

Public Health Assessment

Final Release

QUANTA RESOURCES

EDGEWATER, NEW JERSEY

EPA FACILITY ID: NJD000606442

Prepared by
New Jersey Department of Health
Environmental and Occupational Health Surveillance Program

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

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

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

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Table of Contents

Summary.....	1
Statement of Issues	4
Background.....	4
Site History	5
Industrial History	5
Past ATSDR and NJDOH Activities	7
Environmental Contamination.....	7
Summary of Contaminants of Potential Concern (COPC).....	12
Discussion.....	14
Assessment Methodology.....	14
Completed Pathways	14
Potential Pathways.....	15
Eliminated Pathways	15
Public Health Implications	16
Non-Cancer Health Effects.....	16
Cancer Health Effects	28
Child Health Considerations.....	33
Health Outcome Data	33
Public Comment	33
Conclusions	33
Recommendations	35
Public Health Action Plan (PHAP)	35
Public Health Actions taken by NJDOH and ATSDR	35
Public Health Actions Planned by NJDOH and ATSDR.....	35
References	36
Report Preparation.....	39
Appendix A: Tables and Figures	A-1
Appendix B: Toxicologic Summaries	B-1
Appendix C: Sample Non-cancer Exposure Dose and Lifetime Excess Cancer Risk Calculation	C-1
Appendix D: Summary of Public Comments and Responses	D-1

Summary

Introduction

On January 11, 2001, the U.S. Environmental Protection Agency (USEPA) proposed to add the Quanta Resources Corporation site, Edgewater, Bergen County, New Jersey, to the National Priorities List. Manufacturing activities at the Quanta and adjacent properties and uncontrolled release of hazardous wastes have resulted in the contamination of soil and groundwater. The major contaminants associated with the site are polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and metals. The Agency for Toxic Substances and Disease Registry (ATSDR) and New Jersey Department of Health (NJDOH) released a Public Health Assessment (PHA) based on limited data available in 2002, but a complete evaluation could not be conducted since on- and off- site data were unavailable.

In September 2011, the USEPA released a Record of Decision describing the selected alternative for the soil and groundwater contamination remediation. With the availability of on- and off-site soil and groundwater contamination delineation data, the NJDOH prepared this Public Health Assessment through a cooperative agreement with the ATSDR. The NJDOH held a public comment period from March 4, 2015 through April 5, 2015 to provide an opportunity for interested parties to comment on the draft PHA prepared for the Quanta Resources Corporation Site. Written comments were received from two organizations during the public comment period.

The ATSDR and NJDOH's top priority at this site is to ensure that the community around the site has the best information possible to safeguard its health. It should be noted that this document does not assess the potential inhalation exposure to naphthalene and other volatile organic compounds during remediation. The public health implications of potential exposures during remediation is being evaluated by the NJDOH and ATSDR in a separate health consultation.

Conclusions

The NJDOH and ATSDR have reached five conclusions in this Public Health Assessment on the Quanta Resources Corporation site:

Conclusion 1

The NJDOH and ATSDR conclude that currently there are no ongoing exposures from ingestion of soil contaminants (collected at a depth of 0 to 2 feet) at the Quanta Resources Corporation and adjacent properties that can harm people's health. It should be noted that there is some uncertainty¹ in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

¹ATSDR considers the depth to which soil is likely to be contacted directly is 0-3 inches which differs from the USEPA who considers this depth to be 0-2 feet.

Basis for Conclusion

On- and off-site contaminated soils have been excavated and/or capped and/or fenced. The excavated areas were backfilled with clean fill. Thus, area residents are not being exposed to site-related contaminants via the ingestion pathway.

However, it should be noted that although the interim remedial measures interrupted the exposure pathways, the preferred remedy for the contaminated media has not been implemented yet.

Next Steps

The NJDOH and ATSDR recommend maintaining site access restrictions to prevent access by area residents and trespassers and to ensure the integrity of the remedy.

Conclusion 2

The NJDOH and ATSDR conclude that past exposures to site-related contaminants detected in the soil (a depth of 0 to 2 feet) may have harmed people's health. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Basis for Conclusion

Based on the contaminants detected in the soil (collected at a depth of 0 to 2 feet), the potential for non-cancer adverse health effects associated with past exposures to arsenic, lead, and PCBs were possible in children and adults. Maximum cumulative lifetime excess cancer risks associated with the contaminants detected in the soil (collected at a depth of 0 to 2 feet) were estimated to be as high as three excess cancer cases in 1,000 exposed population. This exposure posed an increased lifetime excess cancer risk, compared to the background risk of cancer from all causes. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Next Steps

Under USEPA oversight, the preferred remedy (as described in the record of decision for the site) should be implemented as soon as feasible.

Conclusion 3

The NJDOH and ATSDR conclude that there were no indoor air exposures² to contaminants at the child care center that could have harmed children's health.

Basis for Conclusion

The adjusted exposure point concentration of tetrachloroethene detected in the indoor air did not exceed the health guideline comparison value. The

²The indoor air exposures discussed were associated with vapor intrusion pathway, not ambient air because of the current remediation work.

calculated lifetime excess cancer risks showed no increase in the estimated cancer risk (one excess cancer case in 100 million individuals exposed).

Next Steps

Following indoor air sampling, the NJDOH and ATSDR recommend maintaining changes to the building ventilation system. The Palisades Child Care Center has relocated to an off-site building in 2012 and currently the building is being demolished.

Conclusion 4

The NJDOH and ATSDR conclude that there were no indoor air exposures to contaminants at the office space that could have harmed people's health.

Basis for Conclusion

The indoor air adjusted exposure point concentration of acrolein is about 35 times lower than the lowest observed adverse effect level; as such, non-cancer adverse health effects from exposures to acrolein are unlikely. The calculated lifetime excess cancer risks showed low estimated cumulative cancer risk (five excess cancer cases in one million individuals exposed, including children).

Conclusion 5

The NJDOH and ATSDR cannot conclude if exposure to the sediment and surface water of the Hudson River harmed people's health.

Basis for Conclusion

The USEPA will address the Hudson River sediment and surface water contamination attributable to the site as a separate operable unit in the future.

Next Steps

The remedial investigation of sediment and surface water should be completed as soon as feasible.

As additional data associated with sediment and surface water become available, the NJDOH and ATSDR will prepare a health consultation to assess the health implications of potential contamination.

For More Information

Questions/Comments about this report should be directed to the NJDOH at (609) 826-4984 or:

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Statement of Issues

On January 11, 2001, the U.S. Environmental Protection Agency (USEPA) proposed to add the Quanta Resources Corporation (QRC) site, Edgewater, Bergen County, New Jersey, to the National Priorities List (NPL). The site was added to the NPL on September 5, 2002. From 1896 to 1974, the site was the location of a coal tar distillation plant. Beginning in 1974, recycling of waste oil including storage, reprocessing, reclamation, and recovery occurred at the site. Due to site activities, the soils, sediment, and groundwater were contaminated with polycyclic aromatic hydrocarbons (PAHs), volatile and semi-volatile aromatic compounds (VOCs and SVOCs), and metals.

In 2002, the Agency for Toxic Substances and Disease Registry (ATSDR) prepared a Public Health Assessment (PHA) for the QRC site based on limited data available at that time; however, a comprehensive evaluation could not be conducted due to insufficient environmental data (ATSDR 2002). The recommendations of the report included collection of additional indoor air samples from a child care center and completion of the Remedial Investigation. In September 2011, after completing the Remedial Investigation/Feasibility Study (RI/FS) for the site, the USEPA released a Record of Decision (ROD) describing the preferred remedy for the soil and groundwater contamination. With the availability of more comprehensive contamination delineation data, the New Jersey Department of Health (NJDOH) prepared this PHA report through a cooperative agreement with the ATSDR. The NJDOH held a public comment period from March 4, 2015 through April 05, 2015 to provide an opportunity for interested parties to comment on the draft Public Health Assessment (PHA) prepared for the Quanta Resources Corporation Site. Written comments were received from two organizations during the public comment period.

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

Background

The QRC site on River Road at the intersection of Gorge Road in Edgewater Borough, Bergen County, New Jersey (see Figure A1, Appendix A), consists of several former industrial facilities that once operated at that location. The former Quanta property, a vacant lot, occupies approximately 5.5 acres of land, and is the remnant of an industrial facility that once covered approximately 15 acres.

The USEPA has organized the site activities into two operable units (OUs) to make it more manageable. The OU1 consists of the land portions of the site and the groundwater (see Figure A2, Appendix A), and OU2 consists of sediments and surface water in the Hudson River. The Hudson River is the only surface water body near the site. The USEPA will address the Hudson River sediment and surface water contamination attributable to the site in OU2 separately. The NJDOH and ATSDR will review and evaluate these data when they are available.

Site History

Before the mid-1800s, the site and surrounding areas were tidal marshlands associated with the Hudson River (CH2M Hill 2008). Development of rail lines and industry along the banks of the Hudson River prompted the systematic filling of these marshlands. After the Civil War, this area became home to some of the earliest chemical operations in New Jersey. Heavy industry began to leave the Borough of Edgewater in the 1960s, marking the beginning of a transformation of Edgewater into a residential community and retail shopping destination.

Industrial History

Of the many industrial operations in the area, three are of particular interest for this Public Health Assessment: Barrett Manufacturing Co., General Chemical Company, and QRC Inc. (CH2M Hill 2008).

From approximately 1872 to 1971, a large portion of the QRC site was used by Barrett Manufacturing Company to process coal tar and to produce paving and roofing materials. The General Chemical Company operated from 1900 to 1957 to produce sulfuric acid using the lead chamber process, which produced waste cinders containing 1% or more of arsenic. In 1974, a portion of the site was leased for waste oil storage and recycling. In 1977, that portion of the site was leased to companies owned and controlled by Russell Mahler, who collected and re-refined waste oil from the eastern United States. In 1980, QRC purchased the assets of Mahler's operations, including the lease at the site. In 1981, the New Jersey Department of Environmental Protection (NJDEP) stopped all activities due to the presence of polychlorinated biphenyls (PCBs) in the waste oil.

In addition, the following industrial operations were located immediately adjacent to the QRC site (see Figures A3 and A4, Appendix A):

Celotex Corporation: The General Chemical Company located just north of the coal tar plant was operated by Celotex Corporation for the manufacture of gypsum wall board from approximately 1967 to 1971. In 1971, the Celotex Corporation leased the industrial space to a variety of smaller enterprises, including a vacuum truck company and a metal reclaiming/refinishing plant.

Spencer-Kellogg & Sons, Inc. (115 River Road): This Company began operations around 1910, manufacturing edible oils such as linseed, castor and coconut oils, at the facility just south of Barrett. Some of the buildings at 115 River Road are the original Spencer-Kellogg buildings, though the tank farm and manufacturing facilities were dismantled in the 1960s.

Lever Brothers: This industrial property had a variety of uses before the Lever Brothers facility, which was built in the 1930s. The Lever Brothers (later known as Unilever) primarily handled soaps and edible oil products. The facility was used for research until about 2003.

Early Regulatory History

At the time when NJDEP closed the facility in 1981, the Quanta property contained 61 aboveground storage tanks (ASTs), at least 10 underground storage tanks (USTs), septic tanks, and underground piping (CH2M Hill 2008). Several removal actions were performed at the site from 1984 to 1988 by a group of potentially responsible parties (PRPs), under USEPA oversight. The removal actions focused on the cleaning and decommissioning of the ASTs and USTs. Several million gallons of product were removed and disposed of or recycled. Some underground piping and shallow soils were also removed.

The site was considered for listing on the National Priorities List (NPL) in the late 1980s but it did not qualify using the Hazard Ranking System. The USEPA retained regulatory responsibility, maintained security fencing, conducted periodic inspections and operated an adsorbent boom system to capture floating oil sheens from the Hudson River mudflat.

The “New” River Road and Redevelopment

Redevelopment of the 115 River Road property began in 1986. It was built out as commercial space, including a child care³ center (Palisades Child Care Center). It has been continuously occupied by about 50 to 60 small commercial businesses (CH2M Hill 2008).

A plan to relocate and expand River Road was proposed in the early 1990s. In 1996, USEPA entered into an Administrative Order on Consent (AOC) with Bergen County and a private developer to allow the County to safely construct a road over a portion of the site. Redevelopment of the Celotex property started in the late 1990s, beginning with a lengthy investigation and remediation phase under the direction of NJDEP.

In 1996, USEPA and one PRP, the Barrett successor company AlliedSignal (now Honeywell), entered into an AOC to improve site security and further investigate the extent of site contamination. A second AOC was signed in 1998 designating steps to investigate and address the ongoing coal tar sheens in the mudflats of the Hudson River. The studies required under these AOCs, along with an ecological risk assessment of Hudson River sediments performed by the USEPA, finally led to the proposal of the site to the NPL in 2001.

In 2000-2002, the Celotex developer found an area contaminated with high concentrations of arsenic (High Concentration Arsenic Area, or HCAA); they petitioned NJDEP to leave it capped in place. The HCAA is located on both the Quanta and former Celotex lots. The developer and the USEPA agreed to leave the capped contaminated “as is” temporarily. An impermeable liner and several feet of fill material have been placed over the HCAA.

³Palisades Child Care Center has relocated to an off-site building in 2012.

NPL Listing and Current Status

On January 11, 2001, USEPA proposed inclusion of the site on the NPL, and on September 9, 2002, USEPA placed the site on the NPL. The RI/FS for OU1 has been performed by the environmental consulting firm CH2M Hill under an AOC with USEPA signed in 2003.

Past ATSDR and NJDOH Activities

The ATSDR and NJDOH prepared a PHA for the QRC site based on limited environmental contamination data (ATSDR 2002). The report recommended collection of additional environmental data, environmental monitoring during construction, remedial investigation of neighboring properties, indoor air sampling of the Palisades Child Care Center and other buildings, and posting warning signs. The NJDOH and ATSDR also recommended maintaining changes to the building ventilation system.

In 2006, the ATSDR prepared a HC for the Celotex site that evaluated the exposure pathways associated with the disposal and processing of Libby vermiculite (ATSDR 2006a). The Celotex site was identified as one of seven New Jersey facilities that received vermiculite ore from the Libby mine. A number of past exposure pathways were identified and evaluated. The report recommendation included asbestos exposure follow-up for workers, maintenance of adequate landfill cap thickness and deed restrictions.

In December 2003, a resident living in the neighborhood of the former Celotex site requested ATSDR to evaluate a potential health concern they felt was associated with dust from redevelopment activities (ATSDR 2008). In response to this request, the exposure pathways associated with dust were evaluated. The results did not show any ongoing residential exposures to dust-related contamination. However, there were completed exposure pathways via the inhalation and incidental ingestion of contaminated dust during past construction activities.

Environmental Contamination

An evaluation of site-related environmental contamination consists of a two-tiered approach: 1) a screening analysis; and 2) a more in-depth analysis to determine public health implications of site-specific exposures (ATSDR 2005). First, maximum concentrations of detected substances are compared to media-specific environmental guideline comparison values (CVs). If contaminant concentrations exceed their CV, it is referred to as a Contaminant of Potential Concern (COPC) and selected for further evaluation. If contaminant levels are found above CVs, it does not mean that adverse health effects are likely, but that a health guideline comparison is necessary to evaluate site-specific exposures. Once exposure doses are estimated, they are compared with health guideline doses to determine the likelihood of adverse health effects.

Environmental Guideline Comparison

There are several CVs available for screening environmental contaminants to identify COPCs (ATSDR 2005, 2013; USEPA 2013a). 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 considered as CVs. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million persons exposed during their lifetimes (78 years).

In the absence of an ATSDR CV, values from other sources may be used to evaluate contaminant levels in environmental media. These include New Jersey Maximum Contaminant Levels (NJMCLs) for drinking water, USEPA MCLs for drinking water and USEPA Regional Screening Levels (RSLs). RSLs are contaminant concentrations corresponding to a fixed level of risk (i.e., a Hazard Quotient⁴ of 1, or lifetime excess cancer risk of one in one million, or 10^{-6} , whichever results in a lower contaminant concentration) in water, air, biota, and soil (USEPA 2011a, USEPA 2013a,b). For soils and sediments, CVs also include the NJDEP Residential Direct Contact Soil Remediation Standards (RDCSCC), Ingestion-Dermal Health Based Criterion (IDHBC) and Inhalation Health Based Criterion (IHBC). Based primarily on human health impacts, these criteria also take into account natural background concentrations, analytical detection limits, and ecological effects (NJDEP 2012).

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

Site Conditions

The QRC site and adjacent areas are contaminated by several former industrial facilities that once operated at the location (CH2M Hill 2008). The industrialized waterfront area had tank farms, industrial unit operation facilities, railway corridors and Hudson River piers (see Figure A4, Appendix A). The site (i.e., OU1) also includes the observed extent of site-related Non Aqueous Phase Liquid (NAPL) and coal tar constituents detected in soil and groundwater, related to former operations.

The site consists of the former QRC Inc. property and any locations to which contamination from the property and former operations have migrated. The presence of

⁴The 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 (USEPA 2013a).

constituents associated with former site operations has been observed in parts of the following areas, which together make up OU1 (see Figure A2, Appendix A):

- QRC property
- former Celotex property
- 115 River Road property
- former Lever Brothers property
- Block 93 (north, central, and south)

Topography, Geology, and Hydrogeology

The Quanta property is generally flat and at a lower elevation than the surrounding properties and River Road. The City Place development, on the former Celotex property, includes residential and commercial space. Substantial filling has raised the ground surface five to over 15 feet above the original Celotex grade. The portions of the property over the HCAA consist of landscaping and a paved roadway. The southern portion of the property is a partially paved and unpaved sloping temporary parking lot. The majority of the 115 River Road property (the former Spencer-Kellogg facility) is improved with a large multi-tenant building and a smaller parking lot/office building. South of the 115 River Road property is the former Lever Brothers property. This property is in the early stages of cleanup and redevelopment. A large parking lot exists on the northeastern portion of the property. The topography here is also flat. Three lots on Block 93 (Lots 1, 3, and the northern portion of Lot 2) are located between Old River Road and River Road and are part of the former Barrett Manufacturing Company property. Topography is generally flat with minimal standing water.

Bedrock at the site is known as the Stockton Formation, composed of a mixture of sandstone, silty mudstone, siltstone, shale, and conglomerate. At the site, the Stockton Formation is overlain by as much as 80 feet of unconsolidated deposits. Several important geologic layers within the unconsolidated deposits are the fill layer at the surface and the silty clay confining unit. The geologic layers vary in thickness across the site. The water table on the QRC property and 115 River Road is quite shallow, within about two feet of the ground surface. The direction of the shallow unconfined groundwater flow (above the confining unit) is generally to the east and south.

Nature and Extent of Contamination

The RI report (CH2M Hill 2008) compiles and presents evaluations of the following data:

- Over 3,600 soil analyses
- Data from 57 groundwater monitoring locations
- Extensive non-aqueous phase liquid (NAPL) “fingerprinting” data
- Soil vapor and indoor and outdoor air analyses and building surveys

The RI for OU1 was conducted with oversight by USEPA. The predominant site contaminants are coal tar constituents and arsenic. The former acid plant, located on the

northern portion of the Quanta property and the southern portion of the former Celotex property, has been demonstrated to be a source of metals, particularly arsenic and lead in soil and groundwater.

Non-Aqueous Phase Liquid (NAPL): Coal tar NAPL is found across the site and is made up of aromatic VOCs and SVOCs (CH2M Hill 2008). There is a broad spectrum of NAPL concentrations at the site. Higher concentrations of NAPL are identified as free-phase NAPL, because in these areas the wastes are concentrated enough to collect as a separate layer in groundwater monitoring wells. Much of the site NAPL is denser than water (dense non-aqueous phase liquid, or DNAPL), so it sinks through rather than floats on the water table. The NAPL is found throughout all the unconfined units, but the silty-clay confining layer under the site has acted as a vertical boundary.

The NAPL constituents extend beyond the lateral extent of NAPL, in the form of staining or odors, and as adsorbed and dissolved-phase VOCs and SVOCs in soil and groundwater (see Figure A5, Appendix A). Most of the NAPL mass at the site is present in six discrete NAPL zones (NZ-1 through NZ-6). The NZ-1 and NZ-2 are close to the surface and are more or less continuous zones of NAPL, whereas the deeper NAPL zones NZ-3 through NZ-6 tend to be present as a series of closely spaced discontinuous lenses separated by NAPL-free zones.

Soil: Soil (a depth of 0 to 2 feet ⁵) samples from the QRC and four adjacent properties were sampled and analyzed for VOCs, SVOCs and metals (CH2M Hill 2008); results indicated the presence of a number of contaminants (see Tables A1 through A5, Appendix A). Maximum concentrations of antimony, arsenic, cadmium, hexavalent chromium, copper, lead, mercury, heptachlor, Aroclor-1242, Aroclor-1254, Aroclor-1260, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, carbazole, chrysene, dibenzo[a,h]anthracene, fluoranthene, indeno[1,2,3-cd]pyrene and naphthalene detected *at the Quanta Resources* soil (a depth of 0 to 2 feet) were above their respective environmental guideline CVs; they were considered as the COPCs for the soil. Environmental guideline CVs for thallium, methoxychlor, isopropyl benzene, methylcyclohexane, 4-nitroaniline, acenaphthylene, dibenzofuran and phenanthrene detected in the soil and the adjacent properties were unavailable.

Maximum concentrations of arsenic, hexavalent chromium, lead, Aroclor-1242, Aroclor-1254, Aroclor-1260, benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene and indeno[1,2,3-cd]pyrene detected at the *former Celotex property* soil (a depth of 0 to 2 feet) were above their respective environmental guideline CVs; they were considered as the COPCs for the soil. Environmental guideline CVs for thallium, acenaphthylene, dibenzofuran and phenanthrene detected in the soil were unavailable.

Maximum concentrations of arsenic, hexavalent chromium, lead, Aroclor-1248, Aroclor-1254, benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene,

⁵Although this assessment is based on the exposures to soils collected from the 0 to 2 feet depth, the ATSDR considers 0 to 3 inches to be the surface soil (the soil to which people are most likely to be exposed).

benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene and indeno[1,2,3-cd]pyrene detected in the soil (a depth of 0 to 2 feet) of *115 River Road property* were above their respective environmental guideline CVs; they were considered as the COPCs. Environmental guideline CVs for isopropyl benzene, methylcyclohexane, acenaphthylene, and dibenzofuran detected in the soil were unavailable.

Maximum concentrations of arsenic, hexavalent chromium, Aroclor-1254, Aroclor-1260, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene, indeno[1,2,3-cd]pyrene and chrysene detected in the soil (a depth of 0 to 2 feet) of the *Lever Brothers property* were above their respective environmental guideline CVs; they were considered as the COPCs. Environmental guideline CVs for acenaphthylene, dibenzofuran, and phenanthrene detected in the soil and the adjacent properties were unavailable.

Maximum concentrations of arsenic, lead, mercury, Aroclor-1248, Aroclor-1254, Aroclor-1260, benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, carbazole, chrysene, fluoranthene, dibenzo[a,h]anthracene, indeno[1,2,3-cd]pyrene and naphthalene detected in the soil (a depth of 0 to 2 feet) of *the Block 93* were above their respective environmental guideline CVs; they were considered as the COPCs. Environmental guideline CVs for thallium, isopropylbenzene, cyclopentane, methylcyclohexane, acenaphthylene, dibenzofuran, and phenanthrene detected in the soil were unavailable.

Indoor Air (via Soil Vapor Intrusion): Vapor intrusion investigations (sub-slab, indoor air and ambient air) have been conducted during the RI at a number of properties⁶, particularly the Palisades Child Care Center and the office spaces located in 115 River Road (see Figure A6, Appendix A) (CH2M Hill 2006). The indoor air sampling was conducted according to the workplan approved by the USEPA. The maximum concentration of none of the contaminants detected in the indoor air of the Child Care Center exceeded the Health Guideline CV (see Table A6, Appendix A). Maximum concentrations of acrolein, benzene, carbon tetrachloride, chloroform, 1,4-dichlorobenzene, naphthalene, tetrachloroethene, and trichloroethene detected at the office space area exceeded the Health Guideline CVs; they were considered as the indoor air COPCs for the office space area (see Table A7, Appendix A).

The PRP group has worked with the owner of 115 River Road to monitor and in some cases modify the building's ventilation system (e.g., increase the fraction of make-up air) to ensure that it can be safely occupied until the preferred remedy for the site is implemented. The NJDOH and ATSDR also recommended maintaining changes to the building ventilation system (ATSDR 2008).

Subsurface Soil: Subsurface soil (a depth of 0 to 10 feet) samples were collected and analyzed for VOCs, SVOCs and metals. Maximum concentrations of 2-methylnaphthalene, anthracene, Aroclor-1254, Aroclor-1260, benzene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, chromium, copper, lead, pyrene, benzo[k]fluoranthene, carbazole, chrysene, dibenzo[a,h]anthracene, fluoranthene,

⁶Annual vapor intrusion monitoring continues at 115 River Road, 163 Old River Road, and 103 River Road.

fluorine, indeno[1,2,3-cd]pyrene, mercury, naphthalene, and selenium were present above their respective environmental guideline CVs (see Table A8, Appendix A); they were considered as the COPCs for the subsurface soil. Environmental guideline CVs for phenanthrene, titanium and zirconium detected in the subsurface soil were unavailable.

Groundwater: Groundwater at the site is classified as a source of potable water, but it was and is not used as a drinking water source. Groundwater samples were collected and analyzed for VOCs, SVOCs and metals (see Table A9, Appendix A). Maximum concentrations of 1,1-dichloroethane, 1,2-dichloroethane, 2,4-dimethylphenol, 2-methylnaphthalene, 4,4'-DDD, 4,4'-DDE, acenaphthene, aldrin, alpha-chlordane, anthracene, Aroclor-1260, total arsenic, arsenic (III), arsenic (VI), benzene, bis(2-ethylhexyl)phthalate, chloromethane, ethylbenzene, fluoranthene, fluorene, lead, methylene chloride, naphthalene, phenol, pyrene, tetrachloroethene, toluene, trichloroethene, vinyl chloride, and xylenes were present above their respective environmental guideline CVs (see Table A9, Appendix A); they were considered as the COPCs for the groundwater.

Summary of Contaminants of Potential Concern (COPC)

The contaminants shown in Table 1 were detected in the environmental media and are designated as the COPCs for the QRC site.

Table 1. Contaminants of Potential Concern (COPCs) by media

Location/ Media	Metals	VOCs/SVOCs
Soil (0-2 feet deep)		
QRC property	Antimony, arsenic, cadmium, copper, mercury, iron, and lead	Heptachlor, Aroclor-1242, Aroclor-1260, benzene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, carbazole, chrysene, dibenz[a,h]anthracene, fluoranthene, indeno[1,2,3-cd]pyrene, and naphthalene
Former Celotex property	Arsenic, chromium, and lead	Aroclor-1242, Aroclor-1254, aroclor-1260, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene
115 River Road property	Arsenic, chromium, and lead	Aroclor-1248, Aroclor-1254, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene
Lever Brothers property	Arsenic	Aroclor-1254, Aroclor-1260, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene

Location/ Media	Metals	VOCs/SVOCs
Block 93 property	Arsenic, lead, and mercury	Aroclor-1248, Aroclor-1254, aroclor-1260, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, carbazole, chrysene, fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, and naphthalene
Sub-Surface Soil (0-10 feet deep)		
	Chromium, copper, lead, mercury, and selenium	2-methylnaphthalene, anthracene, Aroclor-1254, Aroclor-1260, benzene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, carbazole, chrysene, dibenz[a,h]anthracene, fluoranthene, fluorene, indeno[1,2,3-cd]pyrene, and naphthalene
Groundwater		
	Lead and arsenic	1,1-dichloroethane, 1,2-dichloroethane, 2,4-dimethylphenol, 2-methylnaphthalene, 4,4'-DDD, 4,4'-DDE, acenaphthene, aldrin, alpha-chlordane, anthracene, aroclor-1260, benzene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, bis(2-ethylhexyl)phthalate, chloromethane, ethylbenzene, fluoranthene, fluorene, methylene chloride, naphthalene, phenol, pyrene, tetrachloroethene, toluene, trichloroethene, vinyl chloride, and xylenes
Indoor Air (115 River Road)		
Office space		Acrolein, benzene, carbon tetrachloride, chloroform, and 1,4-dichlorobenzene

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

Discussion

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

Assessment Methodology

An exposure pathway is a series of steps starting with the release of a contaminant in environmental media and ending at the interface with the human body (ATSDR 2005). 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 the physicochemical properties of the contaminants detected at the site, dermal exposures were considered to be a minor contributor to the overall exposure dose (i.e., compared to the contribution of ingestion and inhalation exposures) (ATSDR 2005). As such, dermal exposure will not be evaluated in this assessment.

The following exposure pathways (associated with OU1) for individuals who live (or lived) near the site were identified.

Completed Pathways

Incidental ingestion of contaminated soil from Quanta Resources and adjacent properties (past). The soils (a depth of 0 to 2 feet) at the Quanta Resources and adjacent properties are contaminated with SVOCs, PAHs, and metals. Information obtained by the NJDOH from various sources⁷ indicated that in the past, the site was not fenced and area residents had access to the site. The area across the Quanta Resources site (the other side of

⁷Public meetings, NJDOH availability sessions, conversation with long time area residents, NJDEP Case Managers, community action group (CAG) meetings, historical photographs, and information from adjacent sites.

River Road) was densely populated and the residents frequently used the site and adjacent areas to access the Hudson River for fishing, walking and other recreational activities (see Figure A7, Appendix A). Area residents, including children, were exposed to contaminants while engaging in outdoor recreational activities. This scenario includes site visitors.

The most likely receptors who visited the site for recreation were adults and older children (i.e., 6 to 18 years of age) who live (or lived) near the site. Younger children were not expected to visit the on-site areas.

Inhalation of indoor air (present⁸). Air sampling results indicated that the indoor air of the Palisades Child Care Center and adjacent office spaces located at 115 River Road property were contaminated with VOCs. Inhalation of indoor air was an exposure pathway for the occupants including children.

Potential Pathways

Incidental ingestion of contaminated soil from Quanta Resources and adjacent properties (present, future): The contaminated soil from QRC and a number of areas adjacent properties were excavated and disposed of off-site (CH2M Hill 2008). However, the preferred remedy for the site has not been implemented yet (for example, as mentioned earlier, the HCAA has been capped but not removed). As such, the exposure pathway, although unlikely, is considered potential.

Incidental ingestion of contaminated sediment/surface water/biota from Quanta Resources and adjacent properties (past, present, future): The NJDOH, in cooperation with the ATSDR, will prepare a separate health consultation to evaluate contaminant exposures in sediment and surface water and biota when the Remedial Investigation data are available.

Ingestion of Groundwater (past): The area has a long industrial history. The NJDOH and ATSDR have no information or data available that indicated that groundwater was used in the past for potable purposes. As such, the pathway in the past is considered potential.

Inhalation of indoor air (past). Air sampling results indicated that the indoor air of the Palisades Child Care Center and adjacent office spaces located at 115 River Road property were contaminated with VOCs. Inhalation of indoor air was an exposure pathway for the occupants including children. However, since there are no past indoor air data available, this pathway is considered potential.

Eliminated Pathways

Ingestion of Groundwater (current, future): Currently, groundwater at the site is not used for potable purposes. The NJDEP has been requested to establish a groundwater

⁸At the time the indoor air data were collected.

Classification Exception Area (CEA) to prohibit future use of the groundwater within this area, and to restrict the installation of wells (other than for monitoring or remediation purposes) in the area for the duration of the CEA (USEPA 2011b). As such, the current and future status of this pathway is considered eliminated.

Inhalation of indoor air (future). As mentioned earlier, the operation of the building ventilation system was adjusted to ensure that the contaminants levels are below allowable limit until the preferred remedy for the site is implemented. The Palisades Child Care Center relocated from 115 River Road in 2012. As such, the exposure pathway is considered eliminated.

A summary of all exposure pathways identified for the site is presented in Table A10, Appendix A.

Exposure Point Concentration

The exposure point concentration (EPC), or the concentration term in the exposure equation, is derived to reflect a representative concentration at the exposure point or points over the exposure period (USEPA 1989). Consistent with guidance from ATSDR (2005), the 95% upper confidence limit (UCL) of arithmetic mean was used to estimate the EPC. Where the 95% UCL⁹ was greater than the maximum value, the maximum concentration was applied.

Public Health Implications

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

Non-Cancer Health Effects

To assess the possibility of non-cancer health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants that are commonly found at hazardous waste sites (ATSDR 2005). 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) and duration (i.e., acute (less than 14 days); intermediate (15-364 days); or chronic (365 days or more)). MRLs are based largely on toxicological studies in animals and, if available, on reports of human studies. MRLs are usually extrapolated doses from observed effect levels in animal toxicological studies or human studies and

⁹EPC calculations were conducted using USEPA's ProUCL software (USEPA 2007).

adjusted by a series of uncertainty (or safety) factors or using statistical models. In toxicological literature, effect levels include:

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

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

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

Incidental ingestion of contaminated soil from Quanta Resources and adjacent properties (past)

In the past, the manufacturing areas were not fenced; the area residents could access the on-site and adjacent properties (CH2M Hill 2007). Exposures are based on incidental ingestion of contaminated soil; non-cancer exposure doses were calculated using the following formula (ATSDR 2005, 2016):

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

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

C = concentration of contaminant in soil (mg/kg);¹⁰

IR = soil ingestion rate (mg/day);

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

BW = body weight (kg)

It is likely that nearby residents visited the on-site areas for recreational purposes. The most likely visitors/trespassers were adults and older children (i.e., 6 to 18 years old). In this assessment, visitors/trespassers were assumed to be exposed to soil on a given visit. As estimated by the USEPA (CH2M Hill 2007), this assessment used 219 visits per year (or, approximately 4 days/week) for the QRC and adjacent properties.

¹⁰See Appendix C for sample exposure dose calculation.

QRC Property

Based on the maximum concentration of cadmium, chromium, copper, mercury, heptachlor, Aroclor-1242, Aroclor -1254, Aroclor -1260, fluoranthene, and naphthalene detected in the soil and reasonable maximum exposure (RME) scenario, the hazard quotient calculated for children (6 – 11 year) did not exceed 1 (see Table A11, Appendix A); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. Based on the EPC of antimony, arsenic and benzo[a]pyrene detected in the soil (a depth of 0 to 2 feet) (see Table A11, Appendix A) and RME scenario, the hazard quotient calculated for children (6 – 11 year) exceeded one (see Table A11, Appendix A). The EPC of lead detected in the soil (a depth of 0 to 2 feet) (see Table A11, Appendix A) also exceeded the corresponding health guideline CV. The health guideline CVs of benzo[a]anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer adverse health effects associated with exposures to metals and PAHs are evaluated as follows:

Antimony - Ingesting large doses of antimony can cause vomiting. Long-term chronic animal studies have also reported liver damage and blood changes (ATSDR 1992). Although information on the toxic effects of chronic oral exposure to antimony is limited, antimony appears to affect heart muscle, the gastrointestinal tract, and the nervous system. The chronic oral RfD for antimony (0.0004 mg/kg/day) is based on reduced longevity, blood glucose, and altered cholesterol levels of a group of male and female rats in an oral bioassay study. A LOAEL of 0.35 mg/kg/day and an uncertainty factor of 1,000 were used to calculate the oral MRL. Based on the maximum concentration of antimony detected in the soil and RME scenario, the exposure dose calculated (0.00066 mg/kg/day) for children (6 – 11 year) exceeded the MRL (0.0004 mg/kg/day) (see Table A11, Appendix A). However, the calculated child exposure dose is about 530 times lower than the LOAEL (i.e., 0.35 mg/kg/day). As such, non-cancer adverse health effects associated with past exposures of antimony contaminated soil at the QRC property are unlikely. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Arsenic - Arsenic is a naturally occurring element widely distributed in the earth's crust. The MRL for arsenic is set at a level meant to protect against non-cancer health effects, specifically dermal lesions (ATSDR 2007). Chronic exposure to low levels of inorganic arsenic can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling. Organic arsenic compounds are less toxic than inorganic arsenic compounds. A NOAEL of 0.0008 mg/kg/day and an uncertainty factor of 3 were used to calculate the oral MRL.

Based on the estimated EPC of arsenic detected in soil and RME scenario, the chronic exposure dose calculated (i.e., 0.0056 mg/kg/day) for children (6 – 11 year) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table A11, Appendix A) and the NOAEL (i.e., 0.0008 mg/kg/day). Based on the estimated EPC of arsenic detected in soil

and CTE scenario, the chronic exposure dose calculated (i.e., 0.00283 mg/kg/day) for children (6 – 11 year) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table A11, Appendix A) and the NOAEL (i.e., 0.0008 mg/kg/day). As such, there is a potential for non-cancer adverse health effects from exposures to arsenic in soil. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Lead - Accumulation of lead in the body can cause damage to the nervous system, gastrointestinal system, kidneys, and red blood cells (ATSDR 2006b). 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). There is no known safe level of lead exposure. The mean concentration of lead detected in the on-site areas (680 mg/kg) exceeded the NJDEP RDCSCC of 400 mg/kg.

Lead exposures associated with children's intermittent recreational use of lead contaminated on-site areas were evaluated using the USEPA's integrated exposure uptake biokinetic (IEUBK) model (USEPA 1994a). The IEUBK model estimates a plausible distribution of blood lead levels centered on the geometric mean blood lead levels from available exposure information. Blood lead levels are indicators of exposure and are also the most widely used index of internal lead body burdens associated with potential health effects. The model also calculates a probability (or P-value) that children's blood lead levels will exceed a reference level (USEPA 1986; CDC 1991). In using the IUEBK model, the USEPA recommends that the lead concentration in site soil does not result in a 5% probability of exceeding a blood lead concentration of 10 µg/dL (USEPA 1994a; 1994b). The average of lead levels in soils (a depth of 0 to 2 feet) was used as an input value to calculate expected children's blood lead levels due to incidental ingestion. It should be noted that there is some uncertainty because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated blood lead level. The assumptions for the recreational exposure scenario for children aged 72 to 84 months are as follows:

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

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

Table 2 shows the predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the CDC reference value of 5 µg/dL (P₅) for children.

Table 2: Predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the CDC reference value for the QRC property. Exposure Scenario based on site visits (219 days/year)^a

Age (months)	Blood Lead Level ^b (µg/dL)	P ₅ (%) ^c
72 to 84	4.5	42.8

^asoil lead concentration 680 ppm (USEPA 2003a); ^bGeometric mean blood lead level in micrograms per deciliter; ^cprobability of blood lead level >5 micrograms per deciliter

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 72 to 84 months were below the CDC reference value of 5 µg/dL; however, the probabilities of blood lead levels exceeding 5 µg/dL for children ages 72 to 84 months exceeded 5 percent (42.8%). Chronic exposure to lead resulting in blood lead levels even below 10 µg/dL have been suggested to show neurological, behavioral, immunological, and developmental effects in young children. A portion of the pediatric population age 72 to 84 months may have had blood lead levels high enough to potentially result in harmful health effects.

The NJDOH and ATSDR recognize that there are currently no established safe levels for blood lead; the one means to reduce lead exposure is to take steps to reduce contact with lead-contaminated sources.

PAHs and Benzo[a]pyrene - PAHs including benzo[a]pyrene 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 combustion processes produce them, PAHs are widespread in the environment. PAHs have been found to exhibit antiandrogenic¹¹ properties in human cell cultures and are implicated in the loss of fertility in males (Kizu 2003). Non-cancer adverse health effects associated with PAH exposures have been observed in animals but generally not in humans (ATSDR 1995). Non-cancer effects are usually seen at much higher levels than found in the environment. The main potential concern for PAH exposures is for cancer effects.

The chronic past exposure doses for children and adults were calculated based on the EPC of PAHs detected in the soil (a depth of 0 to 2 feet) (see Table A11, Appendix A). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available (Table 3).

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

Table 3: Reference Dose for chronic oral exposure, PAHs¹

PAH	NOAEL ² (mg/kg/day)	RfD ³ (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)

¹Polycyclic Aromatic Hydrocarbons; ²No Observed Adverse Effect Level; ³Reference Dose

The RfDs of these PAHs are based on the NOAEL and are higher than the exposure doses calculated for the PAHs detected in the soil. Based on the 95% UCL of arithmetic mean of benzo[b]fluoranthene (the PAH with the highest concentration, 350 mg/kg), the calculated chronic child exposure dose (0.0013 mg/kg/day) was about 15 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table 11A, Appendix A). As such, non-cancer adverse health effects associated with past exposures of PAH contaminated soil at the QRC property are unlikely in children. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Oral exposures to benzo[a]pyrene has been quantitatively linked to developmental (including developmental neurotoxicity), reproductive, and immunotoxicity in animal studies. In humans, the same effects were reported, which provide qualitative, supportive evidence for health effects associated with benzo[a]pyrene. A benchmark dose (BMD) of 0.092 mg/kg-day and a composite uncertainty factor of 300 were used to calculate the oral RfD for benzo[a]pyrene. Based on the estimated EPC of benzo[a]pyrene detected in soil and RME scenario, the chronic exposure dose calculated (i.e., 0.0011 mg/kg/day) for children (6 – 11 year) exceeded the RfD of 0.0003 mg/kg/day (see Table A11, Appendix A). Based on the estimated EPC of benzo[a]pyrene detected in soil and CTE scenario, the chronic exposure dose calculated (i.e., 0.00011 mg/kg/day) for children (6 – 11 year) did not exceed the RfD of 0.0003 mg/kg/day (see Table A11, Appendix A). The exposure dose based on RME scenario was about 80 times less than the BMD. As such, non-cancer adverse health effects from exposures to benzo[a]pyrene in soil is unlikely. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Celotex Property

Based on the maximum concentration of arsenic, chromium, Aroclor-1242, Aroclor-1260 and benzo[a]pyrene detected in the soil and RME scenario, the hazard quotient calculated for children (6 – 11 year) did not exceed 1 (see Table A11, Appendix A); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. Based on the EPC of Aroclor-1254 detected in the soil (0 to 2 feet depth) (see Table A12, Appendix A) and RME scenario, the hazard quotient calculated for children (6 – 11 year) exceeded 1 (see Table A12, Appendix A). The EPC of lead detected in the soil (a depth of 0 to 2 feet) (see Table A12, Appendix A) also exceeded the corresponding health guideline CV. The health guideline CVs of benzo[a]anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer adverse health effects associated with exposures to metals and PAHs are evaluated as follows:

Lead - The non-cancer adverse health effects associated with exposure to lead were discussed earlier in this section. The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value of 5 µg/dL (P₅) for children are shown in Table 4.

Table 4: Predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value for the Celotex property. Exposure Scenario based on site visits (219 days/year)^a

Age (months)	Blood Lead Level ^b (µg/dL)	P ₅ (%) ^c
72 to 84	6.0	65

^asoil lead concentration 951 ppm (USEPA 2003a); ^bGeometric mean blood lead level in micrograms per deciliter; ^cprobability of blood lead level >5 micrograms per deciliter

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 72 to 84 months were below the CDC reference value of 5 µg/dL; however, the probabilities of blood lead levels exceeding 5 µg/dL for children ages 72 to 84 months exceeded 5 percent (65%). Chronic exposure to lead resulting in blood lead levels even below 10 µg/dL have been suggested to show neurological, behavioral, immunological, and developmental effects in young children. A portion of the pediatric population age 72 to 84 months may have had blood lead levels high enough to potentially result in harmful health effects. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Aroclor-1254 – Maximum hazard quotient calculated for children based on Aroclor-1254 detected in the soil is 65 (see Table A12, Appendix A). 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 2000). 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.

Based on the concentration of Aroclor-1254 detected and RME scenario, the chronic exposure dose (i.e., 0.0013 mg/kg/day) exceeded the MRL (see Table A12, Appendix A). The LOAEL for Aroclor-1254 is 0.006 mg/kg/day and is based on ocular exudate, inflamed and prominent meibomian glands of the eye, distorted growth of finger and toe nails and decreased antibody response. The MRL applies an uncertainty factor of 300 to the LOAEL. Based on the Aroclor-1254 concentration detected and RME scenario, the exposure dose was about 5 times lower than the LOAEL. As such, there was a potential for non-cancer health effects in children from exposures to Aroclor-1254 at the Celotex property. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

PAHs - The non-cancer adverse health effects associated with exposure to PAHs were discussed earlier in this section. The chronic exposure doses for children and adults were calculated (see Table A12, Appendix A). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available and presented earlier in this section.

The RfDs of these PAHs are based on the NOAEL and are higher than the exposure doses calculated for the PAHs detected in the soil. For chrysene (the PAH with the highest concentration, 10.4 mg/kg), the calculated chronic child exposure doses (0.0.000039 mg/kg/day) was about 512 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table A12, Appendix A). As such, non-cancer adverse health effects associated with past exposures to PAH contaminated soil at the Celotex property were unlikely in children. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

115 River Road Property¹²

Based on the estimated EPCs and RME scenario, the hazard quotients calculated for arsenic, chromium, Aroclor-1248, Aroclor-1254 and benzo[a]pyrene were less than 1 (see Table A13, Appendix A); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. The EPC of lead detected in the soil (a depth of 0 to 2 feet) (see Table A13, Appendix A) exceeded the corresponding health guideline CV. The health guideline CVs of benzo[a]anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer

¹²Annual vapor intrusion monitoring continues at 115 River Road, 163 Old River Road and 103 River Road.

adverse health effects associated with exposures to lead and PAHs are evaluated as follows:

Lead - The non-cancer adverse health effects associated with exposure to lead were discussed earlier in this section. The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value of 5 µg/dL (P₅) for children are shown in Table 5.

Table 5: Predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value for the 115 River Road property. Exposure Scenario based on site visits (219 days/year)^a

Age (months)	Blood Lead Level ^b (µg/dL)	P ₅ (%) ^c
72 to 84	3.9	30.4

^asoil lead concentration 557 ppm (USEPA 2003a); ^bGeometric mean blood lead level in micrograms per deciliter; ^cprobability of blood lead level >5 micrograms per deciliter

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 72 to 84 months were below the CDC reference value of 5 µg/dL; however, the probabilities of blood lead levels exceeding 5 µg/dL for children ages 72 to 84 months exceeded 5 percent (30.4%). Chronic exposure to lead resulting in blood lead levels even below 10 µg/dL have been suggested to show neurological, behavioral, immunological, and developmental effects in young children. A portion of the pediatric population age 72 to 84 months may have had blood lead levels high enough to potentially result in harmful health effects. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

PAHs - The non-cancer adverse health effects associated with exposure to PAHs were discussed earlier in this section. The chronic exposure doses for children and adults were calculated (see Table A13). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available and presented earlier in this section.

The RfDs of these PAHs are based on the NOAEL and are higher than the exposure doses calculated for the PAHs detected in the soil. For benzo[k]fluoranthene (the PAH with the highest concentration, 160 mg/kg), the calculated chronic child exposure dose (0.00006 mg/kg/day) was about 33 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table A13, Appendix A). As such, non-cancer adverse health effects associated with past exposures of PAH contaminated soil at the 115 River Road property were unlikely in children and adults. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Lever Brothers Property

Based on the estimated EPCs and RME scenario, the hazard quotients calculated for arsenic, Aroclor-1254, Aroclor-1260 and benzo[a]pyrene for children were lower than 1 (see Table A14, Appendix A); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. The health guideline CVs of benzo[a]anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer adverse health effects associated with exposures PAHs are evaluated as follows:

PAHs - The non-cancer adverse health effects associated with exposure to PAHs were discussed earlier in this section. The chronic exposure doses for children and adults were calculated (see Table A14, Appendix A). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available and presented earlier in this section.

The RfDs of these PAHs are based on the NOAEL and are higher than the exposure doses calculated for the PAHs detected in the soil. For benzo[b]fluoranthene (the PAH with the highest concentration, 18 mg/kg), the calculated chronic child exposure dose (0.000068 mg/kg/day) was about 300 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table A14, Appendix A). As such, non-cancer adverse health effects associated with past exposures of PAH contaminated soil at the Lever Brothers property were unlikely in children and adults. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may underestimate the calculated exposure risk.

Block 93 Property

Based on the estimated EPCs and RME scenario, the hazard quotients calculated for mercury, Aroclor-1248, Aroclor-1254, Aroclor-1260, fluoranthene and naphthalene for children were lower than 1 (see Table A15, Appendix A); as such, past exposures to these contaminants are unlikely to cause non-cancer adverse health effects. Based on the estimated EPCs and RME scenario (see Table A15, Appendix A), the hazard quotients calculated for arsenic and benzo[a]pyrene were 4.4 and 1.5, respectively. The health guideline CVs of benzo[a]anthracene, , benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene are unavailable; they were retained for further evaluation. The non-cancer adverse health effects associated with exposures to arsenic, lead and PAHs are evaluated as follows:

Arsenic - The non-cancer adverse health effects associated with arsenic have been discussed earlier in this section. A NOAEL of 0.0008 mg/kg/day and an uncertainty factor of 3 were used to calculate the oral MRL for arsenic. The chronic exposure dose calculated for children (i.e., 0.0013 mg/kg/day) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table A15, Appendix A). The calculated child exposure dose is about 16 times higher than the NOAEL (i.e., 0.0008 mg/kg/day). As such, non-cancer adverse health effects from

exposures to arsenic in soil were possible. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Lead - The non-cancer adverse health effects associated with exposure to lead were discussed earlier in this section. The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value of 5 µg/dL (P₅) for children are shown in Table 6:

Table 6: Predicted geometric mean blood lead levels and the probability of blood lead levels exceeding the current reference value for the Block 93 property. Exposure Scenario based on site visits (219 days/year)^a

Age (months)	Blood Lead Level ^b (µg/dL)	P ₅ (%) ^d
72 to 84	10.65	94.67

^asoil lead concentration 1960 ppm (USEPA 2003a); ^bGeometric mean blood lead level in micrograms per deciliter; ^cprobability of blood lead level >5 micrograms per deciliter

For the incidental lead ingestion exposure scenario, the model predicted that the geometric mean blood lead levels for children ages 72 to 84 months exceeded the CDC reference value of 5 µg/dL; the probabilities of blood lead levels exceeding 5 µg/dL for children ages 72 to 84 months exceeded 5 percent (94.67%). Chronic exposure to lead resulting in blood lead levels even below 10 µg/dL have been suggested to show neurological, behavioral, immunological, and developmental effects in young children. A portion of the pediatric population age 72 to 84 months may have had blood lead levels high enough to potentially result in harmful health effects. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

PAHs and Benzo[a]pyrene - The non-cancer adverse health effects associated with exposure to PAHs were discussed earlier in this section. The chronic exposure dose for children were calculated (see Tables A15, Appendix A). No health guideline CVs are available for these PAHs; however, the NOAEL, RfD, and associated critical health effects for a number of PAHs are available and presented earlier in this section.

The RfDs of these PAHs are based on the NOAEL and are higher than the exposure doses calculated for the PAHs detected in the soil. For benzo[a]anthracene (the PAH with the highest concentration, 230 mg/kg), the calculated chronic child exposure doses (0.00087 mg/kg/day) was about 23 times lower than the lowest reported RfD for a related PAH (i.e., 0.02 mg/kg/day for naphthalene) (see Table A15, Appendix A). As such, non-cancer adverse health effects associated with past exposures of PAH contaminated soil at the Lever Brothers property were unlikely in children and adults. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

The health effects and the toxicological data of benzo[a]pyrene was summarized earlier in the document. Based on the estimated EPC of benzo[a]pyrene detected in soil and RME scenario, the chronic exposure dose calculated (i.e., 0.00045 mg/kg/day) for children (6 – 11 year) exceeded the RfD of 0.0003 mg/kg/day (see Table A15, Appendix A). The exposure dose based on RME scenario was about 200 (i.e., 0.092/0.00045) times less than the BMD. As such, non-cancer adverse health effects from exposures to benzo[a]pyrene in soil is unlikely. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Inhalation – Indoor Air at 115 River Road (present)

The risks of non-cancer health effects for occupants of the child care center and the office space located at 115 River Road associated with exposure to contaminants were assessed by comparing the 24-hour time-weighted average indoor air EPCs with health guideline CVs (see Table A16, Appendix A). The estimated indoor air tetrachloroethene EPC (i.e., 0.6 $\mu\text{g}/\text{m}^3$) at the Child Care Center was lower than its corresponding health guideline CV (i.e., MRL = 41 $\mu\text{g}/\text{m}^3$), and, therefore, is unlikely to cause adverse non-cancer health effects (see Table A16, Appendix A).

The estimated 24-hour time-weighted average indoor air EPCs of benzene, carbon tetrachloride chloroform, 1,4-dichlorobenzene, tetrachloroethene, and trichloroethene at the office space were lower than their corresponding health guideline CVs (see Table A16, Appendix A), and, therefore, are unlikely to cause adverse non-cancer health effects. The estimated EPC of acrolein at the office space exceeded its health guideline CVs (see Table A16, Appendix A). The health effects associated with acrolein exposure were evaluated as follows:

Acrolein: The estimated 24-hour time-weighted average indoor air EPC of acrolein (0.58 $\mu\text{g}/\text{m}^3$) was higher than the corresponding health guideline CV (see Table A16, Appendix A) (0.02 $\mu\text{g}/\text{m}^3$). Acrolein is a colorless or yellow liquid with a disagreeable odor; it is used as a pesticide to control algae, weeds, bacteria, and mollusks. Animal studies show that breathing acrolein causes irritation to the nasal cavity, lowers breathing rate, and causes damage to the lining of the lungs. A LOAEL of 20 $\mu\text{g}/\text{m}^3$ and an uncertainty factor of 1,000 were used to calculate the inhalation RfC for acrolein.

The estimated 24-hour time-weighted average indoor air EPC of acrolein is about 35 times lower than the LOAEL (20 $\mu\text{g}/\text{m}^3$). As such, non-cancer adverse health effects from exposures to acrolein in indoor air of office space of 115 River Road property are unlikely.

A summary of contaminants in soil having potential for non-cancer health effects is presented in Table 7.

Table 7: Summary of Non-Cancer Health Evaluation – Potential for Health Effects

Exposure Area	Antimony	Arsenic	Lead	PAHs	PCBs
QRC Property	possible	possible	possible	np	np
Celotex	np	np	possible	np	possible
115 River Road	np	np	possible	np	np
Lever Brothers	np	np	np	np	np
Block 93	np	possible	possible	np	np

np=contaminant exposures not above Health Guideline CVs or unlikely to occur based on comparison to NOAEL or LOAEL.

Cancer Health Effects

The site-specific lifetime excess cancer risk (LECR) indicates the cancer-causing potential of contaminants. LECR estimates are usually expressed in terms of excess cancer cases in an exposed population in addition to the background rate of cancer. For perspective, the lifetime risk of being diagnosed with cancer in the United States is 44 per 100 individuals for males, and 38 per 100 for females; the lifetime risk of being diagnosed with any of several common types of cancer ranges between one in 10 and one in 100 (ACS 2018). Typically, health guideline CVs developed for carcinogens are based on one excess cancer case per one million individuals (10^{-6}).

The U.S. Department of Health and Human Services (USDHHS) has assigned cancer classes for contaminants found in hazardous waste site. The cancer classes are defined as follows:

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

Incidental ingestion of contaminated soil from Quanta Resources and adjacent properties (past)

Quanta Resources Property

The cancer classes of the COPCs detected in the soil (a depth of 0 to 2 feet) of the Quanta Resources Property are given in Table A17 (Appendix A). Arsenic, cadmium, chromium and PAHs were the carcinogens found in soil. Exposure doses for cancer evaluation were calculated using the following formula:

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

where, C = concentration of contaminant in soil (mg/kg);¹³

IR = soil ingestion rate (mg/day);

EF = exposure factor representing the site-specific exposure scenario;¹⁴

ED = exposure duration (year);

BW = body weight (kg); and,

AT = averaging time (year).

The California EPA's Office of Environmental Health Hazard Assessment (OEHHA) has developed a relative potency estimate approach for PAHs (OEHHA 2016). Using this approach, the cancer potency of carcinogenic PAHs can be estimated based on their relative potency with reference to benzo[a]pyrene¹⁵. For each of the carcinogenic PAHs, the benzo[a]pyrene equivalents were calculated by multiplying the mean concentration detected with the cancer potency factor (see Table A17). The total benzo[a]pyrene equivalents was obtained by summing each of the individual benzo[a]pyrene equivalents (ATSDR 2016). The USEPA also proposed that cancer risk for chemicals that act with a mutagenic mode of action (MOA) for carcinogenesis can be quantified using age-dependent adjustment factors (ADAFs). The ADAFs are factors by which cancer risk is multiplied to account for increased susceptibility to mutagenic compounds early in life – standard ADAFs are 10 (for ages below 2 years old), 3 (for ages 2 up to 16 years old), and 1 (for ages greater than 16) (ATSDR 2016). Using site specific 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 associated with ingestion of contaminated soil (a depth of 0 to 2 feet) during recreational activities were evaluated (see Table A17, Appendix A). Based on the EPC of the contaminants detected in soil and RME scenario, the calculated cumulative LECRs showed an increase in the estimated cumulative cancer risk (three excess cancer cases in 1,000 individuals) who were exposed to contaminated soil (0 to 2 feet depth). It should be

¹³See Appendix C for sample calculation.

¹⁴An exposure scenario of 219 days/year and a lifetime exposure duration of 30 years was assumed based on default residency time.

¹⁵Dibenz[a,h]anthracene has its own cancer slope factor

noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Celotex Property

LECRs associated with ingestion of contaminated soil (a depth of 0 to 2 feet) during recreational activities were evaluated (see Table A18, Appendix A). Based on the EPCs and RME scenario, the calculated LECRs showed a low estimated cumulative cancer risk (seven excess cancer cases in 10,000 individuals exposed including children) who were exposed to contaminated soil (0 to 2 feet depth). It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

115 River Road Property

LECRs associated with ingestion of contaminated soil (0 to 2 feet depth) during recreational activities were evaluated (see Table A19, Appendix A). Based on the EPCs and RME scenario, the calculated LECRs showed a low estimated cumulative cancer risk (one excess cancer case in 10,000 individuals exposed including children) who were exposed to contaminated soil (a depth of 0 to 2 feet). It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Lever Brothers Property

LECRs associated with ingestion of contaminated soil (a depth of 0 to 2 feet) during recreational activities were evaluated (see Table A20, Appendix A). Based on the EPCs and RME scenario, the calculated LECRs showed low estimated cumulative cancer risk (one excess cancer case in 10,000 individuals exposed including children) who were exposed to contaminated soil (0 to 2 feet depth). It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Block 93 Property

LECRs associated with ingestion of contaminated soil (a depth of 0 to 2 feet) during recreational activities were evaluated (see Table A21, Appendix A). Based on the EPCs, the calculated LECRs showed increase in the estimated cumulative cancer risk (one excess cancer case in 1,000 individuals exposed including children) who were exposed to contaminated soil (a depth of 0 to 2 feet).

Based on the concentrations of PAHs, PCBs, benzene and arsenic detected in the soil and RME scenario, the calculated cumulative highest LECR and highest LECR for

mutagenic contaminants exceeded the cancer risk of one excess cancer case in 1,000,000 (see Table A17 through A21, Appendix A). The highest LECRs are associated with PAHs, benzene and arsenic detected in the on-site area and Aroclor 1254 detected in the Celotex site. The cancer health effects associated with exposures to these contaminants are evaluated as follows:

Polycyclic Aromatic Hydrocarbons (PAHs): The USDHHS has determined that some PAHs may reasonably be expected to be carcinogens. Chronic exposure to mixture of PAHs via inhalation and dermal contact have been found to cause cancer. In laboratory animal tests, some PAHs have caused cancer via inhalation (lung cancer), ingestion (stomach cancer), or dermal contact (skin cancer). Using RME and CTE conditions, the calculated LECR (15 years of exposure as children) for dibenz[a,h]anthracene and BaP and equivalent detected in the on-site areas ranged from 3×10^{-4} to 4.8×10^{-4} and 1.5×10^{-4} to 2.4×10^{-4} , respectively (see Table A22, Appendix A). The CTE and RME cancer risks associated with dibenz[a,h]anthracene and BaP and equivalent exposure for adults exposed for 9 years (i.e., the mean residential occupancy period) and adults exposed for 33 years (i.e., the 95% residential occupancy period) are also presented in the table. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Polychlorinated Biphenyls (PCBs): PCBs are classified as probably carcinogenic, and carcinogenic to humans (group 1) by the USEPA and IARC, respectively. The DHHS has concluded that PCBs may reasonably be anticipated to be carcinogens. Occupational exposures indicate that PCBs were associated with cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. Using RME and CTE conditions, the calculated LECR (15 years of exposure as children) for PCBs detected in the former Celotex property are 3.4×10^{-4} and 1.7×10^{-4} , respectively (see Table A22, Appendix A). The CTE and RME cancer risks associated with benzene exposure for adults exposed for 9 years (i.e., the mean residential occupancy period) and adults exposed for 33 years (i.e., the 95% residential occupancy period) are also presented in the table. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Benzene: The IARC and the EPA have determined that benzene is carcinogenic to humans. Long-term exposure to high levels of benzene in the air can cause leukemia, particularly acute myelogenous leukemia, often referred to as AML. Using reasonable mean exposure (RME) and central tendency exposure (CTE) conditions, the calculated LECR (15 years of exposure as children) for benzene detected in the on-site areas 2.4×10^{-4} and 1.2×10^{-4} , respectively (see Table A22, Appendix A). The CTE and RME cancer risks associated with benzene exposure for adults exposed for 9 years (i.e., the mean residential occupancy period) and adults exposed for 33 years (i.e., the 95% residential occupancy period) are also presented in the table. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

Arsenic: The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans. Ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. Inhalation of inorganic arsenic can cause increased risk of lung cancer. The Department of Health and Human Services (DHHS) and the USEPA have determined that inorganic arsenic is a known human carcinogen. Using reasonable mean exposure (RME) and central tendency exposure (CTE) conditions, the calculated LECR (15 years of exposure as children) for benzene detected in the on-site areas 1.1×10^{-3} and 5.5×10^{-4} , respectively (see Table A22, Appendix A). The CTE and RME cancer risks associated with benzene exposure for adults exposed for 9 years (i.e., the mean residential occupancy period) and adults exposed for 33 years (i.e., the 95% residential occupancy period) are also presented in the table. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (*a depth of 0 to 3 inches*) conditions. This may under- or overestimate the calculated exposure risk.

Inhalation – Indoor Air at 115 River Road (past, present, and future)

The inhalation LECRs associated with indoor air exposures were calculated by using the following formula:

$$\text{Inhalation Cancer Risk} = C \times EF \times \frac{ED}{AT} \times IUR$$

where C = concentration of contaminant in air ($\mu\text{g}/\text{m}^3$);
EF = exposure factor representing the site-specific exposure scenario;
ED = exposure duration (year);
AT = averaging time;
IUR = inhalation unit risk ($\mu\text{g}/\text{m}^3$)⁻¹.

Inhalation unit risk (IUR) is defined as the upper-bound lifetime excess cancer risk estimated to result from continuous exposure to an agent at a concentration of $1 \mu\text{g}/\text{m}^3$ in air.

Palisades Child Care Center: LECRs associated with indoor air were evaluated (see Table A23, Appendix A). Based on the EPC at the Child Care Center located at 115 River Road property, the calculated LECRs showed no expected increase in the estimated cancer risk (one excess cancer case in 100 million individuals exposed including children) who were exposed to contaminants in indoor air at the Child Care Center.

Office Space: LECRs associated with indoor air were evaluated (see Table A23, Appendix A). Based on the EPCs at the office space located at 115 River Road property, the calculated LECRs showed low increase in the estimated cumulative cancer risk (five excess cancer cases in one million individuals exposed including children) who were exposed to contaminants in indoor air of office space.

Child Health Considerations

The NJDOH and ATSDR recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination in their environment. Children are at greater risk than adults from certain types of exposures to hazardous substances. Their lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most important, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

The NJDOH and ATSDR evaluated the potential risk for children resided in the area who may have been exposed to site contaminants. Past child exposure doses associated with antimony, arsenic, PAHs and PCBs detected in the soil exceeded the health guideline CVs. Based on the CDC reference value of 5 µg/dL of lead in blood, adverse health effects associated with past lead exposures were also possible. The adverse health effects from contaminants detected in the indoor air at the child care center were unlikely.

Based on the EPC of contaminants detected in the soil of the QRC and adjacent properties, a cumulative LECR as high as two excess cancer cases in 1,000 was determined for area residents (including children) from past exposures. This exposure posed an increase in cancer risk.

Health Outcome Data

A review of health outcome data (e.g., adverse pregnancy outcomes, cancers, deaths) is conducted to assess the public health impact of completed exposure pathways. However, the size of the exposed population is relatively small, and an evaluation of available health data would be unlikely to produce statistically reliable findings.

Public Comment

The NJDOH held a public comment period from March 4, 2015 through April 5, 2015 to provide an opportunity for interested parties to comment on the draft Public Health Assessment prepared for the Quanta Resources Corporation Site. Written comments were received from two organizations during the public comment period and the comments and responses are given in Appendix D.

Conclusions

Manufacturing activities at the QRC and adjacent properties and uncontrolled release of hazardous wastes have resulted in the contamination of soil and groundwater. There were completed exposure pathways via the incidental ingestion of contaminated soil (a depth of 0 to 2 feet) in the past and inhalation of indoor air. Contaminants of potential concern are metals, VOCs and SVOCs in the soil (a depth of 0 to 2 feet) and VOCs in the indoor air. The exposed population included area residents, trespassers and occupants of

115 River Road property (i.e., the Child Care Center and the office space). The ATSDR and NJDOH have reached the following conclusions in this report:

The NJDOH and ATSDR conclude that currently there are no ongoing exposures from ingestion of soil contaminants (collected at a depth of 0 to 2 feet) at the Quanta Resources Corporation and adjacent properties that can harm people's health.

Contaminated soils have been fenced, excavated and/or capped. The excavated areas were backfilled with clean fill. Thus, area residents are not being exposed to site-related contaminants via the ingestion pathway. However, it should be noted that although the interim remedial measures interrupted the exposure pathways, the preferred remedy for the contaminated media has not been implemented yet. It should be noted that there is some uncertainty¹⁶ in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

The NJDOH and ATSDR conclude that past exposures to site-related contaminants detected in the soil may have harmed people's health. Based on the contaminants detected in the soil (a depth of 0 to 2 feet), the potential for non-cancer adverse health effects associated with past exposures to arsenic, lead and PCBs were possible in children and adults. Maximum cumulative LECRs were associated with the contaminants detected in the soil (a depth of 0 to 2 feet) and estimated to be as high as three excess cancer cases in 1,000 to the exposed population. This exposure poses an increase in cancer risk, compared to the background risk of cancer from all causes. It should be noted that there is some uncertainty in this conclusion because the soil sampling data were from 0 to 2 feet, which may not represent actual soil (a depth of 0 to 3 inches) conditions. This may under- or overestimate the calculated exposure risk.

The NJDOH and ATSDR conclude that there were no indoor air exposures to contaminants at the Child Care Center that could have harmed children's health. The adjusted EPC of tetrachloroethene detected in the indoor air did not exceed the health guideline CV. The calculated LECRs showed no increase in the estimated cancer risk (one excess cancer case in 100 million individuals exposed including children). Following indoor air sampling, the NJDOH and ATSDR recommend maintaining changes to the building ventilation system. The Palisades Child Care Center has relocated to an off-site building in 2012 and currently the building is being demolished.

The NJDOH and ATSDR conclude that there were no indoor air exposures to contaminants at the office space that could have harmed people's health. The indoor air adjusted-EPC of acrolein is about 35 times lower than the LOAEL; as such, non-cancer adverse health effects from exposures to acrolein are unlikely. The calculated LECRs showed low estimated cumulative cancer risk (five excess cancer cases in one million individuals exposed including children).

¹⁶ATSDR considers the depth to which soil is likely to be contacted directly is 0-3 inches which differs from the USEPA who considers this depth to be 0-2 feet.

The NJDOH and ATSDR cannot conclude if exposure to the sediment and surface water of the Hudson River harmed people's health. The USEPA will evaluate the Hudson River sediment and surface water contamination attributable to the site as a separate operable unit in the future.

Recommendations

The NJDOH and ATSDR recommend maintaining site access restriction to ensure integrity of the remedies and to prevent access by area residents and trespassers. OU1 remediation is currently underway. It is recommended that the RI/FS for the OU2 and the overall remedy for the entire site be completed as soon as feasible.

Public Health Action Plan (PHAP)

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

Public Health Actions taken by NJDOH and ATSDR

1. Contamination data collected from the Quanta Resources Corporation site were evaluated by the NJDOH and ATSDR.
2. Representatives of the NJDOH conducted several site visits of the Quanta Resources Corporation.
3. Representatives of NJDOH and ATSDR attended several public meetings and availability sessions.

Public Health Actions Planned by NJDOH and ATSDR

1. Copies of this Public Health Assessment will be made available to concerned residents via the township library and the NJDOH and ATSDR websites.
2. The NJDOH and ATSDR will hold a public availability session to present the findings of this PHA.
3. With the oversight of USEPA, the remedial investigation of the sediment and surface water (i.e., OU2) will be conducted by the potential responsible parties. The NJDOH, in cooperation with the ATSDR, will prepare separate health consultation(s) to evaluate the data.

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Report Preparation

This Public Health Assessment for the Quanta Resources Corporation site, located in Edgewater Borough in Bergen County, New Jersey was prepared by the New Jersey Department of Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented. ATSDR's approval of this document has been captured in an electronic database.

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Appendix A: Tables and Figures

Table A1: Surface Soil (0-2 ft depth) Sampling Results of the Quanta Resources property

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Aluminum	570	7,630	57,000 (EMEG ^d)	No
Antimony	1.2	174	23 (RMEG ^e)	Yes
Arsenic	0.0072	3,900	0.25 (CREG ^f)	Yes
Barium	60.2	305	11,000 (EMEG)	No
Beryllium	0.14	0.53	110 (EMEG)	No
Cadmium	0.67	10.1	5.7 (EMEG)	Yes
Chromium, Trivalent	22.9	24.6	86,000 (RMEG)	No
Hexavalent Chromium	0.81	0.81	51 (EMEG)	No
Cobalt	6.3	42.8	570 (EMEG I ^g)	No
Copper	124	6,170	3,100 (RSL)	Yes
Lead	0.069	7,730	400 (RDCSCC ^h)	Yes
Manganese	54.3	306	2,900 (RMEG)	No
Nickel	5.6	37.8	1,100 (RMEG)	No
Selenium	1.6	27.9	290 (EMEG)	No
Silver	0.47	24.3	290 (RMEG)	No
Thallium	1.7	8.2	NA ⁱ	-
Vanadium	7.7	82.2	390 (RSL ^j)	No
Zinc	139	3,820	17,000 (EMEG)	No
Mercury	2	29.5	14 (RDCSCC)	Yes
4,4'-DDD	0.029	0.56	1.6 (CREG)	No
4,4'-DDE	0.3	1.1	1.1 (CREG)	No
4,4'-DDT	0.078	0.35	1.1 (CREG)	No
Endrin ketone	0.074	0.27	17 (EMEG)	No
Heptachlor	0.35	0.35	0.083 (CREG)	Yes
Methoxychlor	0.32	1.3	290 (RMEG)	-No
Aroclor-1242	0.11	3.2	1.1 (CREG)	Yes
Aroclor-1254	0.047	0.702	1.1 (CREG)	No
Aroclor-1260	0.0073	1.6	1.1 (CREG)	Yes
2-Butanone	0.009	0.015	34,000 (RMEG)	No
Acetone	0.015	1.6	51,000 (RMEG)	No
Acetophenone	254	2.8	5,700 (RMEG)	No
Benzene	0.0007	19	6.8 (CREG)	Yes
Carbon Disulfide	0.001	0.28	5,700 (RMEG)	No
Chloroform	0.003	0.003	570 (EMEG)	No
Cyclohexane	0	0.003	6,500 (RSL)	No
Ethylbenzene	0	50	5,000 (RMEG)	No
Isopropyl Benzene	0.23	3.3	NA	-
Methyl Acetate	0.157	1.1	78,000 (RSL)	No
Methylcyclohexane	0.006	0.006	NA	-

Table A1: (Cont'd.)

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Tetrachloroethene	0.006	0.52	180 (CREG)	No
Toluene	0.002	42	4,600 (RMEG)	No
Trichloroethene	0.005	0.54	5.7 (CREG)	No
Trichlorofluoromethane	12	12	17,000 (RMEG)	No
Xylene (Total)	0.001	21	11,000 (EMEG)	No
1,2-Dichlorobenzene	2.9	2.9	17,000 (EMEG)	No
2,4-Dimethylphenol	1.13	120	1,100 (RMEG)	No
2,4-Dinitrotoluene	17	17	57 (EMEG)	No
2-Methylnaphthalene	0.081	1,200	2,300 (EMEG)	No
2-Methylphenol	0.195	110	2,900 (RMEG)	No
3&4-Methylphenol	0.755	8.84	2,900 (RMEG)	No
4-Methylphenol	2.7	250	2,900 (RMEG)	No
4-Nitroaniline	3.6	29	NA	-
Acenaphthene	0.065	1,300	3,400 (RMEG)	No
Acenaphthylene	0.19	280	NA	-
Anthracene	0.29	940	17,000 (RMEG)	No
Benzo[a]anthracene	1.2	990	0.16 (RSL)	Yes
Benzo[a]pyrene	1.2	941	0.016 (RSL)	Yes
Benzo[b]fluoranthene	1.7	1,100	0.16 (RSL)	Yes
Benzo[g,h,i]perylene	0.87	553	NA	-
Benzo[k]fluoranthene	0.57	632	1.6 (RSL)	Yes
1,1'-Biphenyl	0.042	220	29,000 (RMEG)	No
bis(2-Ethylhexyl)phthalate	0.34	26	35 (RDCSCC)	No
Butylbenzylphthalate	1.13	3.8	11,000 (RMEG)	No
Carpolectum	1.2	1.2	3,100 (RSL)	No
Carbazole	0.089	520	24 (RDCSCC)	Yes
Chrysene	1.2	965	62 (RDCSCC)	Yes
Dibenzo[a,h]anthracene	0.24	220	0.016 (RSL)	Yes
Dibenzofuran	0.052	820	NA	-
Fluoranthene	2.1	2,400	2,300 (RMEG)	Yes
Fluorene	0.066	1,300	2,300 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.74	540	0.16 (RSL)	Yes
Naphthalene	0.24	6,200	1,100 (RMEG)	Yes
Phenanthrene	0.91	4,100	NA	-
Phenol	0.085	190	17,000 (RMEG)	No
Pyrene	2.1	1,700	1,700 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fATSDR Cancer Risk Evaluation Guide for chronic exposure; ^gATSDR Reference Media Evaluation Guide for intermediate exposure for child; ^hNJDEP Residential Direct Contact Soil Cleanup Criteria; ⁱNot available; ^jRegional Screening Level

Table A2: Surface Soil (0-2 ft depth) Sampling Results of the Celotex property

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Aluminum	9,340	19,100	57,000 (EMEG ^d)	No
Antimony	1.05	1.51	23 (RMEG ^e)	No
Arsenic	24.2	167	0.25 (CREG ^f)	Yes
Beryllium	525	1.01	110 (EMEG)	No
Cadmium	0.263	1.49	5.7 (EMEG)	No
Hexavalent Chromium	11.9	153	51 (EMEG)	Yes
Cobalt	7.09	209	570 (EMEG I ^g)	No
Copper	19.2	163	3,100 (RSL ^h)	No
Iron	15,300	38,100	55,000 (RSL)	No
Lead	15.6	951	400 (RDCSCC ⁱ)	Yes
Manganese	200	582	2,900 (RMEG)	No
Nickel	13.6	38.4	1,000 (RMEG)	No
Selenium	21	3.02	290 (EMEG)	No
Silver	0.55	6.08	290 (RMEG)	No
Thallium	0.121	1.37	NA ^j	-
Vanadium	24.1	57.3	390 (RSL)	No
Zinc	38.4	349	17,000 (RMEG)	No
Mercury	0.02	2.64	14 (RDCSCC)	No
4,4'-DDD	0.00451	0.011	1.6 (CREG)	No
4,4'-DDE	0.00845	0.00845	1.1 (CREG)	No
Chlordane (technical)	0.00433	0.018	34 (EMEG)	No
Endosulfan II	0.00996	0.00996	290 (EMEG)	No
Aroclor-1242	0.637	0.637	1.1 (CREG)	Yes
Aroclor-1254	0.045	348	1.1 (CREG)	Yes
Aroclor-1260	0.029	0.621	1.1 (CREG)	Yes
Ethylbenzene	0.756	0.756	5,000 (RMEG)	No
Methyl Acetate	1.64	1.74	78,000 (RSL)	No
Tetrachloroethene	0.459	459	180 (CREG)	No
Xylene (Total)	0.13	1.79	11,000 (EMEG)	No
2-Methylnaphthalene	0.08	248	2,300 (EMEG)	No
Acenaphthene	0.085	3.36	3,400 (RMEG)	No
Acenaphthylene	0.076	0.705	NA	-
Anthracene	0.124	4.59	17,000 (RMEG)	No
Benzo[a]anthracene	0.063	10.4	0.16 (RSL)	Yes
Benzo[a]pyrene	0.083	8.89	0.016 (CREG)	Yes
Benzo[b]fluoranthene	0.206	0.962	0.16 (RSL)	Yes
Benzo[g,h,i]perylene	0.136	5.71	NA	-
Benzo[k]fluoranthene	0.071	5.88	1.6 (RDCSCC)	Yes
1,1-Biphenyl	0.145	0.621	29,000 (RSL)	No
bis(2-Ethylhexyl)phthalate	0.083	1.13	35 (RDCSCC)	No
Butylbenzylphthalate	0.066	1.23	11,000 (RMEG)	No

Table A2: (Cont'd.)

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Carbazole	0.084	2.32	24 (RDCSCC)	No
Chrysene	0.102	10.4	62 (RDCSCC)	Yes
Dibenzo[a,h]anthracene	0.069	2.17	0.016 (RSL)	Yes
Dibenzofuran	0.135	1.85	NA	-
Di-n-butylphthalate	0.064	0.319	5,000 (RMEG)	No
Fluoranthene	0.104	20.8	2,300 (RMEG)	No
Fluorene	0.076	2.49	2,300 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.123	5.07	0.16 (RSL)	Yes
Naphthalene	0.08	14.5	1,100 (RMEG)	No
Phenanthrene	0.064	19.3	NA	-
Pyrene	0.098	18	1,700 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fATSDR Cancer Risk Evaluation Guide for chronic exposure; ^gATSDR Reference Media Evaluation Guide for intermediate exposure for child; ^hRegional Screening Level; ⁱNJDEP Residential Direct Contact Soil Cleanup Criteria; ^jNot available

,Table A3: Surface Soil (0-2 ft depth) Sampling Results of 115 River Road property

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Arsenic	5.3	13	0.25 (CREG ^d)	Yes
Chromium	9.2	101	51 (RMEG ^e)	Yes
Lead	39.1	557	400 (RDCSCC ^f)	Yes
Aroclor-1248	0.47	0.47	1.1 (CREG)	Yes
Aroclor-1254	0.047	0.79	1.1 (CREG)	Yes
Aroclor-1260	0.11	0.12	1.1 (CREG)	No
Acetone	0.016	0.058	51,000 (EMEG ^g)	No
Benzene	0.0008	0.004	6.8 (CREG)	No
Carbon Disulfide	0.002	0.003	5,700 (RMEG)	No
Cyclohexane	0.002	0.002	6,500 (RSL ^h)	No
Ethylbenzene	0.097	0.097	5,000 (RMEG)	No
Isopropylbenzene	0.035	0.035	NA ⁱ	-
Methylcyclohexane	0.014	0.014	NA	-
Tetrachloroethene	0.003	0.003	180 (CREG)	No
Toluene	0.002	0.002	4,600 (RMEG)	No
Xylene (Total)	0.002	0.22	11,000 (EMEG)	No
2-Methylnaphthalene	0.075	4.4	2,300 (EMEG)	No
Acenaphthene	0.051	4.6	3,400 (RMEG)	No
Acenaphthylene	0.3	1.4	NA	-
Anthracene	0.13	12	17,000 (RMEG)	No
Benzo[a]anthracene	0.26	28	0.16 (RSL)	Yes
Benzo[a]pyrene	0.25	25	0.016 (CREG)	Yes
Benzo[b]fluoranthene	0.36	31	0.16 (RSL)	Yes
Benzo[g,h,i]perylene	0.22	15	NA	-
Benzo[k]fluoranthene	0.16	16	1.6 (RDCSCC)	Yes
1,1'-Biphenyl	0.054	0.68	29,000 (RMEG)	No
bis(2-Ethylhexyl)phthalate	0.26	2.9	35 (RDCSCC)	No
Butylbenzylphthalate	27	27	11,000 (RMEG)	No
Carbazole	0.36	3.7	24 (RDCSSCC)	No
Chrysene	0.39	25	62 (RDCSCC)	No
Dibenzo[a,h]anthracene	0.06	4.6	0.016 (RSL)	Yes
Dibenzofuran	0.13	2.1	NA	-
Fluoranthene	0.53	56	2,300 (RMEG)	No
Fluorene	0.069	4	2,300 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.18	15	0.16 (RSL)	Yes
Naphthalene	0.097	12	1,100 (RMEG)	No

Table A3: (Cont'd.)

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
N-Nitrosodiphenylamine	0.12	0.12	76 (CREG)	No
Phenanthrene	0.61	36	NA	-
Pyrene	0.48	47	1,700 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Cancer Risk Evaluation Guide for chronic exposure; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fNJDEP Residential Direct Contact Soil Cleanup Criteria; ^gATSDR Environmental Media Evaluation Guide for chronic exposure; ^hRegional Screening Level; ⁱNot available

Table A4: Surface Soil (0-2 ft depth) Sampling Results of the Lever Brothers property

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Arsenic	5.3	45.3	0.25 (CREG ^d)	Yes
Chromium (III)	14.2	28.9	86,000 (RMEG ^e)	No
Hexavalent Chromium	0.6	4	51 (RMEG)	No
Lead	26.9	166	400 (RDCSCC ^f)	No
Aroclor-1254	0.017	0.37	1.1 (CREG)	Yes
Aroclor-1260	0.014	0.1	1.1 (CREG)	Yes
Acetone	0.019	0.054	51,000 (EMEG ^g)	No
Benzene	0.0006	0.0009	6.8 (CREG)	No
Carbon disulfide	0.003	0.003	5,700 (RMEG)	No
2-Methylnaphthalene	0.25	0.47	2,300 (EMEG)	No
Acenaphthene	0.043	3.5	3,000 (RMEG)	No
Acenaphthylene	0.1	1.2	NA ^h	-
Anthracene	0.13	7.1	17,000 (RMEG)	No
Benzo[a]anthracene	0.49	20	0.16 (RSL ⁱ)	Yes
Benzo[a]pyrene	0.58	20	0.016 (CREG)	Yes
Benzo[b]fluoranthene	0.77	26	0.16 (RSL)	Yes
Benzo[g,h,i]perylene	0.34	13	NA	-
Benzo[k]fluoranthene	0.34	11	1.6 (RSL)	Yes
1,1'-Biphenyl	0.084	0.084	29,000 (RMEG)	No
bis(2-Ethylhexyl)phthalate	0.12	0.15	35 (RDCSCC)	No
Carbazole	0.05	2.2	24 (RDCSCC)	No
Chrysene	0.65	19	62 (RDCSCC)	Yes
Dibenzo[a,h]anthracene	0.065	3.3	0.016 (RSL)	Yes
Dibenzofuran	0.042	1.6	NA	-
Fluoranthene	0.88	35	2,300 (RMEG)	No
Fluorene	0.051	2.8	2,300 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.31	12	0.16 (RSL)	Yes
Naphthalene	0.044	1.6	1,100 (RMEG)	No
Phenanthrene	0.45	20	NA	-
Pyrene	0.95	34	1,700 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Cancer Risk Evaluation Guide for chronic exposure; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fNJDEP Residential Direct Contact Soil Cleanup Criteria; ^gATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^hNot available; ⁱRegional Screening Level

Table A5: Surface Soil (0-2 ft depth) Sampling Results of Block 93 property

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
Aluminum	4,970	15,700	57,000 (EMEG)	No
Antimony	1.23	41	23 (RMEG)	No
Arsenic	3.32	913	0.25 (CREG)	Yes
Barium	49.1	195	11,000 (EMEG)	No
Beryllium	0.24	4.4	110 (EMEG)	No
Cadmium	0.29	1.3	5.7 (EMEG)	No
Chromium (III)	11.9	84.1	86,000 (RMEG)	No
Hexavalent Chromium	0.56	0.56	51 (RMEG)	No
Cobalt	5.6	23	570 (EMEG I)	No
Copper	28.6	472	3,100 (RSL)	No
Iron	16,800	38,300	55,000 (RSL)	No
Lead	17.1	1,960	400 (RDCSCC)	Yes
Manganese	101	828	2,900 (RMEG)	No
Nickel	17.7	173	1,100 (RMEG)	No
Selenium	1.1	28.3	290 (EMEG)	No
Silver	0.615	1.8	290 (RMEG)	No
Thallium	0.127	4.8	NA	-
Vanadium	25.8	95.3	390 (RSL)	No
Zinc	55.7	1,000	17,000 (RMEG)	No
Mercury	0.069	10	14 (RDCSCC)	Yes
4,4-DDT	0.023	0.023	1.1 (CREG)	No
Endosulfan II	0.015	0.015	290 (EMEG)	No
Aroclor-1242	0.051	0.051	1.1 (CREG)	No
Aroclor-1248	0.77	0.77	1.1 (CREG)	Yes
Aroclor-1254	0.065	1.66	1.1 (CREG)	Yes
Aroclor-1260	0.025	3.58	1.1 (CREG)	Yes
Acetone	0.017	0.19	51,000 (EMEG)	No
Benzene	0.0008	13.9	6.8 (CREG)	No
Carbon disulfide	0.002	0.003	5,700 (RMEG)	No
Cyclohexane	3.5	3.5	6,500 (RSL)	No
Cyclopentane	4.8	4.8	NA	-
Ethylbenzene	0.021	40.1	5,000 (RMEG)	No
Isopropylbenzene (Cumene)	15.7	15.7	5,700 (RMEG)	No
Methyl Acetate	0.15	0.15	78,000 (RSL)	No
Methylcyclohexane	0.14	0.14	NA	-
Tetrachloroethene	0.088	0.088	180 (CREG)	No
Toluene	0.731	26.6	4,600 (RMEG)	No
Xylene (Total)	0.12	270	11,000 (EMEG)	No
2,4-Dimethylphenol	3.23	5.63	1,100 (RMEG)	No

Table A5: (Cont'd.)

Contaminant	Minimum Concentration (mg/kg^a)	Maximum Concentration (mg/kg^a)	Environmental Guideline CV^b (mg/kg)	COPC^c
2-Methylnaphthalene	0.336	764	2,300 (EMEG)	No
2-Methylphenol	1.19	5.06	2,900 (RMEG)	No
3&4-Methylphenol	12.6	12.6	2,900 (RMEG)	No
Acenaphthene	0.867	949	3,400 (RMEG)	No
Acenaphthylene	0.443	20.7	NA	-
Anthracene	1.85	1,510	17,000 (RMEG)	No
Benzo(a)anthracene	7.71	396	0.16 (RSL)	Yes
Benzo[a]pyrene	7.8	249	0.016 (CREG)	Yes
Benzo[b]fluoranthene	7.55	260	0.16 (RSL)	Yes
Benzo[g,h,i]perylene	3.98	140	NA	-
Benzo[k]fluoranthene	3.7	185	1.6 (RDCSCC)	Yes
1,1'-Biphenyl	0.026	120	29,000 (RMEG)	No
bis(2-Ethylhexyl)phthalate	0.627	17	35 (RDCSCC)	No
Carbazole	0.35	311	24 (RDCSCC)	Yes
Chrysene	7.4	417	62 (RDCSCC)	Yes
Dibenzo[a,h]anthracene	1.5	49.9	0.016 (RSL)	Yes
Dibenzofuran	0.215	688	NA	-
Fluoranthene	13	2,430	2,300 (RMEG)	Yes
Fluorene	0.511	1,080	2,300 (RMEG)	No
Indeno[1.2,3-cd]pyrene	3.9	130	0.16 (RSL)	Yes
Naphthalene	0.252	1,600	1,100 (RMEG)	Yes
Phenanthrene	5.69	3,620	NA	-
Phenol	6.71	6.71	17,000 (RMEG)	No
Pyrene	11.6	1,530	1,700 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Environmental Media Evaluation Guide for chronic exposure; ^eATSDR Reference Media Evaluation Guide for chronic exposure for child; ^fATSDR Cancer Risk Evaluation Guide for chronic exposure; ^gATSDR Reference Media Evaluation Guide for intermediate exposure for child; ^hNJDEP Residential Direct Contact Soil Cleanup Criteria; ⁱNot available; ^jRegional Screening Level

Table A6: Indoor^a Air Sampling Results of the Palisades Child Care Center located at 115 River Road

Contaminant	Number of Samples	Concentration (µg/m ³) ^b			EPC Calculation Method	Environmental Guideline CVs ^c (µg/m ³)	COPC ^d
		Min.	Max.	EPC			
Dibromochloromethane	4	0.088	0.12	0.12	Max.	NA ^e	-
1,4-Dichlorobenzene	16	0.22	3.7	1.56	95% UCL ^f	60 (MRL ^g)	No
Ethylbenzene	18	0.21	1.6	0.87	95% UCL	260 (MRL)	No
Naphthalene	28	0.2	11	3.79	95% UCL	3.7 (MRL)	No
n-Propylbenzene	8	0.078	0.34	0.265	95% UCL	NA	-
Tetrachloroethene	14	0.15	2.9	1.47	95% UCL	3.8 (CREG ^h)	No
Tetrahydrofuran	9	0.21	1.4	0.85	95% UCL	2,000 (RfC)	No
Trichloroethene	17	0.013	0.31	0.17	95% UCL	0.21 (CREG)	No
1,2,4-Trimethylbenzene	22	0.26	5.1	2.07	95% UCL	60 (RfC)	No
1,3,5-Trimethylbenzene	16	0.13	2.5	0.95	95% UCL	60 (RfC)	-
Toluene	10	1.2	6.1	6.29	95% UCL	3,800 (MRL)	No
Xylenes (Total)	23	0.35	34	11.82	95% UCL	220 (MRL)	No

^aAll indoor air sampling rounds are included; ^bmicrograms per cubic meter; ^cComparison Value; ^dContaminant of Potential Concern; ^eNot Available; ^fUpper Confidence Limit of arithmetic mean; ^gATSDR Minimal Risk Level; ^hATSDR Cancer Risk Evaluation Guide for chronic exposure; ⁱRegional Screening Level

Table A7: Indoor^a Air sampling Results of the Office spaces located at 115 River Road

Contaminant	Number of Samples	Concentration ($\mu\text{g}/\text{m}^3$) ^b			EPC Calculation Method	Environmental Guideline CVs ^c ($\mu\text{g}/\text{m}^3$)	COPC ^d
		Min.	Max.	EPC			
Acrolein	6	0.4	1.9	1.41	95% UCL ^e	0.02 (RfC ^f)	Yes
Benzene	43	0.58	4.3	1.766	95% UCL	0.13 (CREG ^g)	Yes
Carbon tetrachloride	16	0.38	0.5	0.462	95% UCL	0.17 (CREG)	Yes
Chloroform	43	0.065	1.2	0.37	95% UCL	0.043 (CREG)	Yes
1,4-Dichlorobenzene	29	0.16	96	26.89	95% UCL	60 (MRL ^h)	Yes
Ethylbenzene	43	0.24	4.6	1.716	95% UCL	260 (MRL)	No
Naphthalene	41	0.14	8.3	2.16	95% UCL	3.7 (MRL)	No
n-Propylbenzene	6	0.074	0.47	0.367	95% UCL	NA ⁱ	-
Tetrachloroethene	16	0.25	0.65	0.516	95% UCL	3.8 (CREG)	No
Tetrahydrofuran	5	0.32	3	3.07	95% UCL	2,000 (RfC)	-
Toluene	26	1.2	68	12.99	95% UCL	3,800 (MRL)	No
Trichloroethene	36	0.023	1.5	0.331	95% UCL	0.21 (CREG)	No
1,2,4-Trimethylbenzene	16	0.31	2.7	1.281	95% UCL	60 (RfC)	No
1,3,5-Trimethylbenzene	13	0.12	0.89	0.56	95% UCL	60 (RfC)	-
Xylenes (Total)	16	1.38	8.4	5.54	95% UCL	220 (MRL)	No

^aAll indoor air sampling rounds are included; ^bmicrograms per cubic meter; ^cComparison Value; ^dContaminant of Potential Concern; ^eUpper Confidence Level of arithmetic mean; ^fEPA Reference Concentration, ^gATSDR Cancer Risk Evaluation Guide for chronic exposure; ^hATSDR Minimal Risk Level; ⁱNot Available; ^jRegional Screening Level

Table A8: Sub-surface Soil (0-2 ft depth) Sampling Results of the Quanta Resources and adjacent properties

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COPC ^c
	Minimum	Maximum		
1,1'- Biphenyl	0.036	590	29,000 (RMEG ^d)	No
1,1-Dichloroethane	0.13	0.23	NA ^e	-
2,4- Dimethylphenol	0.14	340	1,100 (RMEG)	No
2,4- Dinitrotoluene	0.082	29	57 (EMEG ^f)	No
2- Butanone	0.008	0.018	34,000 (RMEG)	No
2- Methylnaphthalene	0.049	3,490	2,300 (EMEG)	Yes
2- Methylphenol	0.05	260	2,900 (EMEG)	No
2-Nitroaniline	0.16	0.55	NA	-
3&4-Methylphenol	0.0983	292	2,900 (RMEG)	No
4,4'-DDD	0.0026	0.017	1.6 (CREG ^g)	No
4,4'-DDE	0.013	0.013	1.1 (CREG)	No
4,4'-DDT	0.047	0.047	1.1 (CREG)	No
4-Methylphenol	0.099	630	2,900 (RMEG)	No
4-Nitroaniline	9.5	58	NA	-
Acenaphthene	0.042	1.3	3,400 (RMEG)	No
Acenaphthylene	0.0267	402	NA	-
Acetone	0.012	0.066	51,000 (EMEG)	No
Acetophenone	0.245	16.4	5,700 (RMEG)	No
Aluminum	24.8	22,100	57,000 (EMEG)	No
Ammonia	330	355	NA	-
Anthracene	0.0274	2,660	17,000 (RMEG)	Yes
Antimony	5.7	205	23 (RMEG)	No
Aroclor-1242	0.34	0.34	1.1 (CREG)	No
Aroclor-1248	0.043	0.18	1.1 (CREG)	No
Aroclor-1254	0.0071	46.4	1.1 (CREG)	Yes
Aroclor-1260	0.013	16	1.1 (CREG)	Yes
Barium	2.7	400	11,000 (EMEG)	No
Benzaldehyde	0.0891	0.0891	5,700 (RMEG)	No
Benzene	0.0006	255	6.8 (CREG)	Yes
Benzo[a]anthracene	0.042	3,700	0.16 (RSL)	Yes
Benzo[a]pyrene	0.0445	3,500	0.016 (CREG)	Yes
Benzo[b]fluoranthene	0.0278	4,100	0.16 (RSL ^h)	Yes
Benzo[g,h,i]perylene	0.0417	1,900	NA	-
Benzo[k]fluoranthene	0.047	2,000	1.6 (RDCSCC ⁱ)	Yes
Beryllium	0.054	5.8	110 (EMEG)	No
Bis(2-ethylhexyl)phthalate	0.0651	8.5	35 (RDCSCC)	No
Bromoform	0.002	0.002	47 (CREG)	No

Table A8: (Cont'd.)

Contaminant	Concentration (mg/kg ^a)		Environmental Guideline CV ^b (mg/kg)	COPC ^c
	Minimum	Maximum		
Cadmium	0.14	37	5.7 (EMEG)	No
Carbazole	0.047	1,200	24 (RDCSCC)	Yes
Carbon disulfide	0.001	0.65	5,700 (RMEG)	No
Chlorobenzene	1	1	1,100 (RMEG)	No
Chloroform	0.53	2.3	570 (EMEG)	No
Chromium	5.7	105	51 (EMEG)	Yes
Chromium (III)	2.3	105	86,000 (RMEG)	No
Chrysene	0.043	4,200	62 (RDCSCC)	Yes
Cis-1,2-dichloroethene	0.0854	0.13	110 (RMEG)	No
Cobalt	2	69.6	500 (EMEG I ^d)	No
Copper	3.4	4,520	570 (EMEG I)	Yes
Cyanide	8.3	8.3	36 (RMEG)	No
Cyclohexane	0.002	45	6,500 (RSL)	No
Dibenzo[a,h]anthracene	0.045	510	0.016 (RSL)	Yes
Dibenzofuran	0.0308	2,800	NA	No
Di-n-octyl phthalate	3	3	1,100 (RDCSCC)	No
Endosulfan ii	0.012	0.012	290 (EMEG)	No
Endosulfan sulfate	0.0093	0.0093	NA	No
Endrin aldehyde	0.0092	0.0092	17 (EMEG)	No
Ethylbenzene	0.002	174	5,000 (RMEG)	No
Fluoranthene	0.0909	10,000	2,300 (RMEG)	Yes
Fluorene	0.044	4,160	2,300 (RMEG)	Yes
Hexachlorocyclopentadiene	8	8	340 (RMEG)	No
Hexavalent chromium	0.43	4	51 (EMEG)	No
Indeno[1,2,3-cd]pyrene	0.0496	1700	0.16 (RSL)	Yes
Iron	3320	134,000	55,000 (RSL)	Yes
Isopropylbenzene	0.003	30.3	NA	-
Lead	2.6	38,800	400 (RDCSCC)	Yes
Manganese	0.4	987	2,900 (RMEG)	No
Mercury	0.0077	69.2	14 (RDCSCC)	Yes
Methyl acetate	0.192	1.46	78,000 (RSL)	No
Methyl tert-butyl ether	1	1	47 (RSL I)	No
Methylcyclohexane	0.003	190	NA	-
Methylene chloride	0.007	7	3,400 (EMEG)	No
Naphthalene	0.047	10,200	1,100 (RMEG)	Yes
Nickel	2.4	118	1,100 (RMEG)	No
N-nitrosodiphenylamine	1.1	2.6	76 (CREG)	No

Table A8: (Cont'd.)

Phenanthrene	0.0667	11,000	NA	-
Phenol	0.056	460	17,000 (RMEG)	No
Pyrene	0.0878	8,700	1,700 (RMEG)	Yes
Selenium	1	302	290 (EMEG)	Yes
Silver	0.36	25.7	290 (RMEG)	No
Styrene	0.002	57	11,000 (RMEG)	No
Tetrachloroethene	0.094	0.094	180 (CREG)	No
Thallium	1.1	84.2	NA	-
Toluene	0.002	186	4,600 (RMEG)	No
Trans-1,2-dichloroethene	0.0816	0.0816	1,100 (RMEG)	No
Trichloroethene	0.006	0.48	5.7 (CREG)	No
Trichlorofluoromethane	1.4	1.4	17,000 (RMEG)	No
Vanadium	2	55.6	390 (RSL)	No
Vinyl chloride	0.096	0.2	170 (EMEG)	No
Xylenes, total	0.002	348	11,000 (EMEG)	No
Zinc	4.1	4,830	17,000 (RMEG)	No

^amicrograms per kilogram; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Reference Media Evaluation Guide for chronic exposure; ^eNot available; ^fATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^gATSDR Cancer Risk Evaluation Guide for chronic exposure; ^hRegional Screening Level; ⁱNJDEP Residential Direct Contact Soil Cleanup Criteria; ^jATSDR Environmental Media Evaluation Guide for intermediate exposure for child

Table A9: Groundwater Sampling Results of the Quanta Resources and adjacent properties

Contaminant	Concentration (µg/L ^a)		Environmental Guideline CV ^b (µg/L)	COPC ^c
	Minimum	Maximum		
1,1,1-Trichloroethane	0.1	3	14,000 (RMEG)	No
1,1,2-Trichlorotrifluoroethane	0.2	0.3	2,100 (RMEG)	No
1,1'-Biphenyl	0.45	1,100	3,500 (RMEG)	No
1,1-Dichloroethane	0.1	120	63 (EMEG)	Yes
1,2,4-Trichlorobenzene	0.1	13	700 (EMEG)	No
1,2-Dichlorobenzene	0.1	1.1	2,100 (EMEG)	No
1,2-Dichloroethane	0.2	2.9	0.27 (CREG)	Yes
1,2-Dichloropropane	0.2	4.2	630 (EMEG)	No
1,4-Dichlorobenzene	0.4	2.5	490 (EMEG)	No
2,4-Dimethylphenol	2.3	7,400	140 (RMEG)	Yes
2-Butanone	1.1	61	4,200 (RMEG)	No
2-Methylnaphthalene	0.59	8,400	280 (EMEG)	Yes
2-Methylphenol	1	4,200	NA	No
3&4-Methylphenol	2	68.6	NA	No
4,4'-DDD	0.0068	0.17	0.1 (CREG)	Yes
4,4'-DDE	0.0052	0.13	0.071 (CREG)	Yes
4,4'-DDT	0.0065	0.034	0.071 (CREG)	No
4-Methylphenol	2	5,800	NA	No
Acenaphthene	0.252	3,300	420 (RMEG)	Yes
Acenaphthylene	0.21	550	NA	No
Acetone	2.9	470	6,300 (RMEG)	No
Acetophenone	23	30	700 (EMEG)	No
Aldrin	0.032	0.032	0.21 (EMEG)	Yes
Alpha-bhc	0.045	0.048	0.0013 (CREG)	No
Alpha-chlordane	0.0024	0.014	0.069 (CREG)	Yes
Ammonia	35	24,100	NA	No
Anthracene	0.207	1,600	2,100 (RMEG)	Yes
Aroclor-1260	0.59	6.1	0.0078 (CREG)	Yes
Arsenic	0.77	1,590,000	0.016 (CREG)	Yes
Arsenic (III)	13.1	1,830,000	0.016 (CREG)	Yes
Arsenic (VI)	4.8	9,500	0.016 (CREG)	Yes
Benzene	0.1	11,000	0.44 (CREG)	Yes
Benzo[a]anthracene	0.202	1,100	NA	Yes
Benzo[a]pyrene	0.256	800	NA	Yes
Benzo[b]fluoranthene	0.229	920	NA	Yes
Benzo[g,h,i]perylene	0.752	420	NA	Yes
Benzo[k]fluoranthene	0.314	450	NA	Yes
Bis(2-ethylhexyl)phthalate	1.1	43	NA	Yes

Table A9: (Cont'd.)

Contaminant	Concentration (µg/L ^a)		Environmental Guideline CV ^b (µg/L)	COPC ^c
	Minimum	Maximum		
Bromodichloromethane	0.1	0.1	0.39 (CREG)	No
Cacodylic acid	0.523	0.753	NA	No
Caprolactam	4.2	140	3,500 (RMEG)	No
Carbazole	0.64	790	NA	No
Carbon disulfide	0.1	33	700 (RMEG)	No
Chlorobenzene	0.2	1.8	140 (RMEG)	No
Chloroform	0.1	1.7	70 (EMEG)	No
Chloromethane	0.1	21	190 (RSL)	Yes
Chrysene	0.243	1,000	NA	No
cis-1,2-dichloroethene	0.1	16	14 (RMEG)	No
Cyclohexane	0.1	81	70 (RSL)	No
Delta-bhc	0.0044	0.1	0.013 (CREG)	No
Dibenzo[a,h]anthracene	0.347	41	NA	No
Dibenzofuran	0.44	2,200	NA	No
Diethyl phthalate	1.3	19.1	5,600 (RMEG)	No
Endosulfan i	0.0021	0.14	35 (EMEG)	No
Endosulfan ii	0.0071	0.0071	35 (EMEG)	No
Endosulfan sulfate	0.0084	0.02	35 (EMEG)	No
Endrin	0.005	0.51	2.1 (EMEG)	No
Ethylbenzene	0.1	1,300	700 (RMEG)	Yes
Fluoranthene	0.208	3,400	280 (RMEG)	Yes
Fluorene	0.252	2,800	280 (RMEG)	Yes
Gamma-bhc (lindane)	0.0025	0.011	0.013 (CREG)	No
Heptachlor	0.0021	0.016	3.5 (RMEG)	No
Heptachlor epoxide	0.0024	0.0037	0.091 (RMEG)	No
Indeno[1,2,3-cd]pyrene	0.736	390	0.034 (RSL)	No
Isophorone	3	5	1,400 (CREG)	No
Isopropylbenzene	0.1	141	NA	No
Lead	0.083	4,100	15 (AL)	Yes
Methoxychlor	0.16	0.16	35 (RMEG)	No
Methyl tert-butyl ether	0.1	2.1	14 (RSL)	No
Methylcyclohexane	0.1	170	NA	No
Methylene chloride	0.2	10	6.1 (CREG)	Yes
Monomethylarsonic acid	0.066	35	70 (EMEG)	No
Naphthalene	0.251	36,000	140 (RMEG)	Yes
Nitrobenzene	8	8	14 (RMEG)	No
Phenanthrene	0.213	8,300	NA	No

Table A9: (Cont'd.)

Contaminant	Concentration (µg/L ^a)		Environmental Guideline CV ^b (µg/L)	COPC ^c
	Minimum	Maximum		
Phenol	1	3,100	2,100 (RMEG)	Yes
Pyrene	0.226	2,800	210 (RMEG)	Yes
Tetrachloroethene	0.1	9.6	12 (CREG)	Yes
Toluene	0.1	4,800	560 (RMEG)	Yes
trans-1,2-dichloroethene	0.1	0.49	140 (RMEG)	No
Trichloroethene	0.1	460	0.43 (CREG)	Yes
Vinyl chloride	0.1	4.4	0.0086 (CREG)	Yes
Xylenes, total	0.1	3,900	1,400 (EMEG)	Yes

^amicrograms per Liter; ^bComparison Value; ^cContaminant of Potential Concern; ^dATSDR Reference Media Evaluation Guide for chronic exposure; ^eRegional Screening Level; ^fATSDR Environmental Media Evaluation Guide for chronic exposure for child; ^gATSDR Cancer Risk Evaluation Guide for chronic exposure; ^hNot available; ⁱNew Jersey Maximum Contaminant Level; ^jAction Level

Table A10: Major Exposure Pathways for the QRC and adjacent properties

Environmental Pathway	Exposure Scenario(s)	Route of Exposure	Receptor	Location	Pathway Status		
					Past	Present	Future
Surface Soil	Recreation	Ingestion /Dermal	Residents/ Occupants	Quanta Resources Celotex Prop. 115 River Rd. Lever Brothers Block 93	Completed	Potential	Potential
Groundwater	Ingestion	Ingestion		Potential	Eliminated ¹	Eliminated	
Indoor Air	Inhalation	Inhalation		Child Care Center Office Space	Potential	Completed	Eliminated
Sediment/ Surface Water	Ingestion	Ingestion Dermal		Shore Areas	Potential	Potential	Potential
Biota	Ingestion	Ingestion		On-site and adjacent Areas	Potential	Potential	Potential

¹One or more exposure pathway elements were removed

Table A11: Comparison of Surface Soil Exposure Dose associated with the QRC and adjacent properties with the Health Guideline CVs

Contaminants of Potential Concern	EPC (mg/kg) ^a	Estimation Method ^b	Highest Exposure Dose ^c (mg/kg/day)	Health Guideline CV ^d (mg/kg/day)	Hazard Quotient ^d	Potential for Non-cancer Health Effects
Antimony	174	Max	0.00066	0.0004 (MRL ^e)	1.6	Yes
Arsenic	1,500	99% Cheb-m	0.0057	0.0003 (MRL)	18.9	Yes
Cadmium	10.1	Max	0.000038	0.0001 (MRL)	0.4	No
Copper	6,170	Max	0.023	0.04 (RfD ^f)	0.6	No
Lead	680	Average		400 (RDCSCC ^g)		Yes
Mercury	29.5	Max	0.00011	0.0003 (RfD)	0.4	No
Heptachlor	0.35	Max	0.0000013	0.0005 (MRL)	0	No
Aroclor-1242	3.2	Max	0.000012	0.00002 (MRL)	0.6	No
Aroclor-1260	1.6	Max	0.000006	0.00002 (MRL)	0.3	No
Benzene	19	Max	0.000072	0.004 (RfD)	0	No
Benzo[a]anthracene	330	95% App. Gamma	0.0012	NA ^h		-
Benzo[a]pyrene	300	95% App. Gamma	0.0011	0.0003 (RfD)	3.8	Yes
Benzo[b]fluoranthene	350	95% App. Gamma	0.0013	NA		-
Benzo[g,h,i]perylene	160	95% App. Gamma	0.0006	NA		-
Benzo[k]fluoranthene	160	95% App. Gamma	0.0006	NA		-
Dibenz[a,h]anthracene	60	95% App. Gamma	0.00023	NA		-
Indeno[1,2,3-cd]pyrene	170	95% App. Gamma	0.00064	NA		-
Carbazole	140	95% App. Gamma	0.00053	NA		-
Chrysene	320	95% App. Gamma	0.0012	NA		-
Fluoranthene	820	95% App. Gamma	0.0031	0.04 (RfD)	0.1	No
Naphthalene	910	95% Adj. Gamma	0.0034	0.02 (RfD)	0.2	No

^aExposure Point Concentration (milligrams per kilogram); ^bEPC estimation method used by ProUCL; ^cSoil and Sediment Exposure Dose Guidance (ATSDR 2014); ^dComparison Value; ATSDR Minimal Risk Levels; ^fReference Dose; ^gNJDEP Residential Direct Contact Soil Cleanup Criteria; ^hNot Available.

Table A12: Comparison of Surface Soil Exposure Dose associated with the Celotex property with the Health Guideline CV

Contaminants of Potential Concern	EPC (mg/kg) ^a	Estimation Method ^b	Highest Exposure Dose (mg/kg/day) ^c	Health Guideline CV ^d (mg/kg/day)	Hazard Quotient	Potential for Non-cancer Health Effects
Metals						
Arsenic	19	95% App. Gamma	7.2×10^{-5}	0.0003 (MRL ^e)	0.2	No
Chromium	47	95% App. Gamma	1.8×10^{-4}	0.003 (MRL)	0.1	No
Lead	951	Max		400 (RDCSCC ^f)		Yes
Aroclor-1242	0.637	Max	2.4×10^{-6}	0.00002 (MRL)	0.1	No
Aroclor-1254	348	Max	1.3×10^{-3}	0.00002 (MRL)	65	Yes
Aroclor-1260	0.621	Max	2.3×10^{-6}	0.00002 (MRL)	0.1	No
Benzo[a]anthracene	3.2	95% App. Gamma	1.2×10^{-5}	NA ^g		-
Benzo[a]pyrene	3	95% App. Gamma	1.1×10^{-5}	0.0003 (RfD)	0	No
Benzo[b]fluoranthene	2.9	95% App. Gamma	1.1×10^{-5}	NA		-
Benzo[g,h,i]perylene	5.71	Max	2.2×10^{-5}	NA		-
Benzo[k]fluoranthene	5.88	Max	2.2×10^{-5}	NA		-
Dibenzo[a,h]anthracene	0.69	95% App. Gamma	2.6×10^{-6}	NA		-
Indeno[1,2,3-cd]pyrene	1.7	95% App. Gamma	6.4×10^{-6}	NA		-
Chrysene	10.4	Max	3.9×10^{-5}	NA		-

^aExposure Point Concentration (milligrams per kilogram); ^bEPC estimation method used by ProUCL; ^cSoil and Sediment Exposure Dose Guidance (ATSDR 2014); ^dComparison Value; ^eATSDR Minimal Risk Levels; ^fNJDEP Residential Direct Contact Soil Cleanup Criteria; ^gNot Available.

Table A13: Comparison of Surface Soil Exposure Dose associated with the 115 River Road property (Spencer-Kellogg Building) with the Health Guideline CV

Contaminants of Potential Concern	EPC (mg/kg) ^a	Estimation Method ^b	Highest Estimated Exposure Dose ^c (mg/kg/day)	Health Guideline CV ^d (mg/kg/day)	Hazard Quotient	Potential for Non-cancer Health Effects
Metals						
Arsenic	13	Max	4.9 x10 ⁻⁵	0.0003 (MRL ^e)	0.2	No
Chromium	100	Max	3.8 x10 ⁻⁴	0.001 (MRL)	0.1	No
Lead	557	Max		400 (RDCSCC ^f)		Yes
Aroclor-1248	0.47	Max	1.8 x10 ⁻⁶	0.00002 (MRL)	0.1	No
Aroclor-1254	0.79	Max	3 x10 ⁻⁶	0.00002 (MRL)	0.1	No
Benzo[a]anthracene	28	Max	1.1 x10 ⁻⁴	NA ^g		
Benzo[a]pyrene	25	Max	9.4 x10 ⁻⁵	0.0003 (RfD)	0.3	No
Benzo[b]fluoranthene	31	Max	1.2 x10 ⁻⁴	NA		
Benzo[g,h,i]perylene	15	Max	5.7 x10 ⁻⁵	NA		
Benzo[k]fluoranthene	160	Max	6 x10 ⁻⁴	NA		
Dibenz[a,h]anthracene	4.6	Max	1.7 x10 ⁻⁵	NA		
Indeno[1,2,3-cd]pyrene	15	Max	5.7 x10 ⁻⁵	NA		

^aExposure Point Concentration (milligrams per kilogram); ^bEPC estimation method used by ProUCL; ^cSoil and Sediment Exposure Dose Guidance (ATSDR 2014); ^dComparison Value; ^eATSDR Minimal Risk Levels; ^fNJDEP Residential Direct Contact Soil Cleanup Criteria; ^gNot Available.

Table A14: Comparison of Surface Soil Exposure Dose associated with the Lever Brothers property areas with the Health Guideline Comparison Values

Contaminants of Potential Concern	EPC (mg/kg) ^a	Estimation Method ^b	Highest Estimated Exposure Dose ^c (mg/kg/day)	Health Guideline CV ^d (mg/kg/day)	Hazard Quotient	Potential for Non-cancer Health Effects
Metals						
Arsenic	43	95% KM (Chebyshev)	1.6 x10 ⁻⁴	0.0003 (MRL ^e)	0.5	No
Aroclor-1254	0.37	Max.	1.4 x10 ⁻⁶	0.00002 (MRL)	0.1	No
Aroclor-1260	0.1	Max.	3.8 x10 ⁻⁷	0.00002 (MRL)	0	No
Benzo[a]anthracene	14	95% KM (Chebyshev)	5.3 x10 ⁻⁵	NA ^f		-
Benzo[a]pyrene	15	95% KM (Chebyshev)	5.7 x10 ⁻⁵	0.0003 (RfD)	0.2	No
Benzo[b]fluoranthene	18	95% KM (Chebyshev)	6.8 x10 ⁻⁵	NA		-
Benzo[g,h,i]perylene	13	Max.	4.9 x10 ⁻⁵	NA		-
Benzo[k]fluoranthene	7.9	95% KM (Chebyshev)	3 x10 ⁻⁵	NA		-
Dtbenzo[a,h]anthracene	1.7	95% KM (t)	6.4 x10 ⁻⁶	NA		-
Indeno[1,2,3-cd]pyrene	8.8	95% KM (Chebyshev)	3.3 x10 ⁻⁵	NA		-
Chrysene	19	Max.	7.2 x10 ⁻⁵	NA		-

^aExposure Point Concentration (milligrams per kilogram); ^bEPC estimation method used by ProUCL; ^cSoil and Sediment Exposure Dose Guidance (ATSDR 2014); ^dComparison Value; ^eATSDR Minimal Risk Levels; ^fNot Available.

Table A15: Comparison of Surface Soil Exposure Dose associated with Block 93 with the Health Guideline CV

Contaminants of Potential Concern	EPC (mg/kg) ^a	Estimation Method ^b	Estimated Exposure Dose ^c (mg/kg/day)	Health Guideline CV ^d (mg/kg/day)	Hazard Quotient	Potential for Non-cancer Health Effects
Arsenic	350	95% KM (Chebyshev)	1.3 x10 ⁻³	0.0003 (MRL ^e)	4.4	Yes
Lead	1,960	Average		400 (RSCSCC ^f)		Yes
Mercury	5.8	97.5% KM (Chebyshev)	2.2 x10 ⁻⁵	0.0003 (RfD ^g)	0.1	No
Aroclor 1248	0.77	Max	2.9 x10 ⁻⁶	0.00002 (MRL)	0.1	No
Aroclor 1254	0.49	95% KM (t)	1.8 x10 ⁻⁶	0.00002 (MRL)	0.1	No
Aroclor 1260	0.92	95% KM (t)	3.5 x10 ⁻⁶	0.00002 (MRL)	0.2	No
Benzo[a]anthracene	230	95% KM (Chebyshev)	8.7 x10 ⁻⁴	NA ^h		-
Benzo[a]pyrene	120	95% KM (t)	4.5 x10 ⁻⁴	0.0003 (RfD)	1.5	Yes
Benzo[b]fluoranthene	130	95% KM (t)	4.9 x10 ⁻⁴	NA		-
Benzo[g,h,i]perylene	140	Max.	5.3 x10 ⁻⁴	NA		
Benzo[k]fluoranttiene	120	95% KM (Chebyshev)	4.5 x10 ⁻⁴	NA		-
Carbazole	120	95% KM (Chebyshev)	4.5 x10 ⁻⁴	NA		-
Chrysene	220	95% KM (Chebyshev)	8.3 x10 ⁻⁴	NA		-
Dbenzo[a,h]anthracene	22	95% KM (t)	8.5 x10 ⁻⁵	NA		-
Fluoranthene	2,430	Max	9.2 x10 ⁻³	0.04 (MRL)		No
Indeno[1,2,3-cd]pyrene	67	95% KM (t)	25 x10 ⁻⁴	NA		-
Naphthalene	1,200	99% KM (Chebyshev)	4.5 x10 ⁻³	0.02 (RfD)		No

^aExposure Point Concentration (milligrams per kilogram); ^bEPC estimation method used by ProUCL; ^cSoil and Sediment Exposure Dose Guidance (ATSDR 2014); ^dComparison Value; ^eATSDR Minimal Risk Levels; ^fNJDEP Residential Direct Contact Soil Cleanup Criteria; ^gReference Dose ^hNot Available.

Table A16: Indoor Air sampling Results of the Palisades Child Care Center and office space located at 115 River Road property

Contaminant	EPC ($\mu\text{g}/\text{m}^3$) ^a	Adjusted - EPC ^b ($\mu\text{g}/\text{m}^3$)	Environmental Guideline CVs ^c ($\mu\text{g}/\text{m}^3$)	Potential for Non- cancer Effect
Palisades Child Care Center				
Tetrachloroethene	1.47	0.6125	41 (MRL ^d)	No
Office Space				
Acrolein	1.41	0.58	0.02 (RfC ^e)	Yes
Benzene	1.766	0.73	9.6 (MRL)	No
Carbon tetrachloride	0.462	0.2	190 (MRL)	No
Chloroform	0.37	0.154	98 (MRL)	No
1,4-Dichlorobenzene	26.89	11.2	60 (MRL)	No
Tetrachloroethene	0.516	0.21	41 (MRL)	No
Trichloroethene	0.331	0.138	2.1 (MRL I ^f)	No

^amicrograms per cubic meter; ^badjusted for 10 hours exposure time; ^cComparison Value; ^dATSDR Minimal Risk Level for chronic exposures; ^eEPA Reference Concentration; ^fATSDR Minimal Risk Level for intermediate exposures

Table A17: Calculated LECR associated with the Contaminants detected in surface soil at the Quanta on-site areas

Contaminants of Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Mutagenic Mode of Action	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest ^c LECR ^d	Highest LECR (Mutagenic)
Dibenz[a,h]anthracene	60	2	Yes					4.4 x10 ⁻⁴
Benzo[g,h,i]perylene	160	2	No				NA	
Benzo[a]anthracene	330	2	Yes	0.1	33	386.91		6.9 x10 ⁻⁴
Benzo[a]pyrene	300	2	Yes	1	300			
Benzo[b]fluoranthene	350	2	Yes	0.1	35			
Benzo[k]fluoranthene	160	2	Yes	0.01	1.6			
Indeno[1,2,3-cd]pyrene	170	2	Yes	0.1	17			
Chrysene	320	2	Yes	0.001	0.32			
Antimony	1,700	3	No					
Arsenic	1,500	1	No				2.3 x10 ⁻³	
Cadmium	10	1	No				NA	
Copper	6,200	3	No					
Lead	680	3	No					
Benzene	19	1	No				5.2 x10 ⁻⁵	
Heptachlor	0.35	2	No				1.6 x10 ⁻⁶	
Arodor-1242	3.2	2	No				6.6 x10 ⁻⁶	
Arodor-1254	0.702	2	No				1.5 x10 ⁻⁶	
Aroclor-1260	1.6	2	No				3.3 x10 ⁻⁶	

Table A17: (Cont'd)

Contaminants of Concern	EPC (mg/kg)	DHHS^a Cancer Class	Mutagenic Mode of Action	Potency Factor^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest^c LECR^d	Highest LECR (Mutagenic)
Fluoranthene	820	3						
Naphthalene	910	2						
Phenanthrene	1,100	NA						
Carbazole	140	3						
						Sum =	2.3 x10⁻³	1.13 x10⁻³

^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: 219 days/year, 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dLifetime Excess Cancer Risk; ^eNot Applicable

Table A18: Calculated LECR associated with the Contaminants detected in surface soil at the Celotex property areas

Contaminants of Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Mutagenic Mode of Action	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest ^c LECR ^d	Highest LECR (Mutagenic)
Dibenz[a,h]anthracene	0.69	2	Yes					8.1 x10 ⁻⁷
Benzo[g,h,i]perylene	5.71	2	No				NA ^e	
Benzo[a]anthracene	3.2	2	Yes	0.1	0.32	3.58		6.4 x10 ⁻⁶
Benzo[a]pyrene	3	2	Yes	1	3			
Benzo[b]fluoranthene	2.9	2	Yes	0.01	0.029			
Benzo[k]fluoranthene	5.88	2	Yes	0.01	0.058			
Indeno[1,2,3-cd]pyrene	1.7	2	Yes	0.1	0.17			
Chrysene	10.4	2	Yes	0.001	0.01			
Aroclor-1242	0.637	2	No				1.3 x10 ⁻⁶	
Aroclor-1254	348	2	No				7.2 x10 ⁻⁴	
Aroclor-1260	0.621	2	No				1.3 x10 ⁻⁶	
Arsenic	19	1	No				2.9 x10 ⁻⁵	
Chromium	47	1	No				NA	
Lead	951	3	No					
Sum =							7.4 x10 ⁻⁴	7.2 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); ^cAdult exposure scenario: 219 days/year, 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dLifetime Excess Cancer Risk; ^eNot Applicable

Table A19: Calculated LECR associated with the Contaminants detected in surface soil at the 115 River Road property areas

Contaminants of Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Mutagenic Mode of Action	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest ^c LECR ^d	Highest LECR (Mutagenic)
Dibenz[a,h]anthracene	4.6	2	Yes					3.4 x10 ⁻⁵
Benzo[g,h,i]perylene	15	2	No				NA ^e	
Benzo[a]anthracene	28	2	Yes	0.1	2.8	31.21		5.6 x10 ⁻⁵
Benzo[a]pyrene	25	2	Yes	1	25			
Benzo[b]fluoranthene	31	2	Yes	0.01	0.31			
Benzo[k]fluoranthene	160	2	Yes	0.01	1.6			
Indeno[1,2,3-cd]pyrene	15	2	Yes	0.1	1.5			
Arodor-1248	0.47	2	No				9.7 x10 ⁻⁷	
Arodor-1254	0.79	2	No				3 x10 ⁻⁶	
Arsenic	13	1	No				2 x10 ⁻⁵	
Chromium	100	1	No				NA	
Lead	557	3	No					
Sum =							2.3 x10 ⁻⁵	9.0 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: 219 days/year, 100 mg/day ingestion rate, 70 kg body weight and 30 year exposure duration; ^dLifetime Excess Cancer Risk; ^eNot Applicable

Table A20: Calculated LECR associated with the Contaminants detected in surface soil at the Lever Brothers property areas

Contaminants of Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Mutagenic Mode of Action	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest ^c LECR ^d	Highest LECR (Mutagenic)
Dibenz[a,h]anthracene	1.7	2	Yes					1.2 x10 ⁻⁵
Benzo[g,h,i]perylene	13	2	No				NA	
Benzo[a]anthracene	14	2	Yes	0.1	1.4	17.55		3.1 x10 ⁻⁵
Benzo[a]pyrene	15	2	Yes	1	15			
Benzo[b]fluoranthene	18	2	Yes	0.01	0.18			
Benzo[k]fluoranthene	7.9	2	Yes	0.01	0.079			
Indeno[1,2,3-cd]pyrene	8.8	2	Yes	0.1	0.88			
Chrysene	19	2	Yes	0.001	0.019			
Arodor-1248	0.37	2	No				7.6 x10 ⁻⁷	
Arodor-1254	0.1	2	No				2. x10 ⁻⁷	
Arsenic	43	1	No				6.7 x10 ⁻⁵	
Chromium	4	1	No				NA	
Sum =							6.7 x10 ⁻⁵	4.3 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

Table A21: Calculated LECR associated with the Contaminants detected in surface soil at the Block 93 property areas

Contaminants of Concern	EPC (mg/kg)	DHHS ^a Cancer Class	Mutagenic Mode of Action	Potency Factor ^b	BaP Equiv. (mg/kg)	Total BaP Equiv. (mg/kg)	Highest ^c LECR ^d	Highest LECR (Mutagenic)
Dibenzo[a,h]anthracene	22	2	Yes					1.6 x10 ⁻⁴
Benzo[g,h,i]perylene	140	2	No				NA	
Benzo[a]anthracene	230	2	Yes	0.1	23	152.42		2.7 x10 ⁻⁴
Benzo[a]pyrene	120	2	Yes	1	120			
Benzo[b]fluoranthene	130	2	Yes	0.01	1.3			
Benzo[k]fluoranthene	120	2	Yes	0.01	1.2			
Indeno[1,2,3-cd]pyrene	67	2	Yes	0.1	6.7			
Chrysene	220	2	Yes	0.001	0.22			
Carbazole	120	3	No					
Aroclor-1248	0.77	2	No				1.6 x10 ⁻⁶	
Aroclor-1254	0.49	2	No				1 x10 ⁻⁶	
Aroclor-1260	0.92	2	No				1.9 x10 ⁻⁶	
Fluoranthene	2,430	3	No					
Naphthalene	1,200	2	No				NA	
Arsenic	350	1	No				5.4 x10 ⁻⁴	
Mercury	5.8	3	No					
Lead	1,960	1	No					
Sum =							5.4 x10 ⁻⁴	4.3 x10 ⁻⁴

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

^bCancer potency factor relative to benzo[a]pyrene (BaP); ^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

Table A22: LECRs associated with the mutagenic and non-mutagenic contaminants detected in on-site and adjacent areas

Contaminants	Mutagenic/Non-mutagenic	Exposure Condition	Children exposed for 15 years	Adults exposed for 9 years ^a	Adults exposed for 33 years ^b
Quanta On-site					
Dibenz[a,h]anthracene (4.4 x10 ⁻⁴)	Mutagenic	RME ^d	3 x10 ⁻⁴	2.1 x10 ⁻⁵	7.8 x10 ⁻⁵
		CTE ^e	1.5 x10 ⁻⁴	1.1 x10 ⁻⁵	3.9 x10 ⁻⁵
Quanta On-site					
BaP and BaP Equivalent (5 x10 ⁻³)	Mutagenic	RME	4.8 x10 ⁻⁴	3.3 x10 ⁻⁵	1.2 x10 ⁻⁴
		CTE	2.4 x10 ⁻⁴	1.7 x10 ⁻⁵	1.1 x10 ⁻⁴
Celotex					
Aroclor 1254 (7.2 x10 ⁻⁴)	Non-mutagenic	RME	3.4 x10 ⁻⁴	6 x10 ⁻⁵	2.2 x10 ⁻⁴
		CTE	1.7 x10 ⁻⁴	3 x10 ⁻⁵	1.1 x10 ⁻⁴
Quanta On-site					
Benzene (5.2 x10 ⁻⁵)	Non-mutagenic	RME	2.4 x10 ⁻⁴	4.3 x10 ⁻⁶	1.6 x10 ⁻⁵
		CTE	1.2 x10 ⁻⁴	2.2 x10 ⁻⁶	7.9 x10 ⁻⁶
Quanta On-site					
Arsenic (2.3 x10 ⁻³)	Non-mutagenic	RME	1.1 x10 ⁻³	1.9 x10 ⁻⁴	7.1 x10 ⁻⁴
		CTE	5.5 x10 ⁻⁴	9.7 x10 ⁻⁵	3.6 x10 ⁻⁴

^aMean residential occupancy (9 years); ^b95% residential occupancy (33 years); ^cLifetime exposure (78 years); ^dReasonable Maximum Exposure (RME) condition; ^eCentral Tendency Exposure (CTE) condition

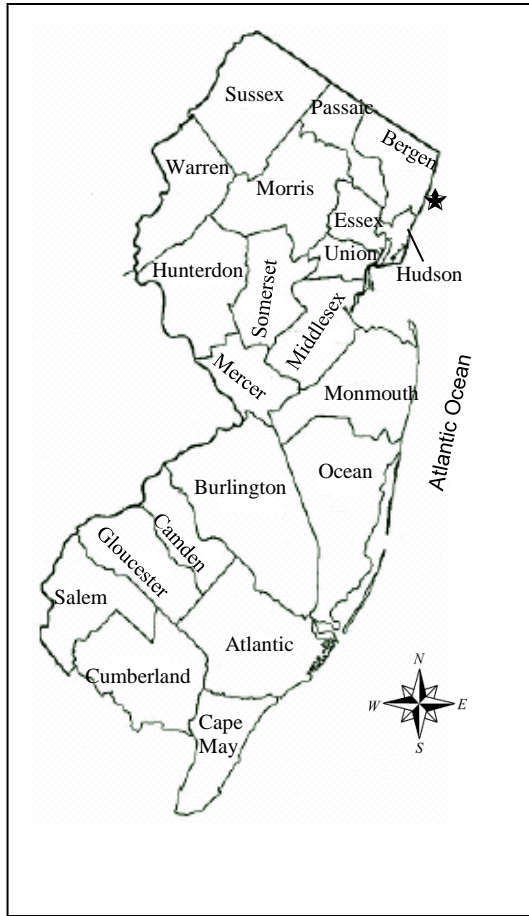


Fig A1: Location of Quanta Res. Corp. Site

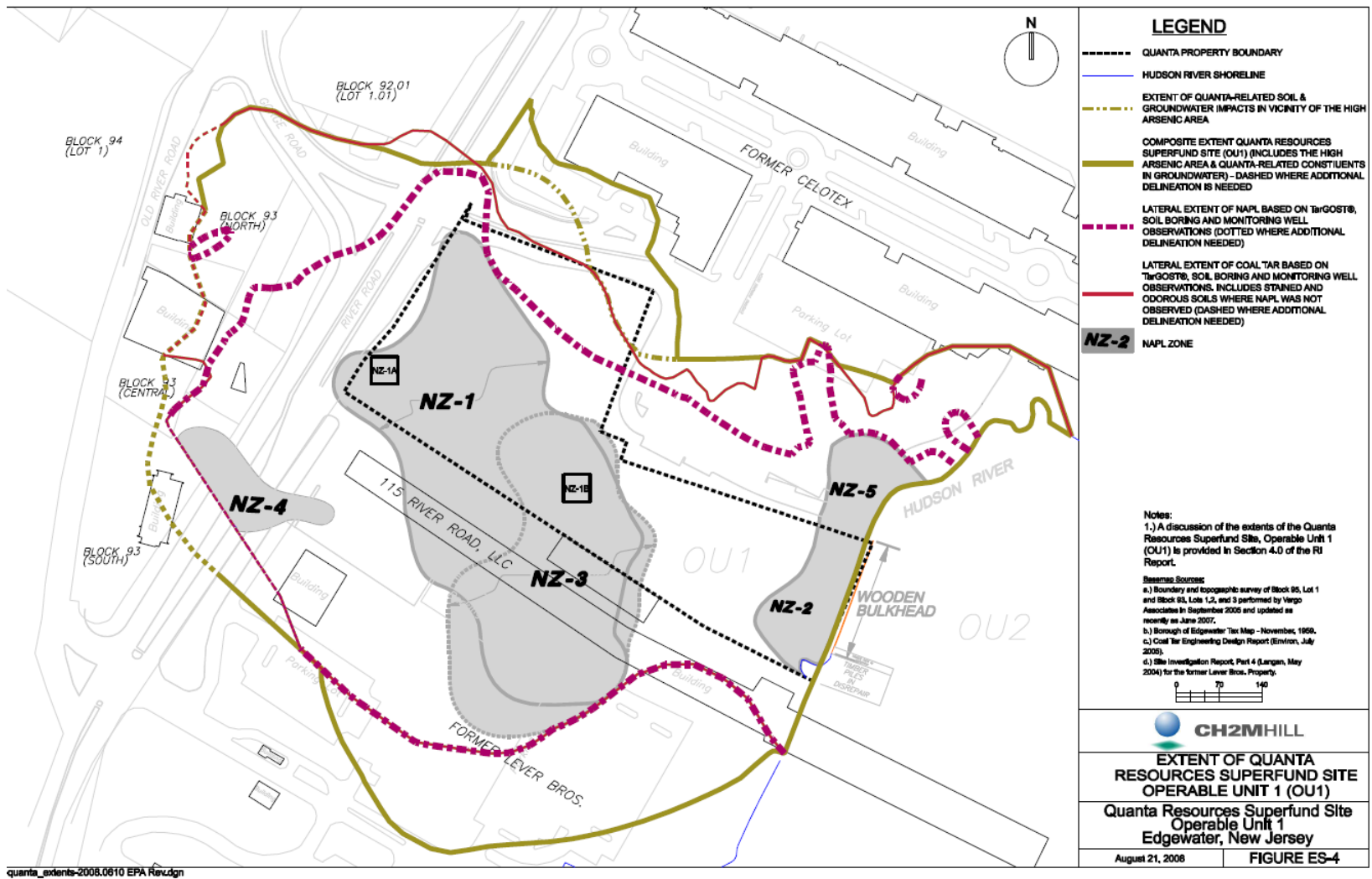
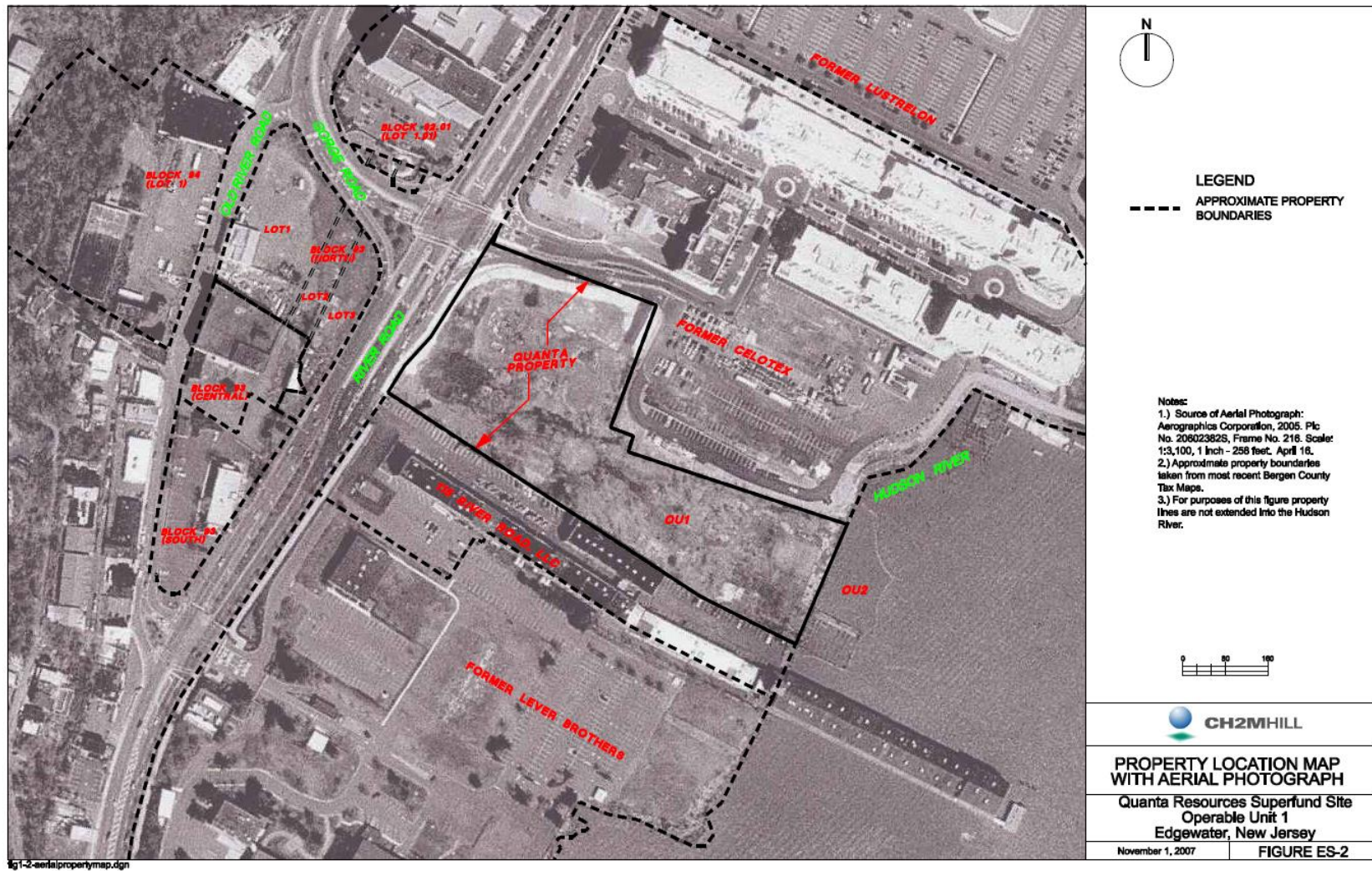


Figure A2: Extent of Quanta Resources Superfund site



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Figure A3: Property location map associated with OU1



Figure A4: Historical Aerial Photo (1970) of the Quanta Resources Corporation and adjacent properties

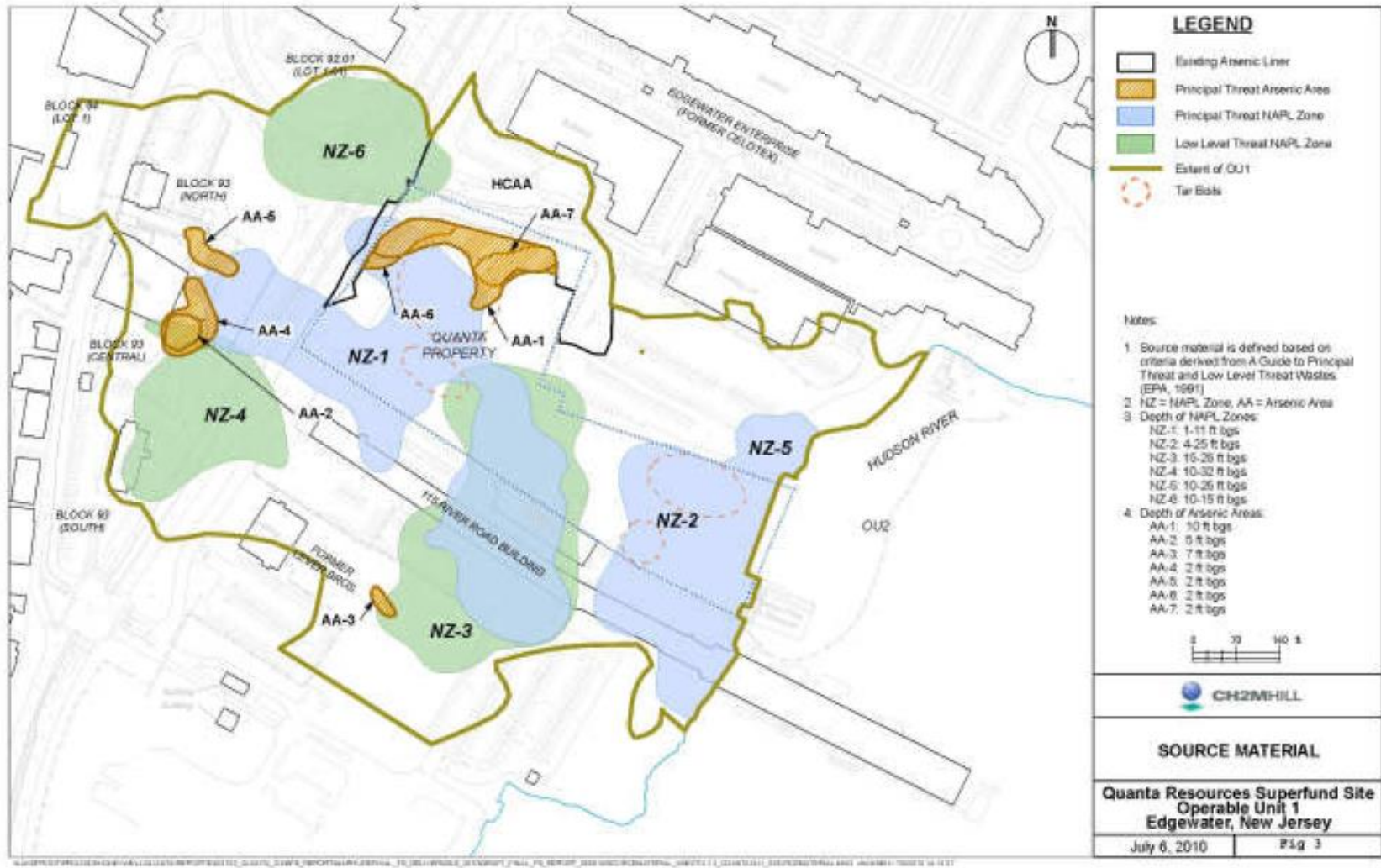


Figure A5: Location of NAPL sources areas

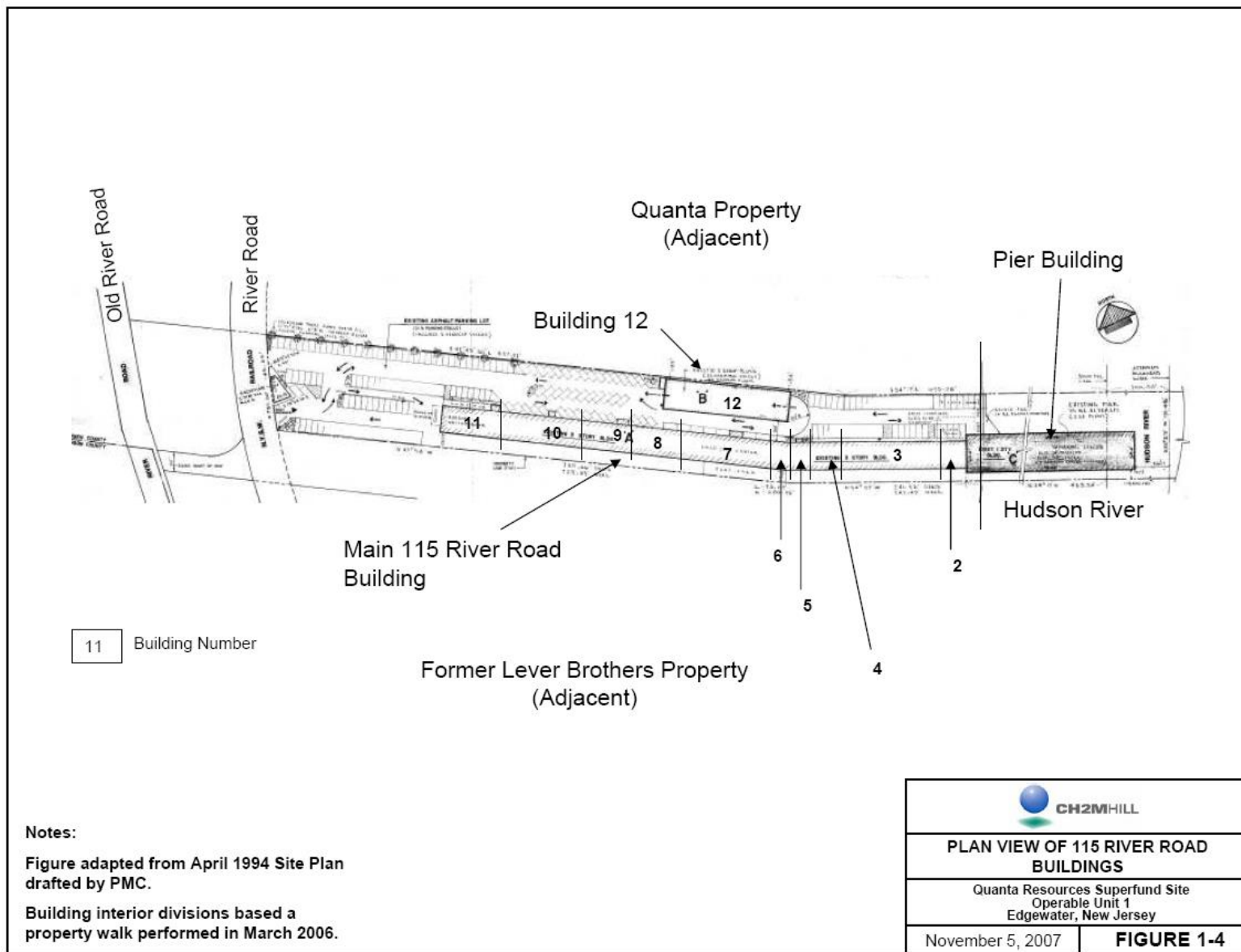


Figure A6: Plan view of 115 River Road Building



Figure A7: Photograph of the area (date between 1986 and 1988)

Appendix B:

Toxicologic Summaries

The toxicological summaries provided in this appendix are based on ATSDR's ToxFAQs (<http://www.atsdr.cdc.gov/toxfaq.html>). Health effects are summarized in this section for the chemicals of concern found off-site in area private wells. The health effects described in the section are typically known to occur at levels of exposure much higher than those that occur from environmental contamination. The chance that a health effect will occur is dependent on the amount, frequency and duration of exposure, and the individual susceptibility of exposed persons.

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

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

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

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

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

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

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

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

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

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

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

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

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

Breathing very high levels of benzene can result in death, while high levels can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death. The major effect of benzene from long-term (365 days or longer) exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease

in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Some women who breathed high levels of benzene for many months had irregular menstrual periods and a decrease in the size of their ovaries. It is not known whether benzene exposure affects the developing fetus in pregnant women or fertility in men. Animal studies have shown low birth weights, delayed bone formation, and bone marrow damage when pregnant animals breathed benzene.

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

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

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

Other studies on occupational exposures to mixtures of phthalate esters containing bis(2-ethylhexyl)phthalate have reported polyneuritis and sensory-motor polyneuropathy with decreased thrombocytes, leukocytes and hemoglobin in some exposed workers. Developmental toxicity studies with rats and mice have shown that bis(2-ethylhexyl)phthalate is fetotoxic and teratogenic when given orally during gestation. Oral exposure has also been shown to result in decreased sperm count in rats.

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

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

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

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

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

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

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

Exposure to high levels of copper will result in the same type of effects in children and adults. We do not know if these effects would occur at the same dose level in children and adults. Studies in animals suggest that young children may have more severe effects than adults, but it is not known if this would also be true in humans. A very small percentage of infants and children are unusually sensitive to copper.

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

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

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

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

DDT, DDE, and DDD DDT (dichlorodiphenyltrichloroethane) is a pesticide once widely used to control insects in agriculture and insects that carry diseases such as malaria. DDT is a white, crystalline solid with no odor or taste. The use of DDT in the US was banned in 1972 because of damage to wildlife, but is still used in some countries. DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) are chemicals similar to DDT that contaminate commercial DDT preparations. DDE has no commercial use.

DDD was also used to kill pests, but its use has also been banned. One form of DDD has been used medically to treat cancer of the adrenal gland.

DDT affects the nervous system. Individuals ingesting large amounts of DDT became excitable and had tremors and seizures. These effects went away after the exposure stopped. No effects were seen in people who took small daily doses of DDT by capsule for 18 months. A study in humans showed that women who had high amounts of a form of DDE in their breast milk were unable to breastfeed their babies for as long as women who had little DDE in the breast milk. Another study in humans showed that women who had high amounts of DDE in breast milk had an increased chance of having premature babies. In animals, short-term exposure to large amounts of DDT in food affected the nervous system, while long-term exposure to smaller amounts affected the liver. Also in animals, short-term oral exposure to small amounts of DDT or its breakdown products may also have harmful reproductive effects.

Studies in DDT-exposed workers did not show increases in cancer. Studies in animals given DDT with the food have shown that DDT can cause liver cancer.

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

Lead Lead is a naturally occurring metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from gasoline, paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. People may be exposed to lead by eating food or drinking water that contains lead, spending time in areas where lead-based paints have been used and are deteriorating, and by working in a job or engaging in a hobby where lead is used. Small children are more likely to be exposed to lead by swallowing house dust or soil that contains lead, eating lead-based paint chips or chewing on objects painted with lead-based paint.

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

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

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

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

The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems.

Short-term exposure to high levels of metallic mercury vapors may cause effects including lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

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

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

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has caused kidney tumors in male mice. The USEPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

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

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

Based on the results from animal studies, the DHHS concluded that naphthalene is reasonably anticipated to be a human carcinogen. The IARC concluded that naphthalene is possibly carcinogenic to humans. The USEPA determined that naphthalene is a possible human carcinogen (Group C) and that the data are inadequate to assess the human carcinogenic potential of 2-methylnaphthalene.

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. PCBs are known human carcinogens according to the USEPA and the International Agency for Research on Cancer (IARC).

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.

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 U.S. 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).

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

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

Tetrachloroethene (PCE) PCE is a manufactured chemical that is widely used for dry cleaning of fabrics and for metal-degreasing. It is a nonflammable liquid at room temperature. It evaporates easily into the air and has a sharp, sweet odor. Most people can smell PCE when it is present in the air at a level of 1 part per million (1 ppm) or more, although some can smell it at even lower levels. People are commonly exposed to PCE when they bring clothes from the dry cleaners.

High concentrations of PCE can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death. Irritation may result from repeated or extended skin contact with it. These symptoms occur almost entirely in work (or hobby) environments when people have been exposed to high concentrations. In industry, most workers are exposed to levels lower than those causing obvious nervous system effects. The health effects of breathing in air or drinking water with low levels of PCE are not known.

Results from some studies suggest that women who work in dry cleaning industries where exposures to PCE can be quite high may have more menstrual problems and spontaneous abortions than women who are not exposed. Results of animal studies, conducted with amounts much higher than those that most people are exposed to, show that PCE can cause liver and kidney damage. Exposure to very high levels of PCE can be toxic to the unborn pups of pregnant rats and mice. Changes in behavior were observed in the offspring of rats that breathed high levels of the chemical while they were pregnant.

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

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 100-1,500 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.

Trichloroethene (TCE) Trichloroethene 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 “known human carcinogen,” and the International Agency for Research on Cancer (IARC) has determined that TCE is “probably carcinogenic to humans.”

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, trichloroethene, and tetrachloroethene. 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 one to pass out, and breathing extremely high levels can cause death.

Some people who have breathed vinyl chloride for several years have changes in the structure of their livers. People are more likely to develop these changes if they breathe high levels of vinyl chloride. Some people who work with vinyl chloride have nerve damage and develop immune reactions. The lowest levels that produce liver changes, nerve damage, and immune reaction in people are not known. Some workers

exposed to very high levels of vinyl chloride have problems with the blood flow in their hands. Their fingers turn white and hurt when they go into the cold.

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

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

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

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

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

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

Appendix C:

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

Sample Non-cancer Exposure Dose Calculation

For QRC areas, the non-cancer exposure dose associated with antimony (See Table A11, Appendix A) was calculated by using the following formula:

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

where C = concentration of arsenic = 1,500 mg/kg

EF = 219 days/365 days

IR = ingestion rate = 200 mg/day

BW = body weight = 31.8 kg

Substituting the values –

$$\begin{aligned} \text{Exposure Dose (mg/kg-day)} &= \frac{1,500 \frac{\text{mg}}{\text{kg}} \times 200 \frac{\text{mg}}{\text{kg}} \times \frac{1 \text{ kg}}{10^6 \text{ mg}} \times \frac{219 \text{ days}}{365 \text{ days}}}{31.8 \text{ kg}} \\ &= 5.6 \times 10^{-3} \text{ mg/kg-day} \end{aligned}$$

$$\text{HQ} = \frac{0.00566}{0.0003} = 18.9 \text{ (see Table A11, Appendix A)}$$

Sample Lifetime Excess Cancer Risk Calculation

For QRC areas, the LECR associated with arsenic contaminated soil ingestion (See Table A17, Appendix A) was calculated by using the following formula:

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

where Σ = sum of exposure as [Child 6 to <11 yr (5 years) + Child 11 to <16 yr (5 years) + child 16 to <21 yr (5 years) + adult 12 yr]

C = concentration of arsenic = 1,500 mg/kg

IR = ingestion rate = 200 mg/day for child and 100 mg/day for adult

EF = 219/365

BW = body weight = Child 6 to <11 yr (31.8 kg) + Child 11 to <16 yr (56.8 kg) + child 16 to <21 yr (71.6 kg) + adult (80 kg)

ED = exposure duration = 33 yr

AT = averaging time = 78 yr

Substituting the values [Soil and Sediment Exposure Dose Guidance (2016)] –

$$\begin{aligned} \text{LECR (unitless)} &= \text{Exposure Dose} \times \text{Cancer Slope Factor} \\ &= 2.3 \times 10^{-3} \end{aligned}$$

Appendix D:

Summary of Public Comments and Responses

Summary of Public Comments and Responses

Quanta Resources Corporation Site Public Health Assessment

The NJDOH held a public comment period from March 4, 2015 through April 5, 2015 to provide an opportunity for interested parties to comment on the draft Public Health Assessment prepared for the Quanta Resources Corporation Site. Written comments were received from two organizations during the public comment period.

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

General Comment:

Comment #1: Conclusion 2 in the draft Public Health Assessment (Report) states that past exposures to site-related contaminants detected in surface soil may have harmed people's health. (Report at 2.) However, the Report's conclusion is based in part on an assumption that in the past, the site was not fenced and area residents frequently visited it for outdoor recreational activities. (Report at 14.) The Report states that the most likely receptors who visited the site for recreation were adults and older children (i.e. 6 to 18 years of age) who lived near the site. Health risks were estimated assuming an individual visited the site 219 days/year (4 days/week) over 30 years, and was exposed to the 95% Upper Confidence Limit (UCL) or maximum concentrations in soil. Report at 17. It is inconceivable to us how an individual could have trespassed on the site 4 days per week for 30 years. The site was either operating or fenced for the bulk of the last 50 years. These conservative health risk assumptions do not comport with the facts regarding the Site and Edgewater's development history. They produce a very biased estimate of the Site risks and possibility of exposure, generating undue concern in the public.

First, prior to 1980, the site was an operating facility and trespassers would not have been tolerated. Second, from 1984 to 1988, the Responsible Parties demolished the site and removed all waste materials and the bulk of the contaminated soil from the surface. Third, we believe the site was fenced sometime in the 1980s when the removal actions were under way. The Remedial Investigation Report itself acknowledges, "the EPA retained regulatory responsibility, maintained security fencing, conducted periodic inspections, and operated an adsorbent boom system" (Report at 6.) All of these steps reduced the likelihood of exposure. Moreover, as to the issue of the age and frequency of trespassers, although some commercial development began in the mid-1980's, the neighborhood was primarily commercial/industrial until the mid-late 1990s and early 2000s, by which time EPA was overseeing the remedial process. Additionally, the Site was not attractive and while it might have enticed the curious for a single visit, there would be no reason why a local resident would continue to revisit a derelict, unattractive industrial site.

The quantitative health risks section should be accompanied, at a minimum, by narrative statements that incorporate information from the site history, which indicates that the

exposure scenario used in the assessment (exposure to the 95% UCL concentrations in soil every day for 219 days/year over 30 years) provides an overly conservative estimate of human health risks.

In addition, we recommend incorporating into the public health assessment other exposure scenarios, based on more realistic exposure scenarios, to provide a range of risks that would be far more representative. Providing narrative information along with quantitative risk estimates, along with providing a range of estimated risks are part of best practices for human health risk assessments. Based on these considerations, the wording of Conclusion 2, that past exposures to site-related contaminants detected in surface soil (0 to 2 feet depth) may have harmed people's health should be revisited.

Response: The exposure scenario used in the public health assessment are selected based on the information collected from the following sources:

- NJDEP Case Managers of this and adjacent sites,
- presentation and discussion(s) at public meetings,
- phone conversation with long time area residents,
- conversation with area residents during availability sessions,
- issues discussed at several community action Group (CAG) meetings,
- information obtained during other site work (e.g., Celotex site), and
- historical aerial photographs.

The area across the site is (and has been) densely populated. The residents used the site and the adjacent areas to access the river for fishing, walking and other recreational activities. As such, exposure assumptions and conclusions of the PHA are consistent with plausible exposure scenario. The cancer risks associated with RME and CTE conditions were evaluated and presented in Table A22 (Appendix A).

Comment #2: Conclusion 3 states that, with respect to the child care center, currently there are no ongoing site-related indoor air exposures to contaminants that can harm children's health. (Report at 2.) However, this conclusion should be revised to state that, as set out in the Report at page 28, there also were not any past exposures at the child care center sufficient to create a health concern. At least as far back as 2001, the Agency for Toxic Substances and Disease Registry (ASTDR) noted that "results of air and soil sample data from the Palisades Child Care Center do not indicate a health concern." See also Public Health Assessment, Quanta Resources Site, September 25, 2002, prepared by New Jersey Department of Health. Further, indoor air monitoring at 115 River Road, including at the child care center space, conducted over the years and shared with both EPA and NJDEP, has not identified any health concerns. (Report at 28.) The conclusion and narrative of the assessment report should be adjusted to note that calculated risks show that there were no expected increases in risks historically to individuals, including children, who may have been exposed to contaminants in the child care center. Specifically, the language should be revised to state that there "currently are no ongoing, and were no past, site-related indoor air exposures to contaminants at the child care center that can harm children's health." Finally, the Report should note that the child care center moved from the 1 IS River Road building in 2012.

Response: Conclusion 3 is based on the indoor air data collected after the interim remedy was implemented. For current and future exposures, the adjusted exposure point concentration of tetrachloroethene detected in the indoor air did not exceed the health guideline comparison value. The calculated lifetime excess cancer risks showed no expected increase in the estimated cancer risk (1 in 100 million individuals exposed). *The past exposures to indoor air contaminants could not be evaluated due to lack of data.* The pathway status is presented in the text as well as in Table A10 (Appendix A).

The relocation date of the child care center was added to this final version of the PHA (see footnote on page 6).

Comment #3: Our organization agrees with Conclusion 1 and 3 but we are not a health agency and therefore, cannot speak to past exposures. As a result, the agency is not at liberty to comment on Conclusions 2 and 5. To further support Conclusion 3, it should be added that the Palisades Child Care Center has since been moved to an off-site building. Our Agency agrees with Conclusion 4 regarding indoor air exposures although in 2014 an IEC condition (NJDEP) was identified in building 9 for elevated naphthalene indoor air concentrations over the NJDEP Rapid Action Level, it has since been remedied and never exceeded an EPA vapor intrusion screening level. It should be noted that annual vapor intrusion monitoring continues at 115 River Road, 163 Old River Road, and 103 River Road. As noted in Conclusion 5, the OU2 Remedial Investigation addressing surface water and sediments of the Hudson River is underway.

Response: Comment acknowledged. A footnote was added to the bottom of page 6 noting that the Palisades Child Care Center relocated to an off-site building in 2012. A footnote was added to the bottom of page 11 noting that annual vapor intrusion monitoring continues at the addresses identified by the commenter.

Comment #4: Our organization's methodology for calculating risk and hazard is different from that of ATSDR. For example, our organization uses different screening levels than ATSDR/NJDOH and generally utilizes a different sampling methodology when sampling specifically for use in the IEUBK model for childhood lead exposures. However, based on the data included in this report and pathways evaluated, we arrive at similar conclusions.

Response: Comment acknowledged.

Comment #5: A final Remedial Investigation for the surface water and sediments of the Hudson River adjacent to the site is underway. Understanding that the PHA is a fluid document, ATSDR has noted that it may need to be updated once a final remedy is selected for these media.

Response: Comment acknowledged.