



Public Health Assessment for

LONG BRANCH MANUFACTURED GAS PLANT SITE
LONG BRANCH, MONMOUTH COUNTY, NEW JERSEY

EPA FACILITY ID: NJD980530471

MAY 13, 2008

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE**

Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR 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 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 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 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
Public Health Services Branch
Consumer and Environmental Health Services
Hazardous Site Health Evaluation Program

Under a cooperative agreement with the
Agency for Toxic Substances and Disease Registry

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Summary

The former Long Branch Manufactured Gas Plant site is located in Long Branch, Monmouth County, New Jersey. In 2002, the Concerned Citizens Coalition of Long Branch expressed concern regarding health effects from past exposures associated with the site. Through a cooperative agreement with the Agency for Toxic Substances and Disease Registry, the New Jersey Department of Health and Senior Services prepared this public health assessment for the former Long Branch Manufactured Gas Plant site.

The former Long Branch Manufactured Gas Plant operated from the 1870s through the 1960s and used coal to produce manufactured gas. Discharge of wastes generated during the manufacturing process resulted in the contamination of on- and off-site areas. Site related contaminants were detected in on-site surface soil, sediment, groundwater, and deeper parts of the underlying aquifer. Characterization and contamination delineation of on-site areas of concern has been conducted and remedial actions were implemented with oversight from the New Jersey Department of Environmental Protection. Currently, there are no completed exposure pathways associated with the on-site contamination; as such, the on-site areas pose ***No Public Health Hazard***.

In the past, there were completed exposure pathways to area residents via the incidental ingestion of contaminated on-site surface soil and sediment and inhalation of indoor air. Potential pathways included past inhalation of ambient air, past and current incidental ingestion of surface water during recreational activities, and ingestion of biota from the Troutman's Creek. Contaminants of concern identified for the site were benzene, ethylbenzene, n-propylbenzene, toluene, xylene, vinyl chloride, bis(2-ethylhexyl)phthalate, polycyclic aromatic hydrocarbons, polychlorinated biphenyls, arsenic, cadmium, lead and mercury. Only one sample result for Aroclor 1242 was available, and non-cancer adverse health effects were found to be possible for children only. Based on the maximum concentrations of arsenic and cadmium detected in surface soil and sediment, potential for non-cancer adverse health effects was found for children only and determined to be low. However, there was a potential for adverse health effects associated with lead exposures to the on-site exposures from the Processing Area and the Southern Site Boundary. Potential health hazards 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 mean and maximum contaminant concentrations. Based on average contaminant concentrations (the more likely exposure scenario), the cumulative lifetime excess risk may have been as high as 7 in 10,000 to the exposed population. As such, based on lifetime excess cancer risks and childhood lead exposure in the past, the site posed a ***Public Health Hazard***.

The characterization and delineation of off-site contamination is currently being conducted. Exposures associated with off-site contaminants will be addressed in a separate health consultation. As such, past, current and future exposures associated with off-site contamination are considered an ***Indeterminate Public Health Hazard***.

Standardized incidence ratios were used for the quantitative analysis of cancer incidence (January 1, 1979, through December 31, 2000) in the area. Although overall cancer incidence was not elevated, brain/central nervous system cancer was elevated in females. Lung cancer in white females was higher than expected while stomach cancer was generally lower than expected. In Census Tract 8056, the area of Long Branch which had the highest potential for exposure, excess levels of esophageal cancer in females, lung cancer in males, and all cancers combined in males was found. Leukemia incidence citywide and in Census Tract 8056 was not higher than expected. While lung cancer incidence was higher in males in Census Tract 8056, lung cancer incidence was not higher than expected for females. The inconsistency between the standardized incidence ratios for males and females for lung cancer and the lack of significant increases in leukemia argue against environmental exposures from the site. Other plausible explanations for any elevated standardized incidence ratios include unmeasured risk factors in the community (e.g., tobacco consumption or occupational exposures) or chance alone.

Other health concerns expressed by the community included possible relationships between site contaminants and asthma, autism, birth defects and respiratory conditions (asthma and bronchitis) in adults, psychological stress associated with relocation, susceptibility to chemical exposures and other diseases such as infections, rashes and lupus. Other than asthma, which may be triggered by outdoor contaminants including particulate matter and psychological stress that may have been a result of relocation, none of the health conditions of concerns were likely to be associated with the site-related contamination.

Recommendations include the completion of the remedial investigation of off-site areas (including residential properties and Troutman's Creek) and the implementation of preventive measures during remedial actions. The New Jersey Department of Health and Senior Services and Agency for Toxic Substances and Disease Registry will prepare health consultation(s) to evaluate the public health implications of contaminants detected in the off-site areas and provide assistance to residents in reducing exposures to contaminants.

Statement of Issues

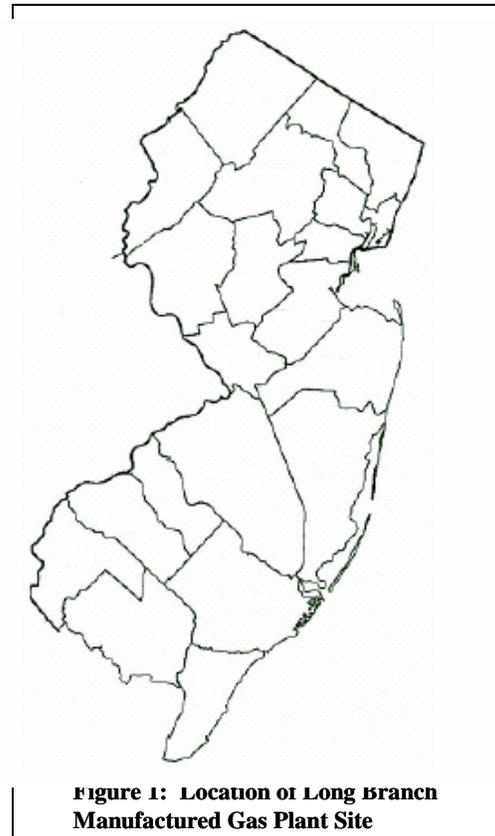
The former Long Branch Manufactured Gas Plant (LBMGP) site is located in Long Branch, Monmouth County, New Jersey, approximately one quarter mile west of the Atlantic Ocean (see Figure 1). The 11.2 acre facility operated from the 1870s through the 1960s and used coal to produce manufactured gas for lighting homes, businesses, and street lamps. Some of the hazardous wastes generated during the manufacturing process were disposed on-site. Results of remedial investigations of the site indicated that both on- and off-site soil and groundwater were contaminated with volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), metals, phenols, and cyanide.

In 1997, site-related contamination was detected during the repair of an underground water main located north of the site. In 2002, the Concerned Citizens Coalition of Long Branch petitioned the federal Agency for Toxic Substances and Disease Registry (ATSDR) regarding health concerns (including cancer) and potential exposures during site remediation activities. The petition was accepted by the ATSDR and the New Jersey Department of Health and Senior Services (NJDHSS), through a cooperative agreement with the ATSDR, prepared a health consultation that evaluated cancer incidence in the community surrounding the site (ATSDR 2003). A second health consultation was also prepared which evaluated exposures to indoor air contamination detected at the Seaview Manor public housing complex (ATSDR 2004).

Through a cooperative agreement with the ATSDR, the 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 on-going. Separate health consultation(s) will be prepared to address exposures associated with off-site contamination.

Background

The LBMGP site is located on Brook Street in Long Branch, Monmouth County, New Jersey. The site is bounded by Long Branch Avenue to the east, Liberty Street to the west (see Figure 2), the former Seaview Manor and Grant Court public housing



complexes to the north and west (see Figure 3), and commercial businesses and Chester A. Arthur housing complex to the south (see Figure 4). The northeast corner of the site is the location of the old Jerry Morgan Park (see Figure 3). Approximately one block north of the site and adjacent to Troutman's Creek is the Long Branch Sewage Treatment plant. In the 1990s, railroad tracks which ran along the southern boundary of the site were removed.

The tidally influenced Troutman's Creek flows through the site in a northeasterly direction and discharges into the Branchport Creek and Shrewsbury River located north of the site (see Photographs 1 and 2). There are tidal fluctuations of two feet on-site and three feet north of the Seaview Avenue bridge. During low tide, the on-site portion of Troutman's Creek has standing water.

Land use in the vicinity of the site is residential and commercial. At the present time there are businesses on the site including the Talco Cash Register Company and the Atlantic Plumbing and Supply Company, as well as a Jersey Central Power and Light Company (JCP&L) electrical substation (see Figure 3). On-site businesses receive municipal water and sewerage services; the closest private potable well is located more than half a mile north of the site (M. Kenney, NJDEP, personal communication, 2005).

As early as 1868, the site was owned by the Long Branch Gas Light Company. In 1895, the company merged with the Consolidated Gas Company and in 1925, with JCP&L (NJDEP 1993). JCP&L operated the site until 1952, when New Jersey Natural Gas (NJNG) purchased the property. NJNG operated the site from 1956 to 1961. From 1962 to 1972, NJNG also operated a liquid propane gas peaking facility (i.e., storage for peak demand) at the site.

The LBMGP used coal to produce manufactured gas for lighting homes, businesses, and street lamps. Generally, there were three processes used to manufacture the fuel from coal: coal carbonization; carburetted water gas; and oil gas (Heritage Research 2004). In coal carbonization, coal gas was produced through the distillation of bituminous coal in heated, anaerobic vessels called retorts. The carburetted water gas process consisted of enriching a form of coal gas, known as water gas, to increase its energy value. The oil gas process did not use coal but, instead, thermo-cracked oil in a steam environment to produce raw gas.

Wastes generated during the manufactured gas process were primarily coal tars containing a variety of hazardous substances including VOCs, PAHs and heavy metals. Sample analysis of this coal tar waste indicated over 4% PAHs and 0.2% VOCs (NUS 1990). Based on available information on LBMGP waste disposal practices, some wastes were sold to outside parties for additional processing, and some were disposed of in on-site marshy areas (later, the location of the old Jerry Morgan Park). There was a transformer storage area located to the south of the old Jerry Morgan Park.

With the increasing availability and use of natural gas, the production of manufactured gas declined and in the 1950s, a portion of the LBMGP site (south of the

Seaview Avenue) was sold to the city of Long Branch. The property was redeveloped as the Seaview Manor public housing complex. Review of Sanborn maps and historic aerial photographs indicated that no manufacturing process structures had been present on this property (ARCADIS 2000). However, there were houses located on this property which were demolished prior to the construction of the public housing complexes.

From 1966 to 1976, the northeast portion of the LBMGP site was leased to the city of Long Branch for use as the (old) Jerry Morgan Park (NJDEP 1993). This property was donated to the city in 1976. During soil excavation activities at the park in August 1983, a tar-like residue was reportedly observed discharging to the surface soil and the excavation was stopped. Soil and groundwater samples collected by the City of Long Branch from the park indicated the presence of coal tar constituents. As a result, the City of Long Branch closed the park to the public in September 1983.

Site Description

The LBMGP site is located within the Atlantic Coastal Plain Physiographic Province. The site elevation is approximately 8 - 10 feet above mean sea level and slopes gently downward to the north and east. The site is underlain by fill material and marsh deposits ranging in thickness from 2 - 4 feet. The marsh deposits consist of silt with fine-to-medium sand, peat, and clay. The fill consists of materials related to both plant operations and the more recent grading of the site with cinders and rubble. The fill material and marsh deposits overlie the Tertiary Vincentown Formation¹, which is primarily sand with some silt or clay. The thickness of the Vincentown Formation ranges from 64 - 79 feet and is underlain by the Tertiary Hornerstown Formation, which consists of clayey glauconitic fine sand (Woodward Clyde Consultants 1986a).

The water table in the vicinity of the site is within five feet of the ground surface. Groundwater closest to the ground surface discharged² into the on-site portion of the Troutman's Creek. In the fill material and marsh deposits underlying the site, groundwater elevations are slightly higher than that in the Vincentown Formation; this suggests that the fill/marsh deposit layer recharges the Vincentown Formation. At the site, Troutman's Creek influences the groundwater flow. The predominant direction of groundwater movement in the Vincentown Formation is to the north toward the confluence of Troutman's Creek and the Shrewsbury River (Woodward-Clyde Consultants 1986a).

¹A formation is a body of rock with distinguishing characteristics apart from surrounding rock layers.

²On-site portion of the Troutman's Creek was re-channelized during remediation. It also included a liner designed to prevent groundwater discharges from entering the Creek.

Demographics

Based on 2000 United States Census data, ATSDR estimates that there are approximately 18,000 individuals living within one mile of the LBMGP site (see Figure 5). Children ages six years and younger represent about 10% of the total population in this area.

Summary of Previous Investigations

On August 22, 1983, the New Jersey Department of Environmental Protection (NJDEP) notified New Jersey public utilities that former MGP sites located throughout the state would be evaluated. In response, JCP&L/NJNG informed the NJDEP of their intention to voluntarily conduct an environmental investigation at the former LBMGP site. From 1984 to 1986, JCP&L/NJNG conducted a Remedial Investigation (RI) of the LBMGP site (Woodward-Clyde Consultants 1985, 1986a). Soil and groundwater samples collected from the site (inclusive of the old Jerry Morgan Park) indicated the presence of PAHs, VOCs, metals, phenols, and cyanide. Sediment and surface water samples were also collected from both on- and off-site sections of Troutman's Creek; results indicated the presence of PAHs and metals.

A Feasibility Study (FS) was also conducted which evaluated various remedial options to address soil, ground and surface water, and sediment contamination at the site (Woodward-Clyde Consultants 1987; NJDEP 1993). The proposed remedy consisted of the excavation and off-site disposal of coal tar waste piles, the removal of underground storage tanks, the removal and disposal of coal tar from a gas holder structure, the capping of the site to eliminate the potential for direct contact with contaminated soils, the channelization of the on-site section of Troutman's Creek to alleviate contaminated groundwater discharge to the creek and the potential for direct contact with contaminated sediment, and the installation of a groundwater recovery and treatment system.

Prior to the implementation of remedial activities, the following studies were also conducted by JCP&L/NJNG between 1987 and 1989:

- supplemental study of off-site soil and sediment contamination (Woodward-Clyde Consultants 1988a);
- feasibility of bioremediation of PAH contaminated soils (Woodward-Clyde Consultants 1988b);
- aquifer pump test (Woodward-Clyde Consultants 1989); and
- delineation of the 100 year floodplain for Troutman's Creek (Langan Engineering 1989).

In December 1989, an on-site reconnaissance survey indicated the potential for public exposure via direct contact with on-site contaminants (NUS 1990). Since the site was improperly secured, illegal disposal and trespassing was possible. In addition, an uncovered, unlined coal tar pile was observed; it was solidified due to the cold weather conditions.

On December 9, 1991, the NJDEP executed an Administrative Consent Order (ACO) directing JCP&L/NJNG to perform the following additional activities: 1) focused RI to assess the limits of site-related contamination; 2) FS to evaluate remedial alternatives; 3) remedial design of the proposed remedy; and 4) implementation of the preferred remedy.

Site Visit

On February 12, 2004 a site visit of the former LBMGP site was conducted. Individuals present during the site visit were Julie Petix and Tariq Ahmed, NJDHSS and a representative of the NJNG. The site visit commenced at 10:00 am. Weather conditions were sunny, cold, and windy with temperatures in the mid 30s.

The representative of the NJNG discussed the site history and the remedial activities conducted by the NJNG to date. He showed the location of the former processing area and described on-going site activities. The remedial action implemented for the Troutman's Creek included rechannelization of the on-site section of the creek to mitigate contaminated groundwater discharge into the creek (see Photograph 3). The locations of the Atlantic Plumbing and Supply (see Photograph 4) and Talco Cash Register Companies, the Seaview Manor and Grant Court public housing complexes (see Photographs 5 and 6), Check Mate and Second Baptist Church Day Care Centers (see Photographs 7 and 8), and the new Jerry Morgan Park were noted. It was observed that the off-site portion of the Troutman's Creek was easily accessible from Seaview Avenue and residential properties located adjacent to the creek.

Community Health Concerns

On a twice monthly basis, the Concerned Citizens Coalition of Long Branch, a local group formed to oversee the remediation of the LBMGP site, meets to discuss site updates. Staff of the NJDHSS and ATSDR have attended these meetings to update coalition members on NJDHSS activities.

Community members have voiced their concerns to the NJDHSS and the ATSDR through the Concerned Citizens Coalition, at public meetings, two Availability Sessions on March 26, 2003, and privately. Concerns expressed included possible relationship between site contaminants and asthma, autism, birth defects and learning disorders in children, respiratory conditions (asthma and bronchitis) in adults, cancer, psychological stress associated with relocation, susceptibility to chemical exposures and other diseases such as infections, rashes and lupus. The community health concerns and the exposures to the indoor air contaminant detected at the Seaview Manor Public Housing Complex were addressed in a health consultation (ATSDR 2004). Indoor air exposures among residents of Seaview Manor Public Housing Complex represented an indeterminate public health hazard. Besides asthma which may be triggered by outdoor contaminants including particulate matter and psychological stress that may have been a result of relocation, none of the other diseases or health conditions of concern, such as bronchitis

and lupus, were likely to be associated with the indoor air levels measured in Seaview Manor.

In preparing the public health assessment for the site, the NJDHSS and ATSDR held two Availability Sessions on May 27, 2004 to provide the Long Branch community with the opportunity to discuss individual health concerns with respect to the LBMGP site. Some residents stated that they and their family members lived in the immediate vicinity of the site all their lives and that the majority of individuals who lived at the Seaview Manor public housing complex were also long time residents. In response to NJDHSS inquiries regarding access to and past use of the site, a number of residents stated that children and adults routinely accessed the site. The old Jerry Morgan Park was the primary recreation area for former Seaview Manor and other area residents. Residents routinely fished Troutman's Creek and ate their catch. Family picnics were held on the on-site portion of Troutman's Creek. NJDHSS also spoke with residents living adjacent to Troutman's Creek across from the former Seaview Manor public housing complex. Concerns expressed included the flooding of their yards and basements during periods of heavy rain. During these periods, debris from the creek washed up in their yards. Some residents have young children; one resident was concerned that his child's health problems (headaches/migraines) may be associated with site-related contamination. The evening Availability Session coincided with a public meeting sponsored by the Environmental Justice Program of the NJDEP. The purpose of the public meeting was to discuss the Environmental Justice petition submitted to the NJDEP by the Concerned Citizens Coalition of Long Branch and to update the community as to the current status of site remedial activities.

On August 19, 2004 NJDHSS staff attended a NJNG sponsored open house held at the administrative building of the Long Branch Housing Authority. The open house was well attended by community members. It was organized into stations where poster-sized photographs, figures and statements were used to provide an overview of past, present and future remedial activities at the site. Concerns expressed by community members included the possible release of contaminants along the truck traffic routes during remediation.

Past ATSDR and NJDHSS Activities

In 2003, a health consultation was prepared for the site to evaluate cancer incidence (ATSDR 2003). The results of analysis provided little evidence that the cancer rate has been affected by the potential exposures to contamination.

A second health consultation was prepared to address the indoor air exposures and health concerns of Seaview Manor public housing complex residents and two off-site child care centers (ATSDR 2004). Since the weather conditions at the time of sampling were not representative of the "worst case scenario", indoor air exposures among residents was considered an indeterminate public health hazard. Concentrations of contaminants detected in the indoor air at the day care centers represented no apparent public health hazard.

Environmental Contamination

An evaluation of site-related environmental contamination consists of a two tiered approach: 1) a screening analysis; and 2) a more in-depth analysis to determine public health implications of site-specific exposures (ATSDR 2005). First, maximum concentrations of detected substances are compared to media-specific environmental guideline comparison values (CVs). If 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 CVs available for 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 Quotient³ 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 NJDEP Residential and Non-Residential Direct Contact Soil Cleanup Criteria (RDSCC, NRDSCC). Based primarily on human health impacts, these criteria also take into account natural background concentrations, analytical detection limits, and ecological effects.

Substances exceeding applicable environmental guideline CVs are identified as COCs and evaluated further to determine whether these contaminants pose a health threat to exposed or potentially exposed receptor populations. In instances where an environmental guideline CV was unavailable, the substance was retained for further evaluation. There are exceptions, however. For example, some naturally occurring substances such as sodium, calcium, potassium, and magnesium are not harmful under most environmental exposure scenarios and may not be retained for further analysis.

³The ratio of estimated site-specific exposure to a single chemical from a site over a specified period to the estimated daily exposure level at which no adverse health effects are likely to occur.

On-Site Contaminants

Surface Soil and Sediment

Hydrogeologic and focused remedial investigations (RIs) of the LBMGP site (Woodward-Clyde 1985; ARCADIS 2000, 2002) identified five on-site areas of concern (AOCs): 1) the manufactured gas processing area (hereinafter referred to as the processing area); 2) the old Jerry Morgan Park; 3) Troutman's Creek (on-site portion); 4) former Seaview Manor public housing complex (between old Jerry Morgan Park and Seaview Avenue); and 5) the southern site boundary. As part of the investigation, twenty test pits (3 - 10 feet in depth) were excavated at the LBMGP site. One test pit was located along the site's southern boundary (upgradient), three in the old Jerry Morgan Park, and the remaining in the processing area. Most of the test pits were described as having an "oily" residue and odor.

Processing Area: Thirteen soil boring samples were collected from the processing area and analyzed for VOCs, semivolatle organic compounds (SVOCs), phenols, cyanide and metals. Results indicated the presence of a number of contaminants including benzene, toluene, ethylbenzene, and xylenes (also known as BTEX compounds), PAHs, phenols, arsenic, lead, chromium, mercury, and cyanide; the range and mean of contaminant concentrations detected are provided in Table 1. Maximum concentrations of PAHs (benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene), and metals (arsenic and lead) were present above their respective environmental guideline CVs. Environmental guideline CVs for acenaphthylene, benzo[g,h,i]perylene, phenanthrene and n-propylbenzene are unavailable.

During waste classification testing (as a disposal requirement), polychlorinated biphenyls (PCBs) (Aroclor 1242) were detected in the non-aqueous phase liquid (NAPL) collected from the southeast portion of the processing area (ARCADIS 2004). Testing results indicated the presence of Aroclor 1242 at a concentration of 307 milligrams per kilogram of soil (mg/kg) (see Table 1). Efforts to obtain further details on the level and extent of on-site PCB contamination were unsuccessful (M. Kleczkowski, ARCADIS, personal communication, December 2004). There was a transformer storage area located to the south of the old Jerry Morgan Park; however, soil analysis for PCBs was not performed in this area (NUS 1990).

Old Jerry Morgan Park: Three soil samples were collected from the park and analyzed for VOCs, SVOCs, phenols, cyanide and metals. Results indicated the presence of a number of contaminants including BTEX compounds, PAHs, phenols, arsenic, lead, chromium, mercury, and cyanide; the range and mean of contaminant concentrations detected are presented in Table 2. Maximum concentrations of PAHs (benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene), vinyl chloride and arsenic detected in the soil exceeded their respective environmental guideline CVs. Environmental

guideline CVs for acenaphthylene, benzo[g,h,i]perylene, and phenanthrene are unavailable.

Contaminated soil (i.e., visibly saturated with coal tar and/or oil) excavated from the old Jerry Morgan Park was moved to an existing waste pile located on the southern portion of the site.

Troutman’s Creek (On-Site Portion): Three sediment samples (upstream, on-site and downstream) were collected from Troutman’s Creek and analyzed for VOCs, SVOCs, phenols, cyanide and metals (see Figure 3). Field personnel noted oily seeps along the banks of the creek and a sheen on the surface water. Results indicated the presence of BTEX, PAHs, arsenic, and selenium (see Table 3) in the sediment. Maximum concentrations of benzo[a]pyrene and arsenic detected in the Troutman’s Creek sediment exceeded their respective environmental guideline CVs. Environmental guideline CVs for acenaphthylene, phenanthrene, and n-propylbenzene are unavailable.

Former Seaview Manor Public Housing Complex Property: Prior to the ACO with the NJDEP, four surface soil samples (0 - 4 inch depth) were collected from the Seaview Manor Housing complex property (Woodward Clyde Consultants, 1988a). Samples were analyzed for VOCs, SVOCs, phenols, cyanide, and lead. The results are shown in the following table:

Contaminant	No. Samples Collected	No. Samples With Detections	Range (mg/kg)
PAHs	4	2	ND - 9.76
Phenols		3	ND - 0.8
Cyanide		3	ND - 1.41
Lead		4	12 - 321

ND = not detected

As previously mentioned, the NJNG entered into an ACO with the NJDEP in December 1991. The ACO included the investigation of soil and groundwater at the former Seaview Manor public housing complex property. Four surface⁴ (0 - 2 feet depth) and three subsurface soil samples (2 - 4 feet depth) were collected from the property; results were below the NJDEP RDCSCC for VOCs, SVOCs and metals. Since results were determined to be within typical urban background levels, remediation of the property was not recommended (Geraghty and Miller 1993, 1994).

During the repair of an underground water main in January 1997, black, stained soil, coal-tar residues and odors were observed at the former Seaview Manor public housing complex parking lot. The NJDEP and NJNG confirmed the contamination to be site-related, and the NJNG arranged for the soil to be removed. Approximately 30 cubic yards of soil were excavated and transported to a disposal facility. Although results of composite soil sample analysis indicated that the excavated soil was non-hazardous, the NJDEP requested further soil investigation be conducted at the public housing property.

⁴Samples collected from 0 - 2 feet depth are classified as surface (NJDEP 2005).

Consequently, soil boring investigations were conducted by the NJNG at the former Seaview Manor public housing property. Surface (0 - 2 feet depth) and subsurface soil samples (> 2 feet depth) were collected using a Geoprobe, hollow stem auger, push probe, and hand auger (ARCADIS 2000). Surface soil samples were analyzed for VOCs, SVOCs, phenols, cyanide, and metals; the range and mean of contaminant concentrations detected are provided in Table 4.

The maximum concentration of PAHs (benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene), benzene, and metals (arsenic, cadmium and lead) exceeded their respective environmental guideline CVs. Although the environmental guideline CVs for acenaphthylene, benzo[g,h,i]perylene, and phenanthrene are unavailable, they were considered as the COCs for the site for further evaluation.

Southern Site Boundary: The southern boundary of the LBMGP site abuts the former Conrail property, which included railroad tracks, a freight depot, and petroleum storage tanks owned by Standard Oil. Results of soil sampling conducted during test pit excavation and (upgradient) groundwater monitoring well installation indicated no apparent site-related contamination (Woodward-Clyde Consultants 1985).

The City of Long Branch owns the former Conrail property and has redeveloped the property as the “new” Jerry Morgan Park. Prior to its redevelopment, an environmental investigation of the property was conducted (Birdsall Engineering 1996). The investigation was limited in scope and included composite soil sampling and analysis for priority pollutant metals and total petroleum hydrocarbons. Although the results of the investigation indicated no contamination above applicable state criteria, the NJDEP requested the delineation of site-related contamination along the southern NJNG property boundary. Between 1997 and 1999, delineation of both the lateral and vertical extent of surface soil contamination along the southern site boundary was conducted (ARCADIS 2000). Sampling was initiated at the property line and “stepped out” to the south in four phases until compliance with the NJDEP RDCSCC was demonstrated. Surface samples from 54 soil borings were collected; the range and mean of contaminant concentrations detected are provided in Table 5.

The maximum concentration of PAHs (benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene), and metals (arsenic and lead) exceeded their respective environmental guideline CVs.

Subsurface Soil

In 1984, thirteen borings were drilled with eleven completed as monitoring wells (B-1, B-2 and MW 1 through 11, see Figure 3) (Woodward-Clyde 1985). Subsurface soil samples were analyzed for VOCs, SVOCs and metals. Results are presented in Table 6. Contaminant concentrations in the subsurface soil were generally lower than those detected in the surface/near surface soil (see Tables 1 and 6).

The maximum concentration of benzene, PAHs (benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene and naphthalene), and metals (arsenic and lead) exceeded their respective environmental guideline CVs. The environmental guideline CVs for acenaphthylene, benzo[g,h,i]perylene, phenanthrene and n-propylbenzene are unavailable; they were considered as the COCs for the site.

Surface Water

Three surface water samples were collected and analyzed for VOCs, SVOCs and metals (Woodward-Clyde 1985). Results are presented in Table 7. The maximum concentration of VOCs (benzene and trichloroethene) and mercury exceeded their respective environmental guideline CVs. The environmental guideline CVs for phenanthrene is unavailable; phenanthrene was considered a surface water COC for the site.

Groundwater

In 1985, the lateral and vertical extent of on-site groundwater contamination was also evaluated (Woodward-Clyde Consultants 1985). Groundwater samples were collected (November 1984 and January 1985) from monitoring wells screened in the shallow fill and the underlying aquifer. During sample collection, an oily substance was encountered at several monitoring wells. Analytical results are presented in Table 8.

The maximum concentration of VOCs (benzene, toluene, ethylbenzene and xylene), bis(2-ethylhexyl)phthalate, PAHs (acenaphthene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, benzo[a]anthracene, chrysene, fluorene, fluoranthene, naphthalene and pyrene) and arsenic exceeded their respective environmental guideline CVs. The environmental guideline CVs for acenaphthylene and phenanthrene are unavailable; they were considered as the COCs for the site.

Ambient/Indoor Air

Between November 1994 and March 2003, NJNG collected on-site ambient (playground, fence line, courtyard and east yard) and crawlspace and indoor air samples from former Seaview Manor Public Housing property and analyzed them for benzene, toluene, ethylbenzene and xylene (BTEX) (see Table 9). The maximum concentration of toluene, ethylbenzene and xylene detected in the indoor air were below their respective environmental guideline CVs. The maximum as well as the mean concentration of benzene detected in the indoor air exceeded the environmental guideline CVs.

In March 2003, NJNG also sampled two off-site childcare facilities (Check Mate Day Care Center on C. P. Williams Place, and the Second Baptist Church Day Care Center, at the intersection of Liberty Street and C. P. Williams Place) for BTEX contamination (see Table 10). The maximum as well as the mean concentration of

benzene detected in the indoor air exceeded the environmental guideline CVs. Completed indoor air exposures to residents were evaluated and discussed in a previous health consultation (ATSDR 2004).

By summer/fall 2004, all Seaview Manor residents were relocated, and buildings were demolished by the NJNG contractors. Currently, the area is being remediated by the NJNG.

Off-Site Contaminants

Site-related contamination which has migrated off-site has been detected in Troutman's Creek. Currently, NJNG is delineating the extent of this site-related contamination (including residential properties located along Seaview Avenue near Troutman's Creek, the Liberty Avenue and the Long Branch Sewerage Authority property). A separate health consultation will be prepared to evaluate exposures associated with this off-site contamination.

Overview of Remedial Actions

Interim remedial measures and remedial actions were implemented at the LBMGP site (NJDEP 1993; Geraghty & Miller 1997; ARCADIS 2004). Interim remedial measures included: the disposal of liquids from gas and oil tanks and gas and relief holders located along the southern site boundary; the excavation of coal tar contaminated soil piles and debris; the removal or closure of underground process and gasoline storage tanks and an aboveground process tank; and the demolition of several structures.

For remedial actions, the cleanup goal was established as the 1) NJDEP Unrestricted Use Criteria or 2) impact to groundwater criteria, whichever was strictest (ARCADIS 2004). Contaminated soil was either removed to accommodate the placement of the cap, or isolated beneath the two foot thick cap. Site remedial actions consisted of the following:

- demolition and disposal of above and below ground structures;
- removal of product from the processing area;
- excavation and off-site disposal of soils impacted by historical operations;
- installation of a minimum two foot soil cap over the entire site;
- installation of replacement stormwater piping;
- abandonment of groundwater monitoring wells;
- excavation and off-site disposal of soil and sediment from the on-site portion of Troutman's Creek;
- rechannelization of the on-site portion of Troutman's Creek;
- installation of a site perimeter security fence (eight foot, barbed wire); and,
- deed restrictions to restrict potential disruption of the cap.

With the exception of asphalt paved areas, the cap was installed over the entire site and FirstEnergy Corporation (formerly GPU Energy) electrical substation. The

electrical substation is fenced, and unpaved areas are covered by six inches of gravel. The purpose of the soil cap and paving is to prevent direct contact with contaminants (ARCADIS 1999). A perimeter air monitoring system was also installed and operated during the periods of intrusive remedial actions (ENSR 2000; Gradient 2003).

Summary of Contaminants of Concern

Surface Soil and Sediment

The maximum concentration of contaminants detected in soil and sediment, along with Environmental Guideline CVs are presented in Tables 1 through 5. The following contaminants in the soil and sediment exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

Location	VOCs	PAHs and PCBs	Metals
Processing Area	n-Propylbenzene	Acenaphthylene, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Chrysene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene, Phenanthrene PCBs ¹	Arsenic, Lead
Old Jerry Morgan Park	Vinyl chloride	Acenaphthylene, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene, Phenanthrene	Arsenic
Troutman's Creek	n-Propylbenzene	Acenaphthylene, Benzo[a]pyrene, Phenanthrene	Arsenic
Former Seaview Manor public housing complex	Benzene	Acenaphthylene, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Chrysene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene, Phenanthrene	Arsenic, Cadmium, Lead
Southern Site Boundary		Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Chrysene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene	Arsenic, Lead

¹PCBs were detected in the processing area during remediation

Subsurface Soil

The maximum concentration of contaminants detected in subsurface soil along with Environmental Guideline CVs is presented in Table 6. The following contaminants in the subsurface soil exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

VOCs	PAHs	Metals
Benzene, n-Propylbenzene	Acenaphthylene, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[g,h,i]perylene, Benzo[k]fluoranthene, Dibenz[a,h]anthracene, Indeno[1,2,3-cd]pyrene, Naphthalene, Phenanthrene	Arsenic, Lead

Surface Water

The maximum concentration of contaminants detected in surface water along with Environmental Guideline CVs is presented in Table 7. The following contaminants in the surface water exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

VOCs	PAHs	Metals
Benzene, Trichloroethene	Phenanthrene	Mercury

Groundwater

The maximum concentration of contaminants detected in groundwater along with Environmental Guideline CVs is presented in Table 8. The following contaminants in the groundwater exceeded their corresponding environmental guideline CVs, and as such, are designated as the COCs for the site:

VOCs	SVOCs and PAHs	Metals
Benzene, Ethylbenzene, Toluene, Xylene	Bis(2-Ethylhexyl)phthalate, Acenaphthylene, Acenaphthene, Benzo[a]anthracene, Benzo[a]pyrene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Chrysene, Fluorene, Fluoranthene, Naphthalene, Phenanthrene, Pyrene	Arsenic

Indoor Air

The maximum and the mean concentration of benzene detected in the indoor air of Seaview Manor Public Housing property (see Table 9) and the day care centers (see Table 10) exceeded its corresponding environmental guideline CVs, and as such, benzene is designated as a COC for the site.

A brief discussion of the general toxicologic characteristics of the COCs 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 LBMGP site were identified as follows (see Table 11):

Completed Pathways

Ingestion of on-site contaminated soil and sediment (past). Surface soils and sediment of the processing area, old Jerry Morgan Park, Seaview Manor public housing complex, properties along the southern site boundary, and the on-site portion of Troutman's Creek were contaminated with PAHs, VOCs, metals, phenols and cyanide. Residents, including children, were exposed to contaminants while living and engaging in outdoor recreational activities at the site (including family picnics held on the on-site portion of Troutman's Creek). This scenario also includes site visitors and trespassers.

Ingestion of surface water from Troutman's Creek (past). Site-related contaminants were detected in surface water of on-site portions of Troutman's Creek. Residents, including children, were exposed to contaminants during outdoor recreational activities including swimming in the Troutman's Creek.

Inhalation of indoor air (past). Results of indoor air sampling performed at the former Seaview Manor public housing complex (via vapor intrusion) and the two off-site day care centers indicated the presence of elevated levels of benzene in the Seaview Manor. Health risks associated with the inhalation pathway were previously evaluated (ATSDR 2004).

Potential Pathways

Potential exposure pathways for the LBMGP site were identified as follows (also summarized in Table 11):

Inhalation of ambient air (past). During the years of plant operation (i.e., prior to 1961), area residents were potentially exposed to VOCs in air due to plant emissions. Since air sampling data prior to 1961 are not known to exist, exposures associated with this pathway could not be evaluated. Future remedial activities which disturb contaminated soils and sediments may release contaminants into the atmosphere.

Ingestion of surface water from Troutman's Creek (present, future). Site-related contaminants have been detected in off-site portions of Troutman's Creek. At the instruction and oversight of the NJDEP, the NJNG is conducting further investigation and delineation of site-related contaminants in Troutman's Creek. Soil, sediment, and surface water samples will be evaluated to address resident concerns regarding contaminant exposures including those associated with creek flooding (September 4, 2004 letter from J. Johnson, NJDEP to J. Wheeler, Concerned Citizens Coalition of Long Branch). The NJDHSS, in cooperation with the ATSDR, will prepare a separate health consultation to evaluate contaminant exposures associated with the recreational use of Troutman's Creek.

Ingestion of biota from Troutman's Creek (past, present, future). Biota (e.g., fish, plants) in Troutman's Creek were exposed to contaminated sediment. Residents routinely fished Troutman's Creek and ate their catch. Since naphthalene and toluene exhibit moderate tendencies to bioconcentrate in the fatty tissues of aquatic animals, COCs may have been introduced into the aquatic food chain (ATSDR 1995). As stated previously, the NJNG is conducting additional investigation and delineation of site-related contaminants in Troutman's Creek. A separate health consultation will be prepared to evaluate exposures associated with the consumption of contaminated biota.

Ingestion of groundwater (past). Exposures to groundwater contamination are unlikely since there are no on-site or nearby off-site private wells drilled into the shallow contaminated aquifer. However, during a May 2004 Availability Session, one longtime

area resident stated that as a young child she recalled her family using a private well for their source of drinking water. No data for this well are available.

Eliminated Pathways

Inhalation of indoor air (present, future). By summer/fall 2004, all Seaview Manor residents were relocated, and buildings were demolished by the NJNG contractors. As such, there are no present or future on-site exposures via this pathway.

Ingestion of groundwater (present, future). Area residents receive their potable water from a public water supply system, and there are no potable wells within a mile radius of the site (M. Kenney, NJDEP, personal communication, 2004). As such, exposures associated with the transport of contaminants in deeper soils through the groundwater ingestion pathway are not possible, and details of groundwater analytical results are not presented in this public health assessment.

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 LBMGP site include the incidental ingestion of soil and surface water and the inhalation of indoor air. Since there is insufficient information available on the nature and magnitude of potential exposures associated with the inhalation of ambient air and the ingestion of groundwater and biota, an evaluation with health guideline CVs could not be conducted.

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) may be used. The RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Ingestion - On-Site Soil

Historically, portions of the LBMGP site were used for residential purposes (pre-1950s housing, Seaview Manor public housing complex) or redeveloped for recreational use (old Jerry Morgan Park). Additionally, residents routinely used the processing area for recreational purposes (i.e., picnicking, hiking and fishing along the on-site portion of Troutman's Creek). Exposures are based on ingestion of contaminated soil; non-cancer exposure doses were calculated using the following formula:

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

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

The following site-specific exposure assumptions (USEPA 1997; NJDEP 2004) were used to calculate past contaminant doses. Estimated number of years exposed is based on information collected during Availability Sessions held with the community:

Exposure Point	Number of Days Exposed Per Year	Number of Years Exposed
Processing Area	108 days (3x per week)	70
Old Jerry Morgan Park	108 days (3x per week)	17
Troutman's Creek	108 days (3x per week)	70
Seaview Manor Public Housing Complex	daily	50
Southern Site Boundary	108 days (3x per week)	70

VOCs

Based on maximum concentrations of vinyl chloride and benzene detected in the surface soil of old Jerry Morgan Park and Seaview Manor public housing complex, respectively (Table 13 and 15), chronic exposure doses calculated for children and adults were lower than the corresponding health guideline CVs. As such, past exposures to vinyl chloride and benzene are unlikely to cause non-cancer adverse health effects. n-Propylbenzene was detected in the surface soil and sediment of the processing area and the Troutman's Creek, respectively. Although no health guideline CV of n-propylbenzene is available, the health effects associated with the exposure to n-propylbenzene is evaluated as follows:

n-Propylbenzene. n-Propylbenzene was detected in the surface soil (0.028 mg/kg) and sediment (0.06 mg/kg) of the processing area and the Troutman's Creek, respectively. n-Propylbenzene occurs as a natural constituent in petroleum and bituminous coal. Exposure to n-propylbenzene at high concentrations can cause adverse effects on the nervous system (neurotoxicity). It can induce confusion, fatigue, irritability, and other behavioral changes. There is little human toxicology data available regarding n-propylbenzene, and no oral exposure studies in humans were found in the literature. Additionally, no MRL or RfD is available. However, the Division of Drinking Water and Environmental Management Branch, California Department of Health Services (2005) proposed a drinking water notification level for n-propylbenzene based on cumene, a structural analog of n-propylbenzene. The notification level derivation used a NOAEL of 110 mg/kg/day, based on histopathological changes and a safety factor of 3,000 to protect against any potential toxic effects in humans. The safety factor includes uncertainty factors of 10 each for interspecies and intraspecies differences, a factor of 3 for extrapolation from six months to chronic duration, and 10 for the many database deficiencies. Using the same NOAEL and safety factor, an oral reference dose for n-propylbenzene may be calculated as 0.037 mg/kg/day.

Based on the concentration of n-propylbenzene detected in surface soil at the processing area and sediment at the Troutman's Creek, the calculated child exposure doses (see Table 12 and 14) were about 430,000 and 200,000 times lower than the calculated reference dose (0.037 mg/kg/day), respectively. As such, non-cancer adverse

health effects associated with n-propylbenzene exposures is not expected in children and adults.

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 combustion processes, PAHs are widespread in the environment. 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 maximum concentrations of PAHs detected in the surface soil, the chronic exposure doses for children and adults were calculated (see Tables 12 through 16); no health guideline CVs are available for these PAHs identified as on-site 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 are shown below:

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

Source: EPA 2006

The RfD's of these PAHs are based on the NOAEL for less serious health effects and are much higher than the exposure doses calculated for the PAHs detected on-site. Based on the maximum concentration of phenanthrene detected in surface soil of the processing area (1,620 mg/kg, see Table 1), the calculated chronic child exposure dose (4.96×10^{-3} mg/kg/day) was about four times lower than the lowest reported RfD (i.e., 0.02 mg/kg/day for naphthalene). The exposure doses associated with the remaining PAHs were up to five orders of magnitude lower than the lowest reported RfD. As such, non-

cancer adverse health effects associated with on-site PAH exposures in the past is unlikely in children and adults (see Table 12).

PCBs

Chronic exposure doses calculated for children and adults based on PCB contamination detected in the processing area were higher than the corresponding health guideline CVs (see Table 1). Aroclor⁵ 1242 was detected in the soil of the processing area during remediation. The most commonly observed non-cancer health effects in individuals exposed to large amounts of PCBs are skin conditions such as acne and rashes (ATSDR 2000a). Occupational exposure studies have shown changes in blood and urine that may indicate liver damage. Animals administered with small exposure doses for several weeks or months developed health effects including anemia, acne-like skin conditions, and liver, stomach, and thyroid gland injuries. Additional health effects in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects.

Since a health guideline CV is unavailable for Aroclor 1242, the RfD for Aroclor 1016 and Aroclor 1254 was used to assess potential non-cancer health effects of Aroclor 1242. Based on the concentration of Aroclor 1242 detected at the processing area, the chronic exposure dose calculated for children and adults (i.e., 0.00094 and 0.00014 mg/kg/day) exceeded the RfD (see Table 12). The NOAEL for Aroclor 1016 is 0.007 mg/kg/day, based on reduced birth weight, and the RfD incorporates an uncertainty factor of 100. The LOAEL and the uncertainty factor for Aroclor 1254 are 0.005 mg/kg/day and 300, respectively and is based on ocular exudate, inflamed and prominent meibomian glands, distorted growth of finger and toe nails and decreased antibody response to sheep erythrocytes. Based on the PCB concentration detected at the Processing Area, the child exposure dose was about seven (based on Aroclor 1016) and five times (based on Aroclor 1254) lower than the NOAEL and LOAEL, respectively. For adults, the exposure dose was about 50 (based on Aroclor 1016) and 35 (based on Aroclor 1254) times lower than the LOAEL. As such, there was a potential for non-cancer health effects in children from exposures to PCBs in the processing area.

Metals

Based on maximum concentrations of arsenic detected in the surface soil of the processing area, old Jerry Morgan Park and southern site boundary and sediment of the Troutman's Creek sediment), the chronic exposure doses calculated for children and adults were lower than the corresponding health guideline CVs. As such, past exposures to arsenic at these locations are unlikely to cause non-cancer adverse health effects.

Based on maximum concentrations of arsenic and cadmium detected in the surface soil of the Seaview Manor Public Housing property, the chronic exposure doses calculated for children exceeded the corresponding health guideline CVs. Health

⁵Aroclors are commercial mixtures of PCBs.

guideline CV for lead is unavailable. A brief evaluation of non-cancer health implications of arsenic, cadmium and lead are presented below.

Arsenic. Arsenic was detected in the soil of the Seaview Manor public housing complex (see Table 4). 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. 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 (less serious effect). Skin contact with inorganic arsenic may cause redness and swelling. Organic arsenic compounds are less toxic than inorganic arsenic compounds (ATSDR 2000b).

Based on the maximum concentration of arsenic detected at the former Seaview Manor public housing complex property, the chronic exposure dose calculated for children (i.e., 0.000468 mg/kg/day) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table 15). The calculated exposure dose is about 1.7 and 30 times lower than the NOAEL (i.e., 0.0008 mg/kg/day) and LOAEL (i.e., 0.014 mg/kg/day), respectively. Additionally, based on the average concentration of arsenic detected (the more likely exposure scenario), the chronic exposure dose (i.e., 0.0000886 mg/kg/day) was lower than the MRL. As such, the likelihood of non-cancer adverse health effects from arsenic exposures are considered low.

Cadmium. Cadmium was detected in the soil of the processing area (see Table 12). Cadmium is a natural element in the earth's crust. All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Long-term exposure to lower levels of cadmium in air, food, or water may lead to the accumulation of cadmium in the kidneys, resulting in possible kidney disease (ATSDR 1999). 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.

Although the maximum chronic exposure dose calculated for children (i.e., 0.000474 mg/kg/day) exceeded the ATSDR MRL of 0.0002 mg/kg/day, the likelihood of non-cancer adverse health effects is considered low due to the following factors: 1) the maximum and the average exposure doses are lower than the RfD (i.e., 0.001 mg/kg/day for food or 0.005 mg/kg/day for water); and 2) based on the average concentration of cadmium detected (the more likely exposure scenario), the calculated chronic exposure dose (i.e., 0.0000143 mg/kg/day) was about an order of magnitude lower than the MRL.

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 maximum concentration of lead detected in the soil of the processing area, Seaview Manor public housing complex, and southern site boundary

exceeded the NJDEP RDCSCC of 400 mg/kg. Since the mean lead concentration detected in the Seaview Manor public housing complex (120 mg/kg) and the Old Jerry Morgan Park (113.3 mg/kg) was well below the RDCSCC, potential for adverse health effects from lead exposures is not expected.

The mean lead concentration detected in the Processing Area (428 mg/kg) and Southern Site Boundary (554 mg/kg) exceeded the RDCSCC. The Processing Area was used for recreational purposes and the Southern Site Boundary area was used as recreational/residential purposes. Lead exposures associated with the intermittent recreational use of lead contaminated on-site areas were evaluated using the USEPA's integrated exposure uptake biokinetic (IEUBK) model (USEPA 1994a). The IEUBK model estimates a plausible distribution of blood lead levels centered on the geometric mean blood lead levels from available exposure information. Blood lead levels are indicators of recent 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 $\mu\text{g}/\text{dL}$) or less (USEPA 1986; CDC 1991). In using the IEUBK model, the USEPA recommends that the lead concentration in site soil does not result in a 5% probability of exceeding a blood lead concentration of 10 $\mu\text{g}/\text{dL}$ (USEPA 1994b). The average lead level in surface soils 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 Processing Area was visited. The visit frequency was three days per week over nine months of the year; exposure during the remaining days of the week was at the residence (Southern Site Boundary area).
2. Model default values were used for all other variables (USEPA 2002) including residential soil and dust.

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

Exposure Scenario		
Age (months)	Three Site Visits Per Week ¹	
	Blood Lead Level ² (µg/dL)	P ₁₀ (%) ³
6 - 12	6	14
12 - 24	6.9	21
24 - 36	6.4	17
36 - 48	6.1	15
48 - 60	5	7
60 - 72	4.2	3
72 - 84	3.7	2

¹weighted paint sludge lead concentration (428 ppm x 3/7) + (554 ppm x 4/7) = 500 ppm (USEPA 2003a); ²Geometric mean lead levels in blood; ³probability of blood lead level > 10 µg/dL

For the incidental soil lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 6 - 84 months ranged between 3.7 and 6.9 µg/dL. However, the probabilities of blood lead levels exceeding 10 µg/dL (the CDC level of concern) for children ages 6 - 60 months exceeded 5 percent. Therefore, there was a potential for adverse health effects associated with lead exposures at the Processing Area and the Southern Site Boundary. An adult blood lead model estimated a geometric mean blood lead level of 2.2 µg/dL (USEPA 2003b).

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 - On-Site Surface Water

Residents, including children, were exposed to contaminants during outdoor recreational activities including swimming in the Troutman's Creek. Exposures are based on ingestion of contaminated surface water; non-cancer exposure doses were calculated using the following formula:

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

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;
 C = concentration of contaminant in water (mg/L);
 IR = water 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 while Swimming (mL/event)	Swimming Frequency (event/year)	Exposure period for swimming (year)	
		Child	Adult
50	39	12	22

Based on maximum concentrations of benzene, trichloroethene and mercury detected in the surface water of the Troutman’s Creek (see Table 17), chronic exposure doses calculated for children and adults were lower than the corresponding health guideline CVs. As such, past exposures associated with swimming in the Troutman’s Creek are unlikely to cause non-cancer adverse health effects.

Inhalation - Indoor Air

Non-cancer adverse health effects from exposure to indoor air contaminants detected in the Seaview Manor public housing property were evaluated (ATSDR 2004). The concentration of the contaminants in the indoor air was lower than the respective health guideline CVs. The NJDHSS and ATSDR classified indoor air exposures among residents of the Seaview Manor public housing property to represent an indeterminate public health hazard.

Due to their proximity to the LBMGP site, indoor air sampling has also been conducted at two nearby child day care centers (ATSDR 2004). BTEX levels were below health guideline CVs. As such, the contaminants are unlikely to cause non-cancer adverse health effects. The NJDHSS and ATSDR also concluded that the indoor air at the day care centers represented no apparent public health hazard.

Cancer Health Effects

The site-specific lifetime excess cancer risk (LECR) indicates the cancer potential of exposure to 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

Ingestion – On-site Soil

The cancer class of the COCs detected in the surface soil and sediment of various areas are given in Tables 18 through 22. The carcinogens found in these environmental media were: PAHs, PCBs and arsenic detected in the surface soil of the Processing Area; PAHs, vinyl chloride and arsenic detected in the surface soil of old Jerry Morgan Park; PAHs and arsenic detected in the sediment of the Troutman’s Creek; PAHs, benzene and arsenic detected in the surface soil of Seaview Manor Public Housing property; and PAHs and arsenic detected along the Southern Site Boundary.

Exposure doses were calculated using the following formula:

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

- 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).

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 18 - 22).

Based on previously described exposure assumptions, LECRs were calculated by multiplying the exposure dose by the cancer slope factor. The cancer slope factor is defined as the slope of the dose-response curve obtained from animal and/or human cancer studies and is expressed as the inverse of the daily exposure dose, i.e., (mg/kg/day)⁻¹. LECRs based on maximum contaminant concentrations detected in the five AOCs are presented in Tables 18 - 22; LECR values in parentheses are based on mean AOCs contaminant concentrations. LECR for cadmium could not be calculated since a cancer slope factor is unavailable (USEPA 2004).

Lead has been classified as a carcinogen by the USDHHS⁶ and USEPA⁷. The carcinogenicity of inorganic lead and lead compounds has been evaluated by the USEPA (USEPA 1986, 1989). It has been 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.

Based on maximum PAHs, PCBs, VOCs and metals concentrations detected in the five AOCs, the resulting LECR for each class of contaminant is presented in Figure 6. For PAHs, for example, the LECRs are 2, 8, and 5 in 10,000 for individuals who lived in and/or participated in recreational activities at the Seaview Manor public housing complex, Processing Area, and Southern Site Boundary, respectively. For the processing area and the southern site boundary, LECRs of 4 and 1 in 10,000 was also indicated at the mean contaminant concentrations (the more likely exposure scenario), respectively (see Figure 7). At the mean PAH contaminant concentration, the LECR for the Seaview Manor public housing complex was reduced by about an order of magnitude.

As previously indicated, the LECRs presented in this report are based on site-specific assumptions that may not be representative of actual individual exposures.

Ingestion - On-Site Surface Water

LECRs associated with ingestion of the Troutman's Creek water during swimming were evaluated (see Table 23). Based on maximum benzene and trichloroethene concentrations, the calculated LECRs were less than one additional cancer case among one million persons exposed which is considered insignificant or no increased risk.

Inhalation - Indoor Air

LECRs associated with indoor air contaminant exposures at the Seaview Manor public housing complex were previously evaluated (see Table 24) (ATSDR 2004). Based on maximum and mean benzene concentrations detected in the indoor air, the calculated LECRs were 2 in 100,000 and 8 in 1,000,000 to the exposed population, respectively.

Past individual exposures to site-related contaminants may be higher than those presented in this Public Health Assessment. That is, individuals may have accessed or frequented more than one AOC on a regular basis. As such, LECRs presented in this report are based on site-specific assumptions that may not be representative of actual individual exposures.

⁶Lead and Lead Compounds are listed in the Eleventh Edition of the Report on Carcinogens as "reasonably anticipated to be human carcinogens" (NTP 2006)

⁷B2; probable human carcinogen

Assessment of Joint Toxic Action of Chemical Mixtures

At the LBMGP site, residents were exposed to PCBs, VOCs, PAHs and metals via direct contact, ingestion and inhalation. 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).

Non-Cancer

To evaluate the risk for non-cancer adverse health effects of chemical mixtures, a hazard index (HI) for the chemicals was calculated (ATSDR 2005). 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 LBMGP site, based on the mean concentration of contaminants detected (the more likely scenario), the HI calculated for children for the soil and sediment (48) was greater than 1 (see Table 25); for adults, it is evident (high PCB concentration detected in soil) that the HI will also be greater than 1. For indoor air exposures, the HI calculated for children was less than 1.0 (see Table 25); as such, it is unlikely that significant additive or toxic interaction would occur.

For chemical mixtures with an HI greater than 1, 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 soil/sediment exposure dose to NOAEL for the contaminants was calculated (see Table 25). Since the exposure dose/NOAEL for PCBs is greater than 0.1, additive or interactive effects of chemical mixtures from exposures to soil/sediment in children and adults is possible. However, an in-depth mixtures evaluation could not be conducted due to unavailability of relevant chemical interaction profiles (consisting of arsenic, benzene, lead, PCBs, n-propylbenzene and vinyl chloride).

Cancer

As measures of probability, individual LECRs can be added. Cumulative LECRs associated with contaminant exposures from inhalation and ingestion are presented in Figure 8. It is evident that past exposures to maximum contaminant concentrations at the Seaview Manor public housing complex property, processing area, and southern site boundary indicated 3, 10 and 7 in 10,000 LECR to the exposed population, respectively.

Based on mean contaminant concentrations, the cumulative LECR was determined to be 0.3, 7 and 2 in 10,000 to the exposed population. Since a cancer slope factor is unavailable for cadmium, a LECR was not included in this evaluation.

Health Outcome Data

The NJDHSS and ATSDR evaluated cancer incidence in the area surrounding the former Long Branch Manufactured Gas Plant site (ATSDR 2003). Total cancer incidence and 13 specific cancer types were evaluated in this investigation. The specific cancers types were selected because they represent cancer groupings that may be more sensitive to the effects of environmental exposure. The New Jersey State Cancer Registry, a population-based cancer incidence registry covering the entire state, was used for the ascertainment of cancer cases. The study period for this investigation was January 1, 1979 through December 31, 2000. Standardized incidence ratios (SIR) were used for the quantitative analysis of cancer incidence. The SIR compares the observed number of cases to an expected number of cases based on average state rates. Males and females, all races combined, were evaluated separately and by two race groups (white and black). Cancer data was evaluated for all of Long Branch and for Census Tract (CT) 8056, the area of Long Branch which had the highest potential for exposure to site contaminants.

Overall, combined cancer incidence was not elevated in Long Branch. The citywide analysis did detect a statistically significant elevation in brain/central nervous system cancer for all females (SIR=1.6), white females (SIR=1.6), and black females (SIR=3.2). Lung cancer in white females (SIR=1.2) was also statistically higher than expected. Stomach cancer was statistically lower than expected for all females (SIR=0.6), white females (SIR=0.6) and black males (SIR=0.8). Pancreatic cancer was also statistically lower for black males (SIR=0.2).

In CT 8056, a statistically significant elevation in all cancers combined was detected for all males (SIR=1.3) and black males (SIR=1.4). A statistically higher number of cancer also was found for esophageal cancer in all females (SIR=5.7) and black females (SIR=4.6). Additionally, lung cancer in all males (SIR=1.6) was statistically higher than expected in CT 8056.

Cancer is a group of more than 100 different diseases (i.e., cancer types and subtypes), each with their own set of risk factors. The multifactorial nature of cancer etiology, where a given disease may have more than one cause, complicates the evaluation of potential risk factors and specific disease outcomes. Benzene and PAHs, two important site contaminants, have been identified as possible risk factors for certain cancer types, primarily leukemia (benzene) and lung cancer (PAHs). In the current analysis, the incidence of leukemia citywide and in CT 8056 was not statistically significantly higher than expected over the study time period. Lung cancer incidence, however, was statistically significantly higher in white females citywide and all males in CT 8056.

While there are multiple risk factors for lung cancer, tobacco smoking is considered the most important risk factor, estimated to account for more than 85% of all lung cancer cases (National Cancer Institute, 1996). Other known risk factors for lung cancer include indoor exposure to radon and environmental tobacco smoke, occupational exposure to asbestos and other cancer-causing agents in the workplace (including radioactive ores; chemicals such as arsenic, vinyl chloride, nickel, chromates, coal products, mustard gas, and chloromethyl ethers; fuels such as gasoline; and diesel exhaust), and exposure to air pollution (American Cancer Society, 2003a).

There is very limited evidence that exposure to environmental contamination is associated with esophageal or stomach cancer risk. Occupational exposure to tetrachloroethylene, the solvent used in dry cleaning, may lead to greater risk of esophageal cancer (American Cancer Society, 2003b). Dry cleaning workers have a higher rate of esophageal cancer. Also, exposure to other chemical fumes may lead to an increased risk of esophageal cancer. However, the most important known risk factors for esophageal cancer are consumption of alcohol and tobacco products, which account for over 80% of the risk of squamous cell carcinoma of the esophagus (American Cancer Society, 2003b). A number of studies have shown an association between esophageal cancer and low socioeconomic status, independent of smoking or drinking, which may be associated with poor nutrition.

The causes of brain/CNS cancer are largely unknown, but a variety of genetic and environmental factors have been suggested (National Cancer Institute, 1996). The only established environmental risk factor for brain/central nervous system cancer is high dose ionizing radiation (American Cancer Society, 2003c). Certain occupations and industrial exposures have also been implicated as possible risk factors for brain/central nervous system cancer including chemists, embalmers, anatomists, precision metal workers, farmers, synthetic rubber and polyvinyl chloride manufacture, refining of crude oil and production of petroleum based chemicals, manufacture of pharmaceuticals, and the nuclear fuels and weapons industry (National Cancer Institute, 1996). Additionally, while there is no conclusive evidence, exposure to electromagnetic fields has also been suggested as a possible risk factor for brain/central nervous system cancer.

While this analysis found some elevations in select cancer types, the results provide inconclusive evidence that the cancer rate in this community has been affected by potential exposures to site contamination. Although no further cancer analyses was recommended based on this evaluation, the NJDHSS and ATSDR did recommend to continue working with community representatives to determine the most appropriate health education and outreach strategies to inform the general population about health issues associated with the environmental contamination.

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 potential health effects associated with exposure to site-related contaminants were evaluated. Based on maximum concentrations of mercury, arsenic and cadmium detected in the soil at the Seaview Manor public housing complex property, exposure doses for children exceeded the respective health guideline CVs, although the likelihood of adverse non-cancer health effects was determined to be low.

Based on the PCB (Aroclor 1242) concentration detected in the processing area, the exposure dose calculated for children exceeded the health guideline CV. Since there is no reported LOAEL available for Aroclor 1242, the exposure dose was compared with the LOAEL for Aroclor 1016 and Aroclor 1254 and found to be only 5 and 30 times lower than the LOAEL, respectively. As such, non-cancer adverse health effects in children are possible. According to the literature, 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 (ATSDR 2003). 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 of children born to and nursed by mothers exposed to increased levels of PCBs were affected. Transplacental transfers of PCBs were also reported.

The maximum concentrations of lead detected in the soil at the Seaview Manor public housing complex property, processing area, and southern site boundary exceeded the NJDEP RDCSCC. The mean lead concentration (the more likely exposure scenario) detected in the Seaview Manor public housing complex was below the NJDEP RDCSCC. No MRL or RfD is available for lead. Accumulation of lead in the body can cause damage to the nervous or gastrointestinal system, kidneys, or red blood cells. 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). Lead exposures associated with on-site contaminated areas were evaluated using the IEUBK model. The model results indicated that there was a potential for adverse health effects associated with lead exposures at the Processing Area and the Southern Site Boundary.

Based on maximum concentrations of PAHs, PCBs, VOCs, and metals detected, 1 in 1,000 cumulative LECR was determined for residents (including children). Based on the mean concentrations of these contaminants, a cancer risk as high as 7 in 10,000 cumulative LECR was determined for individuals including children.

Public Comment

The public comment period for this public health assessment was from April 15, 2007 through June 30, 2007. The comments and the responses are given in Appendix B.

Conclusion

More than 100 years of operation at the former LBMGP site resulted in the generation of hazardous wastes and environmental contamination of on- and off-site areas. Contaminants of concern were PAHs, VOCs, and metals. Although the most contaminated areas are on-site surface soils (including the Seaview Manor public housing complex property), contamination extends into the deeper parts of the underlying aquifer. Characterization and contamination delineation of on-site areas have been conducted and remedial actions were implemented. Currently, the on-site areas pose ***No Public Health Hazard***.

In the past, there were completed exposure pathways to area residents via the incidental ingestion of contaminated on-site surface soil and sediment and inhalation of indoor air. Potential pathways were also identified and included past inhalation of ambient air and past and current ingestion of surface water, biota from Troutman's Creek. Contaminants of concern identified for the site were benzene, ethylbenzene, n-propylbenzene, toluene, xylene, vinyl chloride, bis(2-ethylhexyl)phthalate, polycyclic aromatic hydrocarbons, polychlorinated biphenyls, arsenic, cadmium, lead and mercury. Only one sample result for Aroclor 1242 was available, and non-cancer adverse health effects were found to be possible for children. Based on the maximum concentrations of arsenic and cadmium detected in surface soil and sediment, potential for non-cancer adverse health effects was found for children only and determined to be low. However, there was a potential for adverse health effects associated with lead exposures to the on-site exposures from the Processing Area and the Southern Site Boundary. 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 maximum and mean contaminant concentrations. Cumulative lifetime excess cancer risks (using maximum contaminant concentrations) associated with the site was 1 in 1,000 to the exposed population. Based on average contaminant concentrations (the more likely exposure scenario), the cumulative lifetime excess risk may have been as high as 7 in 10,000 to the exposed population. As such, based on lifetime excess cancer risks and childhood lead exposure in the past, the site posed a ***Public Health Hazard***.

The characterization and delineation of off-site contamination is currently being conducted. Since information necessary to make a public health judgment is lacking, past, current and future exposures associated with off-site contamination are considered an ***Indeterminate Public Health Hazard***.

Recommendations

1. The NJNG, with NJDEP oversight, should complete the remedial investigation of off-site areas. This includes residential properties along Seaview Avenue and off-site surface water, sediment and biota of the Troutman's Creek.
2. NJDEP should continue to require and ensure that air monitoring, dust suppression and odor control measures are implemented during remedial actions.

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 NJDHSS and ATSDR evaluated cancer incidence in the area surrounding the LBMGP site (ATSDR 2003).
2. The NJDHSS and ATSDR evaluated the exposures associated with the indoor air contaminants detected at the Seaview Manor public housing complex property (ATSDR 2004).
3. The NJDHSS and ATSDR have met with residents and the Concerned Citizens Coalition of Long Branch to identify and address community concerns.
4. The NJDHSS and ATSDR held several Availability Sessions for residents to identify community concerns, and to provide information to residents about exposure pathways and the contaminants of concern.
5. The NJDHSS and ATSDR have provided public education materials to residents that describe health aspects of the contaminants.

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. In cooperation with the ATSDR and NJDEP, public meetings will be scheduled to discuss the findings of this report and to address any additional community concerns.
3. As additional off-site contamination data become available, the NJDHSS and ATSDR will prepare health consultation(s) in order to evaluate the public health implications of contaminants detected and provide assistance to residents in reducing exposures to contaminants at the LBMGP site.

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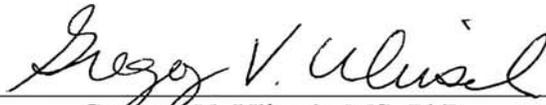
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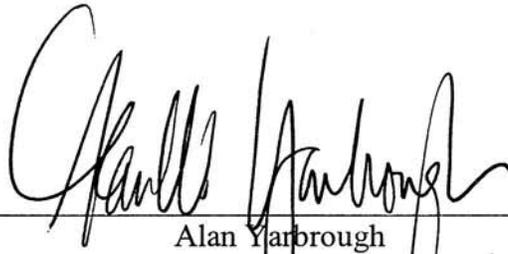
CERTIFICATION

The public health assessment for the former Long Manufactured Gas Plant site, Monmouth 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.



Gregory V. Ulirsch, MS, PhD
Technical Project Officer, CAT, CAPEB, DHAC
Agency for Toxic Substances and Disease Registry

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



Alan Yarbrough
Team Leader, CAT, CAPEB, DHAC
Agency for Toxic Substances and Disease Registry

Table 1: Surface Soil (0-2 ft depth) Sampling Results of the Processing Area

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Volatile Organic Compounds (VOCs)				
Benzene	0.01 - 2.2	0.46	10 (CREG ^d)	No
1,2-Dichloroethene (trans)	0.011	0.01	1,000 (RMEG ^e)	No
Ethylbenzene	0.011 - 12.2	3	5,000 (RMEG)	No
Methylene Chloride	0.016 - 0.028	0.02	3,000 (EMEG ^f)	No
n-Propylbenzene	0.028	0.028	NA ^g	Yes
Styrene	0.06	0.06	10,000 (RMEG)	No
Tetrachloroethene	0.005	0.005	1.2 (RBC ^h)	No
Toluene	0.011 - 3.9	1	10,000 (RMEG)	No
1,1,1-Trichloroethane	0.01	0.01	210 (RDCSCC ⁱ)	No
Trichloroethene	0.004 - 0.012	0.01	1.6 (RBC)	No
Vinyl chloride	0.01	0.01	1 (CREG)	No
Xylenes	23 - 104	46.5	3,000 (RMEG)	No
Polycyclic Aromatic Hydrocarbons (PAHs) and Polychlorinated Biphenyls (PCBs)				
Acenaphthene	1.8 - 110	39.3	3,000 (RMEG)	No
Acenaphthylene	0.77 - 220	50.9	NA	Yes
Anthracene	0.2 - 310	69.8	20,000 (RMEG)	No
Benzo[a]anthracene	0.42 - 390	96.5	0.87 (RBC)	Yes
Benzo[a]pyrene	22 - 140	77.2	0.1 (CREG)	Yes
Benzo[b]fluoranthene	0.75 - 90	48.9	0.87 (RBC)	Yes
Benzo[g,h,i]perylene	1.1 - 15	8.05	NA	Yes
Benzo[k]fluoranthene	1.5 - 65	33.1	0.9 (RDCSCC)	Yes
Chrysene	0.47 - 410	89.4	9 (RDCSCC)	Yes
Dibenz[a,h]anthracene	5.9	5.9	0.087 (RBC)	Yes
Fluoranthene	3.9 - 460	148.7	2,000 (RMEG)	No
Fluorene	0.73 - 560	129.6	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.57- 25	12.8	0.87 (RBC)	Yes
Naphthalene	0.2 - 960	201.7	1,000 (RMEG)	No
Phenanthrene	0.71 - 1620	311	NA	Yes
Pyrene	0.58 - 640	156	2,000 (RMEG)	No
PCBs	307	307	0.4 (CREG)	Yes
Metals				
Arsenic	1.8 - 24	9.22	0.5 (CREG)	Yes
Chromium ^j	23 - 104	46.5	200 (RMEG)	No
Copper	20 - 730	206	3,100 (RBC)	No
Lead	64 - 1,400	428	400 (RDCSCC)	Yes

Table 1: (Cont'd)

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Mercury	0.34 - 1.1	0.7	14 (RDCSCC)	No
Nickel	31 - 72	51.5	1,000 (RMEG)	No
Selenium	1.2 - 3.2	2.15	300 (EMEG)	No
Silver	1.4 - 1.8	1.60	300 (RMEG)	No
Zinc	0.46 - 380	144.9	20,000 (EMEG)	No
Others				
Cyanide	0.93 - 17	8.17	1,000 (RMEG)	No
Phenol	0.13 - 1.7	0.66	20,000 (RMEG)	No

^amilligrams of contaminant per kilogram of soil; ^bComparison Value; ^cContaminant of Concern; ^dATSDR Cancer Risk Evaluation Guide for chronic exposure; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^gNot available; ^hEPA Region 3 Risk-Based Concentration; ⁱNJDEP Residential Direct Contact Soil Cleanup Criteria; ^jBased on Chromium (VI)

Table 2: Surface Soil (0-2 ft depth) Sampling Results of old Jerry Morgan Park

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Volatile Organic Compounds (VOCs)				
Benzene	0.7	0.7	10 (CREG ^d)	No
Chloroform	0.005	0.005	500 (EMEG ^e)	No
Ethylbenzene	0.17	0.17	5,000 (RMEG ^f)	No
Methylene Chloride	0.01 - 0.4	0.2	3,000 (EMEG)	No
Toluene	0.1	0.1	10,000 (RMEG)	No
Trichloroethene	0.1	0.1	1.6 (RBC ^g)	No
Vinyl chloride	1.9	1.9	1 (CREG)	Yes
Xylenes	1.7	1.7	3,000 (RMEG)	No
Polycyclic Aromatic Hydrocarbons (PAHs)				
Acenaphthylene	0.6 - 12	3.9	NA ^h	Yes
Anthracene	0.8 - 1.1	0.9	20,000 (RMEG)	No
Benzo[a]anthracene	0.4 - 6.6	3	0.87 (RBC)	Yes
Benzo[a]pyrene	0.74 - 4.9	2.9	0.1 (CREG)	Yes
Benzo[b]fluoranthene	1.4 - 9.6	6.45	0.87 (RBC)	Yes
Benzo[g,h,i]perylene	0.5 - 3	1.9	NA	Yes
Benzo[k]fluoranthene	1.4 - 9.3	6.4	0.9 (RDCSCC ⁱ)	Yes
Chrysene	0.9 - 8.6	4.5	9 (RDCSCC)	No
Dibenz[a,h]anthracene	0.1 - 0.8	0.5	0.087 (RBC)	Yes
Fluoranthene	0.8 - 9.8	4.37	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.4 - 2.3	1.5	0.87 (RBC)	Yes
Naphthalene	47 - 180	113.5	1,000 (RMEG)	No
Phenanthrene	1.3 - 16	4.9	NA	Yes
Pyrene	1 - 15	6.7	2,000 (RMEG)	No
Metals				
Arsenic	5.5 - 37	15.1	0.5 (CREG)	Yes
Chromium, Total	38 - 100	65.2	200 (RMEG)	No
Chromium (VI)	2-10	5.3	200 (RMEG)	No
Copper	21 - 152	71.2	3,100 (RBC)	No
Lead	19 - 280	113.3	400 (RDCSCC)	No
Mercury	0.3 - 1.8	0.9	14 (RDCSCC)	No
Nickel	34	34	1,000 (RMEG)	No
Selenium	3.2 - 3.8	3.5	300 (EMEG)	No
Zinc	0 - 550	550	20,000 (EMEG)	No
Others				
Cyanide	1.5 - 11	5.4	1,000 (RMEG)	No
Phenol	0.18 - 1.1	0.4	20,000 (RMEG)	No

^amilligrams of contaminant per kilogram of soil; ^bComparison Value; ^cContaminant of Concern; ^dATSDR Cancer Risk Evaluation Guide for chronic exposure; ^eATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^fATSDR Reference Media Evaluation Guide for chronic exposure for child; ^gEPA Region 3 Risk-Based Concentration; ^hNot available; ⁱNJDEP Residential Direct Contact Soil Cleanup Criteria

Table 3: Sediment Sampling Results of Troutman's Creek^a (on-site portion)

Contaminant	Concentration (mg/kg ^b)	Environmental Guideline CV ^c (mg/kg)	COC ^d
Volatile Organic Compounds (VOCs)			
Benzene	0.07	10 (CREG ^e)	No
Ethylbenzene	0.03	5,000 (RMEG ^f)	No
n-Propylbenzene	0.06	NA ^g	Yes
Styrene	0.04	10,000 (RMEG)	No
Xylenes	0.09	3,000 (RMEG)	No
Polycyclic Aromatic Hydrocarbons (PAHs)			
Acenaphthene	0.2	3,000 (RMEG)	No
Acenaphthylene	0.35	NA	Yes
Anthracene	0.22	20,000 (RMEG)	No
Benzo[a]anthracene	0.44	0.87 (RBC ^h)	No
Benzo[a]pyrene	0.48	0.1 (CREG)	Yes
Benzo[b]fluoranthene	0.35	0.87 (RBC)	No
Benzo[k]fluoranthene	0.32	0.9 (RDCSCC ⁱ)	No
Chrysene	0.56	9 (RDCSCC)	No
Fluoranthene	0.65	2,000 (RMEG)	No
Phenanthrene	0.6	NA	Yes
Pyrene	1.1	2,000 (RMEG)	No
Metals			
Arsenic	47	0.5 (CREG)	Yes
Selenium	0.6	300 (EMEG ^j)	No
Others			
Cyanide	0.5	1,000 (RMEG)	No

^aOne sample was collected from the on-site portion of the Troutman's Creek; ^b milligrams of contaminant per kilogram of soil; ^c Comparison Value; ^d Contaminant of Concern; ^e ATSDR Cancer Risk Evaluation Guide for chronic exposure; ^f ATSDR Reference Media Evaluation Guide for chronic exposure for child; ^g Not available; ^h EPA Region 3 Risk-Based Concentration; ⁱ NJDEP Residential Direct Contact Soil Cleanup Criteria; ^j ATSDR Environmental Media Reference Guide for chronic exposure for child

Table 4: Surface Soil (0-2 ft depth) Sampling Results of the Seaview Manor Public Housing Complex Property

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Volatile Organic Compounds (VOCs)				
Acetone	0.03	NA	50,000 (RMEG ^d)	No
Benzene	32	NA	10 (CREG ^e)	Yes
Butylbenzylphthalate	0.04 - 0.06	NA	10,000 (RMEG)	No
Carbon Disulfide	0.007	NA	5,000 (RMEG)	No
Dibenzofuran	0.05 - 2.7	NA	160 (RBC ^f)	No
Diethylphthalate	0.09 - 0.36	NA	40,000 (RMEG)	No
Dimethylphthalate	0.36	NA	10,000 (RDCSCC ^g)	No
Di-n-butylphthalate	0.05 - 0.09	NA	5,000 (RMEG)	No
Di-n-octylphthalate	0.04 - 0.36	NA	1,100 (RDCSCC)	No
Ethylbenzene	0.1 - 5.2	NA	5,000 (RMEG)	No
Methylene Chloride	0.002 - 6.4	NA	3,000 (EMEG ^h)	No
Toluene	0.008	NA	10,000 (RMEG)	No
Xylenes	0.05 - 20	NA	3,000 (RMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
bis(2-Ethylhexyl)phthalate	0.05 - 4.6	1.1	46 (RBC)	No
Polycyclic Aromatic Hydrocarbons (PAHs)				
Acenaphthene	0.04 - 5.8	NA	3,000 (RMEG)	No
Acenaphthylene	0.04 - 16	NA	NA ⁱ	Yes
Anthracene	0.04 - 7.2	NA	20,000 (RMEG)	No
Benzo[a]anthracene	0.04 - 29	NA	0.87 (RBC)	Yes
Benzo[a]pyrene	0.04 - 13	NA	0.1 (CREG)	Yes
Benzo[b]fluoranthene	0.05 - 34	NA	0.87 (RBC)	Yes
Benzo[g,h,i]perylene	0.04 - 2.8	NA	NA	Yes
Benzo[k]fluoranthene	0.04 - 12	NA	0.9 (RDCSCC)	Yes
Carbazole	0.04 - 0.05	NA	32 (RBC)	No
Chrysene	0.04 - 29	NA	9 (RDCSCC)	Yes
Dibenz[a,h]anthracene	0.05 - 0.28	NA	0.087 (RBC)	Yes
Fluoranthene	0.04 - 39	NA	2,000 (RMEG)	No
Fluorene	0.043 - 9.2	NA	2,000 (RMEG)	No
Indeno(1,2,3-cd)pyrene	0.04 - 3.4	NA	0.87 (RBC)	Yes
Naphthalene	0.04 - 59	NA	1,000 (RMEG)	No
2-Methylnaphthalene	0.05 - 33	NA	1,600 (RBC)	No
Phenanthrene	0.04 - 36	NA	NA	Yes
Pyrene	0.02 - 80	NA	2,000 (RMEG)	No

Table 4: (Cont'd.)

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Metals				
Antimony	0.2 - 17	1.3	20 (RMEG)	No
Arsenic	0.4 - 49	9.3	0.5 (CREG)	Yes
Beryllium	0.1 - 1.58	NA	100 (EMEG)	No
Cadmium	0.05 - 49.8	1.5	10 (EMEG)	Yes
Chromium	2.9 - 131	NA	200 (RMEG)	No
Copper	0.6 - 1,430	NA	3,100 (RBC)	No
Lead	3 - 1,170	120	400 (RDCSCC)	Yes
Mercury	0.04 - 4.68	NA	14 (RDCSCC)	No
Nickel	0.4 - 31.5	NA	1,000 (RMEG)	No
Selenium	0.2 - 4.53	NA	300 (EMEG)	No
Silver	0.06 - 1.68	NA	300 (RMEG)	No
Thallium	0.5 - 1.6	NA	2 (RDCSCC)	No
Zinc	8.2 - 8,250	300	20,000 (EMEG)	No
Other				
Cyanide	1.8	NA	1,000 (RMEG)	No
Phenol	13.3 - 15.5	NA	20,000 (RMEG)	No

^a milligrams of contaminant per kilogram of soil; ^b Comparison Value; ^c Contaminant of Concern; ^d ATSDR Reference Media Evaluation Guide for chronic exposure for child; ^e ATSDR Cancer Risk Evaluation Guide for chronic exposure; ^f EPA Region 3 Risk-Based Concentration; ^g NJDEP Residential Direct Contact Soil Cleanup Criteria; ^h ATSDR Environmental Media Evaluation Guide for chronic exposure for child; ⁱ Not available

Table 5: Surface Soil (0-2 ft depth) Sampling Results of the Southern Site Boundary

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COC ^c
	Range	Mean		
Polycyclic Aromatic Hydrocarbons				
Benzo[a]anthracene	0.9 - 44	5.86	0.87 (RBC ^d)	Yes
Benzo[a]pyrene	0.6 - 31	3.94	0.1 (CREG ^e)	Yes
Benzo[b]fluoranthene	0.9 - 32	5.46	0.87 (RBC)	Yes
Benzo[k]fluoranthene	1 - 17	2.87	0.9 (RDCSCC ^f)	Yes
Chrysene	9.1 - 54	22.78	9 (RDCSCC)	Yes
Dibenz[a,h]anthracene	0.72 - 1.9	1.12	0.087 (RBC)	Yes
Fluoranthene	1.3 - 6.3	3.93	2,000 (RMEG ^g)	No
Indeno[1,2,3-cd]pyrene	0.91 - 14	3.07	0.87 (RBC)	Yes
Metals				
Arsenic	21.7 - 86.9	46.58	0.5 (CREG)	Yes
Beryllium	2.2 - 38	9.62	100 (EMEG ^h)	No
Lead	454 - 654	554	400 (RDCSCC)	Yes

^a milligrams of contaminant per kilogram of soil; ^b Comparison Value; ^c Contaminant of Concern;

^d EPA Region 3 Risk-Based Concentration; ^e ATSDR Cancer Risk Evaluation Guide for chronic exposure;

^f NJDEP Residential Direct Contact Soil Cleanup Criteria; ^g ATSDR Reference Media Evaluation Guide for chronic exposure for child; ^h ATSDR Environmental Media Evaluation Guide for chronic exposure for child

Table 6: On-site Sub-Surface Soil (>2 ft) Sampling^a Results of the Long Branch Manufactured Gas Plant site (Number of sample = 13) (1999)

Contaminants	No. of Detections	Concentration (mg/kg ^b)		Environmental Guideline Comparison Value (mg/kg)	COC ^c
		Minimum	Maximum		
Volatile Organic Compounds (VOCs)					
Benzene	7	0.02	68	10 (CREG ^d)	Yes
Ethylbenzene	7	0.018	24	5,000 (RMEG ^e)	No
1,1,-Dichloroethane	3	0.008	4.7	1,000 (RDCSCC ^f)	No
1,2-Dichloroethane (trans)	3	0.003	0.06	1,000 (RMEG)	No
n-Propylbenzene	1	0.02	0.02	NA ^g	Yes
Styrene	1	0.12	0.12	10,000 (RMEG)	No
Tetrachloroethene	1	0.004	0.004	1.2 (RBC ^h)	No
Toluene	6	0.04	12	10,000 (RMEG)	No
1,1,1-Trichloroethane	2	0.007	0.2	210 (RDCSCC)	No
Trichlorofluoromethane	2	0.005	0.05	23,000 (RBC)	No
Vinyl Chloride	2	0.005	0.15	1 (CREG)	No
Xylene (Total)	7	0.036	300	3,000 (RMEG)	No
Semi Volatile Organic Compounds (SVOCs)					
Acenaphthylene	2	3.2	3.4	NA	Yes
Acenaphthene	2	1.2	1.3	3,000 (RMEG)	No
Anthracene	4	0.12	3.2	20,000 (RMEG)	No
Benzo[a]anthracene	4	0.37	5.5	0.87 (RBC)	Yes
Benzo[b]fluoranthene	4	0.29	2.6	0.87 (RBC)	Yes
Benzo[k]fluoranthene	3	0.51	2.7	0.9 (RDCSCC)	Yes
Benzo[a]pyrene	3	0.71	2.9	0.1 (CREG)	Yes
Benzo[g,h,i]perylene	1	1.2	1.2	NA	Yes
Chrysene	4	0.34	6.2	9 (RDCSCC)	No

Table 6: (Cont'd.)

Contaminants	No. of Detections	Concentration (mg/kg ^b)			Environmental Guideline Comparison Value (mg/kg)	COC ^c
		Minimum	Maximum	Mean		
Semi Volatile Organic Compounds (SVOCs)						
Dibenz[a,h]anthracene	1	0.6	0.6	0.6	0.087 (RBC)	Yes
Fluoranthene	5	0.55	7	2.5	2,000 (RMEG)	No
Fluorene	3	0.47	6.2	2.85	2,000 (RMEG)	No
Indeno[1,2,3-cd]pyrene	1	1.3	1.3	1.3	0.87 (RBC)	Yes
Naphthalene	3	6.4	1,100	372.4	1,000 (RMEG)	Yes
Phenanthrene	5	0.46	11	3.06	NA	Yes
Pyrene	5	0.52	10.5	3.5	2,000 (RMEG)	No
Metals						
Arsenic	10	0.95	120	20.8	0.5 (CREG)	Yes
Cadmium	1	1.7	1.7	1.7	10 (EMEG ^d)	No
Chromium	10	9.5	110	47.3	200 (RMEG)	No
Copper	9	10	295	92.6	3,100 (RBC)	No
Lead	7	8.3	530	188	400 (RDCSCC)	Yes
Mercury	7	0.08	1.8	0.5	14 (RDCSCC)	No
Nickel	5	18	68	38.4	1,000 (RMEG)	No
Selenium	10	0.4	14	4.28	300 (EMEG)	No
Silver	3	1.6	2.2	1.8	300 (RMEG)	No
Zinc	10	8.5	200	59.2	20,000 (EMEG)	No
Others						
Cyanide	6	1.3	190	34.5	1,000 (RMEG)	No
Phenols	10	0.06	1.4	0.47	20,000 (RMEG)	No

^a number of samples = 11; ^b milligrams of contaminant per kilogram of soil; ^c Contaminant of Concern; ^d ATSDR Cancer Risk Evaluation Guide for chronic exposure; ^e ATSDR Reference Media Evaluation Guide for chronic exposure for child; ^f NJDEP Residential Direct Contact Soil Cleanup Criteria; ^g Not available; ^h EPA Region 3 Risk-Based Concentration; ⁱ ATSDR Environmental Media Evaluation Guide for chronic exposure for child

Table 7: On-site Surface Water Sampling^a Results of the Long Brnach Manufactured Gas Plant site (1984, 1985)

Contaminant	Concentration (µg/L ^b)			Environmental Guideline Comparison Value (µg/L)	COC ^c
	No. of Detection	Minimum	Maximum		
Volatile Organic Compounds (VOCs)					
Benzene	1	4	4	0.6 (CREG ^d)	Yes
Chloroform	1	1	1	100 (EMEG ^e)	No
Methylene Chloride	2	38	64	600 (EMEG)	No
1,1,2,2-Tetrachloroethane	1	1	1	400 (EMEG)	No
Toluene	1	7	7	2,000 (RMEG ^f)	No
Trichloroethene	2	2	3	1 (NJMCL ^g)	Yes
Xylenes (Total)	1	4	4	2,000 (RMEG)	No
Polycyclic Aromatic Compounds (PAHs)					
Acenaphthene	1	10	10	600 (RMEG)	No
Fluoranthene	1	10	10	400 (RMEG)	No
Naphthalene	3	12	110	200 (RMEG)	No
Phenanthrene	1	29	29	NA ^h	No
Pyrene	1	16	16	300 (RMEG)	No
Metals					
Mercury	1	3.8	3.8	2 (NJMCL)	Yes
Nickel	1	130	130	200 (RMEG)	No
Selenium	2	16	46	50 (EMEG)	No
Zinc	3	20	510	3,000 (EMEG)	No

^anumber of samples = 3; ^bmicrograms of contaminant per liter of water; ^cContaminant of Concern; ^dATSDR Cancer Risk Evaluation Guide for chronic exposure;

^eATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^fATSDR Reference Media Evaluation Guide for chronic exposure for child;

^gNew Jersey Maximum Contaminant Level; ^hNot available

Table 8: On-site Groundwater Sampling^a Results of the Long Branch Manufactured Gas Plant site (1984, 1985)

Contaminant	Concentration (µg/L ^b)			Environmental Guideline Comparison Value (µg/L)	COC ^c
	No. of Detection	Minimum	Maximum		
Volatile Organic Compounds (VOCs)					
Benzene	8	6	31,000	9,184	2 (NJMCL ^d) Yes
Ethylbenzene	9	1	1,800	602	1,000 (RMEG ^e) Yes
Methylene Chloride	2	2	10	6	600 (EMEG ^f) No
Toluene	7	3	24,000	6,176	2,000 (RMEG) Yes
Xylenes (Total)	6	3	11,950	3,833	2,000 (EMEG) Yes
Semivolatile Organic Compounds (SVOCs)					
Bis(2-ethylhexyl)phthalate	2	20	78	49	4.8 (RBC ^g) Yes
Polycyclic Aromatic Compounds (PAHs)					
Acenaphthylene	6	10	2,500	592	NA ^h Yes
Acenaphthene	6	30	1,500	361.6	600 (RMEG) Yes
Anthracene	3	10	2,500	970	3,000 (RMEG) No
Benzo[a]anthracene	1	2,400	2,400	2,400	0.092 (RBC) Yes
Benzo[a]pyrene	1	1,300	1,300	1,300	0.005 (CREG ⁱ) Yes
Benzo[b]fluoranthene	1	1,600	1,600	1,600	0.092 (RBC) Yes
Benzo[k]fluoranthene	1	1,600	1,600	1,600	0.92 (RBC) Yes
Chrysene	1	2,100	2,100	2,100	9.2 (RBC) Yes
Fluorene	5	20	900	295.4	240 (RBC) Yes
Fluoranthene	3	140	4,800	1,813	400 (RMEG) Yes
Naphthalene	11	10	54,000	10,820	200 (RMEG) Yes
Phenanthrene	6	20	18,000	3,464	NA Yes
Pyrene	4	10	7,200	2,032	300 (RMEG) Yes

Table 8: (Cont'd.)

Contaminant	Concentration ($\mu\text{g/L}^b$)			Environmental Guideline Comparison Value ($\mu\text{g/L}$)	COC ^c
	No. of Detection	Minimum	Maximum		
Metals					
Arsenic	5	2	8	3 (EMEG)	Yes
Mercury	1	0.6	0.6	2 (NJMCL)	No
Nickel	4	58	100	200 (RMEG)	No
Selenium	1	2	2	50 (EMEG)	No
Zinc	14	18	600	3,000 (EMEG)	No

^anumber of samples = 11; ^bmicrograms of contaminant per liter of water; ^cContaminant of Concern; ^dNew Jersey Maximum Contaminant Level; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^gEPA Region 3 Risk-Based Concentration; ^hNot available; ⁱATSDR Cancer Risk Evaluation Guide for chronic exposure;

Table 9: Ambient^a, Crawlspace^b and Indoor^c Air sampling Results of the Seaview Manor Public Housing Complex

Contaminant	Concentration ($\mu\text{g}/\text{m}^3$) ^d						Environmental Guideline CVs ^e ($\mu\text{g}/\text{m}^3$)	COC ^f
	Ambient		Crawlspace		Indoor Air			
	Range	Mean	Range	Mean	Range	Mean		
Benzene	0.8 – 13.6	6.8	0.45 – 11.1	1.55	0.86 – 4.5	1.38	0.1 (CREG ^g)	Yes
Ethylbenzene	0.1 – 1	0.5	0.6 – 3.9	1.42	0.43 – 1	0.65	1,000 (RfC ^h)	No
Toluene	0.9 – 11.7	5.52	1 – 8.4	2.84	2.1 – 22	4.01	300 (EMEG ⁱ)	No
Xylene	0.1 – 2.2	1.12	0.48 – 13	2.82	0.65 – 2.8	1.33	434 (EMEG)	No

^aNumber of ambient air sample= 4; ^bNumber of crawlspace sample = 21; ^cNumber of indoor air sample = 20; ^dmicrogram of contaminant per cubic meter of air; ^eComparison Value; ^fContaminant of Concern; ^gATSDR Cancer Risk Evaluation Center for chronic exposure; ^hUSEPA Reference Concentration; ⁱATSDR Environmental Media Evaluation Guide

Table 10: Crawlspace and Indoor Air sampling Results of the Check Mate and Second Baptist Church Day Care Center

Contaminant	Concentration ($\mu\text{g}/\text{m}^3$) ^a						Environmental Guideline CVs ^e ($\mu\text{g}/\text{m}^3$)	COC ^f
	Check Mate Day Care Center			Second Baptist Church Day Care Center				
	Crawlspace ^b		Main Floor ^c	Basement ^d		Mean		
Benzene	0.54	0.54	0.8 – 0.93	0.86	0.57 – 0.61	0.58	0.1 (CREG ^g)	Yes
Ethylbenzene	1.2	1.2	1.8 – 2.3	2.05	2.1 – 3.6	2.63	1,000 (RfC ^h)	No
Toluene	ND ⁱ	ND	ND – 0.43	0.43	ND	ND	300 (EMEG ^j)	No
Xylene	0.65	0.65	1 – 1.2	1.1	ND - 1	1.0	434 (EMEG)	No

^amicrogram of contaminant per cubic meter of air; ^bNumber of samples = 1; ^cNumber of samples = 2; ^dNumber of samples = 3; ^eComparison Value; ^fContaminant of Concern; ^gATSDR Cancer Risk Evaluation Center; ^hUSEPA Reference Concentration; ⁱNot detected; ^jATSDR Environmental Media Evaluation Guide

Table 11: Major Exposure Pathways for the LBMGP Site

Environmental Pathway	Exposure Point	Exposure Scenario(s)	Route of Exposure	Receptor	Location	Pathway Status		
						Past	Present	Future
Soil	Soil	Direct Contact	Ingestion /Dermal	Resident	Seaview Manor Public Housing Complex Property	Completed	Eliminated ¹	Eliminated
					Old Jerry Morgan Park	Completed	Eliminated	Eliminated
					Processing Area	Completed	Eliminated	Eliminated
Surface Water	Incidental Ingestion /Biota	Recreation	Ingestion /Dermal	Resident	Southern Site Boundary	Completed	Eliminated	Eliminated
					Troutman's Creek ²	Completed	Eliminated	Eliminated
Groundwater	Indoor Air	Vapor Intrusion	Inhalation	Resident	Troutman's Creek ²	Completed	Potential	Potential
					Residences	Potential	Eliminated	Eliminated
Air	Indoor/ Ambient Air	Inhalation	Inhalation	Resident	LBMGP and residences	Potential	Not Applicable	Not Applicable

¹One or more exposure pathway elements were removed, ²Sediment exposures; both on- and off-site

Table 12: Comparison of Surface Soil Exposure Dose of the Processing Area with the Health Guideline CVs

Contaminants of Concern	Maximum (mg/kg)	Mean (mg/kg)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Volatile Organic Compounds (VOCs)						
n-Propylbenzene	0.028	0.028	8.57 x 10 ⁻⁸	1.29 x 10 ⁻⁸	NA ^c	NA
Polycyclic Aromatic Hydrocarbons (PAHs) and Polychlorinated Biphenyls (PCBs)						
Acenaphthylene	220	50.9	6.73 x 10 ⁻⁴	1.01 x 10 ⁻⁴	NA	No
Benzo[a]anthracene	390	96.5	1.19 x 10 ⁻³	1.79 x 10 ⁻⁴	NA	No
Benzo[a]pyrene	140	77.2	4.29 x 10 ⁻⁴	6.43 x 10 ⁻⁵	NA	No
Benzo[b]fluoranthene	90	48.9	2.76 x 10 ⁻⁴	4.13 x 10 ⁻⁵	NA	No
Benzo[g,h,i]perylene	15	8.05	4.59 x 10 ⁻⁵	6.89 x 10 ⁻⁵	NA	No
Benzo[k]fluoranthene	65	33.1	1.99 x 10 ⁻⁴	2.98 x 10 ⁻⁵	NA	No
Chrysene	410	89.4	1.26 x 10 ⁻³	1.88 x 10 ⁻⁴	NA	No
Dibenz[a,h]anthracene	5.9	5.9	1.81 x 10 ⁻⁵	2.71 x 10 ⁻⁶	NA	No
Indeno[1,2,3-cd]pyrene	25	12.8	7.65 x 10 ⁻⁵	1.15 x 10 ⁻⁵	NA	No
Phenanthrene	1,620	311	4.96 x 10 ⁻³	7.44 x 10 ⁻⁴	NA	No
PCBs	307	307	9.4 x 10 ⁻⁴	1.4 x 10 ⁻⁴	7 x 10 ^{-5d} (RfD ^e) 2 x 10 ^{-5f} (RfD)	Yes
Metals						
Arsenic	24	9.2	7.35 x 10 ⁻⁵	1.10 x 10 ⁻⁵	3 x 10 ⁻⁴ (MRL ^g)	No
Lead	1,400	428	NA	NA	NA	Yes

^aChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate and 70 kg body weight; ^cNot Available; ^dBased on Aroclor 1016; ^eEPA Reference Dose; ^fBased on Aroclor 1254; ^gATSDR Minimal Risk Level

Table 13: Comparison of Soil Exposure Dose of the Old Jerry Morgan Park with the Health Guideline CVs

Contaminants of Concern	Maximum (mg/kg)	Mean (mg/kg)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Volatile Organic Compounds (VOCs)						
Vinyl chloride	1.9	1.9	5.82 x10 ⁻⁶	8.72 x10 ⁻⁷	2 x10 ⁻⁵ (MRL ^c)	No
Polycyclic Aromatic Hydrocarbons (PAHs)						
Acenaphthylene	12	3.9	3.67E-05	5.51E-06	NA ^d	No
Benzo[a]anthracene	6.6	3	2.02E-05	3.03E-06	NA	No
Benzo[a]pyrene	4.9	2.9	1.50E-05	2.25E-06	NA	No
Benzo[b]fluoranthene	9.6	6.45	2.94E-05	4.41E-06	NA	No
Benzo[g,h,i]perylene	3	1.9	9.18E-06	1.38E-06	NA	No
Benzo[k]fluoranthene	9.3	6.4	2.85E-05	4.27E-06	NA	No
Dibenz[a,h]anthracene	0.8	0.5	2.45E-06	3.67E-07	NA	No
Indeno[1,2,3-cd]pyrene	2.3	1.5	7.04E-06	1.06E-06	NA	No
Phenanthrene	16	4.9	4.90E-05	7.35E-06	NA	No
Metals						
Arsenic	37	15.16	1.13 x10 ⁻⁴	1.70 x10 ⁻⁵	3 x10 ⁻⁴ (MRL)	No

^aChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate and 70 kg body weight; ^cATSDR Minimal Risk Level; ^dNot available

Table 14: Comparison of Sediment Exposure Dose of the Troutman’s Creek with the Health Guideline CVs

Contaminants of Concern	Maximum (mg/kg)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
		Child ^a	Adult ^b		
Volatile Organic Compounds (VOCs)					
n-Propylbenzene	0.06	1.84 x10 ⁻⁶	2.75 x10 ⁻⁸	NA ^c	NA
Polycyclic Aromatic Hydrocarbons (PAHs)					
Acenaphthylene	0.35	1.07 x10 ⁻⁷	1.61 x10 ⁻⁷	NA	No
Benzo[a]pyrene	0.48	1.47 x10 ⁻⁶	2.20 x10 ⁻⁷	NA	No
Phenanthrene	0.6	1.84 x10 ⁻⁶	2.76 x10 ⁻⁷	NA	No
Metals					
Arsenic	47	1.44 x10 ⁻⁴	2.16 x10 ⁻⁵	3 x 10 ⁻⁴ (MRL ^d)	No

^aChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate and 70 kg body weight; ^cNot Available; ^dATSDR Minimal Risk Level

Table 15: Comparison of Surface Soil Exposure Dose of the Seaview Manor Public Housing Property with the Health Guideline CVs

Contaminants of Concern	Maximum (mg/kg)	Mean (mg/kg)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Volatile Organic Compounds (VOCs)						
Benzene	32	1.1	3.05 x10 ⁻⁴	4.57 x10 ⁻⁵	4 x10 ⁻³ (RfD ^c)	No
Polycyclic Aromatic Hydrocarbons (PAHs)						
Acenaphthylene	16	NA ^d	1.52 x10 ⁻⁴	2.29 x10 ⁻⁵	NA	No
Benzo[a]anthracene	29	NA	2.76 x10 ⁻⁴	4.14 x10 ⁻⁵	NA	No
Benzo[a]pyrene	13	NA	1.24 x10 ⁻⁴	1.86 x10 ⁻⁵	NA	No
Benzo[b]fluoranthene	34	NA	3.24 x10 ⁻⁴	4.86 x10 ⁻⁵	NA	No
Benzo[g,h,i]perylene	2.8	NA	2.67 x10 ⁻⁵	4.00 x10 ⁻⁵	NA	No
Benzo[k]fluoranthene	12	NA	1.14 x10 ⁻⁴	1.71 x10 ⁻⁵	NA	No
Chrysene	29	NA	2.76 x10 ⁻⁴	4.14 x10 ⁻⁵	NA	No
Dibenz[a,h]anthracene	0.28	NA	2.67 x10 ⁻⁶	4.00 x10 ⁻⁷	NA	No
Indeno(1,2,3-cd)pyrene	3.4	NA	3.24 x10 ⁻⁵	4.86 x10 ⁻⁵	NA	No
Phenanthrene	36	NA	3.43 x10 ⁻⁴	5.14 x10 ⁻⁵	NA	No
Metals						
Arsenic	49.1	9.3	4.68 x10 ⁻⁴ (8.86 x10 ⁻⁵) ^e	1.33 x10 ⁻⁵	3 x10 ⁻⁴ (MRL ^f)	Yes
Cadmium	49.8	1.5	4.74 x10 ⁻⁴ (1.43 x10 ⁻⁵)	7.11 x10 ⁻⁵	2 x10 ⁻⁴ (MRL)	Yes
Lead	1,170	120	NA	NA	NA	Yes

^aChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate and 70 kg body weight; ^cEPA Reference Dose; ^dNot available; ^eExposure Dose in parentheses is based on Mean Concentration; ^fATSDR Minimal Risk Level

Table 16: Comparison of Surface Soil Exposure Dose of the Southern Site Boundary with the Health Guideline CVs

Contaminants of Concern	Maximum (mg/kg)	Mean (mg/kg)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Polycyclic Aromatic Hydrocarbons (PAHs)						
Benzo[a]anthracene	44	5.86	1.35 x 10 ⁻⁴	2.02 x 10 ⁻⁵	NA ^c	No
Benzo[a]pyrene	31	3.94	9.49 x 10 ⁻⁵	1.42 x 10 ⁻⁵	NA	No
Benzo[b]fluoranthene	32	5.46	9.80 x 10 ⁻⁵	1.47 x 10 ⁻⁵	NA	No
Benzo[k]fluoranthene	17	2.87	5.20 x 10 ⁻⁵	7.81 x 10 ⁻⁶	NA	No
Chrysene	54	22.78	1.65 x 10 ⁻⁴	2.48 x 10 ⁻⁵	NA	No
Dibenz[a,h]anthracene	1.9	1.12	5.82 x 10 ⁻⁶	8.72 x 10 ⁻⁷	NA	No
Indeno(1,2,3-cd)pyrene	14	3.07	4.29 x 10 ⁻⁵	6.43 x 10 ⁻⁶	NA	No
Metals						
Arsenic	86.9	46.58	2.66 x 10 ⁻⁴	3.99 x 10 ⁻⁵	3 x 10 ⁻⁴ (MRL ^d)	No
Lead	654	554	NA	NA	NA	Yes

^aChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate and 70 kg body weight; ^cNot Available; ^dATSDR Minimal Risk Level

Table 17: Comparison of Exposure Dose resulting from ingestion of Surface water from the Troutman’s Creek with the Health Guideline CVs

Contaminants of Concern	Maximum (µg/L)	Mean (µg/L)	Maximum Exposure Dose (mg/kg/day)		Health Guideline CVs (mg/kg/day)	Potential for Non-cancer Health Effects
			Child ^a	Adult ^b		
Volatile Organic Compounds (VOCs)						
Benzene	4	4	1.0 x10 ⁻⁶	3.0 x10 ⁻⁷	0.004 (RfD ^c)	No
Trichloroethene	3	2.5	7.6 x10 ⁻⁷	2.29 x10 ⁻⁷	0.0003 (RfD ^d)	No
Metal						
Mercury	3.8	3.8	9.6 x10 ⁻⁷	2.9 x10 ⁻⁷	0.0003 (RfD)	No

^aChild exposure scenario: 1 days/week, 9 month/year, 0.05 L/day ingestion rate and 21 kg body weight; ^bAdult exposure scenario: 1 days/week, 9 month/year, 0.05 L/day ingestion rate and 70 kg body weight; ^cEPA Reference Dose; ^dEPA Region 3 Reference Dose Oral

Table 18: Calculated LECR associated with the Contaminants detected in surface soil at the Processing Area

Contaminants of Concern	Maximum 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)								
Acenaphthylene	220	3	- ^e	-				
Benzo[a]anthracene	390	2	0.1	39				
Benzo[a]pyrene	140	2	1	140				
Benzo b]fluoranthene	90	2	0.1	9				
Benzo[k]fluoranthene	65	2	0.1	6.5				
Benzo[g,h,i]perylene	15	3	-	-	230.6	1.06 x10 ⁻⁴	7.3	7.33 x10 ⁻⁴ (4.25 x10 ⁻⁴) ^f
Chrysene	410	2	0.01	4.1				
Dibenz[a,h]anthracene	5.9	2	5	29.5				
Indeno[1,2,3-cd]pyrene	25	2	0.1	2.5				
Naphthalene	960	3	-	-				
Phenanthrene	1,620	3	-	-				
PCBs	307	2	-	-	-	1.41 x10 ⁻⁴	2	2.82 x10 ⁻⁴

Table 18: (Cont'd)

Contaminants of Concern	Maximum 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
Volatile Organic Compounds (VOCs)								
n-Propylbenzene	0.028	NA ^g	NA	NA	NA	NA	NA	NA
Metals								
Arsenic	24	1	NA	NA	NA	1.1 x 10 ⁻⁵	1.5	1.65 x 10 ⁻⁵ (6.35 x 10 ⁻⁶)
Lead	1,400	2 ^g	NA	NA	NA	6.41 x 10 ⁻⁴	NA	NA
							Sum =	1.07 x 10⁻³ (7.13 x 10⁻⁴)

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

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 70 year exposure duration; ^dLifetime Excess Cancer Risk; ^eNot Applicable; ^fBased on mean contaminant concentration; ^gCancer Slope Factor for lead is unavailable

Table 19: Calculated LECR associated with the Contaminants detected in surface soil at the old Jerry Morgan Park

Contaminants of Concern	Maximum Conc. (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose (mg/kg/day)		CSF (mg/kg/d) ⁻¹	LECR ^e	
						Child ^c	Adult ^d		Child	Adult
Polycyclic Aromatic Compounds (PAHs)										
Acenaphthylene	12	3	- ^f	-	11.68	5.11 x10 ⁻⁶	1.38 x10 ⁻⁶	7.3	3.73 x10 ⁻⁵	1.01 x10 ⁻⁵ (6.09 x10 ⁻⁶) ^g
Benzo[a]anthracene	6.6	2	0.1	0.66						
Benzo[a]pyrene	4.9	2	1	4.9						
Benzo[b]fluoranthene	9.6	2	0.1	0.96						
Benzo[k]fluoranthene	9.3	2	0.1	0.93						
Benzo[g,h,i]perylene	3	3	-	-						
Dibenz[a,h]anthracene	0.8	2	5	4						
Indeno[1,2,3-cd]pyrene	2.3	2	0.1	0.23						
Phenanthrene	16	3	-	-						
Volatile Organic Compounds (VOCs)										
Vinyl chloride	1.9	1	-	-	-	8.31 x10 ⁻⁷	2.24 x10 ⁻⁷	1.4	1.16 x10 ⁻⁶	3.14 x10 ⁻⁷

Table 19: (Cont'd)

Contaminants of Concern	Maximum Conc. (mg/kg)	DHHS ^a Cancer Class	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Exposure Dose (mg/kg/day)		CSF (mg/kg/d) ⁻¹	LECR ^e	
						Child ^c	Adult ^d		Child	Adult
Metals										
Arsenic	37	1	-	-	-	1.62 x10 ⁻⁵	4.37 x10 ⁻⁶	1.5	2.43 x10 ⁻⁵	6.55 x10 ⁻⁶ (2.69 x10 ⁻⁶)
								Sum =	6.27 x10 ⁻⁵ (3.37 x10 ⁻⁵)	1.69 x10 ⁻⁵ (9.09 x10 ⁻⁶)

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

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cChild exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate, 210 kg body weight and 10 year exposure duration; ^dAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 70 year exposure duration; ^eLifetime Excess Cancer Risk; ^f- Not Applicable; ^gBased on mean contaminant concentration

Table 20: Calculated LECR associated with the contaminants detected in sediment at the Troutman's Creek

Contaminants of Concern	Maximum 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)								
Acenaphthylene	0.35	3	- ^e	-				
Benzo[a]pyrene	0.48	2	1	0.48	0.48	2.20 x10 ⁻⁷	7.3	1.61 x10 ⁻⁶
Phenanthrene	0.59	3	-	-				
Metals								
Arsenic	47	1	-	-	-	2.15 x10 ⁻⁵	1.5	3.24 x10 ⁻⁵
							Sum =	3.39 x10⁻⁵

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

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 70 year exposure duration; ^dLifetime Excess Cancer Risk; ^e - Not Applicable

Table 21: Calculated LECR associated with the contaminants detected in surface soil at the Seaview Manor Public Housing Property

Contaminants of Concern	Maximum Conc. (mg/kg)	DHHS ¹ 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)								
Acenaphthylene	16	3	- ^e	-				
Benzo[a]anthracene	29	2	0.1	2.9				
Benzo[a]pyrene	13	2	1	13				
Benzo[b]fluoranthene	34	2	0.1	3.4				
Benzo[k]fluoranthene	12	2	0.1	1.2	21.13	2.16 x10 ⁻⁵	7.3	1.57 x10 ⁻⁴ (7.04 x10 ⁻⁶) ^f
Benzo[g,h,i]perylene	2.8	3	-	-				
Chrysene	29	2	0.01	0.29				
Indeno[1,2,3-cd]pyrene	3.4	2	0.1	0.34				
Phenanthrene	36	3	NA ^g	NA				
Volatile Organic Compounds (VOCs)								
Benzene	32	1	-	-	-	3.27 x10 ⁻⁵	0.055	1.8 x10 ⁻⁶ (6.17 x10 ⁻⁸)
Metals								
Arsenic	49.1	1	-	-	-	5.01 x10 ⁻⁵	1.5	7.52 x10 ⁻⁵ (1.42 x10 ⁻⁵)

Table 21: (Cont'd.)

Contaminants of Concern	Maximum 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
Metals								
Cadmium	49.8	1	-	-	-	5.08 x10 ⁻⁵	NA	NA
Lead	1,170	2 ^g	-	-	-	1.19 x10 ⁻³	NA	NA
							Sum =	2.34 x10⁻⁴ (2.13 x10⁻⁵)

^aDepartment of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified
^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 70 year exposure duration; ^dLifetime Excess Cancer Risk; ^eNot Applicable; ^fBased on mean contaminant concentration; ^gCancer Slope Factor for lead is unavailable

Table 22: Calculated LECR associated with the Contaminants detected in surface soil along the Southern Site Boundary

Contaminants of Concern	Maximum 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	44	2	0.1	4.4	51.74	2.37 x10 ⁻⁵	7.3	1.73 x10 ⁻⁴ (3.86 x10 ⁻⁵) ^e
Benzo[a]pyrene	31	2	1	31				
Benzo[b]fluoranthene	32	2	0.1	3.2				
Benzo[k]fluoranthene	17	2	0.1	1.7				
Chrysene	54	2	0.01	0.54				
Dibenz[a,h]anthracene	1.9	2	5	9.5				
Dibenz[a]anthracene	1.5	NA ^f	NA	NA				
Indeno[1,2,3-cd]pyrene	14	2	0.1	1.4				

Table 22: (Cont'd)

Contaminants of Concern	Maximum 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
Metals								
Arsenic	86.9	1	- ^g	-	-	3.99 x10 ⁻⁵	1.5	5.99 x10 ⁻⁵ (3.21 x10 ⁻⁵)
Lead	654	2 ^h	-	-	-	3.0 x10 ⁻⁴	NA	NA
							Sum =	2.33 x10⁻⁴ (7.08 x10⁻⁵)

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

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^cAdult exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 70 year exposure duration; ^dLifetime Excess Cancer Risk; ^eBased on mean contaminant concentration; ^fNot Available; ^g-Not Applicable; ^hCancer Slope Factor for lead is unavailable

Table 23: Calculated LECR based on Maximum and Mean Concentration of contaminants detected in the Surface Water

Contaminants of Concern	Maximum (µg/L)	DHHSA Cancer Class	Exposure Dose ^b (mg/kg/day)		CSF ^c (mg/kg/day) ⁻¹	LECR ^d	
			Child	Adult		Child	Adult
Volatile Organic Compounds (VOCs)							
Benzene	4	1	1.75 x10 ⁻⁷	9.62 x10 ⁻⁸	0.055	9.62 x10 ⁻⁹	5.29 x10 ⁻⁹
Trichloroethene	3	2	1.31 x10 ⁻⁷	7.21 x10 ⁻⁸	0.4	5.24 x10 ⁻⁸	2.88 x10 ⁻⁸
Metals							
Mercury	3.8	3	1.66 x10 ⁻⁷	9.13 x10 ⁻⁸	NA ^e	NA	NA
Sum =						6.2 x10⁻⁸ (5.33 x10⁻⁸)^f	3.41 x10⁻⁸ (2.93 x10⁻⁸)

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

^bChild exposure scenario: exposure based on 0.05 L/day ingestion rate and 12 year exposure duration; Adult exposure scenario: exposure based on 0.05 L/day ingestion rate and 22 year exposure duration; ^cCancer Slope Factor; ^dLifetime Excess Cancer Risk; ^eNot available; ^fBased on mean contaminant concentration

Table 24: Calculated LECR based on Maximum and Mean Concentration of Benzene detected in the Indoor Air

Contaminants of Concern	Indoor Air Concentration		DHHS ^a Cancer Class	Exposure Dose ^b		CSFi ^c (mg/kg/day) ⁻¹	LECR ^d	
	Maximum (µg/m ³)	Mean (µg/m ³)		Maximum (mg/kg/day)	Mean (mg/kg/day)		Max.	Mean
Benzene	4.5	1.38	1	9.18 x 10 ⁻⁴	2.18 x 10 ⁻⁴	0.027	2.48 x 10 ⁻⁵	7.6 x 10 ⁻⁶

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

^bAdult exposure scenario: residential exposure based on 50 year exposure duration; ^cEPA Region 3 Cancer Slope Factor (inhalation); ^dLifetime Excess Cancer Risk

Table 25: Multiple Chemical Exposure Analysis for Child: Soil, Sediment and Indoor Air

Contaminant	Child Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	Hazard Index	NOAEL (mg/kg/day)	Dose/NOAEL
Soil/Sediment						
Arsenic	1.42 x10 ⁻⁴	3 x10 ⁻⁴	0.47	48	0.009	0.01
Benzene	1.048 x10 ⁻⁹	4 x10 ⁻³	0		1.2	0
Lead ^a	2.2	10	0.22		NA ^b	NA
PCBs	9.4 x10 ⁻⁴	2 x10 ⁻⁵	47		0.005	0.18
n-Propylbenzene	1.8 x10 ⁻⁶	0.037	0		110	0
Vinyl Chloride	5.82 x10 ⁻⁶	2 x10 ⁻⁵	0.29		0.09	0
Indoor Air						
Contaminant	Exposure Concentration (µg/m ³)	Health Guideline CV (µg/m ³)	Hazard Quotient	Hazard Index	NOAEL (µg/m ³)	Dose/NOAEL
Benzene	1.38	30	0.04	0.05		
Ethylbenzene	0.65	1,000	0			
Toluene	4.01	5,000	0			
Xylene	1.33	100	0.01			

^aBased on blood lead level in µg/dL; ^bNot available



Photograph 1: Troutman's Creek during high tide



Photograph 2: Troutman's Creek during low tide



Photograph 3: Rechannelization of the on-site portion of the Troutman's Creek



Photograph 4: Atlantic Plumbing and Supply building located near the southeast corner of the Long Branch Manufactured Gas Plant site



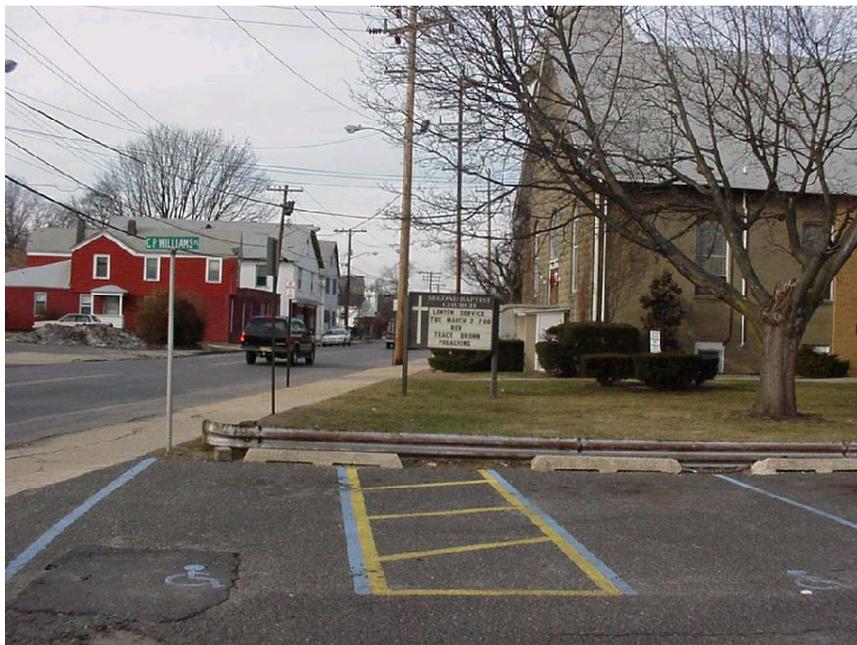
Photograph 5: Seaview Manor public housing complex located to the north of the Long Branch Manufactured Gas Plant site



Photograph 6: Grant Court public housing complex located to the west of the Long Branch Manufactured Gas Plant site



Photograph 7: Check Mate Day Care Center located to the west of the Long Branch Manufactured Gas Plant site



Photograph 8: Second Baptist Church Day Care Center located to the west of the Long Branch Manufactured Gas Plant site

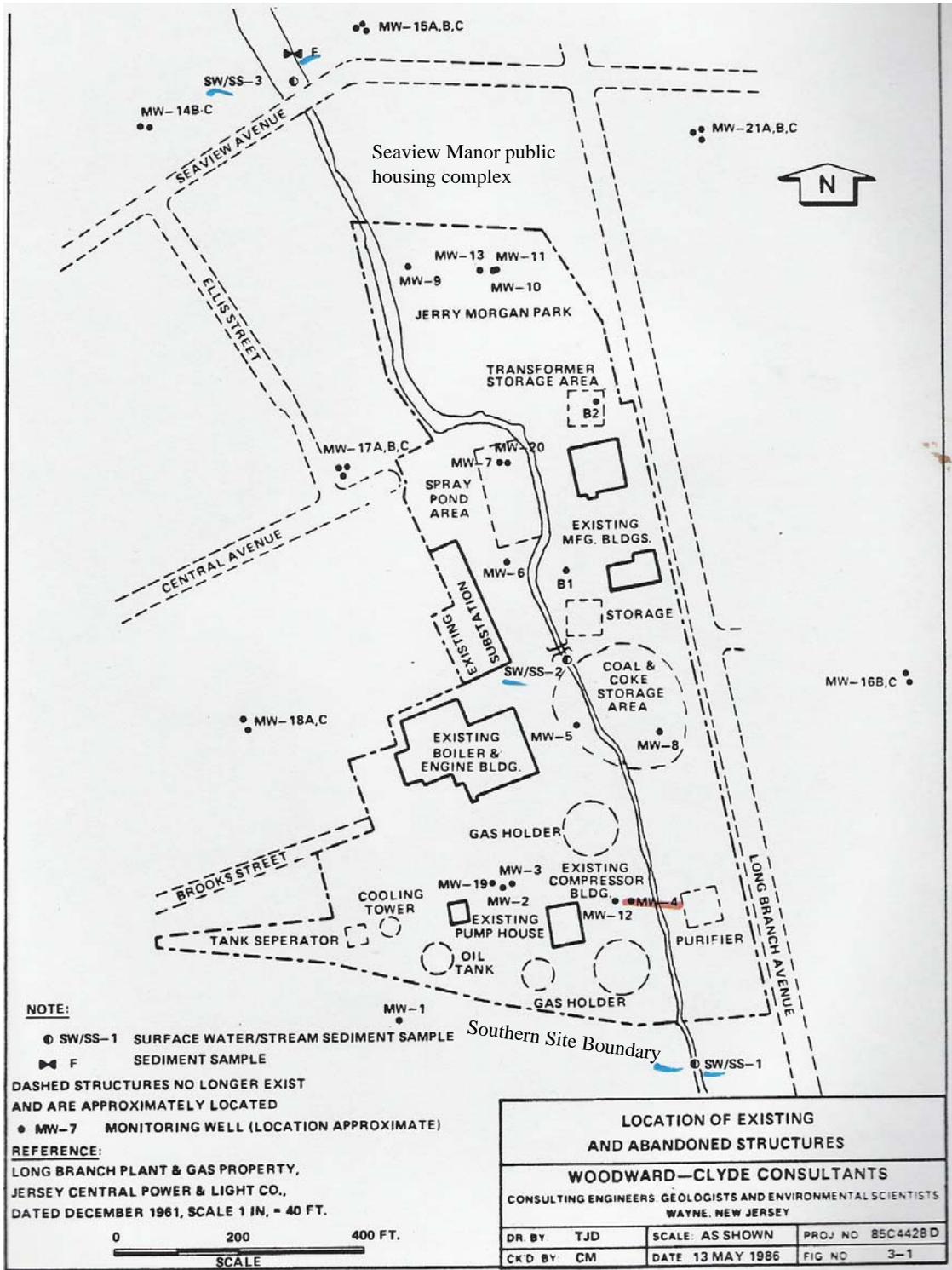


Figure 3: Map of the former Long Branch Manufactured Gas Plant site

(Note: Figure does not represent current conditions)

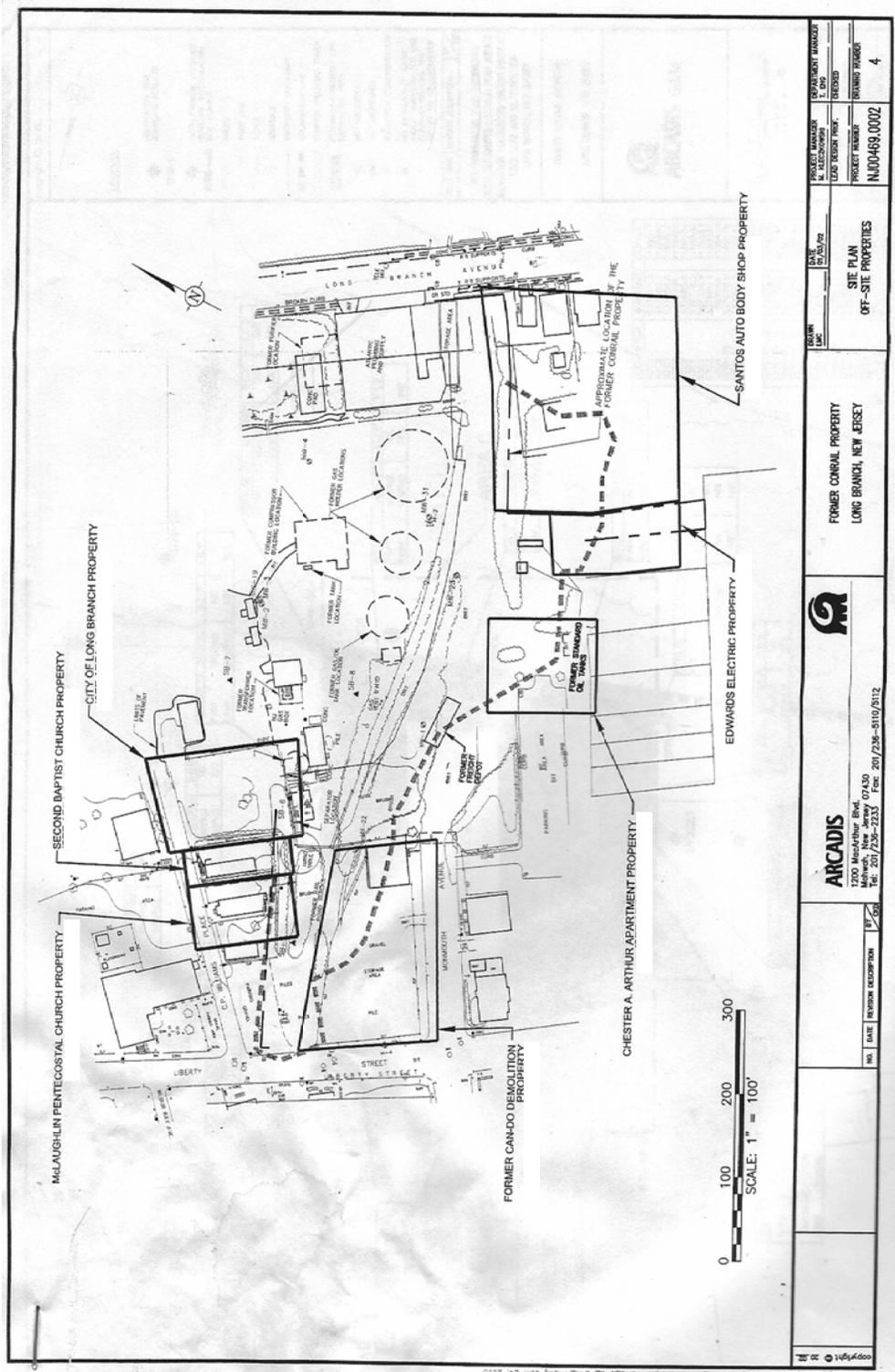


Figure 4: Residential areas near the Southern Site Boundary

Long Branch Manufactured Gas Plant Site

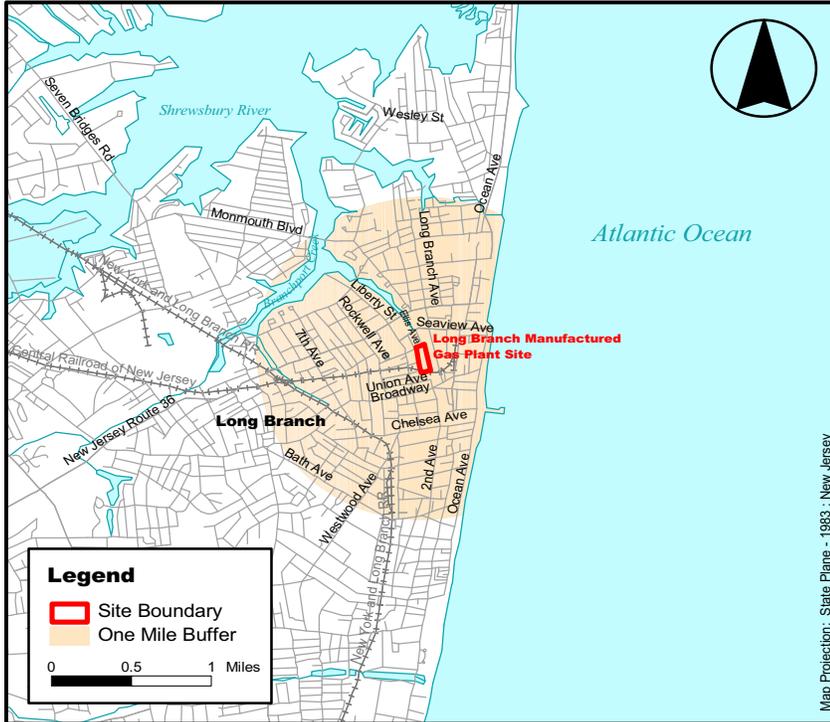
INTRO MAP

Long Branch, New Jersey
EPA Facility ID NJD980530471



Site Location

Monmouth County, New Jersey

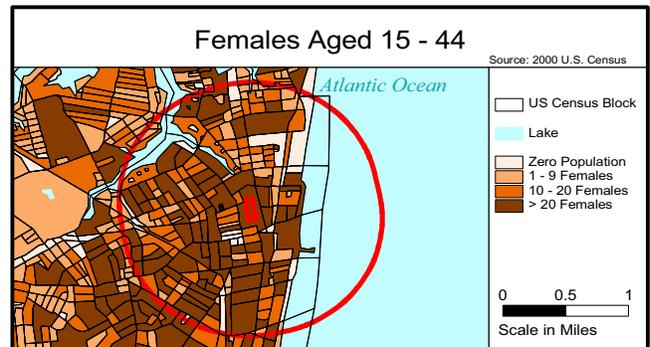
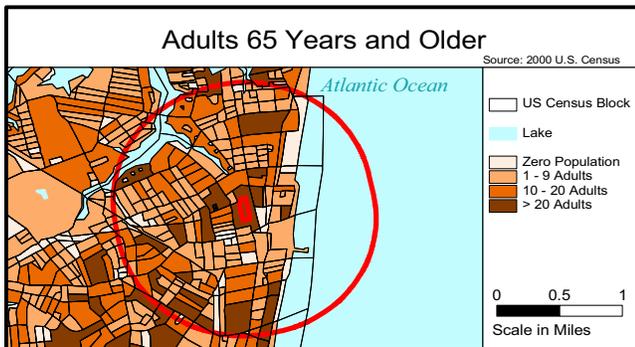
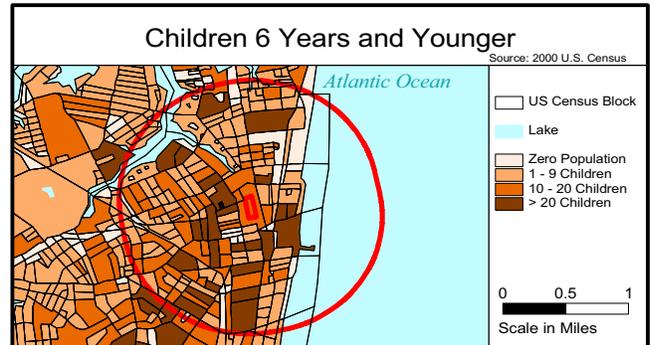
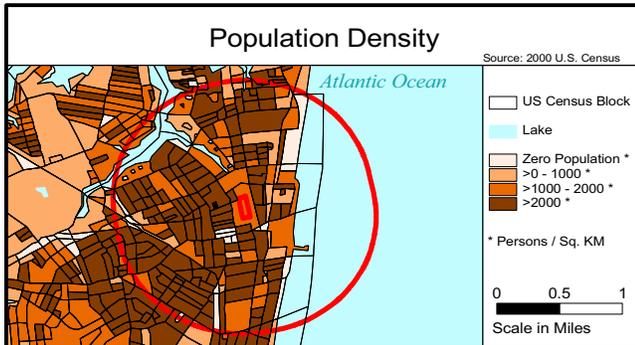


Map Projection: Slate Plane - 1983 - New Jersey

Base Map Source: 1995 TIGER/Line Files

Demographic Statistics Within One Mile of Site*	
Total Population	18402
White alone	10277
Black alone	5075
Am. Indian and Alaska Native alone	83
Asian alone	276
Native Hawaiian and Other Pacific Islander alone	6
Some other race alone	1751
Two or More races	930
Hispanic or Latino	5065
Children Aged 6 and Younger	1955
Adults Aged 65 and Older	2018
Females Aged 15 - 44	4325
Total Housing Units	7365

Demographic Statistics Source: 2000 US Census
 *Calculated using an area-proportion spatial analysis technique



JVA052104



Figure 5: Demographic information of Long Branch Manufactured Gas Plant site based on 2000 U.S. Census

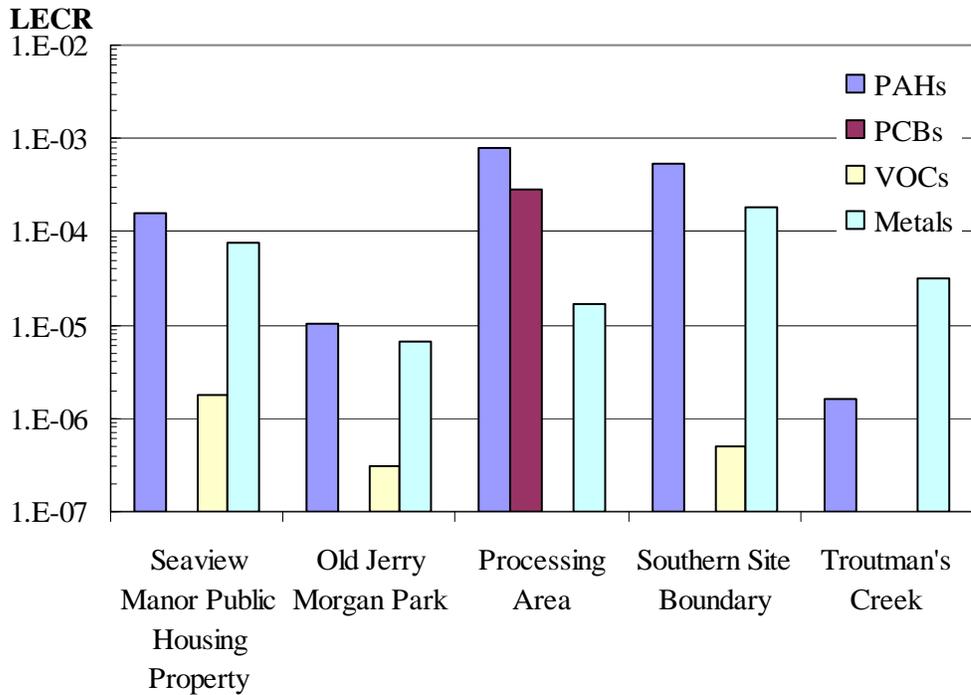


Figure 6: LECR associated with PAHs, VOCs and metals based on Maximum Concentration of Contaminants

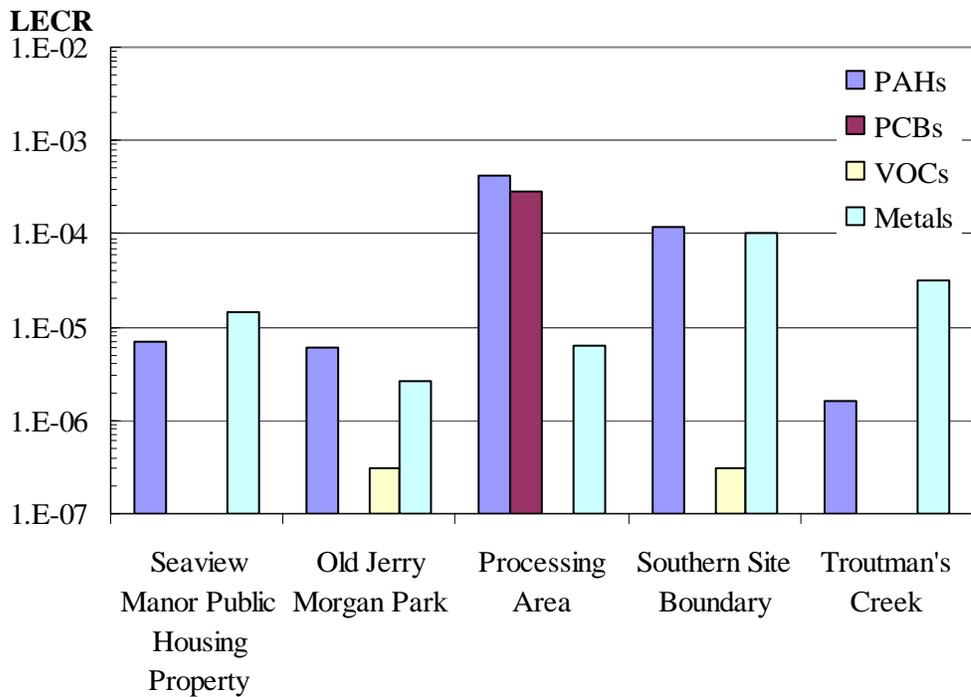


Figure 7: LECR associated with PAHs, VOCs and metals based on Mean Concentration of Contaminants

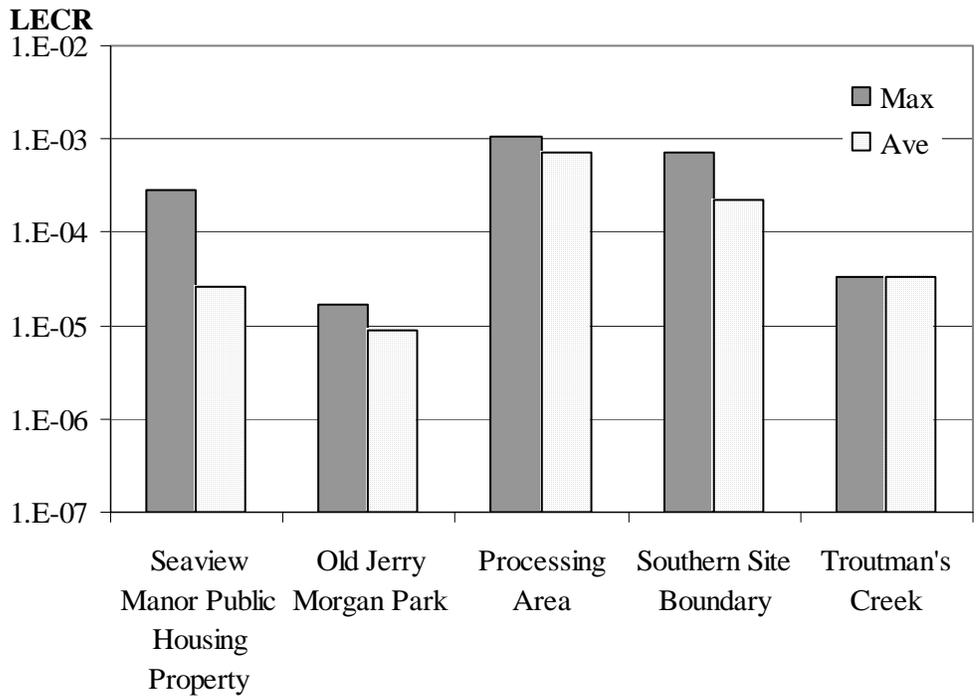


Figure 8: Cumulative LECR based on Maximum and Mean Concentration of Contaminants (includes risk from inhalation exposures to Seaview Manor residents)

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.

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

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

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

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

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

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

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.

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.

Trichloroethylene Trichloroethylene is a nonflammable, colorless liquid with a somewhat sweet odor and a sweet, burning taste. It is used mainly as a solvent to remove grease from metal parts, but it is also an ingredient in adhesives, paint removers, typewriter correction fluids, and spot removers. TCE is slightly soluble in water, and can remain in groundwater for a long time, but it quickly evaporates from surface water, so it is commonly found as a vapor in the air. People can be exposed to TCE by breathing air in and around the home which has been contaminated with TCE vapors from shower water or household products or vapor intrusion, or by drinking, swimming, or showering in water that has been contaminated with TCE.

Breathing small amounts of TCE may cause headaches, lung irritation, dizziness, poor coordination, and difficulty concentrating. Breathing large amounts of TCE may cause impaired heart function, unconsciousness, and death. Breathing it for long periods may cause nerve, kidney, and liver damage. Drinking large amounts of TCE may cause nausea, liver damage, unconsciousness, impaired heart function, or death. Drinking small amounts of TCE for long periods may cause liver and kidney damage, impaired immune system function, and impaired fetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with TCE for short periods may cause skin rashes.

Some studies with mice and rats have suggested that high levels of TCE may cause liver, kidney, or lung cancer. Some studies of people exposed over long periods to high levels of TCE in drinking water or in workplace air have found evidence of increased cancer. The National Toxicology Program has determined that TCE is “reasonably anticipated to be a human carcinogen,” and the International Agency for Research on Cancer (IARC) has determined that TCE is “probably carcinogenic to humans”.

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

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

The US Department of Health and Human Services (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.

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

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

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.

Animal studies have shown that long-term exposure to vinyl chloride can damage the sperm and testes.

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

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.

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

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

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

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 International Agency for Research on Cancer (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.

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/m³) can be highly irritating to the eyes of humans; the threshold for eye irritation has been reported to be 200 ppm (879 mg/m³). 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/m³) 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.

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

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

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

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

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

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

Appendix B

Summary of Public Comments and Responses

Summary of Public Comments and Responses Long Branch Manufactured Gas Plant Site Public Health Assessment

The NJDHSS held a public comment period from April 15, 2007 through June 30, 2007 to provide an opportunity for interested parties to comment on the draft Public Health Assessment prepared for the Long Branch Manufactured Gas Plant 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 considered, 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 stated that the presentation of data in the PHA was inconsistent, confusing, and hard to follow. The tables need footnotes to indicate that numbers in parentheses are based on means, while others are based on maximum concentrations. In addition, the total number of samples and the number of detects should be indicated by compound, in the data tables.

Response: The presentation of the contamination data followed the format of other PHA reports published earlier by the NJDHSS/ATSDR.

In the text and tables, the PHA stated that numbers in parentheses are based on means, while others are based on maximum concentrations.

Generally, the total number of samples and the number of detections are provided in the tables. For some environmental media, the information was unavailable.

Comment #2: The commenter indicated that based on the history of the site, 70 years of exposure in the Processing Area, the Southern Site Boundary (which includes the Conrail area), and Troutman's Creek is highly unlikely.

Response: The exposure duration is the length of time a population has been exposed to site-related contaminants. For the LBMGP site, the contaminant exposure duration was estimated from site history and the information provided by the area residents during availability sessions.

If the site-specific exposure duration information were unavailable, the NJDHSS/ATSDR use 30 years for exposure dose calculation.

Comment #3: *The commenter indicated that the PAH and arsenic exposure dose calculation should include an absorption factor.*

Response: Since the PAH and arsenic desorption characteristics from diet and soil are assumed to be similar between experimental and natural conditions, additional bioavailability factors were not employed to calculate exposure dose.

Comment #4: *The commenter indicated that the PCB concentration used to calculate the exposure dose was based on one sample and it is inappropriate to use just one PCB value as the basis for exposure and risk calculations. Post-excavation soil samples (n=25) collected were all non-detect for PCBs.*

Response: Although previous report (NUS 1990) indicated the existence of a transformer storage area located to the south of the old Jerry Morgan Park, PCB sampling was not conducted. Subsequently, PCBs were detected at a concentration of 307 mg/kg (ARCADIS 2004).

Limited information on PCBs was made available for review. Efforts to obtain levels and the extent of on-site PCB contamination were unsuccessful. As such, the reported value was used for risk calculation.

Post excavation concentration results cannot be used to assess past exposures.

Comment #5: *The commenter recommended that a discussion on acceptable risk levels be added to the PHA; it would be helpful to the reader and add perspective to the analysis.*

Response: Acceptable risk levels and lifetime risk of being diagnosed with cancer in the United States were discussed in the “Cancer Health Effects” section of the PHA report.

Comment #6: *The commenter noted that PAHs are found throughout the environment; exposures to PAHs may occur daily due to their presence in a variety of media such as food, water, air, cigarette smoke, and consumer products (e.g., shampoo). Dietary intake levels of arsenic for children and adults in the US were mentioned. The commenter suggested that exposures to PAHs and arsenic from these sources be evaluated in the LBPHA report.*

Response: Exposure doses of all site-related COCs were calculated and their public health implications were discussed in the PHA. The ATSDR public health assessment process does not investigate/assess health risks associated with lifestyle factors (such as diet, smoking, radon, solar radiation) of residents who were exposed to contaminants. The NJDHSS/ATSDR calculates lifetime *excess* cancer risks regardless of the factors which contribute to the background rate.

However, if the source of the contaminant has not been identified, NJDHSS and ATSDR will indicate that in the report.

Comment #7: *The commenter argued that the PHA found no link between cancer incidence rates in Long Branch and any contaminants at the site. The PHA included an*

epidemiological survey of cancer incidence (January 1, 1979 through December 31, 2000) in Long Branch and in Census Tract 8056, the area of Long Branch with the highest potential for exposure. These findings support the conclusion that past exposures at the site are unlikely to have posed a public health hazard.

Response: While site-specific environmental contamination data is required for conducting health assessments, health outcome data analysis is also considered an important component in the health assessment process. The results of a health outcome evaluation may contribute to a greater understanding of the public health implications associated with contaminant exposures. Although the health outcome data analysis is considered as one of the tools to evaluate potential exposure outcomes, inconclusive and/or negative results do not necessarily indicate no public health hazard.

While the community raised a number of health concerns they believe associated with exposures to site-related contaminants, few health outcomes beyond cancer are reportable. Therefore, the PHA reviewed cancer data to supplement the contaminant exposure data analysis. One of the major limitations to conduct such studies is the difficulty in identifying the actual exposed population. To try to minimize this uncertainty, the population living in the census tract where the facility is located was analyzed separately. And while using the census tract as the definition of exposed, it also likely overestimates the true population at risk (i.e., those actually exposed to the contaminants). This overestimate would result in misclassifying some of the population as exposed, when they are not. The consequence of this type of misclassification is to bias the health outcome analysis toward either being inconclusive or not finding an association.

Evaluation of contaminant exposure data provides a plausible basis for the potential of adverse health affects. Therefore, it is not possible to conclude, as the commenter has, “that past exposures at the site are unlikely to have posed a public health hazard” based on the cancer findings alone. As such, for the LBMGP site, the evaluation of site-related exposures and the public health implications of these exposures are what drove the past hazard designation.

Comment #8: *The commenter indicated that the IEUBK model run did not take into account the 9 months/year exposure in the PHA.*

Response: The Technical Review Workgroup (TRW) has recommended that the IEUBK model be applied to exposures that exceed a minimum frequency of one day per week and duration of three consecutive months (USEPA 1994). Three months is considered to be the minimum exposure to produce a quasi-steady-state blood lead concentration.

Reference

[USEPA] United States Environmental Protection Agency. 1994. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. Office of Emergency and Remedial Response. Washington, D.C., U.S. EPA 540/R-93/081, PB93-963510.

Comment #9: The commenter noted that the PHA IEUBK results appear to be based on outdated default dietary intake factors.

Response: The reviewer is correct. The model results were revised using current dietary intake factors. However, it should be noted that the revised BLLs were within 10% of previous values.

Comment #10: The commenter questioned the use of 0 – 2 feet depth contaminant concentration to evaluate soil exposures.

Response: The PHA used samples collected at 0-2 feet depth as surface samples (NJDEP 2005) for exposure assessment. The soil samples collected from on-site areas during remedial investigation were from 0-2 feet depth; 0-6 inches depth samples were not collected.

Reference

[NJDEP] New Jersey Department of Environmental Protection. 2005. Field Sampling Procedure Manual, August, 2005.

Comment #11: The commenter indicated that USEPA TSCA lead regulations¹ establish the following lead standards for bare residential soil: a hazard standard of 400 ppm (by weight) in play areas based on the play area bare soil sample, and an average of 1,200 ppm in bare soil in the remainder of the yard based on an average of all other samples collected (USEPA, 2001). The commenter recommended that language to this effect be added to the PHA.

Response: The reviewer is correct about the TSCA regulatory requirements. However, residents, during availability sessions, stated that (1) the children and adults routinely accessed the on-site areas, (2) the old Jerry Morgan Park was the primary recreation area for the area residents including the Seaview Manor public housing complex, (3) family picnics were held on the on-site portion of Troutman's Creek and (4) residential areas were located along the Southern Site Boundary (see Figure 4).

Due to long historical recreational use (more than 100 years) of the on-site areas, it is impossible to demarcate whether the contaminated areas were used as play areas or whether the soil was bare. As such, it was assumed that residents including children were exposed to lead contaminated on-site soils/sediments.

Comment #12: The commenter noted that the PHA uses potency factors to evaluate the carcinogenicity of various PAH compounds, relative to BaP. However, although a 1993 USEPA guidance document is cited on p28 of the PHA text, the USEPA-recommended potency factors are not used in the PHA tables (Tables 18-22). The PHA should

¹USEPA TSCA lead regulations apply to housing receiving federal assistance, US Department of Housing and Urban Development (HUD) grants to perform lead hazard control, requirements for lead cleanup under state authorities, etc. (USEPA, 2001).

consistently use currently recommended USEPA potency factors for PAHs in evaluating PAH cancer risks.

Response: The reviewer is correct. The correct reference is provided in the final PHA.

Comment #13: *The commenter noted that the oral bioavailability of soil-bound PAHs is significantly less than the 100% assumed in the Long Branch PHA (e.g., Alexander, 2000; Bordelon et al., 2000; Brainard and Beck, 1992; Goon et al., 1990; Goon et al., 1991; Koganti et al., 1998; Koganli et al., 2001; Reeves et al., 2001; Shor et al., 2004; Stroo et al., 2005; Tang et al., 2006; Van de Wiele et al., 2004; Weyand et al., 1996). Failure to account for the reduced bioavailability of chemicals in soil in human health risk assessments result in an exaggeration of actual risks. It was recommend that ATSDR incorporate an oral bioavailability factor into their exposure and risk calculations in the Long Branch PHA.*

Response: The calculated PAH exposure doses were based on the levels detected in the soil; no bioavailability factor was incorporated. The bioavailability represents the total amount of a substance ingested, inhaled or contacted that actually enters the bloodstream and is available to possibly harm a person.

The reference dose (RfD) reported for acenaphthene (USEPA 1989a), anthracene (USEPA 1989a), fluoranthene (USEPA 1988), fluorine (USEPA 1989b), naphthalene (BCL 1980) and pyrene (USEPA 1989c) are based on animal studies. Animals were gavaged with PAHs and toxicological parameters were monitored. No effort was made to measure the dose/concentration of PAHs at the affected organs. The oral Reference Dose (RfD) was calculated by dividing the body weight normalized PAH daily dose by an uncertainty factor. The bioavailability is implicitly included in the toxicological outcomes which are empirically determined by the bioassay. In addition, since desorption characteristics of PAHs from diet and soil are assumed to be similar, bioavailability factors were not employed to calculate exposure dose.

References

- USEPA. 1989a. Mouse oral subchronic study with acenaphthene. Study conducted by Hazelton Laboratories, Inc., for the Office of Solid Waste, Washington, DC.
- USEPA. 1988. 13-Week mouse oral subchronic toxicity study. Prepared by Toxicity Research Laboratories, Ltd., Muskegon, MI for the Office of Solid Waste, Washington, DC.
- USEPA. 1989b. Mouse oral subchronic toxicity study. Prepared by Toxicity Research Laboratories, LTD., Muskegon, MI for the Office of Solid Waste, Washington, DC.
- [BCL] Battelle's Columbus Laboratories. (1980) Unpublished subchronic toxicity study: Naphthalene (C52904), Fischer 344 rats. Prepared by Battelle Laboratories under NTP Subcontract No. 76-34-106002.
- USEPA. 1989c. Mouse Oral Subchronic Toxicity of Pyrene. Study conducted by Toxicity Research Laboratories, Muskegon, MI for the Office of Solid Waste, Washington, DC.

Comment #14: The commenter noted that the PHA misstated the health effects of PCBs. It was recommended that ATSDR add the levels at which health effects occur.

Response: The information was taken from the ATSDR (2008) website. The recommendation/suggestion was forwarded to the appropriate ATSDR staff.

References

ATSDR Agency for Toxic Substances and Disease Registry. 2008. Accessed on Jan. 23, 2008, at: <http://www.atsdr.cdc.gov/tfacts17.html>.

Comment #15: The commenter inquired about the date that should be associated with the statement “Currently, there are no completed exposure pathways”.

Response: It is the date when all on-site remedial actions were completed.

Comment #16: The commenter inquired about engineering/institutional controls that were implemented at the site to reach the conclusion that there are no present exposure pathways.

Response: The interim and preferred remedial actions implemented for the on-site areas including the selection of cleanup goals, removal of contaminated soil, placement of the cap and deed restrictions were reviewed and summarized in the ‘Overview of Remedial Actions’ section of the PHA. The preferred remedy is designed to address future exposures and is based on various criteria including short and long-term effectiveness and reliability.

Comment #17: The commenter inquired if there have been any changes in the control systems since the time of the determination of no completed pathways.

Response: The on-going monitoring of the long-term effectiveness of the preferred remedy (that was implemented at the site) is the responsibility of the NJDEP.

Comment #18: It was unclear to the commenter whether the expected future discussion about off-site contaminants will contain the same level of detail as this draft Assessment. The commenter asked for a more thorough description of what the “separate health consultation” will contain.

Response: As indicated in the PHA, remedial investigation of off-site areas is being conducted by the NJNG. The past, current and future risks associated with any potential site-related contamination will be evaluated in a separate health consultation with the same level of detail as the in this PHA.

The PHA report identified the levels and extent of all on-site (i.e., Processing Area, old Jerry Morgan Park, on-site portion of the Troutman’s Creek, former Seaview Manor Public Housing complex and southern site boundary) contaminated media.

Comment #19: The commenter questioned the accuracy of information used to develop the PHA document. Because of long history (1880s to 1990s) and extensive remedial work, the commenter was concerned that the PHA may be based on outdated and incomplete information.

Response: The PHA was forwarded to the NJDEP for review and comments. The comments were addressed before the document was released for public comment.

During the public health assessment process, the NJDHSS conducted a comprehensive review of all relevant information including site background, community health concerns, environmental contamination, exposure pathways and remedial actions implemented.

Comment #20: The commenter expressed concern about the future exposures to site-related contamination.

Response: The interim and preferred remedial actions implemented for the site, including the selection of cleanup goals, removal of contaminated soil, placement of the cap and deed restrictions, were reviewed and discussed in the 'Overview of Remedial Actions' section of the PHA. The preferred remedy is designed to address current and future exposures and is based on various criteria including short and long-term effectiveness and reliability.

Comment #21: The PHA follows, to some extent, the format of the remediation process in that it divides the assessment into on site and off site. While there may be some merit to this delineation, it may in some cases mere convenience and simply a fiction. Contaminants that are mobile will not respect the site/off-site distinction.

Response: It is common practice in the remediation industry to divide a site into a number of operable units, depending on the complexity of the problems associated with a site. Operable units may address portions of a site, specific site problems, or initial phases of an action, or may consist of any set of actions performed over time or any actions that are concurrent but located in different parts of a site.

In LBPHA report, based on phases of remedial work completed and being implemented, the contamination was divided into on- and off-site contaminated areas.

Comment #22: The commenter questioned the approach used to assess the health effect of contaminants for which CVs were unavailable.

Response: Section 7.2.1 of the ATSDR guidance manual outlines the procedure for selecting environmental guideline comparison values.

Comment #23: The commenter wanted to know if the PCBs detected in the on-site areas were factored into the PHA.

Response: The non-cancer (see Table 12) and cancer (see Table 18) risk associated with the PCBs detected were evaluated in the PHA. The non-cancer adverse health effects from PCBs exposures were found to be possible for children. For cancer health effects, a lifetime excess cancer risk was 3 in 10,000 to the exposed population.

Comment #24: *The commenter expressed concern about 'deed restriction' as one of the remedial measures implemented at the site.*

Response: According to NJDEP's Technical Requirements for Site Remediation, deed restriction is considered as one of the viable administrative control options that can be implemented at a hazardous waste sites to address contaminant exposure pathways.

Comment #25: *The commenter inquired about the rationale for only evaluating the completed exposure pathways.*

Response: A potential exposure pathway exists when information about one or more of the five elements of an exposure pathway (see Assessment Methodology' section of the PHA) is unavailable or uncertain. Quantitative evaluation is not conducted due to this uncertainty.

Comment #26: *The commenter questioned how the current exposure status was designated as no public health hazard.*

Response: The on-site contaminant exposure pathways were eliminated by the implementation of the remedial actions (see 'Overview of Remedial Actions' section).

Comment #27: *The commenter indicated that on page 1 of the LBPHA report, the typographical emphasis (capitalized, bold-faced and italicized) on the phrase "No Public Health Hazard" is dramatic.*

Response: One of the main objectives of LBMGP PHA is to assess whether/to what extent area residents are being exposed to hazardous substances associated with on-site areas. The results indicated that currently there are no completed exposure pathways associated with the on-site contamination and as such, the on-site areas pose ***No Public Health Hazard***.

The purpose of using bold-faced and italicized letters was not to dramatize but to emphasize the current status of site-related exposures, according to categories established by the ATSDR.

Comment #28: *The commenter indicated that the time-frames associated with exposures need to be separated into unique sections in the Summary and clarified.*

Response: Based on sampling data and knowledge of accessibility of the media to the population, all exposure pathways associated with the LBMGP site were identified; for each pathway, the PHA also determined the pathway status, i.e., completed, potential or eliminated (see Table 11). The implementation of the preferred remedy addresses the current and future exposure pathways (see second paragraph of Summary). The

evaluation of past exposure pathways were discussed in the third paragraph of Summary section.

Comment #29: The commenter expressed reservation about the appropriateness of chemical mixture exposure evaluation conducted in the LBPHA.

Response: The residents were exposed to site-related contaminants via direct contact, ingestion and inhalation. Individual as well as the cumulative/synergistic non-cancer and cancer effects of mixtures of contaminants were evaluated. The concentration of PCBs indicated cumulative/synergistic toxicological effects from exposures to soil/sediment in children and adults. However, an in-depth mixtures evaluation could not be conducted because information on the health effects associated with the interaction of arsenic, benzene, lead, PCBs, n-propylbenzene and vinyl chloride are unavailable in the toxicologic literature.

Although there are uncertainties associated with the chemical mixture risk estimates, the NJDHSS and ATSDR used a toxicological and risk calculation methods developed by the ATSDR for use in PHAs.

Comment #30: The commenter requested that the public health assessment report should evaluate (1) epigenetic effects of chemical exposures, (2) fetal programming and the extraordinary sensitivity of fetuses and infants to chemical exposures and (3) toxicity of additional 32 PAHs being considered by the EPA.

Response: A great deal of toxicological research is being conducted to evaluate the effects of environmental factors (including toxic compounds) on mechanisms such as fetal sensitivity, epigenetic transgenerational imprinting, or intra-generational carcinogenesis. The USEPA and ATSDR have scientific branches which are actively assessing such issues. However, standardized guidance documents are not yet available.

The same can be said for the toxicity evaluation of 32 additional PAH hydrocarbons, in which case toxicity assessment results have not been fully evaluated yet.

Comment #31: The commenter indicated that the PHA was unclear about the procedure followed to link the community health concerns with contaminant exposures.

Response: The community health concerns included asthma, autism, birth defects, learning disorders, respiratory conditions, psychological stress, infections, rashes, and lupus. The lowest observed adverse effects (LOAELs) as well as risk factors for each of the community health concerns were investigated. Asthma may be triggered by outdoor contaminants including particulate matter; and learning disorders may be associated with both indoor and outdoor lead exposures. Based on cancer risks and childhood lead exposures in the past, the site was characterized to pose a public health hazard. Autism, birth defects, respiratory conditions, infections, skin rashes, and lupus were unlikely to be associated with the site-related contamination.

Comment #32: The commenter asked how PHA reached the conclusion that that the psychological stress caused by relocation in Long Branch was not related to the site. One would assume that being removed from one's home under duress would be stressful and might actually lead to increased susceptibility to disease, just as many other serious stresses in life are associated with increased susceptibility to disease and even death. (See, for example, chapter 8 of William R. Clark's book, At War Within; The Double-Edged Sword of Immunity [N.Y.: Oxford University Press, 1995; ISBN 0-19-509286-4.]

Response: The Concerned Citizens Coalition raised the issue of potential psychological stress to residents as a result of unknown (at the time) relocation plans. The NJDHSS and the ATSDR agreed that this was a real risk to residents, and requested that the Monmouth County Division of Mental Health provide appropriate resources to residents upon request. Both NJDHSS and ATSDR understand that unknown and potentially life-altering decisions made by an individual or by others can result in psychological stress which can be transient or long term. If this did occur, it would have been a direct or indirect result of contamination and the resultant relocation of residents. The Community Health Concerns section of the document has been revised to reflect this.

Comment #33: The commenter expressed concern about the potential future exposure pathways at the site.

Response: To remediate and address future exposures associated with on-site contaminants, the NJDEP has been providing oversight for contamination delineation, exposure pathway identification and associated risk assessment, remedy evaluation with respect to short- and long-term effectiveness, and implementation of the preferred remedy. A brief summary of the remedy implemented at the on-site areas were discussed in the "Overview of Remedial Actions" section of the public health assessment report. The implementation of remedy addresses the future exposure pathways.

Comment #34: The commenter expressed concern about exposure from past air emissions from the site and wanted to know why air modeling was not conducted.

Response: Prior to 1961, area residents were potentially exposed to air emissions from the LBMGP. Since air sampling data and information to model contaminant emissions are not known to exist, exposures associated with this pathway could not be evaluated.

Comment #35: The commenter cited ATSDRGM Section 3.1.1.5 regarding demographic information around the site and likely future exposure scenarios.

Response: All past, current and future likely exposure scenarios were identified and their public health implications evaluated in the LBPHA.

Comment #36: The commenter, citing ATSDRGM Section 3.1.2, stated that the LBPHA should discuss "environmental justice." In addition, the "vulnerable populations" should be identified. (See ATSDRGM Sections 6.5 and 6.5.1.).

Response: The NJDHSS is a co-chair and a member of New Jersey Environmental Justice Task Force (EJTF) committee. On March 6, 2004, the Long Branch Concerned Citizens Coalition successfully petitioned the EJTF for recognition as an environmental justice community, with specific concerns relating to the contamination of Troutman's Creek. The NJDHSS provided input into an action plan (NJDEP/NJDHSS 2006) developed to address those community concerns.

Currently, the NJNG is delineating the extent of site-related contamination detected in the off-site areas including the Troutman's Creek. The exposures associated with this off-site contamination will be evaluated in a separate health consultation.

The PHA includes a section specifically on children. In 2004, the NJDHSS/ATSDR prepared a health consultation that addressed the indoor air exposures and health concerns at two off-site child care centers (ATSDR 2004). The indoor air contaminant concentration data were reviewed. Concentrations of contaminants detected in the indoor air at the day care centers represented no apparent public health hazard.

Reference

[NJDEP/NJDHSS] New Jersey Department of Environmental Protection and New Jersey Department of Health and Senior Services 2006] Long Branch Action Plan: An Inter-Agency Report on Cooperative State Action, New Jersey Environmental January Task Force, 2006.

Comment #37: Citing the ATSDRGM Section 3.1.3, the commenter expressed concern over the extent of contamination data presented in the LBPHA.

Response: The level and extent of contamination of environmental media is presented in the "Environmental Contamination" section of the PHA.

Comment #38: The commenter was concerned about the effectiveness of the synthetic liner used to remediate the contamination detected at the on-site areas.

Response: Evaluation of the remedial effectiveness of the liner was not within the scope of this PHA.

Comment #39: The commenter, citing ATSDRGM Section 3.1.4, was concerned about contaminant migration associated with flooding in the off-site areas.

Response: The NJDHSS/ATSDR evaluated the impact of flooding events in the PHA. During the availability sessions, the NJDHSS/ATSDR staff spoke with residents living adjacent to Troutman's Creek across from the former Seaview Manor public housing complex. The residents expressed concerns about flooding of their yards and basements during periods of heavy rain.

As a result, the NJDHSS/ATSDR recommended that the remedial investigation of off-site areas including the residential properties along the Seaview Avenue be completed as soon as feasible.

Comment #40: The commenter, citing ATSDRGM Section 6.1.2, stated that the "site conceptual model" was not presented in the LBPHA report.

Response: Although "site conceptual model" was not specifically discussed in the PHA, it is implicit in the exposure pathway identification.

Comment #41: The commenter cited ATSDRGM Section 6.3.1 and indicated that the natural attenuation of contaminants should be modeled to help people understand what they are dealing with.

Response: Section 6.3.1 of the ATSDRGM discusses "Fate and Transport Processes" with reference to the evaluation of past, current and future exposure pathways. Modeling the natural attenuation of remediated (capped) contaminants at the LBMGP site is not within the scope of the PHA.

Comment #42: The commenter cited ATSDRGM Section 6.4.3 to indicate that the PHA report did not provide a description of how contamination patterns might change over time and space and when people might have or might come in contact with site contaminants.

Response: The past, current and future exposure pathways were identified and evaluated in the "Discussion" section of the PHA report.

Comment #43: The commenter expressed concern about the contamination remaining under the cap. In addition, the quantity of the contaminated media under the cap should also be estimated and provided in the LBPHA.

Response: Assessing the quantity of contaminated media at a hazardous waste site is not within the scope of public health assessment. The reader is referred to the RI for the site.

Comment #44: The commenter specifically was concerned about the conclusion that skin rashes are probably not related to exposure to PAHs.

Response: The NJDHSS/ATSDR relies on toxicologic studies published in peer-reviewed professional journals to assess public health implications of contamination. Although mixtures of carcinogenic PAHs cause skin disorders in human and animals, specific effects in humans of individual PAHs, except for benzo[a]pyrene, have not been reported (ATSDR 1995). A LOAEL of 100 µg (i.e., 0.1 mg) for contact hypersensitivity was identified in animal studies (Klemme *et al.* 1987).

Using a default soil-to-skin adherence factor (0.07 mg/cm²) (USEPA 2008) and a maximum concentration of benzo[a]pyrene detected (140 mg/kg) at the Processing Areas (see Table 1 of PHA), the skin surface exposure concentration may be calculated as follows:

$$\frac{140 \text{ mg BaP}}{\text{kg soil}} * \frac{3 \text{ days}}{7 \text{ days}} * \frac{\text{kg soil}}{10^6 \text{ mg soil}} * \frac{0.07 \text{ mg soil}}{\text{cm}^2 \text{ of skin surface}}$$

$$= 4.2 \times 10^{-6} \text{ mg BaP/cm}^2 \text{ of skin surface}$$

Although regulatory agencies do not have recommended guidelines for evaluating the adverse effects that can occur at the skin surface (i.e., rashes/contact hypersensitivity), the maximum skin surface exposure concentration (i.e., 4.2×10^{-6} mg BaP/cm² of skin surface) was orders of magnitude lower than the levels associated with health effects. As such, the rashes/contact hypersensitivity reported by the community is unlikely to be associated with exposure to site related contaminants.

Reference

Klemme, J.C., H. Mukhtar and C.A. Elmetts (1987). Induction of contact hypersensitivity to dimethylbenz(a)anthracene and benzo(a)pyrene in C3H/HeN mice. *Cancer Research*, 47:6074- 6078.

[USEPA] United States Environmental Protection Agency 2008. Accessed on February 01, 2008 at: <http://www.epa.gov/reg3hscd/risk/human/info/dermalag.htm>.

Comment #45: The commenter was concerned about the deed restriction as one of the components of the preferred remedy. He also expressed concern about the potential disruption of the cap and the resulting future exposure scenario.

Response: Deed restriction was one of the components of preferred remedy selected and implemented for the LBMGP site. Real estate deed restrictions are restrictions on the deed that place limitations on the use of the property.

The NJDEP provided oversight to ensure that the remedy selection and the implementation process is consistent with NJDEP’s Technical Requirements for Site Remediation, which was developed to ensure remedial actions protect human health and the environment. For a complete assessment of the short and long-term effectiveness of the deed restrictions for the on-site areas, the reader is referred to the Feasibility Study for the site.

All work is consistent with NJDEP’s Technical Requirements for Site Remediation, which were developed to ensure remedial actions protect human health and the environment

Comment #46: The commenter was concerned about the “indeterminate” hazard category. He wanted the PHA report to outline the recommendation associated with indeterminate exposure pathways.

Response: The off-site contamination data at the LBMGP site were unavailable at the time the PHA was initiated. Therefore, the NJDHSS/ATSDR recommended that the remedial investigation of off-site areas including the residential properties along the Seaview Avenue be completed as soon as feasible. The off-site contamination data will be evaluated in a separate health consultation.

However, it should be noted that the data on air emissions from the LBMGP site is unavailable and the potential air exposure pathway cannot be evaluated; the pathway remains indeterminate.

Comment #47: The commenter expressed concern about the soil lead concentration detected (i.e., 120 ppm) in the Seaview Manor public housing complex area.

Response: The IEUBK model was used to assess the impact of soil lead levels (120 ppm) for the incidental soil lead ingestion exposure scenario. The model predicted that the geometric mean BLL for children ages 0 - 84 months was 2.1 µg/dL; the probability of blood lead levels exceeding 10 µg/dL (the CDC level of concern) was 0.05% (USEPA recommends that the lead concentration in site soil does not result in a 5% probability of exceeding a BLL of 10 µg/dL). In addition, according to the National Health and Nutrition Examination Survey (1999-2002), the geometric mean and 95% confidence interval of BLL for the U.S. population aged 1 year and older are as follows:

Age group (year)	Survey Years	Geometric Mean (µg/dL)
1 to 5	1999 – 2000	2.23
	2001 – 1002	1.7

The survey results clearly show that the national BLL are similar to those predicted by the IEUBK model. Low-level environmental lead exposure has been associated with subclinical decrements in neurocognitive function in young children. Although in 1991 the CDC established 10 µg/dL as a blood lead concentration of concern in children, no threshold for lead’s effects has yet been identified (NRC, 1993). Recent studies have suggested possible neurodevelopmental effects at blood lead concentrations of less than 10 µg/dL (Lanphear et al., 2000; Canfield et al., 2003); further assessment is ongoing.

Since the geometric mean BLL (2.1 µg/dL) associated with exposure to 120 ppm of soil lead concentration is substantially lower than the CDC level of concern (10 µg/dL) and similar to the national background levels, the PHA stated that a potential for adverse health effects from lead exposures at the Seaview Manor public housing complex is not expected.

Reference

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 Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits with blood lead concentrations < 10 µg/dL in US children and adolescents. Public Health Rep 2000;115:521-9.
 Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 µg/dL. N Engl J Med 2003;348:1517-26.

Comment #48: The commenter expressed concern about the past and future contamination of the Troutman's Creek used by subsistence fishermen and for recreation.

Response: Contamination from the LBMGP has been detected in Troutman's Creek. Currently, NJNG is conducting remedial investigation of site-related contamination (including residential properties located along Seaview Avenue near Troutman's Creek, the Liberty Avenue and the Long Branch Sewerage Authority property).

A separate health consultation will be prepared to evaluate past, current and future exposures associated with this off-site contamination.