1	1.88 x10 ⁻⁶
Benz[a]anthracene	6.04	2	0.1	0.604	8.4	1.83 x10 ⁻⁶	7.3	1.34 x10 ⁻⁵
Benzo[a]pyrene	6.5	2	1	6.5				
Benzo[b]fluoranthene	8.2	2	0.1	0.82				
Indeno[1,2,3-cd]pyrene	2.1	2	5	2.4				
Metals								
Lead	150	NA ^e						
Mercury (total)	195	NA						
							Sum =	1.52 x10 ⁻⁵

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cExposure scenario: 4 days per week during the 13 summer weeks, and three days per week during the 26 spring and fall weeks (NJDEP 2001), 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dCancer Slope Factor; ^eLifetime Excess

Cancer Risk; ^fNot Available

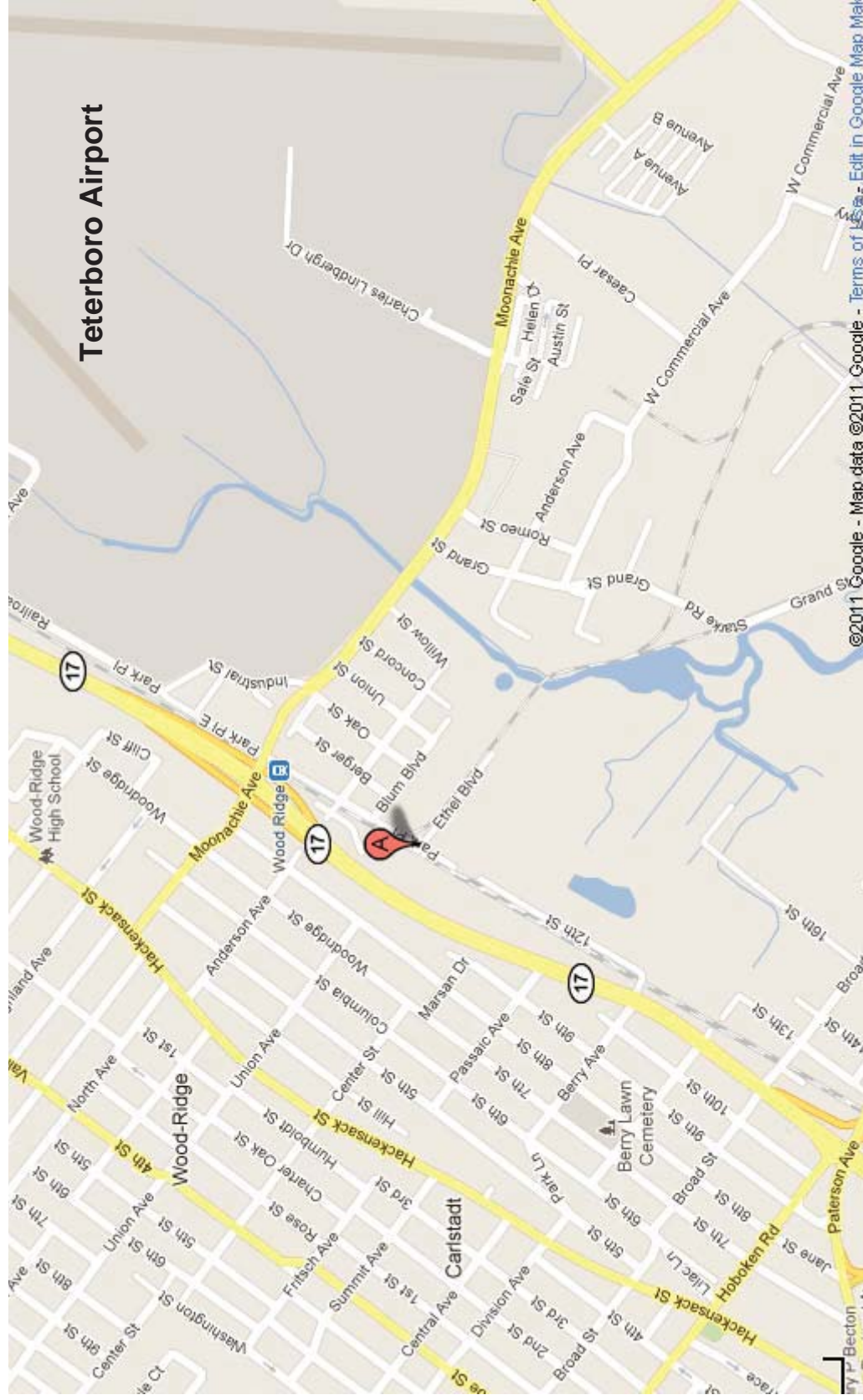
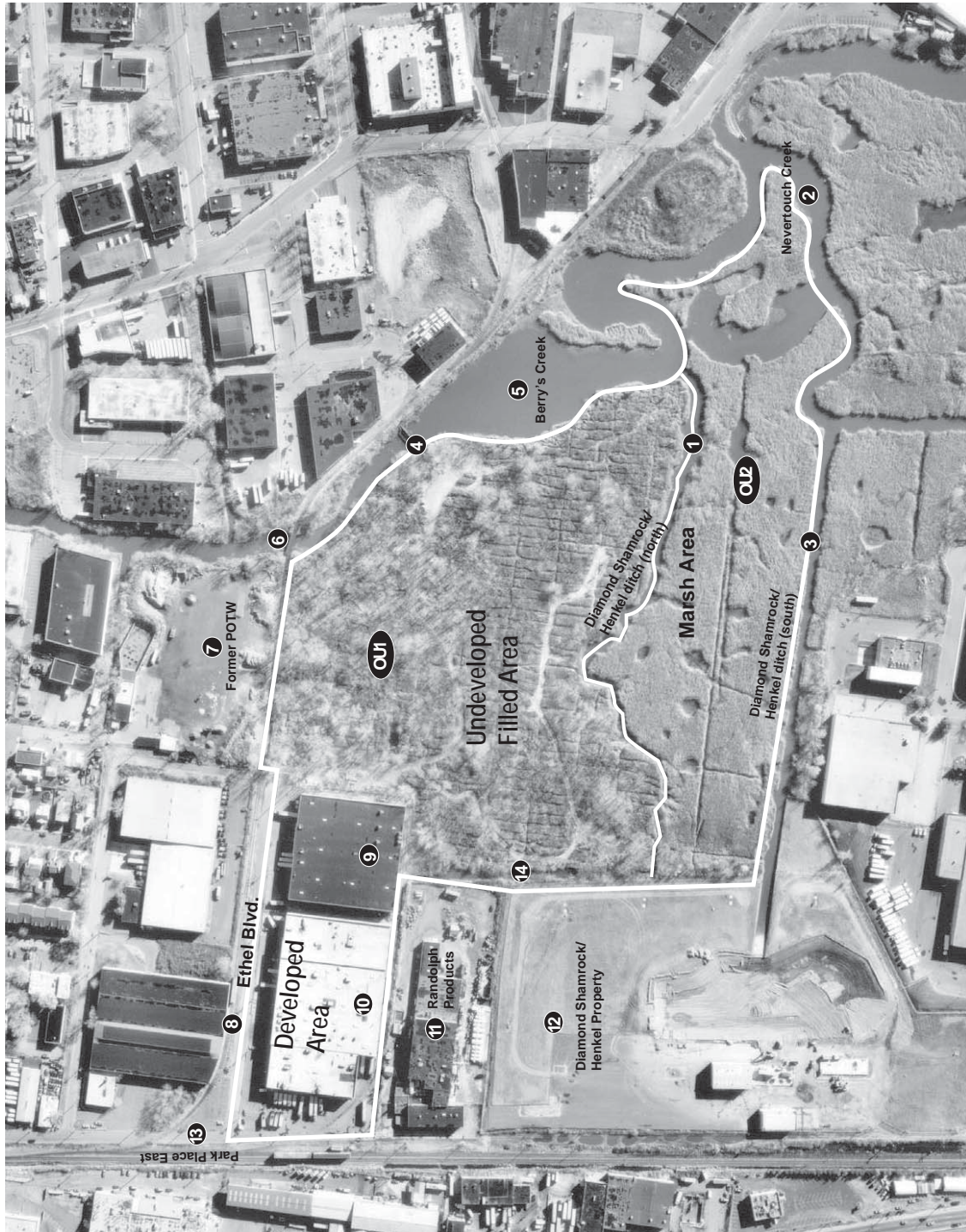


Figure 2: Location of the Ventron/Velsicol site



- LEGEND**
- 1 Diamond Shamrock/Henkel ditch (north)
 - 2 Never Touch Creek
 - 3 Diamond Shamrock/Henkel ditch (south)
 - 4 Tide gate
 - 5 Berry's Creek
 - 6 Railroad bridge
 - 7 Former POTW
 - 8 Ethel Boulevard
 - 9 Wolf warehouse
 - 10 U.S. Life warehouse
 - 11 Randolph Products
 - 12 Diamond Shamrock/Henkel Property
 - 13 Park Place East
 - 14 West ditch
 - Site boundary shown as white line
 - OU1 Operable Unit 1
 - OU2 Operable Unit 2
- 0 200 feet
approximate scale

Figure 3: Site layout map.

Photograph source: James Stewart, Inc. (November 29, 1997)



Photograph source: Intera

LEGEND


- 1 Mercury processing facility
 - 2 Ditch, debris
 - 3 Ditch
 - 4 Ditch partially buried by fill
 - 5 Ditch partially buried by fill
 - 6 Ditch
 - 7 Ditch
 - 8 Recent fill
 - 9 Former oxbow in Berry's Creek
 - 10 Tide gate
 - 11 Disturbed area
-  Site boundary shown as white line



Figure 4. April 7, 1951 aerial photograph.

2010 Population Density Wood-Ridge, Carlstadt, Moonachie

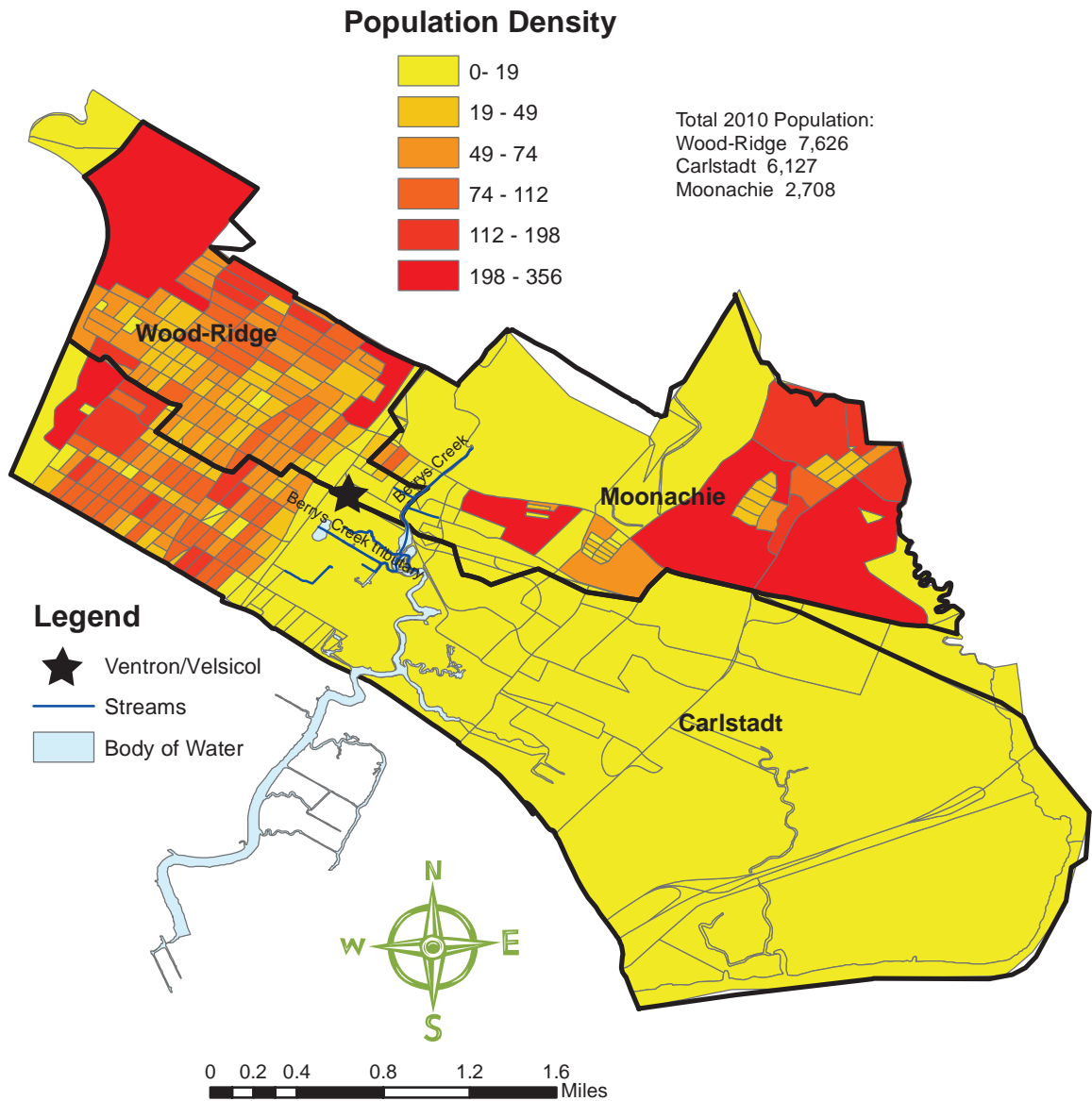
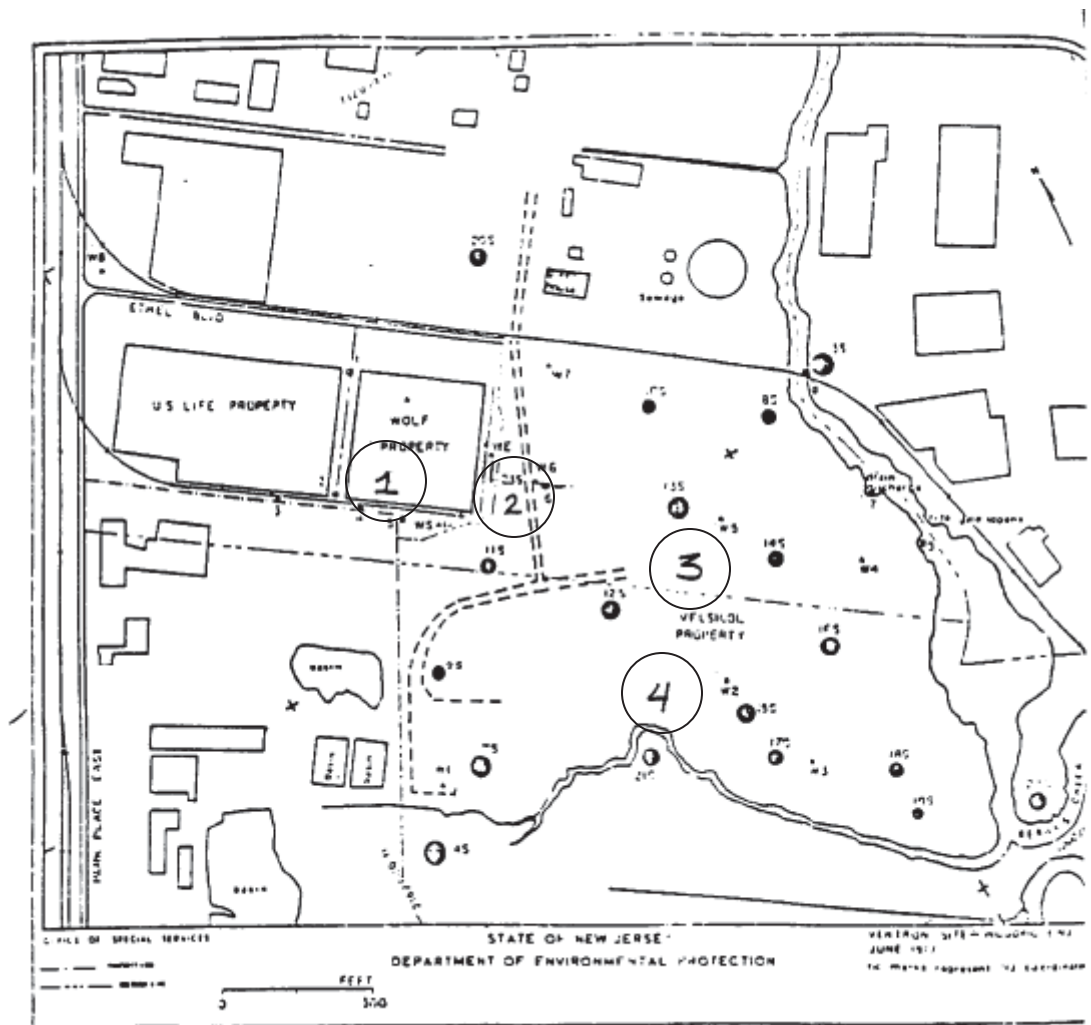
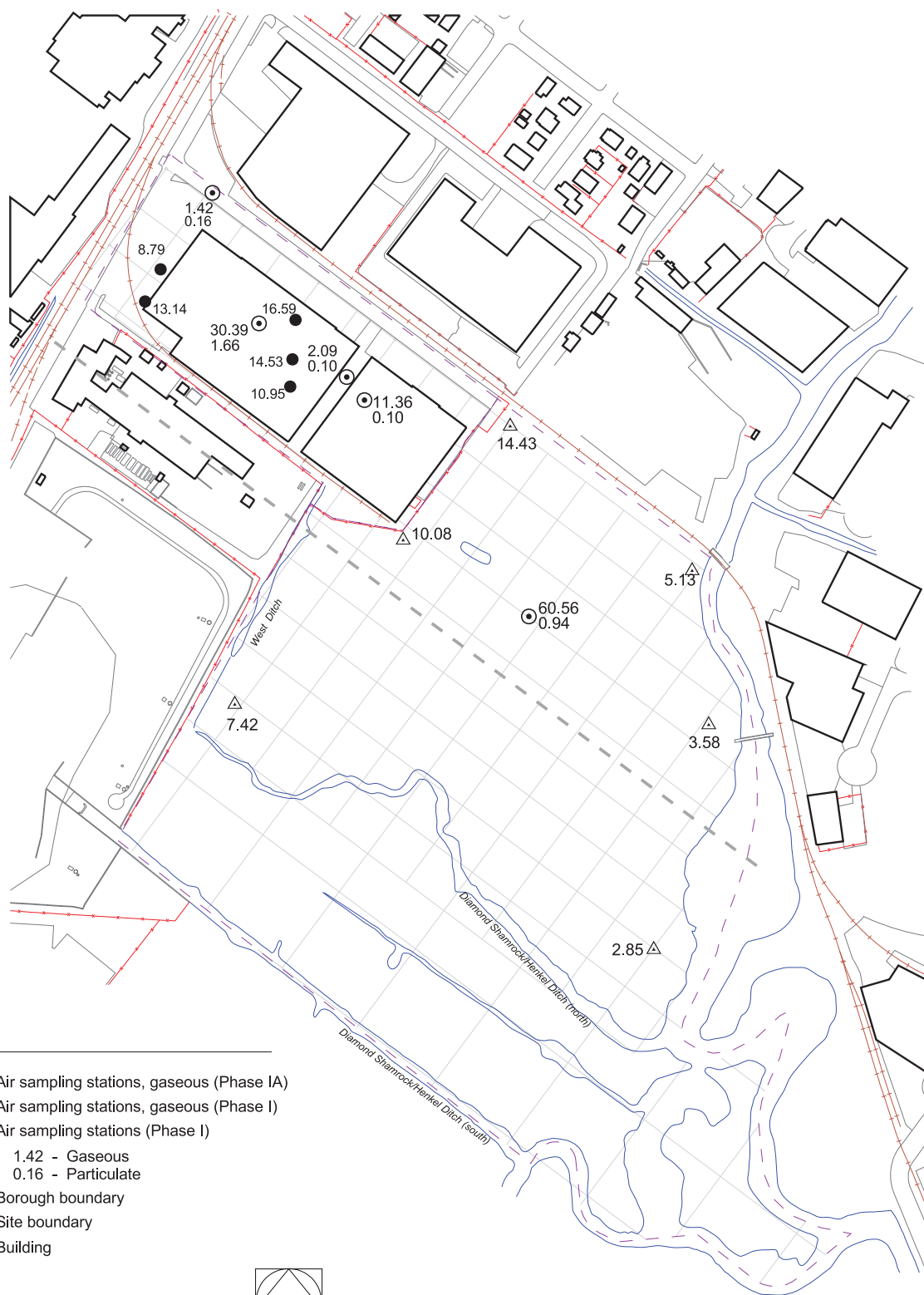


Figure 5: Demographic information for the Ventron/Velsicol site based on 2101 census data



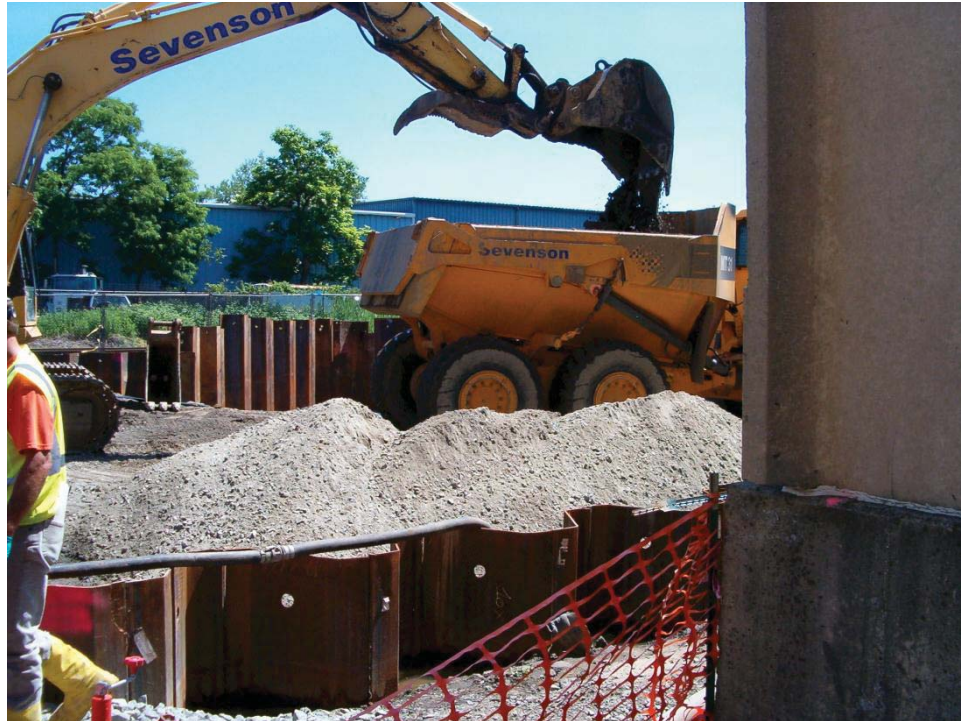
○ Air Sampling Station

Figure 6: Location of air sampling station



Source map survey by: James Stewart, Inc.

Figure 7: Particulate and gaseous mercury concentrations (ng/m^3) in air.



Photograph 1: Excavation of contaminated soil



Photograph 2: Field mercury vapor measurement



Photograph 3: Accessible areas by the public



Photograph 4: Accessible areas by the public



Photograph 5: Accessible areas under bridge



Photograph 6: Tide gate on Berry's Creek

Appendix A

Toxicologic Summaries

The toxicological summaries provided in this appendix are based on ATSDR's ToxFAQs (<http://www.atsdr.cdc.gov/toxfaq.html>). Health effects are summarized in this section for the chemicals of concern found at the site. The health effects described in the 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.

Aluminum. Aluminum occurs naturally and makes up about 8% of the surface of the earth. It is always found combined with other elements such as oxygen, silicon, and fluorine. Aluminum metal is silver-white and flexible. It is often used in cooking utensils, containers, appliances, and building materials. It is also used in paints and fireworks; to produce glass, rubber, and ceramics; and in consumer products such as antacids, astringents, buffered aspirin, food additives, and antiperspirants.

Low-level exposure to aluminum from food, air, water, or contact with skin is not considered harmful. Aluminum, however, is not a necessary substance for our bodies and too much may be harmful. People exposed to high levels of aluminum in air may develop respiratory problems including coughing and asthma from breathing dust. Although some studies show that people with Alzheimer's disease have more aluminum than usual in their brains, the relationship between Alzheimer's disease and aluminum is unknown. Aluminum may cause skeletal problems. Some sensitive people develop skin rashes from using aluminum chlorohydrate deodorants.

The DHHS, the IARC, and the EPA have not classified aluminum for carcinogenicity. Aluminum has not been shown to cause cancer in animals.

Arsenic 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 give you 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. Organic arsenic 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 Benzene is a colorless liquid with a sweet odor. It evaporates into the air very quickly and dissolves slightly in water. It is flammable and is formed from both natural processes and human activities. Benzene is widely used in the United States; it ranks in the top 20 chemicals for production volume. Some industries use benzene to make other chemicals such as plastics, resins, and nylon and synthetic fibers. Benzene is also used to make rubber, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include volcanoes and forest fires. Benzene is also a natural constituent of crude oil, gasoline, and cigarette smoke. Outdoor air contains low levels of benzene from tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions. Indoor air generally contains higher levels of benzene from products such as glues, paints, furniture wax, and detergents.

Breathing very high levels of benzene can result in death, while high levels 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. The major effect of benzene from long-term (365 days or longer) exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Some women who breathed high levels of benzene for many months had irregular menstrual periods and a decrease in the size of their ovaries. It is not known whether benzene exposure affects the developing fetus in pregnant women or fertility in men. Animal studies have shown low birth weights, delayed bone formation, and bone marrow damage when pregnant animals breathed benzene.

The USDHHS 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-ethylhexyl)phthalate Bis(2-ethylhexyl)phthalate is a colorless oily liquid that is extensively used as a plasticizer in a wide variety of industrial, domestic and medical products. It is an environmental contaminant and has been detected in ground water, surface water, drinking water, air, soil, plants, fish and animals.

Animal studies have indicated that the primary target organs are the liver and kidneys; however, higher doses are reported to result in testicular effects and decreased hemoglobin and packed cell volume. The primary intracellular effects of bis(2-ethylhexyl)phthalate in the liver and kidneys are an increase in the smooth endoplasmic reticulum and a proliferation in the number and size of peroxisomes. An epidemiological study reported no toxic effects from occupational exposure to air concentrations of bis(2-ethylhexyl)phthalate up to 0.16 mg/m³. Other studies on occupational exposures to mixtures of phthalate esters containing bis(2-ethylhexyl)phthalate have reported polyneuritis and sensory-motor polyneuropathy with

decreased thrombocytes, leukocytes and hemoglobin in some exposed workers. Developmental toxicity studies with rats and mice have shown that bis(2-ethylhexyl)phthalate is fetotoxic and teratogenic when given orally during gestation. Oral exposure has also been shown to result in decreased sperm count in rats.

Bis(2-ethylhexyl)phthalate is known to induce the proliferation of peroxisomes, which has been associated with carcinogenesis. Dose-dependent, statistically-significant increases in the incidences of hepatocellular carcinomas and combined carcinomas and adenomas were seen in mice and rats exposed to bis(2-ethylhexyl)phthalate in their diet for 103 weeks. An increased incidence of neoplastic nodules and hepatocellular carcinomas was also reported in rats. The EPA has classified antimony as a probable human carcinogen, on the basis of an increased incidence of liver tumors in rats and mice.

Cadmium Cadmium is a natural element in the earth's crust. All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics. Exposure to high levels of cadmium severely damages the lungs and can cause death. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea. Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones. Skin contact with cadmium is not known to cause health effects in humans or animals.

Chromium Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms: chromium(0), chromium(III), and chromium(VI). No taste or odor is associated with chromium compounds. The metal chromium, which is the chromium(0) form, is used for making steel. Chromium(VI) and chromium(III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

Chromium enters the air, water, and soil mostly in the chromium(III) and chromium(VI) forms. In air, chromium compounds are present mostly as fine dust particles which eventually settle over land and water. Chromium can strongly attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate much chromium from water.

Breathing high levels of chromium(VI) can cause nasal irritation, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium(VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin contact with certain chromium(VI) compounds can cause skin ulcers. Allergic reactions consisting of severe redness and swelling of the skin have been noted.

Several studies have shown that chromium(VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer. The WHO has determined that chromium(VI) is a human carcinogen. The USDHHS has determined that certain

chromium(VI) compounds are known to cause cancer in humans. The USEPA has determined that chromium(VI) in air is a human carcinogen.

It is unknown whether exposure to chromium will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to chromium(VI). It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults.

Copper High levels of copper can be harmful. Breathing high levels of copper can cause irritation of nose and throat. Ingesting high levels of copper can cause nausea, vomiting, and diarrhea. Very-high doses of copper can cause damage to liver and kidneys, and can even cause death.

Exposure to high levels of copper will result in the same type of effects in children and adults. We do not know if these effects would occur at the same dose level in children and adults. 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 are a very small percentage of infants and children who are unusually sensitive to copper.

Birth defects or other developmental effects of copper in humans are unknown. Animal studies suggest that high levels of copper may cause a decrease in fetal growth.

The most likely human exposure pathway is through drinking water, especially if the water is corrosive and copper pipes are used for plumbing. One of the most effective ways to reduce copper exposure is to let the water run for at least 15 seconds first thing in the morning before drinking or using it. This reduces the levels of copper in tap water dramatically.

Copper is found throughout the body; in hair, nails, blood, urine, and other tissues. High levels of copper in these samples can show copper exposures. However, these tests can not predict occurrence of harmful effects. Tests to measure copper levels in the body require special equipment.

Human carcinogenicity of copper is unknown. The USEPA has determined that copper is not classifiable as to human carcinogenicity.

Lead 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 swallows 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 Manganese is a naturally occurring metal that is found in many types of rocks. Pure manganese is silver-colored, but does not occur naturally. It combines with other substances such as oxygen, sulfur, or chlorine. Manganese occurs naturally in most foods and may be added to some foods.

Manganese is used principally in steel production to improve hardness, stiffness, and strength. It may also be used as an additive in gasoline to improve the octane rating of the gas. Manganese can be released to the air, soil, and water from the manufacture, use, and disposal of manganese-based products. Manganese cannot break down in the environment. It can only change its form or become attached to or separated from particles. The chemical state of manganese and the type of soil determine how fast it moves through the soil and how much is retained in the soil. The manganese-containing gasoline additive may degrade in the environment quickly when exposed to sunlight, releasing manganese.

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. This combination of symptoms when sufficiently severe is referred to as "manganism". Other less severe nervous system effects such as slowed hand movements have been observed in some workers exposed to lower concentrations in the work place. Nervous system and reproductive effects have been

observed in animals after high oral doses of manganese. The USEPA concluded that existing scientific information cannot determine whether or not excess manganese can cause cancer.

Studies in children have suggested that extremely high levels of manganese exposure may produce undesirable effects on brain development, including changes in behavior and decreases in the ability to learn and remember. We do not know for certain that these changes were caused by manganese alone. We do not know if these changes are temporary or permanent. We do not know whether children are more sensitive than adults to the effects of manganese, but there is some indication from experiments in laboratory animals that they may be.

Studies of manganese workers have not found increases in birth defects or low birth weight in their offspring. No birth defects were observed in animals exposed to manganese.

Mercury 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.

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).

Thallium. Thallium is a bluish-white metal that is found in trace amounts in the earth's crust. It is used mostly in manufacturing electronic devices, switches, and closures, primarily for the semiconductor industry. It also has limited use in the manufacture of special glass and for certain medical procedures. Thallium enters the environment primarily from coal-burning and smelting, in which it is a trace contaminant of the raw materials. Exposure to thallium may occur through eating food contaminated with thallium, breathing workplace air in industries that use thallium, smoking cigarettes, or contact with contaminated soils, water or air.

Exposure to high levels of thallium can result in harmful health effects. A study on workers exposed on the job over several years reported nervous system effects, such as numbness of fingers and toes, from breathing thallium. Studies in people who ingested large amounts of thallium over a short time have reported vomiting, diarrhea, temporary hair loss, and effects on the nervous system, lungs, heart, liver, and kidneys. High exposures can cause death. It is not known what the reproductive effects are from breathing or ingesting low levels of thallium over a long time. Studies in rats exposed to high levels of thallium showed adverse reproductive effects, but such effects have not been seen in people. Animal data suggest that the male reproductive system may be susceptible to damage by low levels of thallium.

The USDHSS, IARC, and the USEPA have not classified thallium as to its human carcinogenicity. No studies are available in people or animals on the carcinogenic effects of breathing, ingesting, or touching thallium.

Toluene. Toluene is a colorless liquid widely used as raw material in the production of organic compounds and as a solvent. It is readily absorbed from the gastrointestinal and respiratory tracts and, to a lesser degree, through the skin. Toluene is distributed throughout the body, with accumulation in tissues with high lipid content. It is metabolized in the liver, primarily to hippuric acid and benzoyl glucuronide, compounds that are rapidly excreted in the urine.

In humans and animals, the primary effect associated with inhalation exposure to toluene is central nervous system (CNS) depression. Short-term exposure of humans to 100-1500 ppm has elicited CNS effects such as fatigue, confusion, incoordination, and impairments in reaction time, perception, and motor control and function. Exposure to concentrations ranging from 10,000-30,000 ppm has resulted in narcosis and deaths. Prolonged abuse of toluene or solvent mixtures containing toluene has led to permanent CNS effects. Exposure to high concentrations of toluene (1,500 ppm) has produced hearing loss in rats. Hepatomegaly and impaired liver and kidney function have been reported in some humans chronically exposed to toluene. Toluene vapors may cause eye irritation, and prolonged or repeated dermal contact may produce drying of skin and dermatitis.

In experimental animals, subchronic inhalation exposure to 2,500 ppm toluene resulted in increased liver and kidney weights (rats and mice), increased heart weights (rats), increased lung weights, and centrilobular hypertrophy of the liver (mice). Chronic inhalation exposure to 600 or 1,200 ppm for 2 years produced degeneration of olfactory and respiratory epithelia of rats and minimal hyperplasia of bronchial epithelia in mice.

Subchronic oral administration of toluene at doses ranging from 312 to 5,000 mg/kg/day produced clinical signs of neurotoxicity at 2,500 mg/kg in rats and mice. Other effects observed at higher doses in rats included increased relative liver, kidney, and heart weights (females only) and necrosis of the brain and hemorrhage of the urinary bladder.

Equivocal evidence shows that exposure to toluene in utero causes an increased risk of CNS abnormalities and developmental delay in humans. Animal studies, in which toluene was administered by inhalation, showed that exposure results in fetotoxicity and delayed skeletal development but does not cause internal or external malformations in rats. An oral study noted an increased incidence of embryonic deaths, cleft palate, and maternal toxicity in mice administered 1 mL/kg toluene during gestation.

An increased incidence of hemolymphoreticular neoplasms was reported in rats exposed to 500 mg/kg of toluene by gavage for 2 years; however, results from two long-term inhalation studies indicate that toluene is not carcinogenic at concentrations up to 1,200 ppm. Based on U.S. Environmental Protection Agency guidelines, toluene was assigned to weight-of-evidence group D, not classifiable as to human carcinogenicity.

Vanadium. Vanadium is a compound that occurs in nature as a white-to-gray metal, and is often found as crystals. It 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. Vanadium is mostly combined with other metals to make alloys. 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. Vanadium is also mixed with iron to make important parts for aircraft engines. Small amounts of vanadium are used in making rubber, plastics, ceramics, and other chemicals.

Exposure to high levels of vanadium can effect lungs, throat, and eyes. Short and long term occupational exposures caused lung irritation, coughing, wheezing, chest pain, runny nose, and a sore throat. These effects stopped soon after they stopped breathing the contaminated air. Similar effects have been observed in animal studies. No other significant health effects of vanadium have been found in human. Data are unavailable to evaluate the ingestion pathway. Animals that ingested very large doses have died. Lower, but still high levels of vanadium in the water of pregnant animals resulted in minor birth defects. Some animals that breathed or ingested vanadium over a long term had minor kidney and liver changes.

No human studies are available on the carcinogenicity of vanadium. No increase in tumors was noted in a long-term animal study where the animals were exposed to vanadium in the drinking water. The DHHS, the IARC, and the EPA have not classified vanadium as to its human carcinogenicity.

Zinc. Zinc is a naturally occurring element. Zinc has many commercial uses as coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass, and bronze. Acute health effects associate with ingesting o flarge doses are stomach cramps, nausea, and vomiting. Low level chronic exposures to zinc can cause anemia and decrease the levels of good cholesterol. Effect of zinc on human reproductive system is unknown; infertility was observed in animal studies at large doses,

Inhaling large amounts of zinc (as dusts or fumes) can cause a specific short-term disease called metal fume fever. Chronic effects of breathing high levels of zinc are unknown. Zinc can cause skin irritation. The DHHS and the IARC have not classified zinc for carcinogenicity. Based on incomplete information from human and animal studies, the EPA has determined that zinc is not classifiable as to its human carcinogenicity.

Appendix B

Sample Non-cancer Exposure Dose and Lifetime Excess Cancer Risk calculation

Sample Non-cancer Exposure Dose calculation

For on-site areas, the non-cancer exposure dose associated with arsenic (See Table 11) was calculated by using the following formula:

$$\text{Exposure Dose (mg/kg-day)} = \frac{C * IR * EF}{BW}$$

where C = concentration of arsenic = 6.2 mg/kg
EF = 130/365
IR = ingestion rate = 200 mg/day
BW = body weight = 17.4 kg

Substituting the values –

$$\begin{aligned}\text{Exposure Dose (mg/kg-day)} &= \frac{6.2 \frac{\text{mg}}{\text{kg}} * 200 \frac{\text{mg}}{\text{day}} * \frac{\text{kg}}{10^6 \text{ mg}} * \frac{130}{365}}{17.4 \text{ kg}} \\ &= 2.53 \times 10^{-5} \text{ mg/kg-day}\end{aligned}$$

Sample LECR calculation

For on-site areas, the LECR associated with arsenic contaminated soil ingestion (See Table 16) was calculated by using the following formula:

$$\text{Exposure Dose (mg/kg-day)} = \frac{C * IR * EF}{BW} * \frac{ED}{AT}$$

where C = concentration of arsenic = 6.2 mg/kg
IR = ingestion rate = 100 mg/day
EF = 130/365
BW = body weight = 17.4 kg
ED = exposure duration = 30 years
AT = averaging time = 70 years

Substituting the values –

$$\begin{aligned}\text{Exposure Dose (mg/kg-day)} &= \frac{C * IR * EF}{BW} * \frac{ED}{AT} \\ &= \frac{6.2 \frac{\text{mg}}{\text{kg}} * 100 \frac{\text{mg}}{\text{day}} * \frac{\text{kg}}{10^6 \text{ mg}} * \frac{130}{365}}{70 \text{ kg}} * \frac{30}{70}\end{aligned}$$

$$= 1.35 \times 10^{-6} \text{ mg/kg-day}$$

$$\textbf{LECR = Exposure Dose * cancer slope factor}$$

$$= 1.35 \times 10^{-6} \text{ mg/kg-day} * 1.5 (\text{mg/kg-day})^{-1}$$

$$= 2.02 \times 10^{-6}$$

Appendix C

Summary of Public Comments and Responses

Summary of Public Comments and Responses Ventron/Velsicol Public Health Assessment

The NJDOH held a public comment period from March 4, 2015 through April 05, 2015 to provide an opportunity for interested parties to comment on the draft Public Health Assessment (PHA) prepared for the Ventron/Velsicol Superfund Site. Written comments were received from two organizations during the public comment period.

The NJDOH and ATSDR used the following steps in preparing responses to all significant public comments received during the public comment period: (1) all comment documents were reviewed and catalogued, and (2) a response was prepared for each comment. Questions regarding this summary or any aspect of this PHA may be addressed to the NJDOH at (609) 826-4984.

General Comment:

Comment# 1. The commenter stated that the scope and nature and timing of the remediation must be made clear to the public so that they understand past versus current conditions and thus can appropriately evaluate statements made concerning exposure potential and risk.

Response: The scope of the PHA was stated in the "Statement of Issues" section of the report. The exposure pathways and the status (i.e., past, present or future) of the exposure pathways were developed based on site history and on- and off-site remedial actions implemented for the site (see Completed, Potential and Eliminated Pathways sections of the draft report). A summary of exposure pathways identified for the site is presented in Table 10.

Comment# 2. The commenter noted as a threshold matter that the Draft PHA distinguished between (a) the Ventron/Velsicol Site and (b) the much larger and more complex Berry's Creek Study Area ("BCSA") that encompasses the watershed within which the Ventron/Velsicol Site as well as several other National Priorities List sites and numerous other contaminated sites are located. The Draft PHA specifies that the focus of this assessment is the former and not the latter, and that the BCSA may later be subject to its own, separate health assessment. The commenter endorses this approach. However, the commenter notes that the Draft PHA incorporates confusing references to Operable Unit 2 or OU-2 as well as statements and data that include descriptions of conditions and data from elsewhere within the BCSA that are not found at the Ventron/Velsicol Site. The commenter suggested that references to such conditions and data are confusing and misleading to the public, and that the PHA should be limited to the 7-acre "developed" portion and the 19-acre "undeveloped" portion of the Site.

Response: The draft PHA evaluated contamination data available for the 38-acre site. The site has been divided into three distinct areas: the 7-acre developed area, the 19-acre undeveloped filled area, and the 12-acre marsh area (also known as OU-2 and which is a part of BCSA). The text has been revised to further clarify this issue in the final version.

Comment# 3. The commenter stated that the Draft PHA was not limited to alleged potential health risks associated with the Ventron/Velsicol Site itself, but, rather, includes statements that only apply to the larger BCSA that is physically different from the Ventron/Velsicol Site and that includes many other unrelated sites. By including statements concerning the larger and much different BCSA, the Draft PHA departs from the purpose of providing information concerning the Site and is likely to cause significant confusion for the public.

Response: The PHA clearly identified the on- and off-site areas/properties that were evaluated in this PHA (see the beginning of “Site Conditions” section). Under USEPA oversight, the remedial investigation of the BCSA (which includes the 12-acre marsh area of this site) is being conducted by the potential responsible parties. The NJDOH, in cooperation with the ATSDR, will prepare separate health consultation(s) to evaluate the exposure pathways associated with BCSA. Further assessment of exposure pathway that were evaluated in this PHA will be conducted in the subsequent health consultation(s), if necessary and/or if additional contamination data become available.

Comment# 4. The commenter stated that the Draft PHA included references to historical contamination that was remedied years ago and therefore cannot present any ongoing potential health risk. These historical references to contamination no longer present are likely to confuse the public, provide the public with outdated and misleading information, and not serve any purpose in guiding future action at the Site.

Response: As emphasized in the ATSDR Public Health Assessment Guidance Manual (PHAGM, 2005), one of the main objectives of the public health assessment process is to evaluate past site-related contaminant exposures pathways. As such, presentation of past contamination data in the PHA are relevant and necessary. The data collected in the past were used to assess the past contaminant exposures.

Comment# 5. The commenter stated that the Draft PHA failed to properly describe and appropriately incorporate the extensive 2009 - 2010 remediation that was carried out across the 7-acre "developed" portion and the 19-acre "undeveloped" portion.

Response: All remedial activities conducted at the site were summarized in the “Previous Investigations/Actions” section of the PHA.

Comment# 6. The commenter noted that the Draft PHA on page 30 stated that a review of health outcomes may be conducted to assess the public health impacts of past completed exposure pathways. Such an assessment, if conducted, would be of no value due to the low likelihood of obtaining statistically valid findings with such a small sample size. As such, any reference to a future assessment of public health outcomes would likely be confusing and inappropriately alarming to anyone described as potentially affected. Therefore, this section should be omitted from the Draft PHA, or at a minimum, the text should be revised to explain that such an assessment would be of no value and should not be performed.

Response: Analysis of health outcome data has been a required part in the public health assessment process. However, the draft PHA clearly states that due of the small size of the

exposed population (i.e., about 80), an evaluation of health data would not produce statistically valid findings.

Comment# 7. The commenter stated that the conclusion 1 should be modified to make clear that the Ventron/Velsicol site poses no current or future health hazard. Conclusion 1 of the draft PHA state does not state clearly enough there is no current or future public health hazard associated with the site. The ATSDR's PHA Guidance makes clear that, where a site, such as the Ventron/Velsicol Site, poses *only* potential past health risks, the PHA should communicate clearly to the surrounding community that there is no current or future public health hazard.

Response: Conclusion 1 of the final PHA was revised to include the word “future”.

Comment# 8. The commenter stated that the conclusion 2 will only confuse the public and should be omitted from the draft PHA.

Response: This conclusion is based on the concentration of mercury detected in the biota in the past and is an integral part of the PHA. Currently, the remedial investigation of the BCSA (including biota evaluation) is being conducted by the potential responsible parties. As additional biota contamination data become available, the NJDOH and ATSDR will prepare a health consultation(s) to assess the health implications of this potential pathways.

Comment# 9. The commenter stated that the conclusion 3 is unnecessarily alarming and fails to meet the objective of providing effective public health hazard communications, and should be removed or at a minimum revised.

Response: The conclusion 3 was based on the contaminants detected in surface soil and ambient air. An assessment of the data indicated that potential for non-cancer adverse health effects associated with past exposures were possible in children and adults. Maximum cumulative lifetime excess cancer risks were estimated to be 1 in 10,000 to the exposed population. As such, any changes to conclusion 3 is not warranted.

Comment# 10. The commenter stated that the range of mean mercury concentrations cited on page 8 of the Draft PHA is for soil and sediment samples, not for biota. The reference should be deleted.

Response: The range of mean mercury concentrations cited on page 8 of the Draft PHA is for soil and sediment samples. The mistake has been corrected in the final version (page 10).

Comment# 11. The commenter stated that the Draft PHA’s conclusions regarding inhalation of mercury are based on data collected in 1978, and are therefore highly uncertain and no longer relevant. First, there may be analytical issues with the data, as the cited method is no longer used; it was replaced by Method 6009 in 1989. Second, the 95th percentile UCLs, commonly used in risk evaluations, are much lower than the maximum values (as shown by the range presented), indicating that there were anomalous spikes in the data that would have skewed the mean upward. Without additional information on the methodology or validation, these data should be used with caution if at all. In NJDEP sampling events in 1989 and 1990, only one air sample had

mercury above detection limits and that exceeded the PHA's environmental guideline of $0.2 \mu\text{g}/\text{m}^3$. Finally, there are more recent air samples collected as part of the Ventron/Velsicol Site RI/FS and on an ongoing basis both inside the warehouse buildings and ambient outside air adjacent to the buildings that provide more accurate and up-to-date information that should be communicated to the public. Since 1997, no air samples have exceeded $0.2 \mu\text{g}/\text{m}^3$, which is even lower than the chronic $0.3 \mu\text{g}/\text{m}^3$ value discussed in discrete comment 3 below. These samples clearly show that there is no risk from inhalation inside or outside of the buildings present on the Site.

Response: We agree with the commenter that the older analytical method for mercury measurement has been replaced by Method 6009 in 1989. As such, the analysis of ambient air data collected in 1978 has been deleted from the revised version, including UCLs mentioned in the comment above.

However, in July 1991, the ATSDR evaluated the potential worker and customer exposure to mercury at the site (ATSDR 1991). Ambient air sampling was conducted using a portable instrument (Jerome meter). Mercury was detected at a concentration of $20 \mu\text{g}/\text{m}^3$ at a point along the railroad tracks adjacent to the fence and $40 \mu\text{g}/\text{m}^3$ at an exhaust vent on the foundation of the furniture distribution center. A review of ambient air data and other relevant information (e.g., LOAEL and the uncertainty factor for mercury, exposure conditions, and the NJDEP Health Study Results) clearly indicated that on-site individuals and nearby residents were exposed to harmful levels of mercury in the past.

Comment# 12. The commenter stated that the estimated risk for inhalation of mercury on page 27 of the Draft PHA is based on comparison to a reference concentration ("RfC") for chronic exposure. The RfC is meant to be protective for people exposed continually over a lifetime. Similarly, ATSDR's environmental guideline comparison value (CV) of $0.2 \mu\text{g}/\text{m}^3$ is a chronic (365 days and longer exposure) value. The exposure scenario in the Draft PHA assumes 130 Site visits per year, or at least approximately one-third of the time assumed in the calculation of the RfC. Therefore, the chronic RfC and CV are inappropriate metrics because they do not correspond to the exposure being assessed. In fact, any trespassers to the Site would have less exposure than workers at the Site, and the National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limit ("REL") for workers is $50 \mu\text{g}/\text{m}^3$, assuming a 40-hour work week. For comparison, a trespasser accessing the Site 130 times a year would need to have remained at the Site for an average of approximately 15 hours per visit to equate to a 40-hour per week, 50 weeks a year worker exposure. Even the maximum 1978 inhalation values (which as discussed above are suspect) are far below the REL. This means that there would not be any inhalation risk even at the overly conservative 130 trespass visits a year.

Response: The mercury product manufacturing on on-site areas ceased in 1974. Subsequently, other businesses (which did not manufacture mercury products) occupied the site. The receptor for this completed exposure pathway are the employees and trespassers, therefore, occupational standards are not appropriate levels to be used for comparison. The receptors for this pathway have been clarified in the final version.

Comment# 13. The commenter stated that the basis for Conclusion 3 (page 2) highlights only arsenic in surface soil as being associated with lifetime excess cancer risks. However, PAH, arsenic, cadmium, and chromium risks were summed to yield the 1 in 10,000 risk estimate and chromium contributed the great majority of even that low level of risk. The Basis for Conclusion should be revised to reflect this. The same revision should be made in the Conclusions section in the last full paragraph on page 31.

Response: The basis for conclusion has been updated in the final version.

Comment# 14. The commenter stated that lifetime excess cancer risk (“LECR”) shown in Table 20 of 1.8×10^{-5} does not match the entry in the table of 1.33×10^{-5} .

Response: The typographical error in Table 20 has been corrected in the final version.

Comment# 15. The commenter stated that in several instances, the descriptions of the Site in the Draft PHA portray the Site before it was remediated, and thus provide an inaccurate account of current conditions at the Site. To avoid confusion and unnecessary alarm, these instances should be modified to make clear that they have been remediated.

Response: Assessment of past contaminant exposures is one of the main requirements of public health assessment (ATSDR 2005). The current condition of the site has been clearly described in the report (see conclusion #1 also).

Comment# 16. The commenter stated that the second full paragraph on page 4 describes the undeveloped portion of the Site as “Approximately 19 acres of filled but not developed land...” and thus makes no mention of the remediation. This description should be modified to read: “Approximately 19 acres of filled but not developed land that has been remediated with a cap under an approved USEPA cleanup plan and which USEPA has determined is suitable for commercial and industrial use....”

Response: There is a description of remediation activities in the Previous Investigations/Actions section which describes the remedial activities that were conducted under EPA’s oversight (including establishing a buffer zone between the capped area and the wetlands).

Comment# 17. The commenter stated that in the “Topography” paragraph on page 10, the phrase “the 7-acre developed area, the 19-acre filled area...” should be modified to acknowledge the completed remediation to read “the 7-acre developed and remediated area, the 19-acre remediated and capped former fill area....”

Response: The remedial activities were described in the “Previous Investigations/Actions” section of the PHA.

Comment# 18. The commenter stated that on page 23 in the paragraph that begins with “Lead:”, the mean concentrations of lead presented in the last sentence are prior to remediation. This should be noted so as not to confuse or unnecessarily alarm the public.

Response: As discussed above, one of the main objectives of a PHA is to evaluate past site-

related contamination.

Comment# 19. The commenter stated that on page 6, when describing NJDOH and ATSDR's 2010 site visit, the PHA states that "[h]uman use of the site appears to be limited to transients and trespassers." Representatives of commenter have been at the Site frequently since well before 2010 and have not observed any issues concerning transients or trespassers or evidence of such Site use. The fence securing the Site is locked with a chain and industrial lock, and the fence is inspected approximately monthly. The Draft PHA appears to rely on a fence that was broken at one point. The statement should be appropriately characterized or eliminated. Further, the statement in the Draft PHA should be expanded to acknowledge that the 2009 – 2010 remediation has left the property in a condition such that there is no unacceptable risk posed to any occasional transient or trespasser.

Response: The statements were based on conversations with individuals during the site visit and several longtime residents who used to visit the on- and off-site areas for recreational (crabbing, fishing) purposes.

Comment# 20. The commenter stated that as expressed in the ATSDR's PHA Guidance, it is important for the public to have confidence in the data upon which the Draft PHA is based. The Draft PHA should note that the work performed under the both the Stipulation and Supplementary Order and the RI/FS referenced on page 5 was conducted under agreement with the environmental agencies and therefore under their direct supervision. Thus, the public can be confident in the work performed and the data collected, upon which this Draft PHA is partially based.

Response: We agree and clearly state in the document that the remediation was conducted with USEPA oversight.

Comment# 21. The commenter stated that on page 17, the paragraph that begins "Incidental ingestion of contaminated soil from off-site (adjacent properties) areas (past, present and future):" is confusing. First, the term "off-site (adjacent properties)" should be specifically defined. Second, it is not specified what data suggest that these properties remain contaminated and thus are current or future risks. Offsite areas were identified and remediated during the 2009 – 2010 remediation conducted under USEPA oversight, so the commenter is not aware of any off-site adjacent areas that fit the description in the Draft PHA, with the exception of adjacent areas of the BCSA (see prior comments regarding the need to clearly separate the BCSA and any future PHA and this PHA for the Ventron/Velsicol Site.) Third, it is not likely that there are current or future exposures associated with these sites since the properties adjacent to the Ventron/Velsicol Site are currently either: 1) included in NJ programs to remediate contaminated sites; 2) being addressed as part of the BCSA investigation; or 3) now developed and covered by concrete, asphalt, or buildings. Accordingly, the text should be revised to indicate that no present health hazard could exist from the incidental ingestion of contaminated soil from off-site (adjacent properties).

Response: The description of off-site properties is provided on page 14 of the final version. The concentrations of contaminants detected are given in Table 9. During the preparation of this

document, the NJDOH staff did not learn of any information regarding the remedial status of these properties.

Comment# 22. The commenter stated that on page 17, in the paragraph that begins “Incidental ingestion of surface water (past):” the source of the statement “Area residents visited the site frequently for recreational purposes” should be provided. If the visits occurred prior to 1974 (or some other date), this should be stated so the public can properly assess what is meant by “past.” Moreover, it is likely that any past recreational activities were related to the BCSA and not the Site, and therefore should be included in any future PHA related to the BCSA.

Response: The statements were based on conversations with individuals during the site visit and several longtime residents who used to visit the on- and off-site areas for recreational (crabbing, fishing) purposes.

Comment# 23. The commenter stated that on page 17, the paragraph that begins “Ingestion of contaminated biota from offsite areas (past):” mentions only mercury contamination. Various USEPA documents indicate that Berry’s Creek also contains PCBs and a variety of other metals and organic contaminants. Thus, limiting the statement to only mercury is not accurate.

Response: The error has been corrected in the final version on page 18.

Comment# 24. The commenter stated that the Draft PHA’s acknowledgements that Berry’s Creek is the subject of a USEPA-supervised Remedial Investigation and will be the subject of a future health consultation should be noted in the summary on page 18, in the paragraph that begins “Ingestion of contaminated biota from off-site areas (present, future).”

Response: It has been noted in the paragraph.

Comment# 25. The commenter stated that on page 20, the sentence “The area residents accessed the site routinely for recreational purposes” should be revised to “Prior to the erection of a security fence in 1979, area residents could have accessed the site for recreational purposes.” Likewise, the reference to the assumed 130 Site visits per year should note that this number was highly conservative and related to the period of time prior to the erection of the fence in 1979. This would be more accurate and would allow the public to place the calculations and assessment in an appropriate (conservative) context.

Response: The suggested change has been made in the final version on page 21.

Comment# 26. The commenter stated that on page 20, in the “Developed Area” paragraph, it should be made clear that the mercury detections in soil at 0 – 2 feet depth were prior to remediation of the Site in 2009 –2010, and that soils have either been excavated and disposed at appropriate off-site facilities or are under concrete, asphalt or soil caps designed and placed under USEPA oversight, so the public can put the calculations in the appropriate context.

Response: The suggested change has been made in the final version on page 22.

Comment# 27. The commenter stated that on page 23, given the description of blood lead levels being slightly above the applicable reference value, it would be clearer if the statement, “Therefore, potential for adverse health effects associated with lead exposures is considered possible” was revised to say only a “slight” or “low” possibility exists, or some statement that acknowledges that the modeled blood levels are only slightly above the reference value. Stating that a potential for adverse health effects associated with lead without acknowledging the slight nature of any possible risk is misleading.

Response: No safe blood lead level in children has been identified. This is the language preferred by the ATSDR and NJDOH reviewers.

Comment# 28. The commenter stated that on page 26, the statement that “Residents, including children, have been exposed . . .” should be revised to “could have been exposed,” or “are assumed to have been exposed.”

Response: The text was revised to address the comment (see page 27).

Comment# 29. The commenter stated that for the same reasons given in comment 21 above, the discussion on page 27 of “Incidental ingestion of contaminated soil from off-site areas” should be modified to refer only to “past” health hazards. Also, the reference to Figure 4 on page 27 to a 1951 aerial photo of the area makes sense only if past health hazards are being discussed.

Response: The text was revised to address the comment (see page 18).

Comment# 30. For the same reasons given in discrete comment 25 above, the reference on page 29 to LECRs associated with ingestion of soil in the Undeveloped area of the Site should clarify that such potential exposures are relevant largely to the period prior to 1979, before the security fence was installed.

Response: The suggested change has been made in the final version.

Comment# 31. The commenter stated that the statement in the second full paragraph on page 30, “Based on the fact that oral and inhalation exposures were close to the adverse effect level (i.e., the LOAEL), there was a potential for non-cancer adverse health effects in children from exposures to mercury” is confusing and unnecessarily alarming. Throughout the Draft PHA, the NOAEL and LOAEL for mercury are described and placed into the appropriate context, but stating here that inhalation exposures were “close” to non-cancer health effect levels is vague and therefore confusing. The statement should make clear that exposure calculations are 6 to 360 times lower than the LOAEL so while there would not be an expectation of adverse health effects, because the levels are not being compared to a NOAEL, the possibility of adverse effects cannot be definitively ruled out. The statement also should be revised to clarify that the calculated exposure levels support at most a “low” potential for non-cancer adverse health effects, as detailed in Appendix A hereto, under very conservative exposure assumptions.

Response: The text was revised to address the comment (see page 31).

Comment# 32. The commenter stated that the sentence “The NJDOH and ATSDR conclude that past exposures to site-related contaminants at the Ventron/Velsicol site may have harmed people’s health” should be edited to clarify that such past exposures are not relevant to the Site since remediation was completed in 2010, and are based on very conservative exposure assumptions.

Response: As discussed above, one of the main objectives of a PHA is to evaluate past site-related contamination. The remedial actions that were implemented at the site and their timeline were summarized in the PHA.

Comment# 33. The commenter agreed with Conclusion 1, but is not a health agency and therefore, does not speak to past exposures (*i.e.*, Conclusions 2 and 3). However, it is important to clarify that with respect to Conclusion 2, the Remedial Investigation in the Berry's Creek Study Area is moving toward completion. The Remedial Investigation is where exposure via consumption of biota will be estimated. Information on current and future exposures via biota consumption in the absence of remediation will be included in the upcoming Baseline Human Health Risk Assessment.

Response: Comment acknowledged. We indicate in the next steps that the RI is being conducted and NJDOH and ATSDR will review the data when available.

Comment# 34. The assessment should clarify the portion of the site to which the conclusions are applicable. Do the conclusions apply to the upland portion of the site (Operable Unit 1) or to the Berry's Creek Study Area (Operable Unit 2)?

Response: We have further clarified the areas described on page 5 and 7.

Comment# 35. A Remedial Investigation for the marsh portion of the site as well as the Berry's Creek waterway is underway. Understanding that the PHA is a fluid document, it may need to be updated once a final remedy is selected for these areas of the site.

Response: Refer to comment #24 and response.

Comment# 27. General- Exposure by consuming biota from the Berry's Creek Study Area include contaminants of potential concern beyond mercury. PCBs, Cr, Pb, Mn and Zn are also considered contaminants of potential concern.

Response: Comment acknowledged.

Comment# 36. The commenter stated that the EPA's methodology for calculating risk and hazard is different from that of ATSDR. For example, EPA uses different screening levels and only uses mean contaminant concentrations as the EPC when assessing lead. All other contaminants use the 95% (or greater) UCL or maximum detected concentration. In this document, ATSDR used mean concentrations for several other contaminants, including PAHs. However, based on the data included in this report and pathways evaluated, we arrive at similar conclusions.

Response: Comment acknowledged.

Comment# 37. Page 3, second paragraph - The Proposed Plan and the Record of Decision were issued by NJDEP with concurrence by USEPA.

Response: This information is added to the final version.

Comment# 38. Page 5, second paragraph - The information on current property ownership should be confirmed and updated as appropriate.

Response: The ownership information was obtained from the site-related reports (see References) reviewed during the preparation of the PHA.

Comment# 39. Page 6, fifth paragraph - The potentially responsible parties have volunteered to provide and post signs warning about the Newark Bay Complex fish consumption advisories.

Response: Comment acknowledged. This has been added to the revised the PHA on page 7.

Comment# 40. Page 10, second paragraph - Operable Unit 2 of the Ventron/Velsicol Site is the Berry's Creek Study Area. The BCSA boundaries are not discussed within the current document, but include the waterways of Berry's Creek as well as the tributaries and adjacent marshes.

Response: The BCSA areas and boundaries are added to the revised version.

Comment# 41. Page 10, third paragraph - The small basin described in this paragraph was eliminated as part of the remediation.

Response: This information is added to the final version as a footnote on the bottom of page 11.

Comment# 42. Page 31, second paragraph of Conclusions - This paragraph should clarify that the findings refer to Operable Unit 1, and not the Berry's Creek Study Area (OU2).

Response: As stated above have further clarified the areas described on pages 4 and 6.