Public Health Assessment

Final Release

SHERWIN-WILLIAMS/HILLIARDS CREEK SITE
GIBBSBORO, CAMDEN COUNTY, NEW JERSEY

EPA FACILITY ID: NJSFN0204181

Prepared by the
New Jersey Department of Health and senior Services

AUGUST 12, 2009

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

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

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Prepared by:

New Jersey Department of Health and Senior Services
Environmental and Occupational Health Surveillance Program

Under a Cooperative Agreement with the
U.S. Department of Health and Human Services
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Summary

Introduction

On April 18, 2006, the United States Environmental Protection Agency proposed to add the Sherwin-Williams/Hilliards Creek site to the National Priorities List. Contaminants, particularly volatile organic compounds and lead have been detected in the groundwater at the former facility and lead and arsenic have been detected in the sediment, floodplain soil and wetlands associated with the Hilliards Creek. The contamination extends from the former facility to the entire length of the Hilliards Creek. Congressional mandate requires that a Public Health Assessment be prepared for sites listed or proposed to be added to the National Priorities List.

Through a Cooperative Agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), the New Jersey Department of Health and Senior Services (NJDHSS) prepared a Public Health Assessment (PHA) of the Sherwin-Williams/Hilliards Creek site.

ATSDR and NJDHSS top priority is to ensure that the community around the site has the best information possible to safeguard its health.

Conclusions

The NJDHSS and ATSDR have reached four conclusions in this Public Health Assessment on the Sherwin-Williams/Hilliards Creek site:

Conclusion 1

NJDHSS and ATSDR conclude that likely lead exposures to area children and lifetime excess cancer risks associated with the sediment and floodplain soils of Hilliards Creek in the past may have harmed people's health.

Basis for Conclusion

In the past, people were exposed to lead contamination by accidentally swallowing soil and sediment from the Hilliards Creek floodplain and adjacent wetlands. The exposures are partially interrupted due to the removal of lead contaminated soil from an area along the United States Avenue near the Bridgewood Lake, one residence located at Kirkwood Road and construction of fence (partial). Children who swallowed sediment from the Hilliards Creek can be at increased risk for elevated blood lead levels. Lead from the sediment can also affect fetuses, if pregnant women swallow the lead-contaminated sediment.

The likelihood of adverse non-cancer health effects in children based on the concentrations of arsenic detected in the soil (0 – 2 feet

	depth) were determined to be low. The lifetime excess cancer risk from exposure to arsenic contamination is estimated to be low, in comparison to the background risk of cancer.
Next Steps	The USEPA should implement permanent remedy for the site as soon as feasible.
Conclusion 2	*The NJDHSS and ATSDR cannot conclude whether past and/or current exposures to potable water, indoor air and consumption of plants, fish and game from the Sherwin-Williams/Hilliards Creek site may have harmed people's health.*
Basis for Conclusion	Potable water, indoor air and biota (i.e., plants, fish and game) data were unavailable at the time the PHA was prepared.
Next Steps	As additional off-site contamination data (e.g., potable water, indoor air, plants, fish and game) become available, the NJDHSS and ATSDR will prepare a health consultation(s) in order to evaluate the public health implications of potential contamination. Until such testing is completed, it is prudent to recommend against consuming biota from the site.
Conclusion 3	*The NJDHSS and ATSDR cannot conclude whether current exposures to soil and sediment at the Sherwin-Williams/Hilliards Creek site could harm people's health.*
Basis for Conclusion	Although Sherwin-Williams implemented interim remedial measures to address the most contaminated on-site media, characterization and delineation of on- and off-site areas have not been completed.
Next Steps	The USEPA should complete the remedial investigation as soon as feasible. As additional soil and sediment contamination data become available, the NJDHSS and ATSDR will prepare a health consultation(s) to assess the health implications of potential contamination.

Conclusion 4	The blood lead levels measured in Gibbsboro children are similar to statewide average levels.
Basis for Conclusion	Childhood blood lead level data from the state Childhood Lead Poisoning Surveillance System for the years 1999 through 2006 was evaluated. A total of 137 Gibbsboro children (age range 0.7 to16.5 years) were tested during this period. No children were found to have a blood lead level above the Centers for Disease Control and Prevention guideline during this time period. The geometric mean blood lead level was 2.9 μg/dL with a 95% confidence interval of 2.7 to 3.1 μg/dL.
Next Steps	A review of other health outcome data is not recommended due to the relatively small size of the impacted population.
For More Information	Copies of this report were made available to concerned residents in the vicinity of the site via the township library and the internet. NJDHSS will provide educational materials on environmental exposures to lead and arsenic to local physicians upon request. Questions about this PHA should be directed to the NJDHSS at (609) 584-5367.

Statement of Issues

On April 18, 2006, the US Environmental Protection Agency (USEPA) proposed to add the Sherwin-Williams/Hilliards Creek site to the National Priorities List (NPL). As required by the 1986 Superfund Amendments and Reauthorization Act (SARA) to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, the Agency for Toxic Substances and Disease Registry (ATSDR) is mandated to conduct a Public Health Assessment for each site listed or proposed to be added to the NPL.

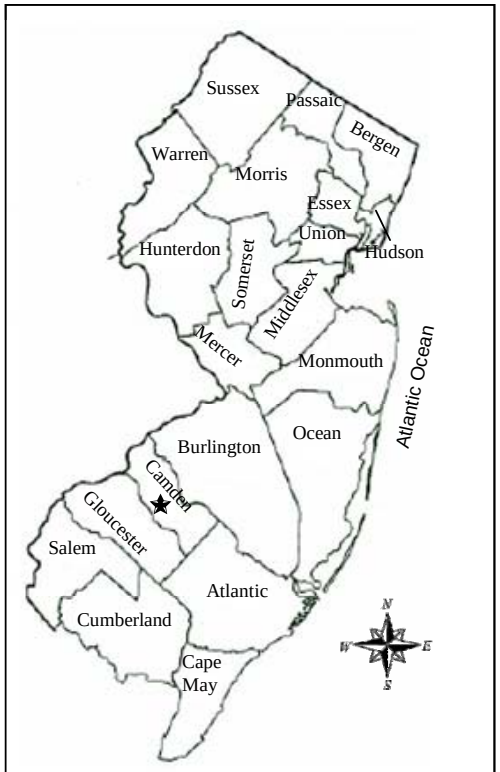

Figure 1: Location of Sherwin-Williams/ Hilliards Creek Site

The Sherwin-Williams/Hilliards Creek site is located in a residential and commercial area of Gibbsboro, Camden County, New Jersey (see Figure 1). The site includes the former facility, the Hilliards Creek and the floodplain, wetland and adjacent residential properties (USEPA 2006). The facility operated from 1849 to 1976 and manufactured primarily white lead paints, varnishes, and lacquer. The John Lucas Company owned the manufacturing plant from 1849 to approximately 1930, when Sherwin Williams acquired control of the company. In 1981, the property was sold to a private developer. While the property was being reconfigured, contractors identified contamination seeping out of the ground. Investigations into the source of the pollution revealed that groundwater underlying the facility buildings and soil surrounding structures, such as above ground raw material storage tanks, waste disposal lagoons and buildings, are contaminated.

The Hilliards Creek is located to the south of the former facility. The creek receives overflow from the Silver Lake and flows through residential areas and the Gibbsboro Nature Preserve (also known as Hilliards Creek Wildlife Refuge), which includes numerous walking trails for area residents. Results of soil and sediment sampling indicated presence of lead contamination in the sediment, floodplain and adjacent residential properties (USEPA 2006). Contaminated sediments were also detected in wetlands associated with the Hilliards Creek.

Through a cooperative agreement with the ATSDR, the New Jersey Department of Health and Senior Services (NJDHSS) prepared this public health assessment in order to determine the public health implications of past, current, and future exposures

associated with on-site contamination. Further investigation and delineation of off-site contamination are ongoing.

Background

Site Description and History

The Sherwin-Williams/Hilliards Creek site is located in a residential and commercial area of Gibbsboro, Camden County (see Figures 2). The site encompasses approximately 60 acres and is bordered to the north by the Silver Lake (see Photograph 1), to the east and west by residential dwellings and small businesses and to the south by the Hilliards Creek, open space and woodlands. Hilliards Creek flows southwesterly through the former facility, under Foster Avenue, then turns west under West Clementon Road, receives the outflow of Bridgewood Lake (see Photograph 2), and continues west to Kirkwood Lake. Approximately 1,000 feet upstream from Kirkwood Lake, Hilliards Creek receives surface water flow from Nichols Creek.

The property was originally developed around the turn of the 19th century as a saw mill, and subsequently, a grain mill (USEPA 2006). In 1851, the John Lucas Company purchased the property and converted the existing mill into a paint and varnish manufacturing facility (see Photograph 3). The plant was expanded at various stages to accommodate new operations such as grinding white lead and colors in oils. The facility included areas for the unloading of raw materials from railroad cars, raw materials tank farms including storage tanks, storage areas for drummed raw materials, an industrial/domestic wastewater disposal system consisting of unlined percolation/settling lagoons, waste disposal areas for paint sludge, and drum cleaning areas (see Figure 3). The Company developed and manufactured oil-based paints, varnish, lacquer, dry colors, and ready-mixed linseed oil paints. Raw materials used included lead oxide, zinc oxide, lead chromate, ferrous sulfate, sulfuric acid, linseed oil, and various solvents. The mixing and processing of raw materials took place in a number of specialized buildings within the facility. In 1930, the Lucas Company merged with Sherwin-Williams of Cleveland, Ohio. Manufacturing operations were terminated at the facility in late 1976 and early 1977. The facility was permanently closed on September 1, 1978 (USEPA 1978).

Wastes generated from the plant were disposed of in Hilliards Creek, the Route 561 Dump Site, and the US Avenue Burn Site (see Figure 4). The US Avenue Burn Site and Route 561 Dump Site have been listed and proposed to be listed in the NPL as separate sites, respectively.

In 1976, Sherwin-Williams conducted a subsurface investigation that included the former lagoon area. The investigation characterized topographic features and subsurface conditions (Alfred McClymont 1979). Sludge material was encountered in the pond, sludge pit, and holding pond ranging from two to five feet in the pond and 15 feet in the

sludge pit and three feet in the holding pond. In 1979, closure of the lagoon area was performed and included removal of 8,096 cubic yards of sludge material.

In 1981, the property was sold to a private developer. Development of the property included demolition and/or renovation of existing structures and construction of new office, manufacturing, warehouse spaces and re-grading of adjacent areas. In 1983, presence of seepage of an "oily substance" ("the petroleum seep") was reported to the New Jersey Department of Environmental Protection (NJDEP). It was reported that the petroleum seep was emanating from the parking lot at the facility, flowing overland to a storm water catch basin in the parking lot and discharging through riprap to the Hilliards Creek. Investigations of the petroleum seep indicated the presence of hazardous substances in the groundwater underlying the former facility and soil surrounding the structures at the plant, such as above ground storage tanks and buildings. The following is a partial list of contaminants detected in the seep sample (NJDEP 1990):

Contaminants	**Concentration (µg/L)[a]**
Benzene	18,000
Toluene	7,750
Sec-Butylbenzene	3,025
p-Xylene	1,170
m-Xylene	7,530
Ethyl Benzene	7,380
Cumene	1,280
n-Propylbenzene	1,580
1,2,3-Trimethylbenzene	5,900
1,3,5-Trimethylbenzene	3,490
Tetrachloroethene	7,605

[a]micrograms of contaminant per liter of water

The buildings are currently being used as office and light industrial operations and are called the Paint Works Corporate Center.

In January, 1990, NJDEP issued a Spill Act Directive requiring the implementation of a Remedial Investigation/Feasibility Study (RI/FS) for the former facility areas (NJDEP 1990). Sherwin-Williams entered into an Administrative Consent Order (ACO) with the NJDEP to conduct the RI/FS. The RI was conducted for the former facility from August 1991 through January 2000; analysis of soil, groundwater and sediment samples found lead and organic contamination (Weston 2001). Seeps located on the facility were identified as an area of Immediate Environmental Concern (IEC). Sherwin-Williams entered into an ACO with the NJDEP to address this IEC. A soil vapor extraction (SVE) system was installed in the area of the seeps, and a free-phase product recovery (FPR) system was installed in the area of former Tank Farm A (see Photograph 4). As of June 20, 2002, a total of 44,785 gallons of free product have been recovered and removed off site for disposal (USEPA 2006). Approximately 8,275 gallons of this total volume collected was primarily product from the product recovery

tank. The remaining 36,510 gallons of product were collected during the groundwater seep response and recovery efforts associated with the SVE and FPR system.

In 1998, lead contamination in Hilliards Creek was identified when sediment samples were collected from the creek to establish reference (background) concentrations (USEPA 2006). One of the sediment samples collected from adjacent to a trail in the Gibbsboro Nature Preserve area contained 221,900 milligrams of lead per kilograms of soil (mg/kg). Subsequently, on September 30, 1999, the USEPA Region 2 and Sherwin-Williams signed an Administrative Order of Consent (AOC) for a removal action that required delineation of the extent of contamination at accessible areas along Hilliards Creek; prevent direct contact with the contamination by use of engineering controls in accessible areas; obtain access; post signs where appropriate; and conduct site inspections on a quarterly basis. The removal action also included sampling of Hilliards Creek and residential properties near Hilliards Creek to delineate the extent of lead-contaminated soil and the installation of a fence around lead-contaminated soil adjacent to Hilliards Creek (see Photograph 5). In response, Sherwin-Williams removed lead contaminated soil from the railroad track area along the United States Avenue (also known as Rail Road site) and constructed a fence around areas in Hilliards Creek containing contaminated sediment. A number of investigations were conducted to determine the source of the lead contamination. The creek and the wetland sediments and the creek's flood plain soils indicated the presence of site-related contaminants. The contamination delineation also included a private residential property (165 Kirkwood Road) located on the flood plain of Hilliards Creek (see Photograph 6). The soil sampling results of the property indicated lead concentrations ranging from non-detect to 38,800 mg/kg. In 2001, soil samples were collected to further delineate of lead contamination in the property. In 2003, an interim removal action was completed consisting of removal of the top 6 inches of soil.

On April 18, 2006, the USEPA proposed to add the Sherwin-Williams/Hilliards Creek site to the National Priorities List (NPL).

Demographics

Using 2000 United States Census data, the ATSDR estimates that there are about 16,000 individuals residing within a one mile radius of the Sherwin-Williams/Hilliards Creek site (see Figure 5).

Site Visit

On November 02, 2006, a site visit of the Sherwin-Williams/Hilliards Creek site was conducted. Individuals present during the site visit were Sharon Kubiak, Glenn Pulliam, and Tariq Ahmed of the NJDHSS, Leah Escobar of the ATSDR, representatives of the EPA, Sherwin-Williams, Inc. and Weston Solutions, Inc. Weston Solutions, Inc., represented Sherwin-Williams, the responsible party (RP) of the former Sherwin-Williams facility.

The site visit started with a presentation by the representative of the Sherwin-Williams, Inc. The site background, location of various contaminated areas and remediation status of contaminated areas were discussed. The Sherwin-Williams representative also indicated that sampling for remedial investigation of on- and off-site areas has not been completed and sampling workplans for the following areas are being developed:

(1) potable wells for residences along United States Avenue,
(2) remaining 200-feet transects located along Hilliards Creek (downstream areas), and,
(3) residential properties adjacent to Hilliards Creek.

During the site visit, the former paint manufacturing buildings (currently know as the "The Paint Works Corporate Center") were visited. The buildings are occupied by various businesses; a pedestrian walk surrounds Silver Lake. The approximate location of the various processing areas, former gas stations, underground, above ground storage tank (UST and AST) areas, railroad tracks and transfer areas and wastewater treatment lagoon areas were pointed out.

The former facility is surrounded by residential properties; the nearest homes are located across United States Avenue (see Photograph 7). Silver Lake discharges into Hilliard Creek through an underground culvert system that crosses under the parking lot between the lake and Foster Avenue. The creek returns to open flow 200 feet south of Foster Avenue. A public school, library, and municipal offices are located approximately 0.2 mile west of the corporate center, along Kirkwood Road. The creek is accessible from residential backyards that lack continuous fencing. The area to the east of the Bridgewood Lake also has walking trails, and a shooting range is located on the southern shore of Bridgewood Lake.

The Hilliards Creek flows from the Silver Lake under the Foster Avenue. One of the groundwater seep areas is located near the headwaters of the Hilliards Creek. The Sherwin-Williams representative showed the SVE and the FPR systems installed to the south of Foster Avenue (see Photograph 8). The site visit also included the Route 561 Dump site, the United States Avenue Burn site and areas to the east of Bridgewood Lake (where lead concentration of 221,900 mg/kg was detected) where soil removal activities were conducted.

Community Health Concerns

The NJDHSS communicated with town administration in order to hold availability sessions for residents to identify community concerns, and to provide information to residents about exposure pathways and the contaminants of concern. The town administration did not express any interest to hold availability sessions.

The NJDHSS staff had informal conversation with two of the individuals. One of the individuals was born and lived in a house located to the east of the US Avenue Burn

site for 18 years. The individual stated that the children used to use the facility areas and the Hilliards Creek for recreation; they used to trespass the areas to go to school. The US Avenue Burn site was used by the 4H Club for various outdoor activities. The area lakes were used by local resident for fishing.

The other individual is in his 50s and lives in the next town located to the south of Gibbsboro Wildlife Refuge. During 1970s and 1980s, he and his friends frequently visited the Hilliards Creek and adjacent lakes for swimming and fishing. Due to the construction of new homes along Kirkwood Road, the lake and creek water quality deteriorated and the fishing declined.

Past ATSDR/NJDHSS Involvement

In 1999, the ATSDR and NJDHSS prepared a health consultation for Hilliards Creek (ATSDR 1999a). ATSDR and NJDHSS evaluated the analytical data collected in 1998 where lead was detected at 221,900 mg/kg in a sediment sample collected in the Hilliards Creek Wildlife Refuge. The ATSDR and NJDHSS concluded that an urgent health hazard exists to children and adults who use the refuge. The area where the sediment sample was collected was expected to be visited frequently because a trail in the refuge brought visitors to the sampling location.

The ATSDR and NJDHSS have completed two health consultations and one public health assessment for the US Avenue Burn and the Route 561 Dump sites. Two data reviews were also completed for the sites.

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. First, maximum concentrations of detected substances are compared to media-specific environmental guideline comparison values (CVs). If concentrations exceed the environmental guideline CV, these substances, referred to as Contaminants of Concern (COC), are selected for further evaluation. Contaminant levels above environmental guideline CVs do 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 environmental guideline CVs available for the screening environmental contaminants to identify COCs. These include ATSDR Environmental Media Evaluation Guides (EMEGs) and Reference Media Evaluation Guides (RMEGs). EMEGs are estimated contaminant concentrations that are not expected to result in adverse noncarcinogenic health effects. RMEGs represent the concentration in water or

soil at which daily human exposure is unlikely to result in adverse noncarcinogenic effects. If the substance is a known or a probable carcinogen, ATSDR's Cancer Risk Evaluation Guides (CREGs) were also considered as comparison values. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). In the absence of an ATSDR CV, other comparison values may be used to evaluate contaminant levels in environmental media. These include New Jersey Maximum Contaminant Levels (NJMCLs) for drinking water, and USEPA Region 3 Risk-Based Concentrations (RBCs). RBCs are contaminant concentrations corresponding to a fixed level of risk (i.e., a hazard quotient[1] of 1, or lifetime excess cancer risk of one in one million, whichever results in a lower contaminant concentration) in water, air, biota, and soil. For soils and sediments, other CVs include the New Jersey Residential and Non-Residential Direct Contact Soil Cleanup Criteria (RDCSCC, NRDCSCC). Based primarily on human health impacts, these criteria may also take into account natural background concentrations, analytical detection limits, and ecological effects.

Substances exceeding applicable environmental guideline CVs were identified as COCs and evaluated further to determine whether these contaminants pose a health threat to exposed or potentially exposed receptor populations.

Site Conditions

The Sherwin-Williams/Hilliards Creek site is located to the south of Route 561 in Gibbsboro, Camden County (Figure 3). The site is situated within the Atlantic Coastal Plain Physiographic Province (Weston 2001). The site areas are underlain by the Pleistocene sands of the Pennsauken Formation. The Pennsauken Formation outcrops in irregular patches in the Gibbsboro area and ranges in thickness from a few feet to 30 feet. The Formation consists of medium to coarse-grained quartz sand, gravel, and clay. Groundwater at the site occurs in two distinct zones: the shallow zone (30 to 40 feet thick) and a deeper zone (total thickness unknown). The two zones are separated by a silt unit that acts as a confining layer. Depth to groundwater is between 1 to 15 feet below ground surface. The horizontal direction of ground flow is generally to the south-southwest. At the site, the creeks and the lakes act as discharge zones for shallow groundwater.

The former facility is situated within a naturally occurring topographic depression. The central part this topographic low is defined by a series of ponds and lakes. Surface water runoff generated from the former facility areas flows into the Silver Lake (see Figure 3). Overflow from the Silver Lake discharges directly into Hilliards Creek through a series of storm sewers and culverts. Surface water runoff generated from the areas located to the east of United States Avenue flow towards the Haney Run and White Sands Branch, which ultimately discharges to Bridgewood Lake. The lake also receives surface water runoff from the areas located west of the United States Avenue, including the former landfill area (i.e., the US Avenue Burn site). Bridgewood

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

Lake discharges directly to the main branch of Hilliards Creek. The Hilliards Creek flows through residential areas and the Gibbsboro Nature Preserve, which includes walking trails for area residents and eventually discharges into the headwaters of the Cooper River located approximately three quarters of a mile southwest of the site.

On-site Contamination

As mentioned the Sherwin-Williams/Hilliards Creek site includes the former facility, the Hilliards Creek and the floodplain, wetland and adjacent residential properties.

Former Facility Area

Soil and Groundwater: In 1990, Sherwin-Williams entered into an Administrative Consent Order (ACO) with NJDEP to conduct Remedial Investigation/Feasibility Study for the former plant area. The RI/FS (Weston 2001) included collection of environmental samples from areas identified as: former Tank Farm areas A and B, groundwater seeps and the former lagoon area (see Figure 3). Samples were analyzed for Priority Pollutant Volatile Organic Compounds plus 15 non-target compounds (PP VOA+15), PP Base neutral compounds (PP BNA), lead, chromium and barium. The range, mean, standard deviation and median of the contaminants detected in the surface (0 – 2 feet) and subsurface soil (greater than 2 feet depth) and groundwater were presented in Tables 1, 2 and 3, respectively. Maximum concentrations of benzo[a]pyrene and lead detected in surface soil (see Table 1), lead detected in subsurface soil (see Table 2) and benzene, 1,2-dichloroethene, ethylbenzene, methylene chloride, vinyl chloride, xylenes, bis(2-ethylhexyl)phthalate, 2-methylnapthalene, naphthalene, pentachlorophenol, and lead detected in the groundwater (see Table 3) exceeded their respective environmental guideline CVs; they are considered as the COCs for the former facility areas.

Surface water samples collected from the head waters of Hilliards Creek did not indicate presence of organic pollutants.

Hilliards Creek

Sediment and soil: In 1991, for conducting the RI for the facility areas, three sediment and surface water samples were collected from the Hilliards Creek near Foster Avenue (USEPA 2006). Results indicated the presence of numerous metals and SVOCs including PAHs, di-n-octyl phthalate, dibenzofuran, pentachlorophenol in the sediment. Surface water results indicated presence of benzene, xylenes, phenols, aluminum, arsenic, chromium, copper, lead, magnesium, manganese, vanadium, and zinc.

In November 1998, the USEPA collected sediment, soil and surface water samples from Hilliards Creek (92 transects, every 50 feet) between Foster Avenue and Hilliards Road (see Figure 3) to determine the extent of lead contamination within the creek and the flood plain. Three sets of samples were collected from each of the transects: the north bank, the south bank, and the center of the creek. Each set of samples consisted of two composite samples collected at depths of approximately 0 to 2 inches

below ground surface (bgs) and 1 to 1.5 feet bgs. Soil samples were collected on the north and south flood plains of creek at distances of 7 and 12 feet from the north and south bank.

Blue-stained material, believed to be paint, was observed in the sediments of Hilliards Creek and in soil adjacent to the creek (USEPA 2006). One sediment sample collected from the creek containing blue-stained material (paint) contained lead (68,000 mg/kg) and arsenic (1,280 mg/kg). Three waste samples collected from the banks of Hilliards Creek contained lead up to 65,000 mg/kg, chromium up to 38,500 mg/kg, barium up to 2,870 mg/kg and arsenic up to 759 mg/kg .

In June 28, 1999, 155 soil samples were collected to define the extent of lead in the soil adjacent to Hilliards Creek (USEPA 2006). The samples were analyzed on site using X-ray Fluorescence (XRF). Sixteen samples were analyzed for metals. Lead was detected in silt-rich soils in Hilliards Creek flood plain. Lead concentrations decreased to a nondetectable level in areas up slope from the flood plain. The highest lead concentrations were detected in samples containing blue-green clayey material. Subsequently (December 1999), lead-contaminated soil from the 100-year flood plain of Hilliards Creek were collected at 16 transects (200 feet apart) between Clementon-Gibbsboro Road and Hilliard Road. Soil samples were collected from the north and south banks of the creek at each transect at 30-feet intervals along each transect. The sampling included 165 Kirkwood Road, a residential property located on the flood plain of Hilliards Creek. Hilliards Creek bisects the backyard the property. All samples were analyzed for lead (see Table 4). Both the maximum and mean concentration of lead detected in the soil exceeded the environmental guideline CVs, however, since more comprehensive contamination data were collected subsequently, these data were not used for this public health assessment.

In 2004, 39 sediment (0 – 2 inches) samples (from Gibbsboro-Clementon Road to downstream areas) were collected from the creek and the wetlands areas (USEPA 2006). The samples were analyzed for SVOCs, arsenic and lead; the range, mean, median and the standard deviation of the detected concentrations were presented in Table 5. Background samples from the area were also collected; three times the highest concentrations of SVOCs, arsenic and lead detected in the background samples were used to establish the background concentrations for the Hilliards Creek and wetland areas. The reported results included those that exceeded the background concentration. Maximum concentrations of benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, arsenic and lead exceeded their respective environmental guideline CVs; they were considered as the COCs for the site. However, since the data were exceedance values with respect to background levels and more comprehensive contamination data were collected subsequently, these data were not used for health risk assessment.

In 2005, approximately 350 soil and sediment samples were collected from the Hilliards Creek and wetlands areas as part of the remedial investigation (Sherwin-Williams, 2005). The samples were analyzed for the full list of analytical parameters

(i.e., VOCs, SVOCs, pesticides and polychlorinated biphenyls and metals) except for soil samples obtained from the 0 – 6 inch interval, which were not analyzed for VOCs. The range, mean, standard deviation and median of the contaminants detected in the sediment, surface (0 – 2 feet) and subsurface soil (greater than 2 feet depth) were presented in Tables 6, 7 and 8, respectively. Maximum concentrations of benzo[a]anthracene, benzo[b]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, antimony, arsenic, cadmium, chromium, copper, lead, selenium, vanadium and zinc detected in the sediment (see Table 6); 4,4'-DDD, arochlor 1254 and 1260 benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, alumimum, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, lead, manganese, nickel, selenium, silver, thallium, vanadium and zinc detected in the soil (see Table 7); 4,4'-DDD, 4,4'-DDE, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene arsenic, lead, manganese and mercury (see Table 8) detected in subsurface soil exceeded their respective environmental guideline CVs. They were considered as the COCs for the site.

Surface Water: In 2004, 13 surface water samples were collected from the Hilliards creek (USEPA 2006). The samples were analyzed for SVOCs, arsenic and lead; the range, mean, median and the standard deviation of the detected concentrations were presented in Table 9. Maximum concentrations of arsenic and lead detected in the surface water exceeded their respective environmental guideline CVs; they were retained as the COCs for further evaluation.

Off-site Areas

Potable Wells: A well search was performed (Weston 2001) to determine whether wells used for potable water purposes existed in the vicinity of the site. The survey identified seven domestic wells within one-half mile of the site. A number of these potable wells are located to the east of United States Avenue. Water quality data for these well are unavailable. As mentioned earlier, the Sherwin-Williams is developing workplan for sampling these wells.

Municipal Supply Wells: Potable water to the Gibbsboro area is provided by the New Jersey American Water Company (Weston 2001). Three production wells serve the Gibbsboro area and all are located on Linden Avenue. The nearest production well used by the company is located two miles southwest of the site and is positioned at a depth of over 450 feet, within the Raritan-Magothy formation.

Contaminants of Concern: Summary

Former Plant Areas: The maximum concentration of contaminants detected in soil (0 -2 feet and greater than 2 feet depth) and groundwater, along with Environmental Guideline CVs are presented in Tables 1, 2 and 3. The following contaminants exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

VOCs	SVOCs	Metals
On-site Soil (0 – 2 feet depth)		
	Benzo[a]pyrene	Lead
On-site subsurface soil		
	Benzo[a]pyrene	Lead
On-site Groundwater		
Benzene, 1,2-Dichloroethene, Ethylbenzene, Methylene Chloride, Vinyl Chloride, Xylenes	bis(2-Ethylhexyl)phthalate, 2-Methylnapthalene, Naphthalene, Pentachlorophenol	Lead

Hilliards Creek: The maximum concentration of contaminants detected in the Hilliards Creek sediment, soil (0 – 2 and greater than 2 feet depth) and surface water, along with Environmental Guideline CVs are presented in Tables 6 through 9. The following contaminants exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

VOCs	SVOCs	Metals
Hilliards Creek Sediment		
	Benzo[a]anthracene, Benzo[b]fluoranthene, Benzo[a]pyrene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene	Antimony, Arsenic, Cadmium, Chromium, Copper, Lead, Selenium, Vanadium, Zinc
Hilliards Creek Floodplain – Soil (0 -2 feet depth)		
	4,4'-DDD, Arochlor 1254 and 1260 Benzo[a]anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Benzo[a]pyrene, Indeno[1,2,3-cd]pyrene	Alumimum, Antimony, Arsenic, Barium, Beryllium, Cadmium, Chromium, Cobalt, Copper, Lead, Manganese, Nickel, Selenium, Silver, Thallium, Vanadium, Zinc
Hilliards Creek Floodplain - Subsurface Soil		
	4,4'-DDD, 4,4'-DDE, Benzo[a]anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene Benzo[a]pyrene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene	Arsenic, Lead, Manganese, Mercury
Hilliards Creek Surface Water		
		Arsenic, Lead

A brief discussion of the toxicologic characteristics of the COC is presented in Appendix A.

Discussion

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

Based on sampling data, results and knowledge of accessibility of the media to the population, exposure pathways for individuals who live (or lived) in the area of the Sherwin-Williams/Hilliards Creek site were identified as follows (see Table 10):

Completed Exposure Pathways

Ingestion of on-site contaminated soil from former facility areas (past, current, future). Soils (0 - 2 feet depth) at the former facility are contaminated with benzo[a]pyrene and lead. Residents, including children, were and are currently being exposed to contaminants while living and engaging in outdoor recreational activities at the site (Area near Tank Farm B, Building 67). This scenario also includes site visitors and trespassers.

Ingestion of contaminated soil from Hilliards Creek floodplain and sediment from adjacent wetlands (past, current, future). Site-related contaminants were detected in the

floodplain soils of Hilliards Creek and sediment of adjacent wetlands. Area residents reported to have accessed these areas in the past for recreational purposes (swimming and wading in the Hilliards Creek and adjacent areas). The exposures associated with these areas are partially interrupted due to the removal of lead contaminated soil from an area along the United States Avenue, one residence located at Kirkwood Road and construction of fence (partial) around the contaminated area located to the north of the Hilliards Creek. The remedial investigation workplan of on- and off-site areas are being developed by Sherwin-Williams. Therefore, residents including children, were and are potentially being exposed to contaminants during outdoor recreational activities.

Ingestion of surface water from Hilliards Creek (past, current, future). Site-related contaminants have been detected in the Hilliards Creek surface water. Residents, including children, were and are exposed to contaminants during outdoor recreational activities including swimming and wading in the Hilliards Creek.

Potential Exposure Pathways

Potential exposure pathways for the Sherwin-Williams/Hilliards Creek site were identified as follows (also summarized in Table 10):

Inhalation of indoor air (past, present, future). The on-site groundwater sampling results indicated the presence of free phase VOCs. Currently the on-site buildings are occupied by various businesses. A number of residences are also located to the east of United States Avenue. Employees and residents may have been or currently being exposed to groundwater contaminants in the indoor air of the buildings via vapor intrusion. Volatile chemicals in groundwater can migrate through subsurface soils and into indoor air spaces of overlying buildings (USEPA 2002a; NJDEP 2005a). The vapor intrusion pathway may be important for buildings with or without a basement. Vapors can accumulate in occupied spaces to concentrations that may pose safety hazards, health effects, or aesthetic problems (e.g., odors). On- and off-site indoor air data is needed to evaluate the public health implication of this pathway.

Ingestion of groundwater from potable wells (past, present, future). Although most area residents receive water from a public water supply system, a number of residences near the former facility (across the United States Avenue) have potable wells on the property. As mentioned earlier, Sherwin-Williams is obtaining access agreements with the residents and developing workplan for sampling these wells. Off-site potable well water quality data is needed to evaluate the public health implication of this pathway.

Ingestion of biota from Hilliards Creek (past, present, future). Biota (e.g., fish, game and plants) in Hilliards Creek, Kirkwood Lake and adjacent areas were exposed to contaminated soil and sediment. It is possible that area residents grew plants/vegetable in the adjacent areas and fished at the Hilliards Creek and Kirkwood Lake and ate their catch. Since the contaminants detected in the sediment may bioconcentrate in the plants and in the fatty tissues of aquatic animals, COCs may have been introduced into the food

chain. Biota data is needed to evaluate the public health implication of this pathway.

Exposure Point Concentration

Probability of exposure to contaminants is used as the basis for determining health risks at hazardous waste sites. Although the maximum concentration of contaminants is used to identify COCs, it would be inappropriate to calculate site risk based on the maximum concentration of contaminants. Due to inherent uncertainties associated with this approach, one single concentration is unlikely to represent the contamination at the entire site. Alternatively, a 'conservative estimate' of the mean chemical concentration, known as the exposure point concentration (EPC), in an environmental medium can be used to effectively represent a concentration at a hazardous waste sites. Unless there is site-specific evidence to the contrary, an individual receptor is assumed to be equally exposed to media within all portions of the exposure area over the time frame of the public health assessment.

In 1992, the USEPA recommended that the 95 percent upper confidence limit (UCL) of the arithmetic mean should be used as the EPC (USEPA 1992). Subsequently, USEPA developed a software package, ProUCL® (USEPA 2002, USEPA 2004, USEPA 2007) that uses rigorous parametric and nonparametric statistical methods on site data sets to estimate risk assessment parameters of interest, such as the EPC. For the Sherwin-Williams/Hilliards Creek site, the ProUCL 4.0 was used to estimate the surface soil and surface water EPCs. In the case of adequate data unavailability, an arithmetic mean is used as the EPC.

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 the epidemiologic and toxicologic literature and often include uncertainty or safety factors to ensure that they are amply protective of human health.

Completed human exposure pathways associated with the Sherwin-Williams/Hilliard Creek site include the incidental ingestion of soil and surface water from the former facility and Hilliards Creek. Since there is insufficient information available on the nature and magnitude of potential exposures associated with the off-site potable wells, inhalation of indoor air and the ingestion of biota, the public health implications of these potential pathways could not be determined at this time.

Non-Cancer Health Effects

To assess non-cancer health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants that are commonly found at hazardous waste sites. An MRL is an estimate of the daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of adverse, non-cancer health effects. MRLs are developed for a route of exposure, i.e., ingestion or inhalation, over a specified time period, e.g., acute (less than 14 days); intermediate (15 - 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, observed 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 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 effect levels in experimental studies. When MRLs for specific contaminants are unavailable, other health based comparison values such as USEPA Reference Dose (RfD). 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.

Ingestion - On-Site Soil

Based on informal conversation with the area residents, it is likely that the on-site areas and the Hilliards Creek and adjacent areas were frequently accessed by the area residents. Exposures are based on incidental ingestion of contaminated soil during recreational activities; non-cancer exposure doses were calculated using the following formula:

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

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;
C = concentration of contaminant in surface soil (mg/kg);
IR = soil ingestion rate (kg/day);

EF = exposure factor representing the site-specific exposure scenario; and,
BW = body weight (kg).

The following site-specific exposure assumptions (USEPA 1997) were used to calculate past contaminant doses. Estimated number of years exposed is based on information collected from past site documents.

Exposure Point	Number of Days Exposed Per Year	Number of Years Exposed
Plant Areas and Hilliards Creek floodplain	78 days (3x per week for six months per year)	30

Benzo[a]pyrene - Benzo[a]pyrene is one of a group of compounds called polycyclic aromatic hydrocarbons (PAHs). PAHs are formed as a result of incomplete combustion of organic materials. Many industrial products contain PAHs, including coal tar, roofing tar, and creosote. No acute or chronic MRL have been derived for benzo[a]pyrene because no adequate human or animal dose-response data are available that identify threshold levels for appropriate non-cancer health effects. However, intermediate duration oral MRLs of 0.4 mg/kg/day have been derived for fluoranthene and fluorene; both were based on LOAELs of 125 mg/kg/day for increased relative liver weight in male mice (ATSDR 1995). Based on the UCL of the arithmetic mean of benzo[a]pyrene detected in former facility surface soil[2] (0 – 2 feet depth), the estimated child and adult dose of 1.2×10^{-7} and 1.89×10^{-8} mg/kg/day, respectively (see Table 11) are several orders of magnitude lower than the MRL of 0.4 mg/kg/day. Therefore, it is unlikely that non-cancer adverse health effects would occur in children or adults. This determination takes into account that PAHs have similar physical, chemical, and toxicological characteristics.

Lead. Accumulation of lead in the body can cause damage to the nervous or gastrointestinal system, kidneys, or 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). The UCL of the arithmetic mean of lead detected in the on-site areas (995 mg/kg) exceeded the NJDEP RDCSCC of 400 mg/kg; residents, including children, were and are currently being exposed to contaminants while living and engaging in outdoor recreational activities at or near the site.

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 1994b). The IEUBK model estimates a plausible

[2]Although the samples collected from 0 - 2 feet depth are considered surface soil (NJDEP 2005b), the ATSDR consider 0 – 3 inches to be the surface soil; incidental ingestion exposures are more likely to be associated with soils from 0 – 3 inches below ground surface

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 (or P_{10}) that children's blood lead levels will exceed a level of concern. Health effects of concern have been determined to be associated with childhood blood lead levels at 10 micrograms of lead per deciliter of blood (or µg/dL) or less (USEPA 1986; CDC 1991). In using the IUEBK 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 1994c). The UCL of the arithmetic mean of lead levels in soils (0 – 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 six to 84 months are as follows:

1. Children were exposed to soil containing lead each time the former facility areas were visited. The visit frequency was three days per week over six months of the year.
2. Model default values were used for all other variables (USEPA 2002b) including residential soil and dust.

The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are shown in the following table:

Exposure Scenario		
Age (months)	**Three Site Visits Per Week[a]**	
	Blood Lead Level[b] (µg/dL)	**P_{10} (%)[c]**
6 -12[d]	6.8	20.8
12 - 24[d]	7.7	29.4
24 - 36	7.2	24.8
36 - 48	6.9	21.8
48 - 60	5.7	12.2
60 - 72	4.9	6.5
72 - 84	4.4	4

[a]weighted soil lead concentration (995 ppm x 3/7 + 200 ppm x 4/7) = 540 ppm (USEPA 2003a); [b]Geometric mean lead levels in blood; [c]probability of blood lead level > 10 µg/dL; [d]the exposure pathway is mainly dust

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 6 - 84 months were below the level of concern (10 µg/dL); however, the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 72 months exceeded 5 percent. Therefore, potential for adverse health effects associated with lead exposures is considered low. An adult blood lead model estimated a geometric mean blood lead level among adult workers and 95th percentile of blood lead level among fetuses are 2.3 µg/dL and 6.9 µg/dL, respectively (USEPA 2003b). As such, potential for adverse health effects to adults associated with lead exposures at the Hilliards Creek area is considered low.

It is important to note that the IEUBK model should not be used for exposure periods of less than three months, or in which a higher exposure occurs less than once per week or varies irregularly.

Ingestion – Hilliards Creek Soil

Residents, including children, were exposed to contaminants during outdoor recreational activities in the Hilliards Creek floodplain areas. Exposures are based on ingestion of contaminated soil; non-cancer contaminant exposure doses were calculated using the following formula:

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

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

Based on the UCL of the arithmetic mean of 4,4'-DDD, Arochlor 1254 and 1260, aluminum, antimony, barium, beryllium, cadmium, chromium, cobalt, copper, manganese, nickel, selenium, vanadium and zinc detected in the soil (0 – 2 feet depth) of Hilliards Creek (see Table 12), chronic exposure doses calculated 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. The health guideline CVs of benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene are unavailable. Based on the UCL of the arithmetic mean of arsenic detected in the soil (0 – 2 feet depth) of Hilliards Creek (see Table 12), chronic exposure doses calculated for children exceeded the health guideline CVs. The UCL of the arithmetic mean of lead detected in the soil[3] of Hilliards Creek (4108 mg/kg) exceeded the NJDEP RDCSCC of 400 mg/kg. The non-cancer adverse heath effects associated with exposures to these contaminants are evaluated as follows:

PAHs. Benzo[a]anthracene, benzo[b]fluoranthene, benso[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene belong to a group of compounds called polycyclic aromatic hydrocarbons (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 1995). 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 they are produced by

[3]The ProUCL analysis indicated the data 585,000 mg/kg an outlier and was excluded from UCL analysis.

combustion processes, PAHs are widespread in the environment. Typical urban background concentrations of a few PAHs are as follows (ATSDR 1995):

Contaminant	Urban Background Soil Concentration (mg/kg)
Benzo[a]anthracene	0.169 - 59
Benzo[a]pyrene	0.165 – 0.22
Benzo[b]fluoranthene	15 - 62
Benzo[g,h,i]perylene	0.9 - 47

PAHs have been found to exhibit antiandrogenic properties in human cells cultures and are implicated in the loss of fertility in males (Kizu 2003). Non-cancer adverse health effects associated with PAH exposures has been observed in animals but generally not in humans (ATSDR 1995).

Based on the UCL of the arithmetic mean of PAHs detected in the Hilliards Creek soil (0 – 2 feet depth), the chronic exposure doses for children and adults were calculated (see Tables 12); no health guideline CVs are available for the PAHs identified as the COCs. However, the NOAEL, RfD and associated critical health effects for a number of PAHs (i.e., acenaphthene, anthracene, fluoranthene, fluorene, naphthalene and pyrene) are available and is 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)

Source: EPA 2006

The RfD's of these PAHs are based on the NOAEL for less serious health effects and are much higher than those calculated for the on-site PAHs. Based on the UCL of the arithmetic mean of PAHs (benzo[a]anthracene, benzo[b]fluoranthene, benso[k]fluoranthene, benzo[a]pyrene and indeno[1,2,3-cd]pyrene) detected in soil (0 – 2 feet depth) of the Hilliards Creek surface soil (see Table 12), the calculated chronic child

exposure doses (1.1 $x10^{-6}$ mg/kg/day to 1.05 $x10^{-5}$ mg/kg/day) were about 1,900 to 17,000 times lower than the lowest reported RfD (i.e., 0.02 mg/kg/day for naphthalene). In addition, the concentrations of PAHs detected at the site are similar to those reported in the urban soil. As such, non-cancer adverse health effects associated with the PAH exposures in the past is unlikely in children and adults.

Arsenic. Arsenic is a naturally occurring element widely distributed in the earth's crust. The MRL for arsenic is set at a level meant to protect against non-cancer health effects, specifically dermal lesions (ATSDR 2000a). Chronic exposure to low levels of inorganic arsenic 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 less toxic than inorganic arsenic compounds.

Based on the UCL of arsenic detected in soil, the chronic exposure dose calculated for children (i.e., 0.0009 mg/kg/day) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table 12). The calculated child exposure doses are about the same as the NOAEL (i.e., 0.0008 mg/kg/day), respectively. As such, non-cancer adverse health effect associated with exposure to arsenic detected in the Hilliards Creek floodplain soil (0 – 2 feet depth) is not expected.

Lead. The UCL of the arithmetic mean of lead detected in the on-site areas (4,108 mg/kg) exceeded the NJDEP RDCSCC of 400 mg/kg. Residents, including children, were and are currently being exposed to contaminants while living and engaging in outdoor recreational activities.

As mentioned earlier, lead exposures associated with the intermittent recreational use of lead contaminated areas were evaluated using the USEPA's integrated exposure uptake biokinetic (IEUBK) model (USEPA 1994b). The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are shown in the following table:

Exposure Scenario		
Age (months)	**Three Site Visits Per Week[a]**	
	Blood Lead Level[b] (µg/dL)	**P_{10} (%)[c]**
6 -12[d]	15.5	82
12 - 24[d]	18	89
24 - 36	17	87
36 - 48	16.5	86
48 - 60	14	77
60 - 72	12	65
72 - 84	10.7	56

[a]weighted soil lead concentration (4,108 ppm x3/7 + 200 x4/7) = 1,875 ppm (USEPA 2003a); [b]Geometric mean lead levels in blood; [c]probability of blood lead level > 10 µg/dL; [d]the exposure pathway is mainly dust

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 6 - 84 months were above the level of concern (10 µg/dL); the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 48 months exceeded 5 percent. Therefore, potential for adverse health effects associated with lead exposures at the Hilliards Creek area is considered high. An adult blood lead model estimated a geometric mean blood lead level among adult workers and 95th percentile of blood lead level among fetuses are 4.2 µg/dL and 12.8 µg/dL, respectively (USEPA 2003b). As such, potential for adverse health effects to adults associated with lead exposures at the Hilliards Creek area is also high.

Ingestion – Hilliards Creek Surface Water

Residents, including children, have been exposed to surface water contaminants during outdoor recreational activities in the Hilliards Creek. In order to assess exposures from incidental ingestion of surface water contaminants, an exposure dose was calculated using the following formula:

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

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).

The following exposure assumptions (USEPA 1997) were used to calculate contaminant doses.

Incidental Ingestion Rate (mL)	Number of Days Exposed Per Year	No. of years Exposed
50	78 days (3x per week for six months per year)	30

The estimated exposure dose was compared to health guideline CVs. Based on the concentration of arsenic detected in the surface water, the chronic exposure doses calculated for adults and children were lower than the corresponding health guideline CVs (see Table 13). Although the maximum concentration of lead exceeded AL, it should be noted that the AL is based on 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, past exposures associated with incidental ingestion of surface water from the Hilliards Creek are unlikely to cause non-cancer adverse health effects.

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 46 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 approximately between 1 in 100 and 10 in 100 (SEER 2005). Typically, health guideline CVs developed for carcinogens are based on a lifetime risk of one excess cancer case per 1,000,000 individuals. ATSDR considers estimated cancer risks of less than one additional cancer case among one million persons exposed as insignificant or no increased risk (expressed exponentially as 10^{-6}).

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

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

Exposure doses for cancer risk assessment were calculated using the following formula:

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

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 duration (year);
BW = body weight (kg); and,
AT = averaging time (year).

Ingestion – On-site Soil

The cancer class of the COCs detected in the soil (0 – 2 feet depth) of on-site areas is given in Table 14. The table shows that benzo[a]pyrene detected in the soil (0 – 2 feet depth) have the potential to cause cancer among exposed populations.

Based on previously described exposure assumptions, LECR was calculated by multiplying the benzo[a]pyrene 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., $(mg/kg/day)^{-1}$. Based on the UCL of the arithmetic mean concentrations of

benzo[a]pyrene detected in the on-site soil, the calculated LECR was six in 100,000,000 which is considered as insignificant or no increased risk.

Lead has been classified as a carcinogen by the USDHHS[4] and the USEPA[5]. The carcinogenicity of inorganic lead and lead compounds has been evaluated by the USEPA (USEPA 1986, 1989). The USEPA has determined that data from human studies are inadequate for evaluating the carcinogenicity of lead, but there are sufficient data from animal studies which demonstrate that lead induces renal tumors in experimental animals. In addition, there are some animal studies which have shown evidence of tumor induction at other sites (i.e., cerebral gliomas; testicular, adrenal, prostate, pituitary, and thyroid tumors). A cancer slope factor has not been derived for inorganic lead or lead compounds, so no estimation of LECR can be made for lead exposure.

Ingestion – Hilliards Creek Soil

The cancer class of the COCs detected in the floodplain soil (0 – 2 feet depth) of Hilliards Creek is given in Table 15. The table shows that PAHs, 4,4'-DDD, Aroclor 1254 and 1260, beryllium, cadmium, chromium and nickel detected in the soil (0 – 2 feet depth) have the potential to cause cancer among exposed populations.

The USEPA has developed a relative potency estimate approach for PAHs (USEPA 1993). 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 maximum 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 Tables 15).

Based on previously described exposure assumptions, LECR were calculated by multiplying the exposure dose by the cancer slope factor. LECRs based on the UCL of the arithmetic mean concentrations detected in the soil (0 – 2 feet depth) are presented in Table 15. Based on the UCL of the arithmetic mean concentrations of 4,4'-DDD, Arochlor 1254 and 1260 detected in the floodplain soil, the calculated LECR was considered as insignificant or no increased risk.

For PAHs, based on the UCL of the arithmetic mean detected in the floodplain soil (0 – 2 feet depth), the LECRs is 5 in 1,000,000, for individuals who lived in and/or participated in recreational activities in the floodplain areas. For arsenic, based on the UCL of the arithmetic mean concentration detected in the floodplain soil (0 – 2 feet depth), the LECRs are 1 in 10,000, respectively, for individuals who lived in and/or participated in recreational activities in the floodplain areas.

[4]Lead and Lead Compounds are listed in the Eleventh Edition of the Report on Carcinogens as "reasonably anticipated to be human carcinogens" (NTP 2006)
[5]Probable human carcinogen (B2)

USEPA reviewed the carcinogenicity information available for beryllium for oral exposures but did not estimate a value; information on the carcinogenicity of chromium by oral exposure in humans was unavailable; limited epidemiologic studies have indicated that exposure to cadmium in food or drinking water is not carcinogenic and quantitative estimate of carcinogenic risk from oral exposure to copper was not assessed (USEPA 2007). The cancer risk associated with lead exposures are discussed earlier.

Ingestion – Hilliards Creek Surface Water

The cancer class of the COCs detected in the Hilliards Creek surface water is given in Table 16; both arsenic and lead are carcinogens. Based on the maximum arsenic concentration, the LECR is 3 in 1,000,000 to the exposed population. The cancer risk associated with lead exposures are discussed earlier.

Assessment of Joint Toxic Action of Chemical Mixtures

At the Sherwin-Williams/Hilliard Creek site, residents were exposed to PAHs and metals via incidental ingestion. Although toxicological effects associated with site-related contamination were evaluated individually, the cumulative or synergistic effects of mixtures of contaminants may increase their public health impact. This depends upon the specific contaminant, its pharmacokinetics, and toxicity in the receptor population. Research on the toxicity of mixtures indicates that adverse health effects are unlikely when the mixture components are present at levels well below their individual toxicological thresholds (ATSDR 2005).

To assess the risk for non-cancer adverse health effects of chemical mixtures, the hazard indexes (HI) and the ratio of exposure dose to NOAEL for the contaminants was calculated (see Appendix B for details). The results indicated that potential exists for additive or interactive effects of chemical mixtures from exposures to floodplain soil of the Hilliards Creek, particularly for neurological effects associated with co-exposure to lead and arsenic.

As measures of probability, individual LECRs can be added. Based on calculated UCL of arithmetic mean contaminant concentrations, cumulative ingestion exposures indicated a cancer risk of approximately one excess cancer cases per 10,000 individuals. Since the weight of evidence scores for carcinogenic interaction of PAHs and arsenic are unavailable (see Appendix B for details), the potential cancer health hazard due to interaction could not be evaluated.

Health Outcome Data

Based on a review of data, completed exposure pathways existed among area residents who used the former facility and the Hilliards Creek area for recreation. Exposures may have continued for years until fences were installed and the pathway was

partially interrupted. Due to the exposure to lead in the floodplain soil and sediment, data on childhood blood lead tests was evaluated for the community. NJDHSS obtained all available information from the NJDHSS' Childhood Lead Poisoning Surveillance System for the years 1999 through 2006.

A review of other health outcome data (e.g., adverse pregnancy outcomes, cancers, deaths) may be conducted to assess the public health significance of these completed exposure pathways. However, due to the small number of individuals exposed, an evaluation of other available health data is unlikely to produce interpretable results.

Childhood Lead Exposure

Because of the potential for exposure to lead in contaminated site media, data on blood lead tests were evaluated for Gibbsboro children. Information from the NJDHSS' Childhood Lead Poisoning Surveillance System is summarized below.

Blood lead is an excellent indicator of exposure to lead. Current state regulations, in accordance with federal Centers for Disease Control and Prevention (CDC) guidelines, require health care providers to do a blood lead test on all one and two year old children. This is the age at which lead poisoning is most damaging to the developing nervous system. State regulation requires all clinical laboratories to report the results of all blood lead tests to the NJDHSS. Prior to July 1999, only blood lead tests above 20 micrograms per deciliter (µg/dL) were reportable. While the current CDC blood lead guideline is 10 µg/dL, all blood-lead test data are reportable to the NJDHSS' Childhood Lead Poisoning Prevention Surveillance System.

Data from the Childhood Lead Poisoning Prevention Surveillance System was reviewed for the period July 1999 through February 2007 for Gibbsboro. A total of 137 Gibbsboro children were tested during this period. The age range for children tested was 0.7 to16.5 years. The range of blood lead levels in Gibbsboro children was 0.2 to 8.0 µg/dL. No children were found to have a blood lead level above the CDC guideline during this time period. The geometric blood lead average was 2.9 µg/dL with a 95% confidence interval of 2.7 to 3.1 µg/dL. The blood lead levels measured in Gibbsboro children are similar to statewide average levels.

Child Health Considerations

The NJDHSS 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 NJDHSS and ATSDR evaluated the potential risk for children residing in the area who were exposed to site contaminants. Although the exposures doses calculated for children based on the maximum concentrations of aluminum, antimony, arsenic, cadmium, chromium, copper and vanadium detected in the soil (0 – 2 feet depth) exceeded the health guideline CVs, likelihood of adverse non-cancer health effects in children were determined to be low. Based on the UCL of the arithmetic mean lead contamination in the Hilliards Creek floodplain soils (0 – 2 feet depth), exposures were found to have the potential to cause non-cancer adverse health effects in children.

The potential cancer health effects associated with exposure to site-related contaminants were evaluated. Based on the the UCL of the arithmetic mean concentrations of contaminants detected, the calculated LECRs were estimated to be approximately one excess cancer cases per 10,000 (including exposure to children).

Public Comment

The public comment period for this PHA was from October 21, 2008 through November 22, 2008. The comments and the responses are given in Appendix C.

Conclusion

More than 120 years of operation of paint manufacturing have resulted in the generation of hazardous wastes and environmental contamination of on- and off-site areas including area residences. Contaminants of concern identified for the site were benzene, 1,2-dichloroethene, ethylbenzene, methylene chloride, vinyl chloride, xylenes, bis(2-ethylhexyl)phthalate, 2-methylnapthalene, naphthalene, pentachlorophenol and lead detected in the groundwater at the former facility and 4,4'-DDD, 4,4'-DDE, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, Arochlor 1254 and 1260, alumimum, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, lead, manganese, nickel, selenium, silver, thallium, vanadium and zinc detected in the Hilliards Creek sediment and soil. The ATSDR and NJDHSS reached four important conclusions in this PHA:

NJDHSS and ATSDR conclude that likely lead exposures to area children and lifetime excess cancer risks associated with the sediment and floodplain soils of Hilliards Creek in the past may have harmed people's health. In the past, there were completed exposure pathways to area residents via the incidental ingestion of contaminated soil (0 – 2 feet depth) in the facility areas, the Hilliards Creek floodplain and sediment in the adjacent wetlands. The exposures associated with these pathways are partially interrupted due to the removal of lead contaminated soil from an area along the United

States Avenue, one residence located at Kirkwood Road and fence (partial) around the contaminated area located to the north of the Hilliards Creek. Potential pathways also included inhalation of indoor air, ingestion of groundwater from potable wells and biota from Hilliards Creek. Although the maximum exposure doses for aluminum, antimony, arsenic, cadmium, chromium copper and vanadium exceeded their respective health guideline CVs for children, the likelihood of potential non-cancer adverse health effects was determined to be low. The potential for adverse health effects other than cancer associated with past lead exposures is possible. The potential health hazard due to additive or interactive effects of chemical mixtures may be greater than estimated by the endpoint-specific hazard index. For cancer health effects, lifetime excess cancer risks were calculated based on the UCL of the arithmetic mean contaminant concentrations. Cumulative lifetime excess cancer risks associated with the site was one in 10,000 to the exposed population, which is considered to be low in comparison to the background risk of cancer. As such, based on likely lead exposures to the children and lifetime excess cancer risks associated with the sediment and floodplain soils of the Hilliards Creek in the past, the site contaminants may have harmed people's health. Although this assessment is based on the exposures to soils collected from the 0 – 2 feet depth, the ATSDR considers 0 – 3 inches to be the surface soil (the soil to which people are most likely to be exposed).

The NJDHSS and ATSDR cannot conclude whether past and/or current exposures to potable water, indoor air and consumption of plants, fish and game from the Sherwin-Williams/Hilliards Creek site may have harmed people's health. Potable water, indoor air and biota (i.e., plants, fish and game) data were unavailable at the time the PHA was prepared.

The NJDHSS and ATSDR cannot conclude whether current exposures to soil and sediment at the Sherwin-Williams/Hilliards Creek site could harm people's health. Although Sherwin-Williams implemented interim remedial measures to address the most contaminated on-site media, characterization and delineation of on- and off-site areas have not been completed.

The blood lead levels measured in Gibbsboro children are similar to statewide average levels. Childhood blood lead level data from the state Childhood Lead Poisoning Surveillance System for the years 1999 through 2006 were evaluated. A total of 137 Gibbsboro children (age range 0.7 to16.5 years) were tested during this period. No children were found to have a blood lead level above the CDC guideline during this time period. The geometric mean blood lead was 2.9 μg/dL with a 95% confidence interval of 2.7 to 3.1 μg/dL. The blood lead levels measured in Gibbsboro children are similar to statewide average levels.

Recommendations

1. Sherwin-Williams, with USEPA oversight, should characterize the potential contamination of local biota, particularly fish and game consumed by area residents.

2. Sherwin-Williams, with USEPA oversight, should characterize the potential contamination of indoor air of the residences located near the former facility and the office buildings on the facility.

3. The USEPA should continue to require and ensure that all contaminated areas remain fenced, post signs where appropriate; and conduct site inspections on a quarterly basis.

4. The Sherwin-Williams, with USEPA oversight, should complete the remedial investigation and remediation of on- and off-site areas including the residential properties as soon as feasible. Consideration should be given to collect soil samples from 0 – 3 inches depth so that the concentrations represent the incidental ingestion exposure pathway.

Public Health Action Plan (PHAP)

The purpose of a PHAP is to ensure that this 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 ATSDR and NJDHSS to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by the NJDHSS and the ATSDR are as follows:

Public Health Actions Undertaken by NJDHSS and ATSDR

1. The ATSDR and NJDHSS evaluated the exposures associated with the lead contaminated area identified in the Hilliards Creek Wildlife Refuge.

2. The NJDHSS and ATSDR communicated with town officials in order to hold availability sessions for residents to identify community concerns, and to provide information to residents about exposure pathways and the contaminants of concern.

Public Health Actions Planned by NJDHSS and ATSDR

1. Copies of this Public Health Assessment will be provided to concerned residents in the vicinity of the site via the township library and the Internet.

2. Former and current area residents concerned about potential exposures to site-related contamination should be examined by their personal physicians. Upon request, NJDHSS will provide educational materials on environmental exposures, arsenic and lead to local physicians and other medical personnel to assist them in this evaluation.

3. In cooperation with the USEPA, public meetings will be scheduled to discuss the findings of this report and to determine and address any additional community concerns.

4. The NJDHSS and ATSDR are currently evaluating childhood blood lead level data from the state Childhood Lead Poisoning Surveillance System for the years 1999 through 2006 and the results will be incorporated into the public comment draft report.

5. As additional off-site contamination data (e.g., from private wells located along the United States Avenue, remaining 200-feet transects located along Hilliards Creek, and, residential properties adjacent to Hilliards Creek) become available, the NJDHSS and ATSDR will prepare health consultation(s) in order to evaluate the public health implications of potential contamination.

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CERTIFICATION

The public health assessment for the Sherwin-Williams/Hilliards Creek site, Gloucester County, New Jersey was prepared by the New Jersey Department of Health and Senior Services under a cooperative agreement with the Agency for Toxic Substances and Disease Registry. It is in accordance with approved methodology and procedures existing at the time the health assessment were initiated. An editorial review was conducted by the cooperative agreement partner for this document.

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The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this health consultation and concurs with its findings.

Alan Yarbrough
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Agency for Toxic Substances and Disease Registry

Table 1: Soil (0-2 feet depth) sampling results of former facility areas

Contaminant	No. of Detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Volatile Organic Compounds (VOCs)				
1,12,2-Tetrachloroethane	1	ND[d] - 0.05	2,000 (EMEG[e])	No
1,2-Dichloroethene	1	ND - 0.019	1,000 (RMEG[f])	No
2-Butanone	1	ND - 0.024	30,000 (RMEG)	No
Chloroform	1	ND - 0.003	500 (EMEG)	No
Ethylbenzene	6	ND - 5.5	5,000 (RMEG)	No
Tetrachloroethene	1	ND - 0.019	1.2 (RBC[g])	No
Toluene	1	ND - 0.009	4,000 (RMEG)	No
Trichloroethene	4	ND - 0.55	1.6 (RBC)	No
Xylene (total)	4	ND - 37	30,000 (EMEG)	No
Semivolatile Organic Compounds (SVOCs)				
2.4-Dimethylphenol	2	ND - 4.1	1,000 (RMEG)	No
2-Methylnaphthalene	6	ND - 4.5	2000 (EMEG)	No
Acenapthylene	1	ND - 0.21	NA[h]	
Acenaphthene	1	ND - 0.098	3,000 (RMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
Anthracene	2	ND - 0.18	20,000 (RMEG)	No
Benzo[a]anthracene	3	ND - 0.53	0.87 (RBC)	No
Benzo[a]pyrene	3	ND - 0.46	0.1 (CREG[i])	**Yes**
Benzo[b]fluoranthene	3	ND - 0.53	0.87 (RBC)	No
Benzo[g,h,i]perylene	1	ND - 0.23	NA	
Benzo[k]fluoranthene	3	ND - 0.42	8.7 (RBC)	No
bis(2-Ethylhexyl)phthlate	3	ND - 0.15	46 (RBC)	No
Butyl benzyl phthalate	1	ND - 0.058	10,000 (RMEG)	No

Table 1: (Contd.)

Semi Volatile Organic Compounds (SVOCs)				
Chrysene	3	ND - 0.62	87 (RBC)	No
Dibenzofuran	2	ND - 0.13	NA	
Di-n-Butylphthalate	1	ND - 0.043	5,000 (RMEG)	No
Fluoranthene	3	ND - 1	2,000 (RMEG)	No
Fluorene	3	ND - 0.19	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	1	ND - 0.24	0.87 (RBC)	No
Semi Volatile Organic Compounds (SVOCs)				
Naphthalene	6	ND - 10	1,000 (RMEG)	No
Pentachlorophenol	1	ND - 0.33	50 (EMEG)	No
Phenanthrene	3	ND - 0.7	NA	
Pyrene	3	ND - 0.81	2,000 (RMEG)	No
Metals				
Barium	12	6.8 – 2,940	30,000 (EMEG)	No
Chromium	12	1.7 - 90.7	200 (RMEG)	No
Lead	12	1.6 – 2,070	400 (RDCSCC[j])	**Yes**

[a]Number of Samples = 12 ; [c]Comparison Value; [d]Contaminant of Concern; [b]Not detected; [e]ATSDR Environmental Media Evaluation Guide; [f]ATSDR Reference Media Evaluation Guide; [g]USEPA Region 3 Risk-Based Concentration; [h]Not Available; [i]ATSDR Cancer Risk Evaluation Guide; [j]NJDEP Residential Direct Contact Soil Cleanup Criteria

Table 2: Subsurface soil (greater than 2 feet depth) sampling results of former facility areas

Contaminant	No. of Detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Volatile Organic Compounds (VOCs)				
1,1,1-Trichloroethane	7	ND[d] - 0.06	1,000,000 (EMEG[e])	No
2-Butanone	1	ND - 22	30,000 (RMEG[f])	No
Benzene	2	ND - 3.2	10 (CREG[g])	No
Chloroform	7	ND - 0.06	500 (EMEG[h])	No
Ethylbenzene	11	ND - 1,300	5,000 (RMEG)	No
Methylene Chloride	1	ND - 0.017	3,000 (EMEG)	No
Toluene	7	ND - 24	4,000 (RMEG)	No
Trichloroethene	8	ND - 0.19	1.6 (RBC[i])	No
Xylene (total)	13	ND - 6,900	30,000 (EMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
2.4-Dimethylphenol	1	ND - 0.042	1,000 (RMEG)	No
2-Methylnaphthalene	9	ND - 18	2000 (EMEG)	No
Acenapthylene	1	ND - 0.04	NA[j]	
Anthracene	1	ND - 0.058	20,000 (RMEG)	No
Benzo[a]anthracene	2	ND - 0.27	0.87 (RBC)	No
Benzo[a]pyrene	3	ND - 0.28	0.1 (CREG)	No
Benzo[b]fluoranthene	3	ND - 0.37	0.87 (RBC)	No
Benzo[k]fluoranthene	3	ND - 0.27	8.7 (RBC)	No
bis(2-Ethylhexyl)phthalate	1	ND - 0.06	46 (RBC)	No
Benzoic Acid	1	ND - 0.073	200,000 (RMEG)	No
Butylbenzyl phthalate	1	ND - 0.05	10,000 (RMEG)	No
Chrysene	4	ND - 0.27	87 (RBC)	No

Table 2: (Contd.)

Semi Volatile Organic Compounds (SVOCs)				
Di-n-Butylphthalate	2	ND - 0.09	5,000 (RMEG)	No
Fluoranthene	3	ND - 0.38	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	1	ND - 0.087	0.87 (RBC)	No
Naphthalene	8	ND - 92	1,000 (RMEG)	No
Phenanthrene	3	ND - 0.19	NA	
Pyrene	3	ND - 0.41	2,000 (RMEG)	No
Barium	18	2 - 379	30,000 (EMEG)	No
Chromium	18	2.2 - 39.2	200 (RMEG)	No
Lead	18	1.8 - 680	400 (RDCSCC[k])	**Yes**

[a]Number of Samples = 18; [b]Comparison Value; [c]Contaminant of Concern; [d]Not Detected; [e]ATSDR Environmental Media Evaluation Guide for intermediate exposures; [f]ATSDR Reference Media Evaluation Guide; [g]ATSDR Cancer Risk Evaluation Guide; [h]ATSDR Environmental Media Evaluation Guide; [i]USEPA Region 3 Risk-Based Concentration; [j]Not Available; [k]NJDEP Residential Direct Contact Soil Cleanup Criteria

Table 3: Shallow Groundwater Sampling Results of former facility areas

Contaminant	No. of Detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Volatile Organic Compounds (VOCs)				
Acetone	3	ND[d] – 2,900	9,000 (RMEG[e])	No
Benzene	12	ND – 3,200	0.6 (CREG[f])	**Yes**
2-Butanone	1	ND – 110	6,000 (RMEG)	No
Carbon disulfide	2	ND – 16	1,000 (RMEG)	No
1,2-Dichloroethene	6	ND – 630	200 (RMEG)	**Yes**
Ethylbenzene	15	ND – 2,700	1,000 (RMEG)	**Yes**
Methylene Chloride	5	ND – 1,600	600 (EMEG[g])	**Yes**
Styrene	1	ND – 650	2,000 (RMEG)	No
Toluene	2	ND – 38	800 (RMEG)	No
Vinyl Chloride	1	ND – 63	30 (EMEG)	**Yes**
Xylene	13	ND – 12,000	6,000 (EMEG)	**Yes**
Semivolatile Organic Compounds (SVOCs)				
4-Chloroaniline	1	ND – 3	40 (RMEG)	No
Diethylphthalate	1	ND – 2	8,000 (RMEG)	No
2,4-Dimethylphenol	1	ND – 24	200 (RMEG)	No
bis(2-Ethylhexyl)phthalate	17	ND – 110	4.8 (RBC[h])	**Yes**
4-Methylphenol	1	ND – 1	180 (RBC)	No
2-Methylnapthalene	10	ND – 810	400 (EMEG)	**Yes**
Naphthalene	14	ND – 5,200	200 (RMEG)	**Yes**
Pentachlorophenol	6	ND – 18	10 (EMEG)	**Yes**
2,4,5-Trichlorophenol	1	ND – 12	1,000 (RMEG)	No

Table 3: (Contd.)

Metals				
Barium	26	16 - 130	6,000 (EMEG)	No
Chromium	1	ND – 3	30 (RMEG)	No
Lead	24	ND – 50	15 (AL[i])	**Yes**
Phenolics	21	ND – 450	3,000 (RMEG)	No

[a]Number of Samples = 26; [b]Comparison Value; [c]Contaminant of Concern; [d]Not Detected; [e]ATSDR Reference Media Evaluation Guide; [f]ATSDR Cancer Risk Evaluation Guide; [g]ATSDR Environmental Media Evaluation Guide; [h]USEPA Region 3 Risk-Based Concentration; [i]NJDEP Action Level for Lead

Table 4: Concentration of lead detected in the sediment of Hilliards Creek (1999)

Contaminants	No. of Detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Metals				
Lead (Sediment)	106	69.6 – 80,700	400 (RDCSCC[d])	**Yes**
Lead (Soil)	46	24 – 16,300	400 (RDCSCC)	**Yes**

[a]Number of Sediment and Samples = 106 and 46; [b]Comparison Value; [c]Contaminant of Concern; [d]NJDEP Residential Direct Contact Soil Cleanup Criteria

Table 5: Concentration of contaminants detected in the wetland soil (0 – 2 inches) and sediment (0 – 2 inches) of the Hilliards Creek (2004)

Contaminants	No. of Detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Semi Volatile Organic Compounds (SVOCs)				
Benzo[a]anthracene	3	ND[d] – 2.6	0.87 (RBC[e])	**Yes**
Benzo[a]pyrene	3	ND – 3.4	0.1 (CREG[f])	**Yes**
Benzo[b]fluoranthene	5	ND – 7.5	0.87 (RBC)	**Yes**
Benzo[g,h,i]perylene	2	ND – 3	NA[g]	
Benzo[k]fluoranthene	2	ND – 1.9	0.9 (RDCSCC[h])	**Yes**
Chrysene	4	ND – 3.6	9 (RDCSCC)	No
Fluoranthene	5	ND – 7.1	2,000 (RMEG[i])	No
Phenanthrene	3	ND – 3.1	NA	
Pyrene	5	ND – 7.3	2,000 (RMEG)	No
Metals				
Arsenic	36	ND – 1,110	0.5 (CREG)	**Yes**
Lead	35	ND – 9,140	400 (RDCSCC)	**Yes**

[a]Number of Samples = 39; [b]Comparison Value; [c]Contaminant of Concern; [d]Not Detected; [e]USEPA Region 3 Risk-Based Concentration; [f]ATSDR Cancer Risk Evaluation Guide; [g]Not Available; [h]NJDEP Residential Direct Contact Soil Cleanup Criteria; [i]ATSDR Reference Media Evaluation Guide

Table 6: Concentration of contaminants detected in the Hilliards Creek sediment (0 -2 feet depth) (2005)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Volatile Organic Compounds				
1,2-Dichlorobenzene	1	ND[d] – 0.006	20,000 (EMEG[e])	No
2-Butanone	131	ND – 0.23	30,000 (RMEG[f])	No
Acetone	88	ND – 0.81	50,000 (RMEG)	No
Bromomethane	1	ND – 0.022	70 (RMEG)	No
Carbon disulfide	44	ND – 0.033	5,000 (RMEG)	No
Chloromethane	10	ND – 0.017	520 (RDCSCC[g])	No
cis-1,2-Dichloroethene	1	ND – 0.001	20,000 (EMEG I[h])	No
Dichloromethane	37	ND – 0.026	3,000 (RMEG)	No
Ethylbenzene	6	ND – 0.07	5,000 (EMEG)	No
Isopropylbenzene	1	ND – 0.11	5,000 (RMEG)	No
Methyl Acetate	35	ND – 0.18	NA[i]	
Methylcyclohexane	4	ND – 4.4	NA	
Toluene	28	ND – 0.12	4,000 (RMEG)	No
Xylenes	8	ND – 0.36	10,000 (RMEG)	No
Pesticides/PCBs				
4,4'-DDD	66	ND – 0.25	3 (CREG[j])	No
4,4'-DDE	48	ND – 0.09	2 (CREG)	No
4,4'-DDT	14	ND – 0.26	2 (CREG)	No
alpha-Chlordane	49	ND – 0.05	30 (EMEG C[k])	No
Arochlor 1254	31	ND – 0.79	1 (EMEG)	No
Arochlor 1260	29	ND – 0.3	1 (EMEG [l])	No
beta-BHC	15	ND – 0.035	0.52 (RDCSCC L[m])	No

Table 6: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Pesticides/PCBs				
Dieldrin	8	ND – 0.017	3 (EMEG)	No
Endrin	3	ND – 0.012	20 (EMEG)	No
Endrin Aldehyde	9	ND – 0.009	20 (EMEG[n])	No
Endrin Ketone	5	ND – 0.012	20 (EMEG)	No
gamma-BHC (Lindane)	4	ND – 0.005	0.52 (RDCSCC L)	No
gamma-Chlordane	47	ND – 0.04	30 (EMEG C)	No
Heptachlor Epoxide	17	ND – 0.013	0.7 (RMEG)	No
Semivolatile Organic Compounds				
1,1'-Biphenyl	13	ND – 0.55	3,000 (RMEG)	No
2-Methylnaphthalene	21	ND – 0.72	2,000 (EMEG)	No
4-Chloroaniline	2	ND – 0.33	200 (RMEG)	No
4-Methylphenol	3	ND – 0.15	390 (RBC[o])	No
Acenaphthene	47	ND – 1.8	3,000 (RMEG)	No
Acenaphthylene	4	ND – 0.13	NA	
Acetophenone	14	ND – 0.12	5,000 (RMEG)	No
Anthracene	68	ND – 4.3	20,000 (RMEG)	No
Benzaldehyde	79	ND – 1.6	NA	
Benzo[a]anthracene	113	ND – 11	0.87 (RBC)	**Yes**
Benzo[b]fluoranthene	113	ND – 9.7	0.87 (RBC)	**Yes**
Benzo[g,h,i]perylene	94	ND – 2.2	NA	
Benso[k]fluoranthene	112	ND – 7.9	8.7 (RBC)	No
Benzo[a]pyrene	111	ND – 10	0.1 (CREG)	**Yes**
Benzyl butyl phthalate	14	ND – 0.46	10,000 (RMEG)	No
bis(2-Chloroethyl)ether	2	ND – 0.11	NA	
bis(2-Ethylhexyl)phthalate	71	ND – 10	46 (RBC)	No

Table 6: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Semivolatile Organic Compounds				
Carbazole	58	ND – 1	32 (RBC)	No
Chrysene	124	ND – 13	87 (RBC)	No
Dibenz[a,h]anthracene	68	ND – 1.2	0.087 (RBC)	**Yes**
Dibensofuran	31	ND – 0.89	NA	
Di-n-butylphthalate	29	ND – 0.17	5,000 (RMEG)	No
Fluoranthene	128	ND – 21	2,000 (RMEG)	No
Fluorene	51	ND – 1.7	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	95	ND – 2.4	0.87 (RBC)	**Yes**
Naphthalene	26	ND – 0.59	1,000 (RMEG)	No
Phenanthrene	115	ND – 16	NA	
Pyrene	130	ND – 34	2,000 (RMEG)	No
Metals				
Alumimum	151	ND – 30,900	100,000 (EMEG I)	No
Antimony	46	ND – 43.5	20 (RMEG)	**Yes**
Arsenic	148	ND – 4,460	20 (EMEG)	**Yes**
Barium	152	ND – 8,530	30,000 (EMEG)	No
Beryllium	130	ND – 4.4	100 (EMEG)	No
Cadmium	144	ND – 17.1	10 (EMEG)	**Yes**
Chromium	152	ND – 3,430	200 (RMEG)	**Yes**
Cobalt	115	ND – 14.3	500 (EMEG I)	No
Copper	142	ND – 1,400	500 (EMEG I)	**Yes**
Lead	151	ND – 39,200	400 (RDCSCC)	**Yes**
Manganese	153	4.5 - 266	3,000 (RMEG)	No
Mercury	68	ND – 3.8	14 (RDCSCC)	No
Nickel	146	ND – 45.2	300 (EMEG)	No
Selenium	21	ND – 8.2	5.5 (RBC)	**Yes**

Table 6: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Metals				
Silver	9	ND – 4.4	200 (EMEG I)	No
Sodium	17	ND – 932	20,000 (EMEG)	No
Thallium	7	ND – 9.8	100,000 (EMEG I)	No
Vanadium	153	1.6 - 81.2	20 (RMEG)	**Yes**
Zinc	152	ND – 2,770	20 (EMEG)	**Yes**
Other				
Cyanide	75	ND – 791	1,000 (RMEG)	No

[a]Number of Samples = 153; [b]Comparison Value; [c]Contaminant of Concern; [d]Not Detected; [e]ATSDR Environmental Media Evaluation Guide; [f]ATSDR Reference Media Evaluation Guide; [g]NJDEP Residential Direct Contact Soil Cleanup Criteria; [h]EMEG for intermediate exposures; [i]Not Available; [j]ATSDR Cancer Risk Evaluation Guide; [k]EMEG based on Chlordane; [l]Based on Aroclor 1254; [m]RDCSCC based on Lindane; [n]EMEG based on Endrin; [o]USEPA Region 3 Risk-Based Concentration

Table 7: Concentration of contaminants detected in the soil (less than 2 feet depth) of the Hilliards Creek (2005)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[c] (mg/kg)	COC[d]
Volatile Organic Compounds (VOCs)				
1,2-Dichlorobenzene	1	ND – 0.0006	20,000 (EMEG[e])	No
2-Butanone	12	ND – 0.048	30,000 (RMEG[f])	No
Acetone	27	ND – 0.32	50,000 (RMEG)	No
Benzene	5	ND – 0.39	10 (CREG[g])	No
Chloroform	1	ND – 0.26	500 (EMEG)	No
cis-1,2-Dichloroethene	5	ND – 0.52	20,000 (EMEG I[h])	No
Cyclohexane	1	ND – 0.0012	NA[i]	
Dichloromethane	7	ND – 0.0047	3,000 (EMEG)	No
Ethylbenzene	3	ND – 0.44	5,000 (RMEG)	No
Methyl Acetate	2	ND – 16	NA	
Methylcyclohexane	10	ND – 33	NA	
Styrene	8	ND – 0.2	10,000 (RMEG)	No
Tetrachloroethene	1	ND – 0.011	1.2 (RBC[j])	No
Toluene	13	ND – 0.83	4,000 (RMEG)	No
Xylenes	6	ND – 2.1	10,000 (RMEG)	No
trans-1,2-Dichloroethene	1	ND – 0.11	20,000 (RMEG I[k])	No
Trichlorofluo-romethane	1	ND – 0.001	20,000 (RMEG)	No
Pesticides/PCBs				
4,4'-DDD	117	ND – 3.5	3 (CREG)	**Yes**
4,4'-DDE	123	ND – 0.42	2 (CREG)	No
4,4'-DDT	92	ND – 0.22	2 (CREG)	No

Table 7: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[c] (mg/kg)	COC[d]
Pesticides/PCBs				
Aldrin	2	ND – 0.0044	2 (EMEG)	No
alpha-BHC	1	ND – 0.0024	0.52 (RDCSCC L[l])	No
appha-Chlordane	65	ND – 0.053	30 (EMEG[m])	No
Aroclor-1248	1	ND – 0.13	1 (EMEG)	No
Arochlor 1254	17	ND – 1.4	1 (EMEG)	**Yes**
Arochlor 1260	64	ND – 3.1	1 (EMEG)	**Yes**
beta-BHC	7	ND – 0.023	0.52 (RDCSCC L)	No
delta-BHC	1	ND – 0.005	0.52 (RDCSCC L)	No
Dieldrin	44	ND – 0.077	3 (EMEG)	No
Endosulfan II	29	ND – 0.04	100 (EMEG)	No
Endosulfan Sulfate	26	ND – 0.022	100 (EMEG E[n])	No
Endrin	6	ND – 0.051	20 (EMEG)	No
Endrin Aldehyde	48	ND – 0.085	1 (EMEG En[o])	No
Endrin Ketone	9	ND – 0.1	20 (EMEG)	No
gamma-BHC (Lindane)	2	ND – 0.018	0.52 (RDCSCC)	No
gamma-Chlordane	62	ND – 0.11	30 (EMEG C[p])	No
Heptachlor Epoxide	8	ND – 0.028	0.7 (RMEG)	No
Methoxychlor	9	ND – 0.05	300 (RMEG)	No
Semi Volatile Organic Compounds (SVOC)				
1,1'-Biphenyl	11	ND – 1	3,000 (RMEG)	No
2,4-Dimethylphenol	2	ND – 0.07	1,000 (RMEG)	No
2-Methylnaphthalene	65	ND – 3.3	2,000 (EMEG)	No
2-Methylphenol	3	ND – 0.33	390 (RBC)	No

Table 7: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[c] (mg/kg)	COC[d]
Semi Volatile Organic Compounds (SVOC)				
3,3'-Dichlorobenzidine	1	ND – 0.01	2 (CREG)	No
4-Chloroaniline	8	ND – 1.4	200 (RMEG)	No
4-Methylphenol	26	ND – 2.4	390 (RBC)	No
Acenaphthene	127	ND – 10	3,000 (RMEG)	No
Acenaphthylene	92	ND – 2.5	NA	
Acetophenone	8	ND – 0.13	NA	
Anthracene	162	ND – 21	20,000 (RMEG)	No
Benzaldehyde	14	ND – 0.34	NA	
Benzo[a]anthracene	205	ND – 29	0.87 (RBC)	**Yes**
Benzo[b]fluoranthene	208	ND – 29	0.87 (RBC)	**Yes**
Benzo[g,h,i]perylene	190	ND – 16	NA	
Benso[k]fluoranthene	199	ND – 27	8.7 (RBC)	**Yes**
Benzo[a]pyrene	203	ND – 29	0.1 (CREG)	**Yes**
Benzyl butyl phthalate	14	ND – 0.2	NA	
bis(2-ethylhexyl)phthalate	108	ND – 15	46 (RBC)	No
Cyclohexanone	31	ND – 0.36	300,000 (RMEG)	No
Carbazole	147	ND – 11	32 (RBC)	No
Chrysene	207	ND – 29	87 (RBC)	No
Dibenz[a,h]anthracene	153	ND – 4.4	0.087 (RBC)	
Dibenzofuran	92	ND – 7.1	NA	
Di-n-butylphthalate	33	ND – 0.18	5,000 (RMEG)	No
Di-n-Octylphthalate	5	ND – 0.52	20,000 (RMEG)	No
Fluoranthene	210	ND – 68	2,000 (RMEG)	No

Table 7: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[c] (mg/kg)	COC[d]
Semi Volatile Organic Compounds (SVOC)				
Fluorene	120	ND – 11	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	190	ND – 17	0.87 (RBC)	**Yes**
Naphthalene	67	ND – 15	1,000 (RMEG)	No
Pentachlorophenol	43	ND – 1.8	50 (EMEG)	No
Phenanthrene	206	ND – 62	NA	
Pyrene	211	ND – 41	2,000 (RMEG)	No
Metals				
Alumimum	233	ND – 6,520,000	100,000 (EMEG I)	**Yes**
Antimony	150	ND – 1,380	20 (RMEG)	**Yes**
Arsenic	211	ND – 3,130	20 (EMEG)	**Yes**
Barium	232	ND – 177,000	30,000 (EMEG)	**Yes**
Beryllium	213	ND – 499	100 (EMEG)	**Yes**
Cadmium	196	ND – 1,100	10 (EMEG)	**Yes**
Chromium	228	ND – 4,760	200 (RMEG)	**Yes**
Cobalt	213	ND – 1,150	500 (EMEG I)	**Yes**
Copper	225	ND – 33,700	500 (EMEG I)	**Yes**
Lead	221	ND – 585,000	400 (RDCSCC)	**Yes**
Manganese	234	ND – 18,000	3,000 (RMEG)	**Yes**
Mercury	193	ND – 11.2	14 (RDCSCC)	No
Nickel	215	ND – 5,660	1,000 (RMEG)	**Yes**
Selenium	137	ND – 2,540	300 (EMEG)	**Yes**
Silver	122	ND – 32.4	300 (RMEG)	No
Thallium	56	ND – 3.8	5.5 (RBC)	No

Table 7: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[c] (mg/kg)	COC[d]
Metals				
Vanadium	229	ND – 18,000	200 (EMEG I)	**Yes**
Zinc	234	ND – 59,800	20,000 (EMEG)	**Yes**
Other				
Phenol	3	ND – 0.5	20,000 (RMEG)	No
Cyanide	182	ND – 353	1,000 (RMEG)	No

[a]Number of Samples = 252; [b]Comparison Value; [c]Contaminant of Concern; [d]Not Detected; [e]ATSDR Environmental Media Evaluation Guide; [f]ATSDR Reference Media Evaluation Guide; [g]ATSDR Cancer Risk Evaluation Guide; [h]EMEG for intermediate exposures; [i]Not Available; [j]USEPA Region 3 Risk-Based Concentration; [k]RMEG for intermediate exposures; [l]NJDEP Residential Direct Contact Soil Cleanup Criteria for Lindane; [m]Based on EMEG for Chlordane; [n]Based on EMEG for Endosulfan; [n]Based on EMEG for Endrin

Table 8: Concentration of contaminants detected in the subsurface soil (> 2 feet depth) of the Hilliards Creek (2005)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Volatile Organic Compounds (VOC)				
2-Butanone	4	ND[d] – 0.02	30,000 (RMEG[e])	No
Acetone	9	ND – 0.052	50,000 (RMEG)	No
Carbon disulfide	1	ND – 0.002	5,000 (RMEG)	No
Chloroform	1	ND – 0.001	500 (EMEG)	No
Cyclohexane	1	ND – 0.0043	NA[f]	
Dichloromethane	4	ND – 0.007	3,000 (EMEG[g])	No
Ethylbenzene	1	ND – 0.013	5,000 (RMEG)	No
Isopropylbenzene	1	ND – 0.025	5,000 (RMEG)	No
Methylcyclohexane	2	ND – 0.012	NA	
Styrene	4	ND – 0.71	10,000 (RMEG)	No
Tetrachloroethene	5	ND – 0.011	1.2 (RBC[h])	No
Trichloroethene	4	ND – 0.002	1.6 (RBC)	No
Pesticides/PCBs				
4,4'-DDD	8	ND – 8.7	3 (CREG[i])	**Yes**
4,4'-DDE	9	ND – 2.8	2 (CREG)	**Yes**
4,4'-DDT	10	ND – 0.011	2 (CREG)	No
Dieldrin	1	ND – 0.00056	3 (EMEG)	No
Endosulfan II	1	ND – 0.00038	100 (EMEG)	No
Endrin Ketone	1	ND – 0.01	20 (EMEG[j])	No
Methoxychlor	3	ND – 0.003	300 (RMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
2-Methylnaphthalene	8	ND – 0.11	2000 (EMEG)	No
4-Methylphenol	1	ND – 0.009	390 (RBC)	No

Table 8: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Semi Volatile Organic Compounds (VOC)				
Acenaphthene	11	ND – 1	3,000 (RMEG)	No
Acenaphthylene	9	ND – 0.3	NA	
Anthracene	16	ND – 1.7	20,000 (RMEG)	No
Benzo[a]anthracene	21	ND – 5.6	0.87 (RBC)	**Yes**
Benzo[b]fluoranthene	21	ND – 4.8	0.87 (RBC)	**Yes**
Semi Volatile Organic Compounds (SVOCs)				
Benzo[g,h,i]perylene	18	ND – 2.2	NA	
Benzo[k]fluoranthene	20	ND – 4.8	8.7 (RBC)	No
Benzo[a]pyrene	21	ND – 5.2	0.1 (CREG)	**Yes**
bis(2-ethylhexyl)phthalate	4	ND – 0.1	46 (RBC)	No
Cyclohexanone	1	ND – 0.048	300,000 (RMEG)	No
Carbazole	12	ND – 0.84	32 (RBC)	No
Chrysene	21	ND – 6.2	87 (RBC)	No
Dibenz[a,h]anthracene	13	ND – 0.91	0.087 (RBC)	**Yes**
Dibenzofuran	9	ND – 0.38	NA	
Diethylphthalate	1	ND – 0.064	40,000 (RMEG)	No
Di-n-butylphthalate	5	ND – 0.034	5,000 (RMEG)	No
Di-n-Octylphthalate	1	ND – 0.029	20,000 (RMEG)	No
Fluoranthene	21	ND – 14	2,000 (RMEG)	No
Fluorene	11	ND – 1	2,000 (RMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
Indeno[1,2,3-cd]pyrene	19	ND – 2.4	0.87 (RBC)	**Yes**
Naphthalene	8		1,000 (RMEG)	No

Table 8: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Semi Volatile Organic Compounds (VOC)				
Pentachlorophenol	2	ND – 0.29	50 (EMEG)	No
Phenanthrene	19	ND – 8	NA	
Pyrene	21	ND – 7.3	2,000 (RMEG)	No
Metals				
Alumimum	26	ND – 4,140	100,000 (EMEG I[k])	No
Antimony	5	ND – 2.1	20 (RMEG)	No
Arsenic	22	ND – 25.5	20 (EMEG)	**Yes**
Barium	25	ND – 1,020	30,000 (EMEG)	No
Beryllium	23	ND – 0.3	100 (EMEG)	No
Cadmium	18	ND – 4.9	10 (EMEG)	No
Chromium	24	ND – 55.1	200 (RMEG)	No
Cobalt	19	ND – 3.3	500 (EMEG I)	No
Copper	24	ND – 43.7	500 (EMEG I)	No
Metals				
Lead	20	ND – 440	400 (RDCSCC[l])	**Yes**
Manganese	27	ND – 12,200	3,000 (RMEG)	**Yes**
Mercury	15	ND – 15.9	14 (RDCSCC)	**Yes**
Nickel	25	ND – 15.8	1,000 (RMEG)	No
Selenium	16	ND – 1.1	300 (EMEG)	No
Silver	5	ND – 0.36	300 (RMEG)	No
Thallium	9	ND – 1.6	5.5 (RBC)	No
Vanadium	26	ND – 14.7	200 (EMEG I)	No
Zinc	26	ND – 400	20,000 (EMEG)	No

Table 8: (Contd.)

Contaminant	No. of detection[a]	Concentration Range (mg/kg)	Environmental Guideline CVs[b] (mg/kg)	COC[c]
Other				
Cyanide	10	ND – 8.6	1,000 (RMEG)	No

[a]Number of Samples = 28; [b]Standard Deviation, for contaminants with standard deviation greater than the mean, the data are relatively widely dispersed and the mean is a rather poor representation of the full data set; [c]Comparison Value; [d]Contaminant of Concern; [e]ATSDR Reference Media Evaluation Guide; [f]Not Available; [g]ATSDR Environmental Media Evaluation Guide; [h]USEPA Region 3 Risk-Based Concentration; [i]ATSDR Cancer Risk Evaluation Guide; [j]Based on EMEG for Endrin; [k]ATSDR Environmental Media Evaluation Guide for intermediate exposures; [l]NJDEP Residential Direct Contact Soil Cleanup Criteria

Table 9: Concentration of contaminants detected in the Hilliards Creek surface water (2004)

Contaminants	No. of Detection[a]	Concentration Range (µg/L)	Environmental Guideline CVs[c] (µg/L)	COC[d]
Metals				
Arsenic	1	30.1	0.02 (CREG[e])	**Yes**
Lead	4	12.4 – 29	15 (AL[f])	**Yes**

[a]Number of Samples = 4; [b]Standard Deviation; [c]Comparison Value; [d]Contaminant of Concern; [e]ATSDR Cancer Risk Evaluation Guide; [f]NJDEP Action Level for Lead

Table 10: Summary of Exposure Pathways

Medium	Point of Exposure	Exposure Route	Exposed Population	Exposure Pathway Classification		
				Past	Present	Future
Surface soils	Former Plant and Hilliard Creek	Ingestion, skin	Residents, hunters, recreators	Completed	Potential[a]	Potential[a]
Surface water	Hilliard Creek	Ingestion, skin	Residents, hunters, recreators	Completed	Potential[a]	Potential[a]
Groundwater	Off-site (Potable wells)	Ingestion, inhalation, skin	Residents	Potential	Potential	Potential
	Indoor Air			Potential	Potential	Potential
Biota	Hilliards Creek and Lakes	Ingestion	Residents, hunters, recreators	Potential	Potential	Potential

[a]partilly interrupted due to the construction of fence

Table 11: Comparison of Surface Soil Exposure Dose from the former facility with the Health Guideline CVs

Contaminants of Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline CVs[c] (mg/kg/day)	Potential for Non-cancer Health Effects
			Child[a]	Adult[b]		
Semivolatile Organic Compounds (SVOCs)						
Benzo[a]pyrene	0.062	Arithmetic Mean	1.2×10^{-7}	1.89×10^{-8}	NA[d]	No
Metals						
Lead	995	ProUCL	2.02×10^{-3}	3.03×10^{-4}	NA[e]	**Yes**

[a]Child exposure scenario: 3 days/week, 6 month/year, 200 mg/day ingestion rate and 21 kg body weight; [b]Adult exposure scenario: 3 days/week, 6 month/year, 100 mg/day ingestion rate and 70 kg body weight; [c]Comparison Value; [d]Not Available; [e]The non-cancer health effects of lead were evaluated by using the IEUBK model

Table 12: Comparison of surface soil exposure dose from the Hilliards Creek with the Health Guideline CVs

Contaminants of Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline CVs[c] (mg/kg/day)	Potential for Non-cancer Health Effects
			Child[a]	Adult[b]		
Semivolatile Organic Compounds (SVOCs)						
4,4'-DDD	0.57	ProUCL	1.17×10^{-6}	1.76×10^{-7}	5.0×10^{-4} (RfD[d])	No
Benzo[a]anthracene	3.55		7.23×10^{-6}	1.08×10^{-6}	NA[e]	
Benzo[b]fluoranthene	5.14		1.05×10^{-5}	1.57×10^{-6}	NA	
Benso[k]fluoranthene	3.92		7.98×10^{-6}	1.2×10^{-6}	NA	
Benzo[a]pyrene	4.04		8.22×10^{-6}	1.23×10^{-6}	NA	
Indeno[1,2,3-cd]pyrene	2.24		4.56×10^{-6}	6.84×10^{-7}	NA	
Arochlor 1254	0.66		1.34×10^{-6}	2.01×10^{-7}	2×10^{-5}[f] (RfD)	No
Arochlor 1260	1.08		2.2×10^{-6}	3.3×10^{-7}		
Metals						
Alumimum	263,035	ProUCL	5.35×10^{-1}	8.03×10^{-2}	2 (MRL I[g])	No
Antimony	108.4		2.21×10^{-4}	3.31×10^{-5}	0.0004 (RfD)	No
Arsenic	485.8		9.89×10^{-4}	1.48×10^{-4}	0.0003 (MRL[h])	**Yes**

Table 12: (Contd.)

Contaminants of Concern	Exposure Point Concentration (mg/kg)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline CVs[c] (mg/kg/day)	Potential for Non-cancer Health Effects
			Child[a]	Adult[b]		
Metals						
Barium	7,743	ProUCL	1.58×10^{-2}	2.36×10^{-3}	0.6 (MRL)	No
Beryllium	20.45		4.16×10^{-5}	6.24×10^{-6}	0.002 (MRL)	No
Cadmium	46.8		9.52×10^{-5}	1.43×10^{-5}	0.0002 (MRL)	No
Chromium	637.5		1.3×10^{-3}	1.95×10^{-4}	0.003 (RfD)	No
Cobalt	51.7		1.05×10^{-4}	1.58×10^{-5}	0.01 (MRL I)	No
Copper	1403		2.86×10^{-3}	4.28×10^{-4}	0.01 (MRL I)	No
Lead	4,108[i]		5.15×10^{-2}	7.72×10^{-3}	NA[j]	**Yes**
Manganese	1124		2.29×10^{-3}	3.43×10^{-4}	0.05 (RfD)	No
Nickel	234.1		4.76×10^{-4}	7.15×10^{-5}	0.02 (RfD)	No
Selenium	114.2		2.23×10^{-4}	3.49×10^{-5}	0.005 (MRL)	No
Vanadium	734.4		1.49×10^{-3}	2.24×10^{-4}	0.003 (MRL I)	No
Zinc	2,723		5.54×10^{-3}	8.31×10^{-4}	0.3 (MRL)	No

[a]Child exposure scenario: 3 days/week, 6 month/year, 200 mg/day ingestion rate and 21 kg body weight; [b]Adult exposure scenario: 3 days/week, 6 month/year, 100 mg/day ingestion rate and 70 kg body weight; [c]Comparison Value; [d]EPA Reference Dose; [e]Not Available; [f]Based on Aroclor 1254; [g]ATSDR Minimal Risk Level for intermediate exposures; [h]ATSDR Minimal Risk Level; [i]The ProUCL analysis indicated 585,000 mg/kg to be an outlier; [j]The non-cancer health effects of lead were evaluated by using the IEUBK model

Table 13: Comparison of Surface Water Exposure Dose of the Hilliards Creek with the Health Guideline CVs

Contaminants of Concern	Exposure Point Concentration (μg/L)	Estimation Method	Estimated Exposure Dose (mg/kg/day)		Health Guideline CVs[c] (mg/kg/day)	Potential for Non-cancer Health Effects
			Child[a]	Adult[b]		
Metals						
Arsenic	30.1	Maximum[d]	1.5×10^{-5}	4.6×10^{-6}	0.0003 (MRL[e])	No
Lead	29	Maximum[f]			15 (AL[g])	**Yes**

[a]Child exposure scenario: 3 days/week, 6 month/year, 50 mL/day ingestion rate and 21 kg body weight; [b]Adult exposure scenario: 3 days/week, 6 month/year, 50 mL/day ingestion rate and 70 kg body weight; [c]Comparison Value; [d]One Sample collected; [e]ATSDR Minimal Risk Level; [f]Estimated value by ProUCL exceeded the maximum; [g]NJDEP Action Level for Lead

Table 14: Calculated LECR associated with the contaminants detected in the Hilliards Creek surface soil

Contaminants of Concern	Exposure Conc. (mg/kg)	DHHS[a] Cancer Class	Exposure Dose[b] (mg/kg/day)	CSF[c] $(mg/kg/d)^{-1}$	LECR[d]
Polycyclic Aromatic Compounds (PAHs)					
Benzo[a]pyrene	0.062	2	8.1×10^{-9}	7.3	5.9×10^{-8}
Metals					
Lead	995	2[e]			
				Sum =	5.9×10^{-8}

[a]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [b]Adult exposure scenario: 3 days/week, 6 month/year, 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; [c]Cancer Slope Factor; [d]Lifetime Excess Cancer Risk; [c]Cancer Slope Factor is unavailable

Table 15: Calculated LECR associated with the contaminants detected in the Hilliards Creek floodplain surface soil

Contaminants of Concern	Exposure 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 $(mg/kg/d)^{-1}$	LECR[d]
Polycyclic Aromatic Compounds (PAHs)								
Benzo[a]anthracene	3.55	2	0.1	2.9	5.52	7.23×10^{-7}	7.3	5.28×10^{-6}
Benzo[b]fluoranthene	5.14	2	0.1	2.9				
Benso[k]fluoranthene	3.92	2	0.1	2.7				
Benzo[a]pyrene	4.04	2	1	29				
Indeno[1,2,3-cd]pyrene	2.24	2	0.1	1.7				
4,4'-DDD	0.57	2				7.45×10^{-8}	0.34	2.54×10^{-8}
Arochlor 1254	0.66	2				8.63×10^{-8}	2	1.73×10^{-7}
Arochlor 1260	1.08	2				1.41×10^{-7}	2	2.83×10^{-7}
Metals								
Alumimum	263,035	3						
Antimony	108.4	3						
Arsenic	485.8	1				6.35×10^{-5}	1.5	9.53×10^{-5}
Barium	7,743	3						
Beryllium	20.45	1[e]						
Cadmium	46.8	1[f]						
Chromium	637.5	1[g]						

Table 15: (Contd.)

Contaminants of Concern	Exposure 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 $(mg/kg/d)^{-1}$	LECR[d]
Cobalt	51.7	3						
Copper	1403	3						
Lead	4,108	2[h]						
Manganese	1124	3						
Nickel	234.1	2[i]						
Selenium	114.2	3						
Vanadium	734.4	3						
Zinc	2,723	3						
							Sum =	**1.01×10^{-4}**

[a]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [b]Cancer potency factor relative to benzo[a]pyrene (BaP); [c]Adult exposure scenario: 3 days/week, 6 month/year, 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; [d]Lifetime Excess Cancer Risk; [e]Cancer Slope Factor is unavailable for nickel; [f]Limited epidemiologic studies have indicated that exposure to cadmium in food or drinking water is not carcinogenic; [g]Information on the carcinogenicity of chromium by oral exposure in humans was unavailable; [h]Cancer Slope Factor is unavailable for lead; [i]Cancer Slope Factor is unavailable for nickel

Table 16: Calculated LECR associated with the contaminants detected in the Hilliards Creek surface water

Contaminants of Concern	Exposure Conc. (μg/L)	DHHS[a] Cancer Class	Exposure Dose[b] (mg/kg/day)	CSF $(mg/kg/d)^{-1}$	LECR[c]
Metals					
Arsenic	30.1	1	1.96×10^{-6}	1.5	2.9×10^{-6}
Lead	29	2[d]			

[a]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [b]Adult exposure scenario: 3 days/week, 6 month/year, 50 mL/day ingestion rate, 70 kg body weight and 30 year exposure duration; [c]Lifetime Excess Cancer Risk; [d]Cancer Slope Factor is unavailable

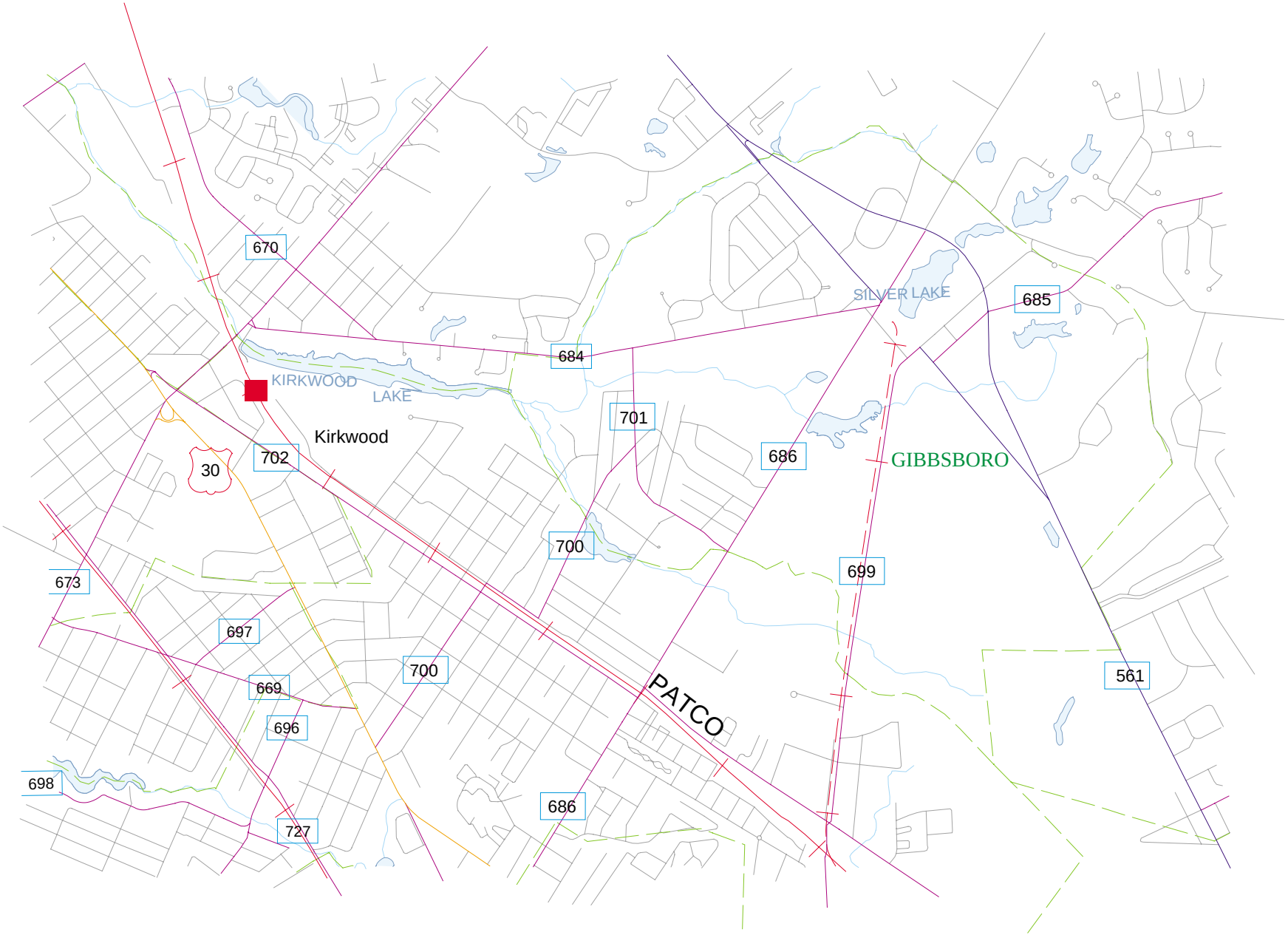


Figure 2: Location of Sherwin-Williams/Hilliards Creek site (not to scale)

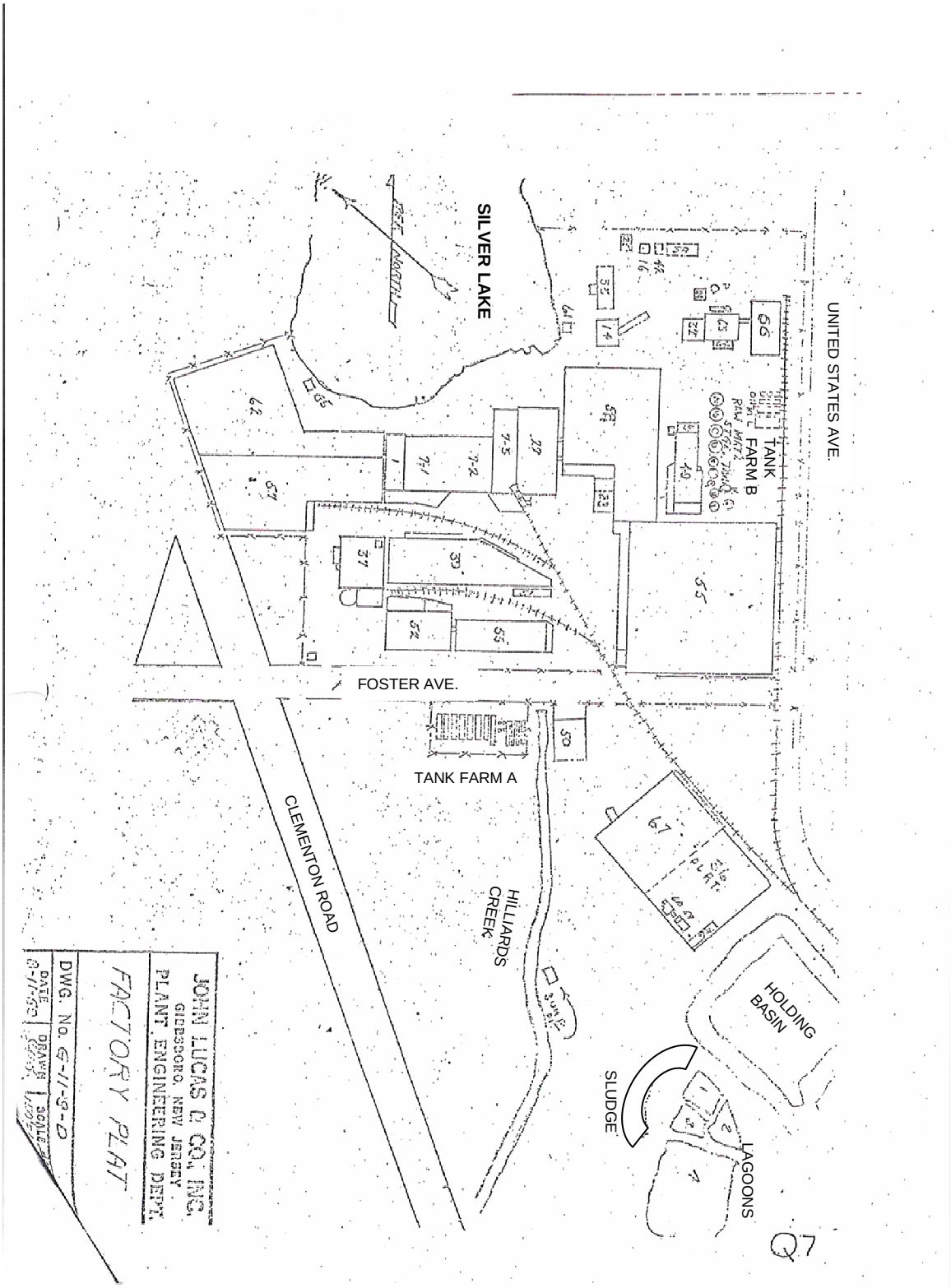


Figure 3: Facility Buildings and areas of concern identified for the Sherwin-Williams/Hilliard Creek site

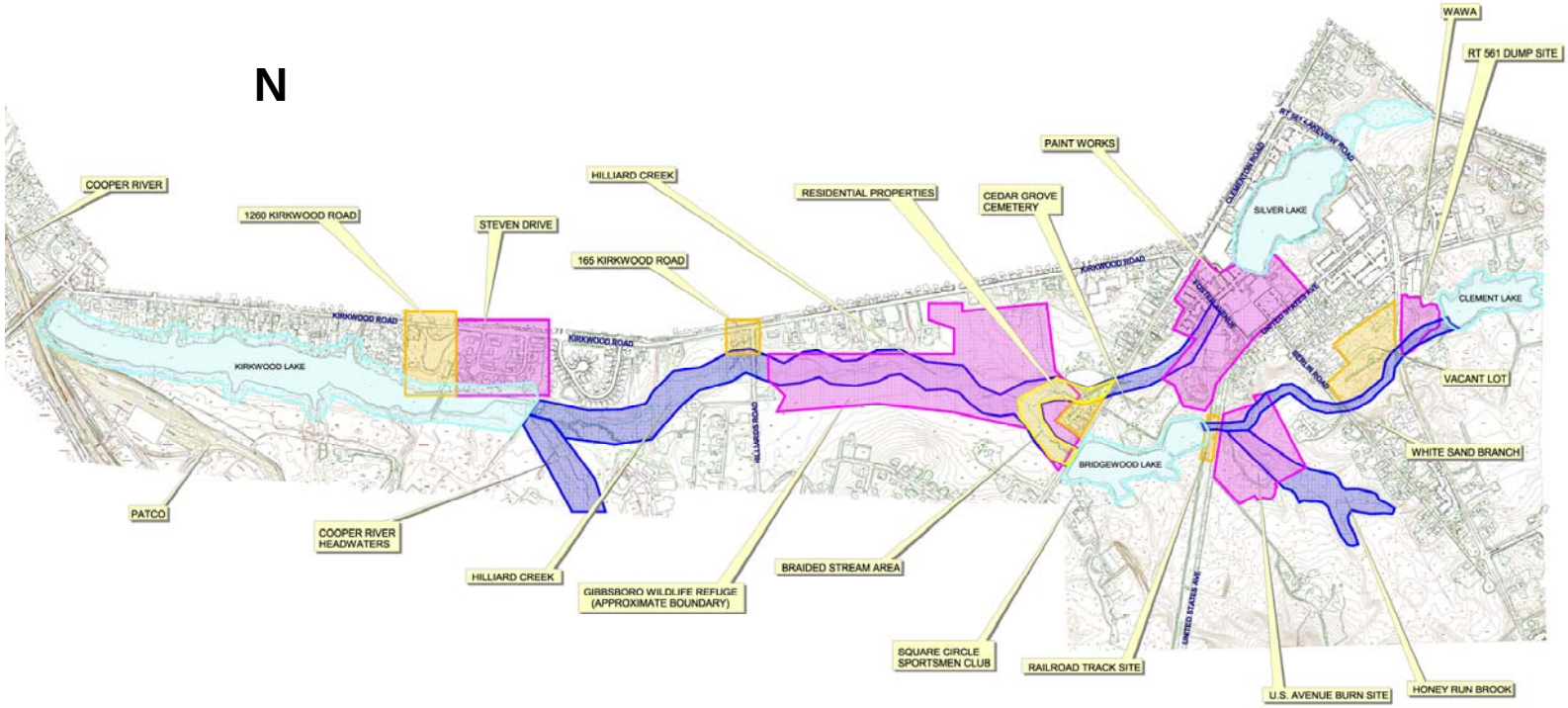
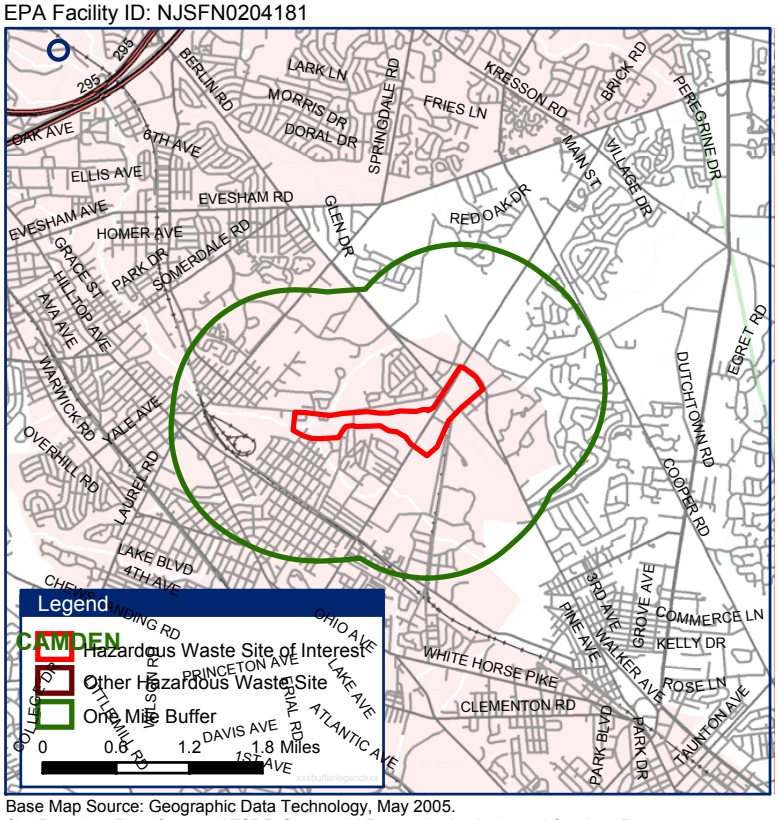


Figure 4: Reference Map of the Sherwin-Williams/Hilliards Creek site (not to scale)

Sherwin-Williams/Hilliards Creek
Gibbsboro, NJ

EPA Facility ID: NJSFN0204181



Base Map Source: Geographic Data Technology, May 2005.
Site Boundary Data Source: ATSDR Geospatial Research, Analysis, and Services Program, Current as of Generate Date (bottom left-hand corner).
Coordinate System (All Panels): NAD 1983 StatePlane New Jersey FIPS 2900 Feet

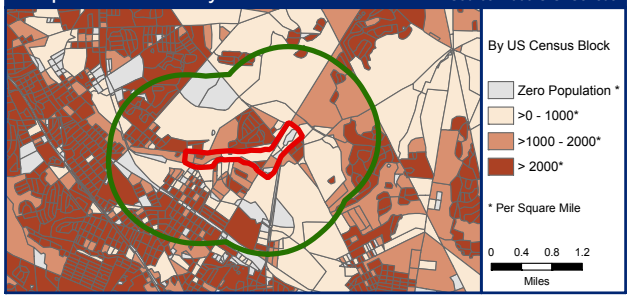
Site Location: Camden County, NJ

Demographic Statistics
Within One Mile of Site*

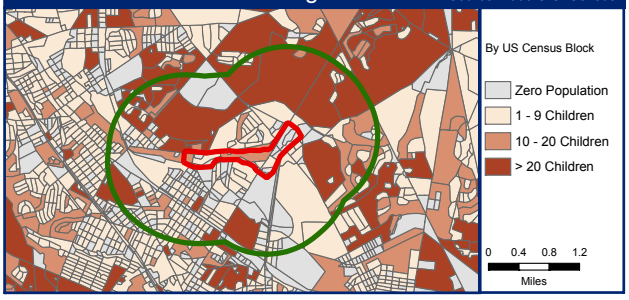
Total Population	15,892
White Alone	11,566
Black Alone	2,416
Am. Indian & Alaska Native Alone	42
Asian Alone	1,341
Native Hawaiian & Other Pacific Islander Alone	3
Some Other Race Alone	168
Two or More Races	357
Hispanic or Latino**	629
Children Aged 6 and Younger	1,377
Adults Aged 65 and Older	1,809
Females Aged 15 to 44	3,600
Total Housing Units	6,871

Demographics Statistics Source: 2000 U.S. Census
* Calculated using an area-proportion spatial analysis technique
** People who identify their origin as Hispanic or Latino may be of any race.

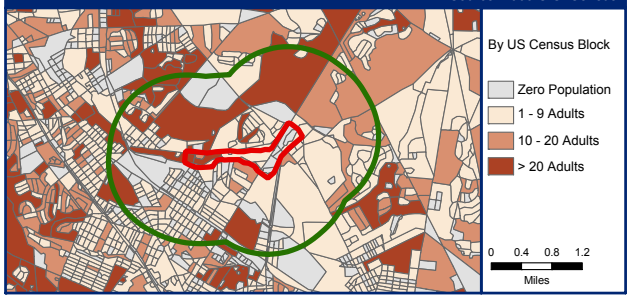
Population Density
Source: 2000 U.S. Census



Children 6 Years and Younger
Source: 2000 U.S. Census



Adults 65 Years and Older
Source: 2000 U.S. Census



Females Aged 15 to 44
Source: 2000 U.S. Census

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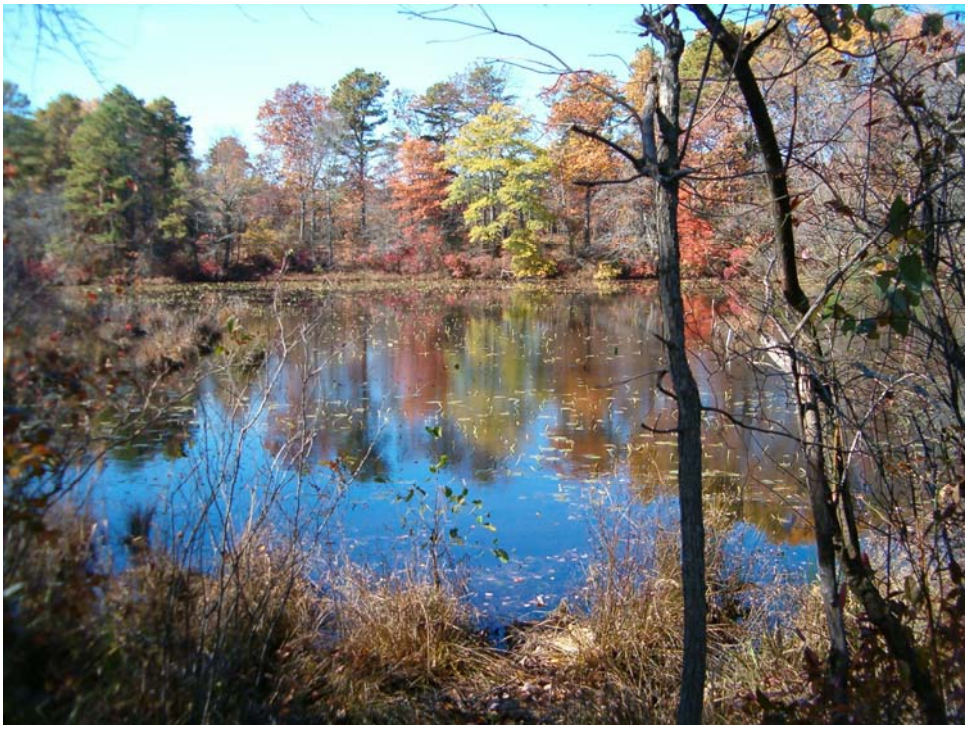
Figure 5: Demographic Information of the Sherwin-Williams/Hilliards Creek site

Photograph 1: The Silver Lake and the former paint manufacturing facility



Photograph 2: The Bridgewood Lake located to the south of the facility

Photograph 3: Location of the Tank Farm A (currently a parking lot)

Photograph 4: The FPR system located near Foster Avenue

Photograph 5: Fenced area located to the west of the school

Photograph 6: The Hilliard Creek flowing through the residential backyard

Photograph 7: The residences across the US Avenue near the facility

Photograph 8: The SVE system located near Foster Avenue

Appendix A

Toxicologic Summaries

The toxicological summaries provided in this appendix are based on ATSDR's ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html). 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.

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

Breathing high levels of 1,2-dichloroethene can cause nausea, drowsiness, and tiredness; breathing very high levels is fetal. Livers and lungs were the target organs in chronic exposures studies in animals. Lower doses of cis-1,2-dichloroethene caused effects on the blood(such as decreased numbers of red blood cells) and liver. Exposure to 1,2-dichloroethene has not been shown to affect fertility in people or animals. The US Environmental Protection Agency (EPA) has determined that 1,2-dichloroethene is not classifiable as to its human carcinogenicity.

DDT, DDE, and DDD. DDT (dichlorodiphenyltrichloroethane) is a pesticide once widely used to control insects in agriculture and insects that carry diseases such as malaria. DDT is a white, crystalline solid with no odor or taste. The use of DDT in the U.S. was banned in 1972 because of damage to wildlife, but is still used in some countries. DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) are chemicals similar to DDT that contaminate commercial DDT preparations. DDE has no commercial use. DDD was also used to kill pests, but its use has also been banned. One form of DDD has been used medically to treat cancer of the adrenal gland.

DDT affects the nervous system. Individuals ingesting large amounts of DDT became excitable and had tremors and seizures. These effects went away after the exposure stopped. No effects were seen in people who took small daily doses of DDT by capsule for 18 months. A study in humans showed that women who had high amounts of a form of DDE in their breast milk were unable to breast feed their babies for as long as women who had little DDE in the breast milk. Another study in humans showed that women who had high amounts of DDE in breast milk had an increased chance of having premature babies. In animals, short-term exposure to large amounts of DDT in food affected the nervous system, while long-term exposure to smaller amounts affected the liver. Also in animals, short-term oral exposure to small amounts of DDT or its breakdown products may also have harmful reproductive effects. Studies in DDT-exposed workers did not show increases in cancer. Studies in animals given DDT with the food have shown that DDT can cause liver cancer.

The Department of Health and Human Services (DHHS) determined that DDT may reasonable be anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC) determined that DDT may possibly cause cancer in humans. The EPA determined that DDT, DDE, and DDD are probable human carcinogens.

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.

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

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

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

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

Lung cancer has been observed in some studies of rats that breathed high levels of antimony. No human studies are available. The DHHS, the International Agency for Research on Cancer, and the EPA have not classified antimony as to its human carcinogenicity.

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 DHHS, and the EPA have determined that inorganic arsenic is a human carcinogen

Barium. Barium is a silvery-white metal which exists in nature only in ores containing mixtures of elements. It combines with other chemicals such as sulfur or carbon and oxygen to form barium compounds. Barium compounds are used by the oil and gas industries to make drilling muds, which make it easier to drill through rock by keeping the drill bit lubricated. They are also used in paint, bricks, ceramics, glass, and rubber.

The health effects of the different barium compounds depend on water solubility or in the stomach contents. Barium compounds that do not dissolve well, such as barium sulfate, are not generally harmful. Barium has been found to potentially cause gastrointestinal disturbances and muscular weakness when people are exposed to it at levels above the EPA drinking water standards for relatively short periods of time. Some

people who eat or drink amounts of barium above background levels found in food and water for a short period may experience vomiting, abdominal cramps, diarrhea, difficulties in breathing, increased or decreased blood pressure, numbness around the face, and muscle weakness. Eating or drinking very large amounts of barium compounds that easily dissolve can cause changes in heart rhythm or paralysis and possibly death. Animals that drank barium over long periods had damage to the kidneys, decreases in body weight, and some died.

The DHHS and the IARC have not classified barium as to its carcinogenicity. The EPA has determined that barium is not likely to be carcinogenic to humans following ingestion and that there is insufficient information to determine whether it will be carcinogenic to humans following inhalation exposure.

Beryllium. Beryllium is a naturally occurring, hard, grayish metal naturally found in mineral rocks, coal, soil, and volcanic dust. Beryllium compounds are commercially mined, and the beryllium is purified for use in nuclear weapons and reactors, aircraft and space vehicle structures, instruments, x-ray machines, and mirrors. Beryllium ores are used to make specialty ceramics for electrical and high-technology applications. Beryllium alloys are used in automobiles, computers, sports equipment (golf clubs and bicycle frames), and dental bridges.

The adverse effect of beryllium through inhalation depends on the extent of exposure. Air levels greater than 1000 $\mu g/m^3$ can result in an acute condition. This condition resembles pneumonia and is called acute beryllium disease. Individuals can (1-15%) become sensitive to beryllium and may develop an inflammatory reaction in the respiratory system called chronic beryllium disease (CBD). CBD can occur many years after exposure to higher than normal levels of beryllium (greater than 0.5 $\mu g/m^3$). This disease can make you feel weak and tired, and can cause difficulty in breathing, anorexia, weight loss, and may also lead to right side heart enlargement and heart disease in advanced cases. Some people who are sensitized to beryllium may not have any symptoms. Ingestion of beryllium has not been reported to cause adverse effects in humans because very little beryllium is absorbed from the stomach and intestines. Ulcers have been seen in dogs ingesting beryllium in the diet. Beryllium contact with skin that has been scraped or cut may cause rashes or ulcers.

Long term exposure to beryllium can increase the risk of developing lung cancer in people. The DHHS and the IARC have determined that beryllium is a human carcinogen. The EPA has determined that beryllium is a probable human carcinogen. EPA has estimated that lifetime exposure to 0.04 $\mu g/m^3$ beryllium can result in a one in a thousand chance of developing cancer.

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 DHHS has determined that benzene is a known human carcinogen. Long-term exposure to high levels of benzene in the air can cause leukemia, cancer of the blood-forming organs.

Bis(2-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/m^3. 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 DHHS has determined that certain chromium (VI) compounds are known to cause cancer in humans. The EPA 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.

Cobalt. Cobalt is a naturally occurring element found in rocks, soil, water, plants, and animals. Cobalt is used to produce alloys used in the manufacture of aircraft engines, magnets, grinding and cutting tools, artificial hip and knee joints. Cobalt compounds are also used to color glass, ceramics and paints, and used as a drier for porcelain enamel and paints. Radioactive cobalt is used for commercial and medical purposes. 60Co (read as cobalt sixty) is used for sterilizing medical equipment and consumer products, radiation therapy for treating cancer patients, manufacturing plastics, and irradiating food. 57Co is used in medical and scientific research.

Cobalt can benefit or harm human health. Cobalt is beneficial for humans because it is part of vitamin B12. Exposure to high levels of cobalt can result in lung and heart effects and dermatitis. Liver and kidney effects have also been observed in animals exposed to high levels of cobalt. Exposure to high levels of radioactive cobalt can damage cells. Individuals may experience acute radiation syndrome that includes nausea, vomiting, diarrhea, bleeding, coma, and even death.

Nonradioactive cobalt has not been found to cause cancer in humans or animals following exposure in food or water. Cancer has been shown, however, in animals that breathed cobalt or when cobalt was placed directly into the muscle or under the skin. Based on the laboratory animal data, the IARC has determined that cobalt and cobalt compounds are possibly carcinogenic to humans. Exposure to high levels of cobalt radiation can cause changes in the genetic materials within cells and may result in the development of some types of cancer.

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 EPA has determined that copper is not classifiable as to human carcinogenicity.

Ethylbenzene. Ethylbenzene is a colorless, flammable liquid with a pungent odor. Ethylbenzene is commonly used as a solvent, chemical intermediate in the manufacture of styrene and synthetic rubber and as an additive in some automotive and aviation fuels. Occupational exposure to ethylbenzene may occur during production and conversion to polystyrene and during production and use of mixed xylenes. The general public can be exposed to ethylbenzene in ambient air as a result of releases from vehicle exhaust and cigarette smoke.

Ethylbenzene can be absorbed through the lungs, digestive tract, and skin. It also crosses the placenta. The liver is the major organ of ethylbenzene metabolism. In humans the major metabolites of ethylbenzene are mandelic acid (64 to 70%) and phenylglyoxylic acid (25%); however, these compounds are only minor metabolites in laboratory animals. Excretion occurs primarily in the urine.

Ingestion of sublethal amounts of ethylbenzene is likely to cause central nervous system (CNS) depression, oro-pharyngeal and gastric discomfort, and vomiting; however, specific experimental data are not available. Animal studies indicate that the primary target organs following chronic oral exposures are likely to be the liver and kidney. The oral RfD for chronic exposures is based on increased weight and histopathological changes in the liver and kidneys of rats.

Acute exposures to high atmospheric concentrations of ethylbenzene may cause eye and respiratory tract irritation and CNS effects (e.g., coordination disorders, dizziness, vertigo, narcosis, convulsions, pulmonary irritation, and conjunctivitis). Concentrations of 1,000 ppm (434 mg/m^3) can be highly irritating to the eyes of humans; the threshold for eye irritation has been reported to be 200 ppm (879 mg/m^3). No evidence is available to suggest that occupational exposures to ethylbenzene result in chronic toxic effects; however, histopathological changes in the liver and kidney have been observed in experimental animals following prolonged inhalation exposures. Laboratory studies also indicate that exposure to ethylbenzene (4,340 mg/m^3) during gestation results in adverse developmental effects in rats (skeletal variants) and rabbits (reduced number of live offspring per litter).

No epidemiological information is available on the potential carcinogenicity of ethylbenzene in humans following oral or inhalation exposures. A statistically significant

increase in total malignant tumors was observed in female rats dosed orally with ethylbenzene; however, because of study limitations, these results cannot be considered conclusive. Although ethylbenzene has been tested by NTP in a two-year rodent bioassay, the results of that study are not yet available. Ethylbenzene is placed by EPA in Group D, not classifiable as to human carcinogenicity, based on a lack of data in humans and animals.

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 DHHS 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. Organic manganese compounds include pesticides, such as maneb or mancozeb, and methylcyclopentadienyl manganese tricarbonyl (MMT), a fuel additive in some gasolines.

Chronic occupational exposures to manganese can result in mental and emotional disturbances and slow and clumsy body movements. This combination of symptoms is a disease called "manganism." Manganism occurs due to injury in a part of the brain that helps control body movements. Exposure to high levels of the metal may also cause respiratory problems and sexual dysfunction. The EPA has determined that manganese is not classifiable as to human carcinogenicity.

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 EPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

Methylene Chloride Methylene chloride is a colorless liquid with a mild, sweet odor. It is used as an industrial solvent and as a paint stripper. It may also be found in some aerosol and pesticide products and is used in the manufacture of photographic film. The most likely way to be exposed to methylene chloride is by breathing contaminated air.

Breathing in large amounts of methylene chloride may cause dizziness, nausea, and tingling or numbness of fingers and toes. A person breathing smaller amounts of methylene chloride may become less attentive and less accurate in tasks requiring hand-eye coordination. We do not know if methylene chloride can affect the ability of people to have children or if it causes birth defects. Some birth defects have been seen in animals inhaling very high levels of methylene chloride.

We do not know if methylene chloride can cause cancer in humans. An increased cancer risk was seen in mice breathing large amounts of methylene chloride for a long time. The DHHS has determined that methylene chloride can be reasonably anticipated to be a cancer-causing chemical, and the EPA has determined that methylene chloride is a probable cancer-causing agent in humans.

Naphthalene and 2-Methylnapthalene. Naphthalene is a white solid that evaporates easily. Fuels such as petroleum and coal contain naphthalene. The major commercial use of naphthalene is in the manufacture of polyvinyl chloride (PVC) plastics. Its major consumer use is in moth repellents and toilet deodorant blocks. 1-Methylnaphthalene is naphthalene-related compounds. 2-Methylnaphthalene is used to make other chemicals such as dyes, resins, and vitamin K.

Exposure to large amounts of naphthalene may damage or destroy red blood cells. This condition is called hemolytic anemia. Some symptoms of hemolytic anemia are fatigue, lack of appetite, restlessness, and pale skin. Exposure to large amounts of naphthalene may also cause nausea, vomiting, diarrhea, blood in the urine, and a yellow color to the skin. In animal studies, chronic lifetime exposures to naphthalene have shown to develop irritation and inflammation of nose and lungs. Mice fed food containing 1-methylnaphthalene for most of their lives had part of their lungs filled with an abnormal material. It is unclear if naphthalene causes reproductive effects in animals. No human studies for 2-methylnaphthalene is available.

Based on the results from animal studies, the DHHS concluded that naphthalene is reasonably anticipated to be a human carcinogen. The IARC concluded that naphthalene is possibly carcinogenic to humans. The EPA determined that naphthalene

is a possible human carcinogen (Group C) and that the data are inadequate to assess the human carcinogenic potential of 2-methylnaphthalene.

Nickel. Nickel is a very abundant natural element. Pure nickel is a hard, silvery-white metal and can be combined with other metals, such as iron, copper, chromium, and zinc, to form alloys. These alloys are used to make coins, jewelry, and items such as valves and heat exchangers. Most nickel is used to make stainless steel. Nickel can combine with other elements such as chlorine, sulfur, and oxygen to form nickel compounds. Many nickel compounds dissolve fairly easy in water and have a green color. Nickel compounds are used for nickel plating, to color ceramics, to make some batteries, and as substances known as catalysts that increase the rate of chemical reactions.

The most common harmful health effect of nickel in humans is an allergic reaction. Approximately 10-20% of the population is sensitive to nickel. People can become sensitive to nickel through contact with the skin for a long time. Once sensitized to nickel, further contact may produce skin. Less frequently, sensitive individuals may have asthma attacks following exposure to nickel. Some sensitized people react when they consume food or water containing nickel or breathe dust containing it. Long term occupational inhalation exposures have resulted in chronic bronchitis and reduced lung function. Ingestion of water containing high amounts of nickel caused stomach ache and adverse effects on blood and kidneys. Damage to the lung and nasal cavity has been observed in rats and mice breathing nickel compounds. Eating or drinking large amounts of nickel has caused lung disease in dogs and rats and has affected the stomach, blood, liver, kidneys, and immune system in rats and mice, as well as their reproduction and development.

Cancers of the lung and nasal sinus have resulted from occupational exposures to dust containing high levels of nickel. The DHHS has determined that nickel metal may reasonably be anticipated to be a carcinogen and that nickel compounds are known human carcinogens. The IARC has determined that some nickel compounds are carcinogenic to humans and that metallic nickel may possibly be carcinogenic to humans. The EPA has determined that nickel refinery dust and nickel subsulfide is 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-cd0pyrene, 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 DHHS has determined that some PAHs may reasonably be expected to be carcinogens. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

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

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

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

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

from breast milk. Transplacental transfers of PCBs were also reported In most cases, the benefits of breast-feeding outweigh any risks from exposure to PCBs in mother's milk.

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

Pentachlorophenol Pentachlorophenol is a manufactured chemical that does not occur naturally. Pure pentachlorophenol exists as colorless crystals. Impure pentachlorophenol (the form usually found at hazardous waste sites) is dark gray to brown and exists as dust, beads, or flakes. Pentachlorophenol was widely used as a pesticide and wood preservative. Since 1984, the purchase and use of pentachlorophenol has been restricted to certified applications (such as a wood preservative for utility poles, railroad ties, and wharf pilings) and unavailable to the general public

Occupational studies show that exposure to high levels of pentachlorophenol can cause very high fever, profuse sweating, and difficulty breathing. The body temperature can cause injury to various organs and tissues, and even death. Liver effects and damage to the immune system have also been observed in humans exposed to high levels of pentachlorophenol for a long time. In animal studies, exposure to high doses of pentachlorophenol showed damage to the thyroid and reproductive system. Some of the harmful effects of pentachlorophenol are caused by the other chemicals present in technical grade pentachlorophenol.

Although there is sufficient evidence of carcinogenicity in animals, relevant human data is considered inadequate. Increases in liver, adrenal gland, and nasal tumors have been found in laboratory animals exposed to high doses of pentachlorophenol. The EPA has determined that pentachlorophenol is a probable human carcinogen and the IARC considers it possibly carcinogenic to humans.

Selenium. Selenium is a naturally occurring mineral element that is distributed widely in nature in most rocks and soils. Most processed selenium is used in the electronics industry, but it is also used: as a nutritional supplement; in the glass industry; as a component of pigments in plastics, paints, enamels, inks, and rubber; in the preparation of pharmaceuticals; as a nutritional feed additive for poultry and livestock; in pesticide formulations; in rubber production; as an ingredient in antidandruff shampoos; and as a constituent of fungicides. Radioactive selenium is used in diagnostic medicine.

Acute exposures cause nausea, vomiting, and diarrhea. Chronic oral exposure to high concentrations of selenium compounds can produce a disease called selenosis. The major signs of selenosis are hair loss, nail brittleness, and neurological abnormalities (such as numbness and other odd sensations in the extremities). Animal studies have shown that very high amounts of selenium can affect reproductive system. The IARC has determined that selenium and selenium compounds are not classifiable as to their

carcinogenicity to humans. The EPA has determined that one specific form of selenium, selenium sulfide, is a probable human carcinogen.

Silver. Silver is a naturally occurring element. It is found in the environment combined with other elements such as sulfide, chloride, and nitrate. Silver is used to make jewelry, silverware, electronic equipment, and dental fillings. Chronic exposure to high levels of silver may result in a condition called arygria, a blue-gray discoloration of the skin and other body tissues. Chronic lower-level exposures may also cause silver to be deposited in the skin and other parts of the body; however, this is not known to be harmful. Inhalation exposures to high levels have resulted in breathing problems, lung and throat irritation, and stomach pains. Skin contact with silver can cause mild allergic reactions such as rash, swelling, and inflammation in some people. Information on the effect of silver on reproduction or development is unavailable.

No studies are available on the human carcinogenicity of silver. The only available animal study showed both positive and negative results when silver was implanted under the skin. The EPA has determined that silver is not classifiable as to human carcinogenicity.

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 DHHS, IARC, and the EPA 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.

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.

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

Breathing high levels of vinyl chloride can cause dizziness. Breathing very high levels can cause you to pass out, and breathing extremely high levels can cause death.

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

It has not been proven that vinyl chloride causes birth defects in humans, but studies in animals suggest that vinyl chloride might affect growth and development. Animal studies also suggest that infants and young children might be more susceptible than adults to vinyl chloride-induced cancer. Animal studies have shown that long-term exposure to vinyl chloride can damage the sperm and testes.

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

Xylenes. Xylene is a colorless, sweet-smelling easily flammable liquid. It occurs naturally in petroleum and coal tar and is formed during forest fires. Xylene is used as a solvent and in the printing, rubber, and leather industries. It is also used as a cleaning agent, a thinner for paint, and in paints and varnishes. It is found in small amounts in airplane fuel and gasoline.

Xylene affects the brain. High levels from exposure for short periods (14 days or less) or long periods (more than 1 year) can cause headaches, lack of muscle coordination, dizziness, confusion, and changes in one's sense of balance. Exposure of people to high levels of xylene for short periods can also cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; problems with the lungs; delayed reaction time; memory difficulties; stomach discomfort; and possibly changes in the liver and kidneys. It can cause unconsciousness and even death at very high levels.

Studies of unborn animals indicate that high concentrations of xylene may cause increased numbers of deaths, and delayed growth and development. In many instances, these same concentrations also cause damage to the mothers. It is unknown if xylene harms the unborn child if the mother is exposed to low levels of xylene during pregnancy.

The IARC has determined that xylene is not classifiable as to its carcinogenicity in humans. Human and animal studies have not shown xylene to be carcinogenic, but these studies are not conclusive and do not provide enough information to conclude that xylene does not cause cancer.

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

Assessment of Joint Toxic Action of Chemical Mixtures

Non-Cancer

In the Sherwin-Williams/Hilliard Creek site, residents were exposed to contaminants detected in the former facility, floodplain soil and wetland sediments associated with the Hilliards Creek. Although the evaluation of health effects associated with individual chemicals for specific pathways was conducted earlier, the exposure to chemical mixtures should be considered. Exposure to multiple chemicals with similar toxicological characteristics may increase their public health impact (ATSDR 2005). The severity of the impact depends on the particular chemicals being ingested, pharmacokinetics, and toxicity in children and adults.

To evaluate the risk for non-cancer adverse health effects of chemical mixtures, a hazard index (HI) for the chemicals was calculated (ATSDR 2004). The hazard index is defined as the sum of the hazard quotients (i.e., estimated exposure dose of a chemical divided by applicable health guideline CV). If the HI is less than 1.0, it is highly unlikely that significant additive or toxic interaction would occur, so no further evaluation is necessary. If the HI is greater than 1.0, then further evaluation is necessary. For the Sherwin-Williams/Hilliard Creek site, based on the mean concentration of contaminants detected (the more likely scenario), the HI calculated for children for the floodplain soil (4) was greater than 1 (see Table B1) which indicated that further evaluation is necessary. For adults, based on the mean concentration of contaminants detected (the more likely scenario), the HI (0.55) was less than 1(see Table B1); as such, it is unlikely that significant additive or toxic interaction would occur.

For chemical mixtures with an HI greater than 1.0, the estimated doses of the individual chemicals are compared with their NOAELs or comparable values. If the dose of one or more of the individual chemicals is within one order of magnitude of its respective NOAEL, then potential exists for additive or interactive effects. The ratio of exposure dose to NOAEL for the contaminants was calculated (see Table B1). Since the potential exists for additive or interactive effects of chemical mixtures from exposures to lead and arsenic in children, an in-depth mixtures evaluation is required using ATSDR's *Guidance Manual for the Assessment of Joint Action of Chemical Mixtures* (2004).

The flow chart in Figure B1 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential non-cancer impact of joint toxic action (ATSDR 2004). Since toxicological profiles dealing with the mixture of chemicals detected in floodplain sediment is unavailable, a component approach is employed (Step 3, Figure B1). The hazard quotients of antimony, arsenic, chromium and lead in the floodplain soil were at least 0.1; they were selected as component of concern. Physiologically-based pharmacokinetic/pharmacodynamic (PBPK/PD) model is unavailable for the mixture (Step 4, Figure B1). The critical effects of the components of concern are as follows (Step 5, Figure B1):

Antimony	Arsenic	Chromium (IV)	Lead
Lifespan	***Dermal lesions*** **Cardiovascular** **Hematological** Renal **Neurological** **Cancer**	Hematological Hepatic Renal Neurological Testicular	***Neurological*** **Hematological** **Cardiovascular** Renal Testicular

[a]The basis for the MRL or health assessment approach is bolded and italicized; other sensitive effects are bolded; and less sensitive effects in common across two or more metals, or known to be affected synergistically by another metal in the mixture, are listed without bold or italics

Hazard indexes were then calculated using target organ toxicity dose (TTD) method for components with different critical effects (Step 6b, Figure B1). The magnitude of the hazard index shows potential neurological, dermal, renal, cardiovascular, hematological, testicular health effects in children due to additivity (see Table B2). As such, further evaluation of interaction (Step 7b, Figure B1) is indicated.

Binary weight of evidence (BINWOE) scores relevant to the route, duration, and endpoint for the three chemical are available (ATSDR 2004); the BINWOE scores for antimony are unavailable. The predicted direction of joint toxic action for neurological effects is greater than additive for the effect of lead on arsenic, arsenic on lead, chromium(VI) on arsenic, and less than additive for the effect of arsenic on chromium(VI) (see Table B3). The remaining two BINWOE scores were indeterminate due to a lack of toxicological and mechanistic data. Thus, the potential health hazard may be somewhat greater than estimated by the endpoint-specific hazard index for neurological effects (i.e., 3.52 for children). The impact of interaction on potential health hazard is summarized as follows:

Health Effect	Impact of interaction
Neurological	Higher
Dermal	Indeterminate
Renal	Lower
Cardiovascular	Little Impact
Hematological	Lower
Testicular	Lower

Cancer

The flow chart in Figure B2 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential cancer impact of joint toxic action (ATSDR 2004). The cancer risk estimate associated with the former facility soil, wetland sediment and floodplain soil of the Hilliards Creek are presented in Table 14 through 16. Since the estimated risks are greater than 1×10^{-6} for PAHs and arsenic (Step 3, Figure B2, see Table 15), they were selected as component of concern. Since physiologically-based pharmacokinetic/pharmacodynamic (PBPK/PD) model is unavailable for the mixture (Step 4, Figure B2), the sum of the cancer risks based on

mean concentration of contaminants (5.03×10^{-5}) is less than 1.0×10^{-4} (Step 4, Figure B2), further evaluation of interaction (Step 6a, Figure B1) using qualitative weight-of-evidence (WOE) scoring approach is required. Since the WOE scores for carcinogenic interaction of PAHs and arsenic are unavailable, the potential cancer health hazard due to interaction and/or additivity could not be evaluated.

Table B1: Multiple Chemical Exposure Analysis for Child: Floodplain Soil of the Hilliards Creek

Contaminant	Child Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	Hazard Index	NOAEL (mg/kg/day)	Dose/NOAEL
Child						
Aluminum	0.07	2	0.035	4	62	0.03
Antimony	0.00007	0.0004	0.17		0.35[a]	0.001
Arsenic	0.00048	0.0003	1.6		0.0008	0.375
Cadmium	0.000015	0.0002	0.075		0.0021	0.09
Chromium	0.0006	0.003	0.2		2.5	0.001
Copper	0.0004	0.01	0.04		0.042	0.23
Lead[b]	18.64	10	1.8		NA[c]	-
Vanadium	0.0002	0.003	0.06		3	0.001
Adult						
Aluminum	0.01	2	0.005	0.55		
Antimony	0.00001	0.0004	0.025			
Arsenic	0.00007	0.0003	0.02			
Cadmium	0.000002	0.0002	0.01			
Chromium	0.00009	0.003	0.03			
Copper	0.00007	0.01	0.007			
Lead[b]	4.4	10	0.44			
Vanadium	0.00003	0.003	0.01			

[a]Based on LOAEL; [a]Based on blood lead level in µg/dL; [b]Not available

Table B2: Target Organ Toxicity Dose modification of HI Analysis: Components with different critical effects in Children

	Exposure Dose (mg/kg/day)	Neurological	Dermal	Renal	Cardiovascular	Hematological	Testicular
Arsenic	0.00048	1.60	0.60	0.01	1.60	0.80	NA[a]
Chromium (VI)	0.0006	0.06		0.06		0.20	0.12
Lead	18.64[b]	1.86		0.55	1.86	1.86	0.47
Hazard Index =		**3.52**	**0.60**	**0.61**	**3.46**	**2.86**	**0.59**

; [a]Not available; [b]Blood lead levels in μg/dL

Table B3: Matrix of BINWOE Determinations for Simultaneous Oral Exposure to Chemicals of Concern

		On Toxicity of		
		Lead	Arsenic	Chromium(VI)
Neurological Toxicity				
Effect of	Lead		**>IIIB (+0.23)**	? (0)
	Arsenic	**>IIB (+0.50)**		**<IIIC2ii (-0.06)**
	Chromium(VI)	? (0)	**>IIIC (=0.10)**	
Dermal Toxicity				
Effect of	Lead		**? (0)**	NA
	Arsenic	NA		NA
	Chromium(VI)	NA	**>IIIC(+0.10)**	
Renal Toxicity				
Effect of	Lead		**<IIIB (-0.23)**	? (0)
	Arsenic	**<IIIB (-0.23)**		**<IIIB2ii (-0.14)**
	Chromium(VI)	? (0)	**<IIIB2ii (-0.14)**	
Cardiovascular Toxicity				
Effect of	Lead		**? (0)**	NA
	Arsenic	**? (0)**		NA
	Chromium(VI)	? (0)	**>IIIC (+0.10)**	
Hematological Toxicity				
Effect of	Lead		**<IIIB (-0.23)**	? (0)
	Arsenic	**<IIIB (-0.23)**		**<IIIC2ii (-0.06)**
	Chromium(VI)	? (0)	**>IIIC (+0.10)**	
Testicular Toxicity				
Effect of	Lead		NA	? (0)
	Arsenic	? (0)		**<IIIC2ii (-0.06)**
	Chromium(VI)	? (0)	NA	

BINWOE scheme (with numerical weights in parentheses) condensed from ATSDR (2001a, 2001b):
DIRECTION: = additive (0); > greater than additive (+1): < less than additive (-1); ? indeterminate (0)
MECHANISTIC UNDERSTANDING:
I: direct and unambiguous mechanistic data to support direction of interaction (1.0);
II: mechanistic data on related compounds to infer mechanism(s) and likely direction (0.71);
III: mechanistic data do not clearly indicate direction of interaction (0.32).
TOXICOLOGIC SIGNIFICANCE:
A: direct demonstration of direction of interaction with toxicologically relevant endpoint (1.0);
B: toxicologic significance of interaction is inferred or has been demonstrated for related chemicals (0.71);
C: toxicologic significance of interaction is unclear (0.32).
MODIFYING FACTORS:
1: anticipated exposure duration and sequence (1.0);
2: different exposure duration or sequence (0.79);
a: *in vivo* data (1.0); b: *in vitro* data (0.79);
i: anticipated route of exposure (1.0); ii different route of exposure (0.79).

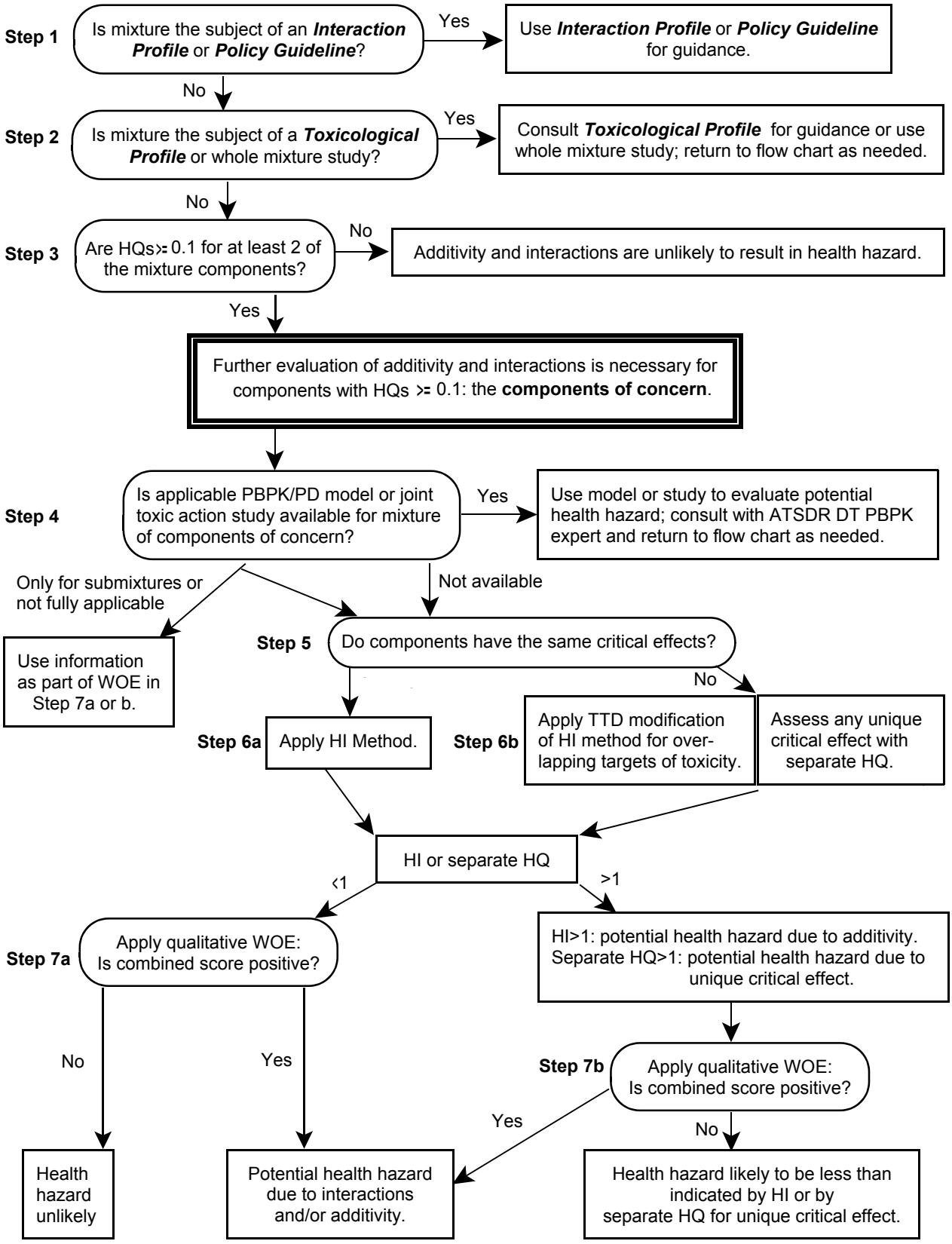


Figure B1: Exposure-Based Assessment of Joint Toxic Action of Chemical Mixtures: Non-Cancer Effects

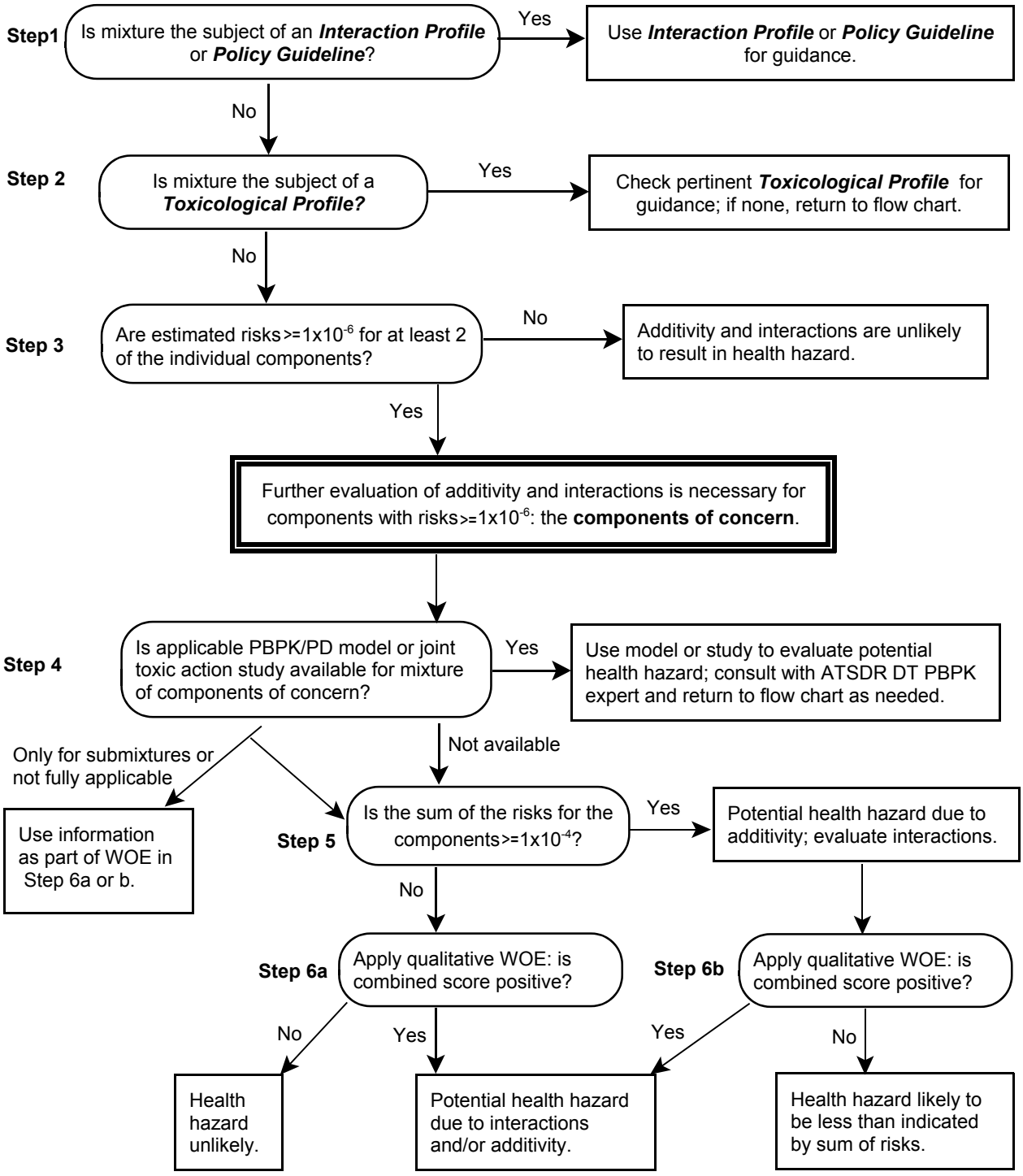


Figure B2: Exposure-Based of Joint Toxic Action of Chemical Mixtures: Cancer Effects

Appendix C

Summary of Public Comments and Responses

Summary of Public Comments and Responses
Sherwin-Williams/Hilliards Creek Site Public Health Assessment

The NJDHSS held a public comment period from October 21, 2008 through November 22, 2008 to provide an opportunity for interested parties to comment on the draft Public Health Assessment prepared for the Sherwin-Williams/Hilliards Creek Site. Written comments were received by the NJDHSS during the public comment period.

The NJDHSS and ATSDR followed 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, (2) the material was organized for content (comments addressing similar issues may have been combined), and (3) a response was prepared for each comment.

Questions regarding this summary or any aspect of this Public Health Assessment may be addressed to the NJDHSS at (609) 584-5367.

Comment #1: The commenter suggested that (1) the geographic reach of the recommendations be extended downstream along Hilliards Creek at least into Kirkwood Lake and perhaps even farther downstream depending on the levels of contaminants found, and, (2) the recommendations be expanded to include the residents of the Borough of Lindenwold.

Response**:** According to USEPA Region 2, the Kirkwood Lake was sampled in Fall 2007 and Spring 2008, due to contamination previously detected within downstream portions of Hilliards Creek and the surrounding wetlands. The contaminants were mostly lead and arsenic in the soil and sediments. As a result, the Kirkwood Lake is now considered to be a part of the Sherwin-Williams/Hilliards Creek Site.

The exposure pathways associated with the contamination detected in the Kirkwood Lake will be evaluated in a separate health consultation.

Comment #2: The commenter requested that the PHA include a blood lead evaluation of children in Lindenwold. As part of its public health assessment, DHSS examined the records of its Childhood Lead Poisoning Prevention Surveillance System for July 1999 through February 2007 in Gibbsboro and found no children with blood lead levels above the Center for Disease Control guideline of 10 micrograms per deciliter. We recommend that DHSS determine whether this is the case with the children of Lindenwold.

Response**:** The following is a blood lead evaluation of children in Lindenwold.

The concentration of lead in blood is an excellent indicator of exposure to lead. Current state regulations, in accordance with federal Centers for Disease Control and Prevention (CDC) guidelines, require health care providers to perform a blood lead test on all one and two year old children. This is the age at which lead poisoning is most damaging to the developing nervous system. New Jersey State regulation requires all

clinical laboratories to report the results of all blood lead tests to the NJDHSS. Prior to July 1999, only blood lead tests above 20 micrograms of lead per deciliter of blood (μg/dL) were required to be reported. While the current CDC blood lead guideline is 10 μg/dL, all blood-lead test data are reportable to the NJDHSS' Childhood Lead Poisoning Prevention Surveillance System.

For the purpose of this evaluation, all blood lead data were requested from the Department's Childhood Lead Poisoning Prevention Surveillance System for Lindenwold over the period January 1999 to December 2008.

A total of 2,311 blood lead tests from Lindenwold children were available during the survey period. The age range for the blood lead tests was under one month of age to 16.5 years of age, with an arithmetic average age of 2.8 years. The distribution of tests by sex for the Lindenwold blood lead was 47.3% female, 51.7% male, and 1.0% unknown.

The geometric mean of blood lead was 2.29 μg/dL with a 95% confidence interval of 2.24 μg/dL to 2.35 μg/dL. A total of 11 (0.5%) of these results were over the CDC blood lead guideline, with a maximum concentration of 20 μg/dL. A review of the addresses of the 11 children with elevated blood lead results indicated that they do not live near Hilliards Creek. As such, it is unlikely that the site contamination may be the source of the children's elevated blood lead levels. The principal source of lead exposure for children in the United States is household dust and soil contaminated by leaded paint.

In conclusion, the average blood lead level in children living in Lindenwold, and the proportion of blood lead levels above CDC guideline were similar to statewide levels. This evaluation does not provide any evidence that Lindenwold children have been adversely affected by contamination from the Sherwin Williams-Hilliards Creek site in Gibbsboro.

Comment #3: The commenter stated that the PHA only references approximately 350 samples collected in 2005. The PHA fails to use all of the data collected to date as there is no discussion of the additional sampling activities and evaluation of those analytical results contained within this document.

Response**:** The PHA reviewed all available and relevant environmental contamination data to evaluate the completed and potential human exposure pathways. The references (presented at the end of the PHA) are a partial list of site-related documents reviewed. Many more samples than those collected in 2005 are discussed and referenced in the document (pages 10-12).

Comment #4: The commenter stated that the PHA is incorrect in stating that residents could currently be exposed to soil from the 0-2 foot depth interval at the Paint Works facility. The facility is being used as commercial/industrial office park, and as such, residents do not recreate at the facility. In addition, exposure to contaminated soil is not possible because the facility is covered by pavement, or landscaped (grassy) areas.

Response***:*** The PHA specifically referred to the unpaved on-site areas located to the south of Foster Avenue (i.e., between US Avenue and Clementon Road).

Comment #5: The commenter stated that ingestion of surface water from Hilliard Creek is not a realistic exposure pathway. The creek is too narrow and shallow for swimming or wading. The PHA fails to note that the Wildlife Refuge next to Hilliard Creek is completely fenced off, and has an alarm, preventing access to this area by the public. As such, exposure to soil in this area is not possible by the public and the fence should not be described as "partial".

Response***:*** Although the fence and the alarm system were installed recently to prevent resident access to the contaminated area, the contaminated areas including the Bridgewood Lake (see Figure 2 of PHA) were used for recreational purposes (i.e., swimming, wading, and fishing) in the past.

The contamination delineation has not been completed to date. As such, the fence for controlling resident access was described as "partial".

Comment #6: The commenter stated that the current owner and the USEPA conducted indoor air sampling of the office buildings and adjacent residential properties in 2007 and 2008. The PHA did not discuss or evaluate these sampling results.

Response***:*** At the time the PHA was prepared, the indoor data were unavailable. However, the ATSDR and the NJDHSS is aware of indoor air sampling by the owner and USEPA.

The indoor air data will be evaluated in a separate health consultation.

Comment #7: The commenter stated that Sherwin-Williams has collected two rounds of potable water samples during 2006-2008 time-frame. The PHA did not discuss or evaluated these sampling results.

Response***:*** At the time the PHA was prepared, the potable water sampling results were unavailable. However, the ATSDR and the NJDHSS are aware of water sampling data. The potable well data will be evaluated in a separate health consultation.

Comment #8: Page 15 – Potential Exposure Pathways – Ingestion of biota from Hilliards Creek (past, present, future)

Based on discussions with the NJDEP and USEPA, Sherwin-Williams conducted a fish sampling program in Kirkwood Lake during November 2002 and presented the results in a report entitled "Fish Collection and Tissue Analysis Report – Kirkwood Lake – Camden County, NJ" dated February 10, 2003.

Although heavy metals were detected in fish tissue, the report concluded that the fish do not present a health risk to human consumers, with the exception of mercury, which is a

known statewide problem unrelated to this site. Kirkwood Lake has signs posted warning against fish consumption due to mercury contamination. Due to the narrow width and shallow depth of Hilliard Creek, it is unlikely that the creek supports a fish population for sport fishing, and unlikely that people fish there.

There is no discussion of these sampling activities and evaluation of the analytical results contained within this document.

Response: The ATSDR and NJDHSS is aware of the report titled"Fish Collection and Tissue Analysis Report – Kirkwood Lake – Camden County, NJ" dated February 10, 2003. As indicated earlier, the Kirkwood Lake was not a part of the site and as such the results were not included/summarized in the draft PHA.

It should be noted that once biota sampling results from the Hilliards Creek area is available, all biota data will be evaluated in a separate health consultation.

Comment #9: The commenter indicated that an incorrect soil lead EPCs was used in the IEUBK model. According to USEPA, the correct soil lead input to the IEUBK model is the arithmetic mean soil lead concentration, not the UCL on the mean (USEPA, 1994, page 1-18). The soil lead EPCs used in the Adult Lead Model should also be the arithmetic mean concentration.

Response: The USEPA (2009) also states the following:

"The IEUBK model can use an upper confidence limit (UCL); however, the interpretation of the model results is somewhat different if a UCL is used. If an arithmetic mean (or average) is used, the model provides a central point estimate for risk of an elevated blood lead level. If a UCL is used, the model result would be interpreted as a more conservative estimate of the risk of an elevated blood lead level".

For Sherwin-Williams/Hilliards Creek site, the ATSDR and the NJDHSS used a conservative exposure point concentration of lead to assess childhood and adult lead exposure risks.

Reference

[USEPA 2009] United States Environmental Protectoin Agency. 2009. Accessed at http://www.epa.gov/oerrpage/superfund/programs/lead/ieubkfaq.htm on January 27, 2009.

Comment #10: The commenter stated that the exposure frequency assumed for the IEUBK model was three days per week for six months per year (page 19). The blood lead modeling with the IEUBK model accounted for the three days per week by using a weighted soil EPC; however, the modeling failed to incorporate an exposure frequency of six months per year. The PHA should have followed USEPA's Guidance for Assessing Intermittent or Variable Exposures at Lead Sites (USEPA, 2003). This guidance states that when calculating seasonal exposures in the IEUBK model, the exposure can be

calculated for each year of exposure as if there were no exposure during the previous year, and then the results can be averaged across years (USEPA, 2003a; Appendix A, Example 5). The PHA should have used this method of averaging, because the site exposures occur only six months per year. The lead risks are overestimated because the modeling did not account for the seasonal exposure.

Response: The modeled blood lead levels presented in the draft PHA were generated without allowing time for the blood lead levels to return to baseline during the six months following exposure.

As suggested by the commenter, using seasonal exposures and assuming no exposure during the previous year (USEPA, 2003a; Appendix A, Example 5), the predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are as follows:

Exposure Scenario		
Age (months)	**Three Site Visits Per Week[a]**	
	Blood Lead Level[b] (µg/dL)	**P_{10} (%)[c]**
6 -12[d]	6.8 (6.8)[d]	20.8 (20.8)
12 - 24	7.2 (7.7)	26.5 (29.4)
24 - 36	6.5 (7.2)	18.1 (24.8)
36 - 48	6 (6.9)	14.5 (21.8)
48 - 60	4.8 (5.7)	5.8 (12.2)
60 - 72	4 (4.9)	2.8 (6.5)
72 - 84	3.6 (4.4)	1.5 (4)
Mean		**12.85 (17.07)**

[a]weighted soil lead concentration (995 ppm x 3/7 + 200 ppm x 4/7) = 540 ppm (USEPA 2003a); [b]Geometric mean lead levels in blood; [c]probability of blood lead level > 10 µg/dL; [d]the corresponding number without allowing the blood lead levels to return to baseline as presented in the draft PHA

It is evident that by allowing time for the blood lead levels to return to baseline, the mean P_{10} value decreases from 17.07% to 12.85%. Since both numbers are greater than 5%, the conclusions remain unchanged.

Comment #11: The commenter stated that the lead risks presented in the PHA have been overestimated, and that they are contradicted by the actual blood lead data from Gibbsboro for the period July 1999 through February 2007. Since the PHA itself reviews data showing there is no evidence that children in Gibbsboro have elevated blood lead levels, it is unlikely they have (or had) a potential for adverse health effects from exposure to lead in soil.

Response: The modeled blood lead level presented in the public health assessment is associated with the 'historic exposure situation' that existed in the past and would occur in future if the site is allowed to be used without any control/restriction. The modeling objectives were clarified in the final version.

The report did not attempt to assess the lead exposures associated with currently accessible contaminated areas.

It should be noted that it is incorrect to conclude that because current children blood lead levels are relatively low that there was/is no health concern.

Comment #12: The commenter stated that the PHA correctly evaluates lead risks by modeling blood lead levels. However, a hazard quotient for lead should not be calculated in Appendix B (Assessment of Joint Toxic Action of Chemical Mixtures). Lead does not have a published MRL or RfD, therefore, a hazard quotient for lead can not be calculated. It is not correct to calculate an improvised "hazard quotient" for lead based on the ratio of the modeled blood lead level to the target blood lead level of 10 µg/dL. Lead should be removed from Tables B1 and B2, as the invalid "hazard quotient" for lead contributes 80% of the total hazard index.

Response: In the absence of an MRL or EPA RfD, the ATSDR and NJDHSS used the CDC blood lead level guideline of 10 µg/dL to calculate the hazard quotient.

Comment #13: The commenter stated that in Tables 11 and 12, the entry for lead should be footnoted, because the CV is listed as "NA", thus the conclusion that there is a potential for non-cancer health effects from lead must be based on the blood lead modeling, and not on the estimated exposure dose calculated in the table.

Response: The suggested change was incorporated in the final version.

Comment #13: The commenter stated that the Figure 3 incorrectly depicts the locations of Tank Farm A and B. The locations should be reversed.

Response: The label was corrected in the final version.

Comment #14: The commenter stated that the Photograph 3 is actually a picture of the former Tank Farm A area and not Tank Farm B as incorrectly noted.

Response: The label was corrected in the final version.

Comment #15: The commenter stated that the PHA does not do a good job of communicating risk to the public. The purported purpose of the PHA is to support the listing of the site on the NPL. Therefore, it is not clear why the report assesses human health risks without reviewing all available information, and based on "past" conditions, particularly when such conditions no longer exist because interim remedial actions have been conducted. The report should focus on evaluation of public health risk from site conditions as they exist in 2008. However, since current data has not been reviewed, the report concludes that currently the site poses an "Indeterminate Public Health Hazard". This language is imprecise and could be misinterpreted by the public.

Response: The purpose of preparing a PHA for a site is not to support the listing of the site to the NPL but to assess potential impact of site-related contamination on public health. According to the Public Health Assessment Guidance Manual, purpose of a public health assessment is defined as:

"The evaluation of data and information on the release of hazardous substances into the environment in order to assess any [past], current, or future impact on public health, develop health advisories or other recommendations, and identify studies or actions needed to evaluate and mitigate or prevent human health effects (42 Code of Federal Regulations, Part 90, published in 55 Federal Register 5136, February 13, 1990)".

In order to address the past exposures, part of the draft PHA is based on "past" conditions, although such conditions may not exist because some interim remedial actions have been conducted.

At the time the draft PHA was prepared, sampling was still on-going, and some pathways could not be evaluated due to lack of contamination data. As additional contamination data become available, the ATSDR and NJDHSS will prepare health consultation(s) to evaluate the public health implications of potential contamination.

References

ATSDR. 2007. Toxicological Profile for Lead. http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf.

USEPA, Technical Review Workgroup for Lead. 1994. "Guidance manual for the Integrated Exposure Uptake Biokinetic Model for lead in children." Report to US EPA, Office of Emergency and Remedial Response (Research Triangle Park, NC) OERR Publication 9285.7-15-1 ; EPA540-R-93-081 ; NTIS PB93-963510. February. Link to Chapter 1: http://www.epa.gov/superfund/health/contaminants/lead/ products/ch01.pdf

USEPA. 2003. Assessing Intermittent or Variable Exposures at Lead Sites. EPA-540-R-03-008. OSWER 9285.7-76. http://www.epa.gov/superfund/lead/products/twa-final-nov2003.pdf