Public Health Assessment

Final Release

EIGHTEEN MILE CREEK CORRIDOR AND

DOWNSTREAM REACHES

CITY OF LOCKPORT, NIAGARA COUNTY, NEW YORK

EPA FACILITY ID: NYN000206456

Prepared by New York State Department of Health

DECEMBER 18, 2015

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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Prepared by:

New York State Department of Health Center for Environmental Health Under Cooperative Agreement with the U.S. Department of Health and Human Services Agency for Toxic Substances and Disease Registry

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SUMMARY

INTRODUCTION

The New York State Department of Health (DOH) and Agency for Toxic Substances and Disease Registry (ATSDR) want to provide the community around Eighteen Mile Creek with the best information possible about how contaminants in the creek between Lockport and Olcott, New York could affect their health. The public was invited to review the draft of this public health assessment (PHA) during the public comment period, which ran from August 19, 2014 to September 30, 2014. New York State Department of Health did not receive any comments on the draft PHA.

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 mandates that a public health assessment be conducted for each site that is proposed for inclusion on the federal National Priorities List (NPL) by the United States Environmental Protection Agency (EPA). The NPL is EPA's list of the nation's most contaminated hazardous waste sites, also known as Superfund sites. On September 16, 2011, EPA proposed to place the Eighteen Mile Creek site on the NPL and on March 15, 2012, EPA officially listed the Eighteen Mile Creek site on the NPL.

Land use surrounding Eighteen Mile Creek includes a mix of agricultural, recreational, residential, and active and abandoned commercial and industrial properties. The properties also include disposal areas, millraces, millponds, and several bridges, culverts and dams. The "corridor" portion of the creek in the City of Lockport flows through and out of the abandoned commercial and industrial properties, as well as a small residential neighborhood along Water Street.

Some residents use Eighteen Mile Creek for recreation and catch and eat fish from the creek. There are access points and places on the creek where people have been observed fishing. The DOH maintains a "Don't eat ANY fish" advisory for the full length of Eighteen Mile Creek (including waters above and below Burt Dam [DOH 2014]).

Environmental sampling data include chemical contamination data for sediments, surface water, soil, and fill materials in and along the creek. Elevated levels of polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and heavy metals (particularly lead) are found throughout the corridor and length of the creek. Areas of unrestricted access to the creek and former industrial properties increase the risk of exposure to these contaminants. Some residential properties also had elevated contaminant levels resulting from sediment deposition during high water events.

The New York State Department of Environmental Conservation (DEC) issued Records of Decision (RODs) in March 2006 and March 2010 to address PCB and metal

contamination at the Flintkote property (which borders the creek and Mill Street) and in the creek corridor, respectively. DEC did not implement either remedy; however, in 2011 DEC requested that the United States Environmental Protection Agency (EPA) consider the site for inclusion on the National Priorities List (NPL). In March 2012, EPA listed the site on the NPL and became the lead agency for the Site.

The site, as defined by the EPA, includes the Eighteen Mile Creek from the New York State Canal, in Lockport, New York, to the creek's discharge into Lake Ontario at Olcott, New York. The creek receives waters from both the New York State Barge Canal (the Erie Canal) and urban upland watershed sources. The DEC previously subdivided the Eighteen Mile Creek Corridor site into six Operable Units (OUs). The EPA subdivided the site in a different manner and is addressing it in three OUs. EPA OU-1 addresses nine contaminated residential properties impacted by flooding and contaminated sediment deposition from the creek, and the demolition of an old industrial building at the former Flintkote property on Mill Street. EPA OU-2 addresses contaminated creek sediment and several other contaminated properties in the creek corridor. EPA OU-3 addresses contaminated sediment in the creek north of the corridor to Lake Ontario.

CONCLUSION 1

Eating fish taken from Eighteen Mile Creek could harm people's health if they do not follow DOH's fish consumption advisories.

BASIS FOR DECISION

Contaminated sediments in Eighteen Mile Creek have impacted fish species. If consumed, fish in Eighteen Mile Creek contain PCBs at levels that could harm people's health. DOH has a "Don't eat <u>ANY</u> fish" advisory for all people for the full length of Eighteen Mile Creek (including waters above and below Burt Dam). Fish advisories for Eighteen Mile Creek and other New York State water bodies are available on the DOH website (DOH 2014).

CONCLUSION 2

DOH and ATSDR conclude that prolonged contact with Eighteen Mile Creek sediments and contaminated fill materials in sediments in the corridor area could harm people's health.

BASIS FOR DECISION

The highest level of total PCBs in creek sediments is estimated to pose a moderate risk for noncancer health effects and the highest level of benzo[a]pyrene poses a moderate risk for cancer based on exposure to the sediments through incidental ingestion and

dermal contact if the sediment are contacted during recreational activities such as wading in the creek. However, long-term significant exposure to the sediments with elevated contaminant levels appears unlikely because these sediment samples are from deeper waters and/or from locations in the creek corridor that are inaccessible due to vegetation and steep creek embankments.

There are several accessible areas where contact with sediments and fill is possible, through fishing, wading and other recreational activities. Accessible areas include the mill pond areas and locations downstream of the corridor specifically developed for fishing access. Property owners along the creek corridor have attempted to secure contaminated areas from trespassers and recreational fishermen, but there is evidence of trespass.

CONCLUSION 3

The DOH and ATSDR conclude that past contact with sediment and soil in residential backyards on Water Street (EPA OU-1) could have harmed people's health. Actions taken by EPA in the fall of 2013 (installation of a clean soil cover over contaminated soil) followed by relocation of residents in 2014 prevents future residents or trespassers from contacting contaminated soil in the backyards on Water Street.

BASIS FOR DECISION

Repeated, long-term past exposure to arsenic and chromium in surface soil and sediment is estimated to pose a moderate risk for cancer, and repeated long-term exposure to PCBs at the creek bank locations (where the highest levels were found at the residential properties) is estimated to pose a moderate risk for noncancer health effects. In addition, past exposure to elevated lead levels in soil and sediment in several residential yard areas could result in increased exposure of children and adults to lead through incidental ingestion. Based on results using the EPA Integrated Exposure Uptake Biokinetic Model (IEUBK), this increased exposure could result in increases in blood lead levels.

CONCLUSION 4

The DOH and ATSDR conclude that prolonged contact with surface soil while trespassing onto the former Flintkote plant property and long-term exposure to surface soil at Upson Park could harm people's health. Parts of the Flintkote property also constitute a physical hazard.

BASIS FOR DECISION

Exposure to PAHs in surface soil at the Flintkote Plant property poses a moderate increased risk of cancer, assuming someone trespassed onto the property for two days per week, six months per year for 11 years, and was exposed to the soil contaminants by incidental ingestion and skin contact. With fencing around the property to restrict access, the cancer risk may be lower since it is unlikely that an individual would trespass onto the property with the same frequency and duration assumed by the exposure scenario.

Based on the available samples, the highest level of total PCBs at Upson Park may be localized to a specific area and therefore may not be representative of potential exposures for the entire park. However, repeated, long-term exposure to the highest level of total PCBs in surface soil at Upson Park is estimated to pose a moderate risk for noncancer health effects. The estimated exposure is only 29 times lower than the lowest exposure that caused immune toxicity in laboratory animals.

Elevated lead levels in soil at the Flintkote property and at Upson Park could result in increased exposure of children and adults to lead through incidental ingestion. Based on results using the EPA Integrated Exposure Uptake Biokinetic Model (IEUBK), this exposure could result in increases in blood lead levels.

GENERAL RECOMMENDATIONS

For those people using or living along the Eighteen Mile Creek corridor and downstream, the DOH and ATSDR recommend measures to reduce exposure to contaminated soils and sediments. People recreating in or living around the creek can reduce the risk of exposure to chemical contaminants by avoiding the creek sediments and unfenced fill areas along the corridor, particularly after periods of high water flow, when new sediment may be deposited or existing sediment may be scoured. Since the greatest exposure to the contaminants is by contacting sediments or soils at the creek bank, or accessing shoreline fill areas, avoid any activity that would result in contacting these areas of contamination. If people do contact sediments, washing hands would reduce exposure, especially before eating. If people get sediments on more than just their hands and arms, it may also be helpful to take a shower to wash off the creek mud. If people walk in the shoreline areas, remove shoes upon entering homes to reduce the potential for tracking sediment inside.

Do not eat any fish taken from Eighteen Mile Creek. Follow the DOH consumption advisory "Don't eat <u>ANY</u> fish" for the entire Eighteen Mile Creek.

DOH and ATSDR further recommend that EPA maintain access restrictions to the former Flintkote property and evaluate the extent of PCB and lead contamination of

surface soil at Upson Park. Based on the evaluation at Upson Park, additional exposure reduction measures may be warranted.

NEXT STEPS

The DOH has already provided exposure reduction advice in letters written to homeowners of Water Street after soil in their yards was sampled in 2002. Six privately owned properties on Water Street have been acquired by the federal government and EPA has relocated residents. As an interim remedial measure, EPA placed clean soil over the properties to reduce the potential for exposures while residents awaited relocation.

- Additional investigations are planned by the EPA. ATSDR and DOH will work with DEC and EPA on any plans for further evaluation of the nature, extent and possible sources of contamination in the Eighteen Mile Creek corridor. ATSDR and DOH will evaluate data as they become available to determine whether additional actions are needed to reduce people's exposure to contamination in the creek or other areas of the site.
- 2. ATSDR and DOH will work with EPA as it determines whether additional measures (e.g., investigations, sampling, or remedial actions) are needed in the corridor, and whether measures will be needed in the downstream portion of Eighteen Mile Creek.

FOR MORE INFORMATION

If you have questions about the environmental investigation of Eighteen Mile Creek, please contact the EPA Field Office of Niagara at (716) 551-4410. If you have questions about this final public health assessment or other health concerns about this site, please contact Mr. Matthew Forcucci of the DOH at 716-847-4501.

PURPOSE AND HEALTH ISSUES

The purpose of this public health assessment (PHA) is to evaluate human exposure pathways and health risks for contaminants from the Eighteen Mile Creek site in Niagara County, New York. This PHA fulfills the congressional mandate that a public health assessment be conducted for every site proposed for inclusion on the federal National Priorities List (NPL). The Eighteen Mile Creek site was proposed for inclusion on the NPL on September 16, 2011 and the United States Environmental Protection Agency (EPA) added it to the NPL on March 15, 2012.

In 1985, the EPA's Great Lakes Program Office designated a portion of Eighteen Mile Creek as the "Eighteen Mile Creek Area of Concern (AOC)" (EPA, 2014). The portion of the creek that is the AOC starts just downstream of Burt Dam, and extends to its outlet to Lake Ontario in Olcott Harbor. However, all of Eighteen Mile Creek and its watershed are considered a "source area of concern." The creek was designated as an AOC because of water quality and sediment problems associated with past industrial and municipal discharge practices upstream of the AOC. Contaminants from the creek sediments have impacted Lake Ontario (EPA, 2014).

BACKGROUND

Site Description and History

Eighteen Mile Creek, in the heart of Niagara County, is surrounded by six residential townships, and many citizens own creek-front property. Portions of the creek are used extensively for fishing, boating, and recreation. During operation, the New York State Barge Canal discharges approximately 50 cubic feet of water per second into the East and West Branches of the creek. During dry periods, the New York State Barge Canal provides a significant portion of the creek's flow. The portion of the creek in the City of Lockport flows through and out of several abandoned commercial and industrial properties, as well as a small residential neighborhood and is referred to as the "corridor." The creek corridor itself consists of approximately 10.6 acres between Clinton and Harwood Streets in the City of Lockport (Appendix A, Figure 1). The corridor is bounded by Water Street, residential properties and vacant land to the west, Clinton Street to the south, Mill Street to the east and commercial property to the north. The topography of the site is relatively flat with a steep downward slope toward Eighteen Mile Creek and the millrace, which bisects the former Flintkote property.

Eighteen Mile Creek north of the New York State Barge Canal originates from East and West branches. Water from the East branch originates at the spillway in the Barge Canal near the Mill Street Bridge where canal water joins with water from the culverted section of Eighteen Mile Creek south of the canal. This water flows north under the Barge Canal toward Clinton Street. Water from the West branch originates from the dry dock on the north side of the Barge Canal and also flows north toward Clinton Street. Water from the East and West Branches converges south of Clinton Street and flows under the street to a mill pond. The mill pond is formed by the Clinton Street Dam on the former United Paperboard Company property. Water from Eighteen Mile Creek eventually discharges to Lake Ontario in Olcott, New York, which is about 13 miles north of the site (Appendix A, Figure 2).

In the Eighteen Mile Creek Corridor Area, there are four distinct geologic units. Topsoil is often encountered above fill material, but is absent in some areas of the site. Where encountered, the thickness of the topsoil layer is usually less than 0.2 feet. Fill material is often present and consists primarily of various colored ash and cinder material containing glass, coal, coke, slag, buttons, metal, ceramic, rubber and brick. Where encountered, the thickness of the fill material ranges from 0.9 to 24.9 feet. A glaciolacustrine deposit sits directly over bedrock, and ranges in thickness from 0.1 to more than 28 feet. Finally, Dolostone bedrock with interbedded clay underlies the southern portion of the site and red and white sandstone underlies the northern portion of the site. Depth to bedrock at the site ranges from 1.6 to more than 28 feet, with the greater depths generally associated with the thicker fill areas.

Groundwater in the area occurs in both the overburden and upper fractured bedrock, and flows toward Eighteen Mile Creek. Saturated conditions are not encountered in the overburden soils at the northern portion of the site east of Eighteen Mile Creek and at the southern portion of the site west of the creek. Groundwater in these areas is confined to the upper bedrock. As groundwater flows toward Eighteen Mile Creek, it discharges from the bedrock into the overburden along the creek. Groundwater continues to flow within the overburden and discharges to Eighteen Mile Creek and the millrace.

Operable Units

EPA has subdivided the Eighteen Mile Creek Corridor into three Operable Units (OUs) (Appendix A, Figure 3). An OU represents a portion of the site that for technical or administrative reasons can be addressed separately to eliminate or mitigate a release, threat of release or exposure pathway resulting from the site contamination. The OUs at the Eighteen Mile Creek Corridor Site are defined as follows:

Operable Unit 1 (OU-1) - Water Street Properties and Former Flintkote Building

OU-1 consists of nine adjoining residential properties approximately 2.25 acres in size and located on the east side of Water Street (Appendix A, Figure 3). They are bounded to the north by the Water Street Section of the former Flintkote Plant Site, to the east by Eighteen Mile Creek, to the south by Olcott Street, and to the west by Water Street. Six of the properties are privately owned. Houses are located on five of the six private properties. The three other properties, which are empty lots, are owned by the City of Lockport. All of the properties have been impacted by fill material eroding onto the properties from the Water Street Section of the Flintkote property, and by the deposition of contaminated creek sediments during flooding events. Consequently, the properties have elevated levels of PCBs and other contaminants, including lead and chromium.

The EPA issued a Record of Decision (ROD) for OU-1 on September 30, 2013. The ROD called for cleanup of nine residential properties located on Water Street. The six privately owned properties have since been acquired by the federal government. Residents from the five homes have been relocated and the houses will be demolished. As an interim remedial measure, EPA placed clean soil over the properties to reduce potential exposures while the residents awaited relocation. Approximately 5,800 cubic yards of contaminated soil will be excavated from the nine properties and will be transported off-site to an appropriate receiving facility.

OU-1 also includes the former Flintkote property, which is approximately 6.0 acres in size and consists of the parcels at 198 and 300 Mill Street. The section of 300 Mill Street parcel between Eighteen Mile Creek and the millrace is referred to as the Island. A small portion of the property extends between the creek and the residential properties on Water Street and is referred to as the Water Street Section.

The Flintkote Company began operations as a manufacturer of felt and felt products in 1928, when the property was purchased from the Beckman Dawson Roofing Company. In 1935, Flintkote began production of sound-deadening and tufting felt for installation and use in automobiles. Manufacturing of this product line was continued at Flintkote until December 1971, when operations ceased and the plant closed. The disposal history of the Flintkote Company is largely unknown, although aerial photographs suggest that disposal of fill on the island was occurring by 1938. It has also been reported that ash resulting from the burning of municipal garbage was dumped on the property. The fill material on the 198 Mill Street parcel and Island is consistent with such a source.

The September 2013 ROD calls for the demolition of the former Flintkote building at 300 Mill Street. The building is dilapidated and poses a potential physical hazard, and some of the building material is also contaminated with lead and PAHs. The demolition work is expected to be completed by the spring of 2015, and removal of the building will allow the EPA to complete the characterization of the property, which was started by the New York State Department of Environmental Conservation (DEC). EPA has also secured the property with a fence which will be maintained to keep trespassers out.

Operable Unit 2 (OU-2) – Creek Sediments and Corridor Properties

EPA's OU-2 investigation addresses contamination in creek sediment and at several properties in the corridor (Appendix A, Figure 3). These properties include:

The Creek and Millrace in the Corridor

The creek and millrace in the corridor consists of approximately 4,000 linear feet of contaminated creek and millrace sediment located between the New York State Barge Canal and Harwood Street. This area has been impacted by fill material eroding into the creek from the former Flintkote property to the White Transportation property (located at 30 to 40 Mill Street), and by direct discharges to the creek from the various facilities that operated at these operable units.

Former United Paperboard Property

This portion of OU-2 consists of the former United Paperboard Company property at 62 and 70 Mill Street and two adjoining parcels separated by Olcott Street. The property is about 4.8 acres in size and is bounded to the north by the former Flintkote Plant site, to the east by Mill Street, to the south by Clinton Street and to the west by Water Street and residential properties. The United Paperboard Company operated at this location between the late 1880s and early 1890s as a lumber company, and as a paper company from the late 1890s until at least 1948. The history of the property after 1948 is unknown. The disposal history of the United Paperboard Company property is unknown, although ash similar to that at the former Flintkote property is observed at the surface in many locations. Coal ash from the power plant east of Mill Street and operated by the United Paperboard Company may also have been disposed of on the United Paperboard Company property along Eighteen Mile Creek. The portion of the property near the Clinton Street/Mill Street intersection is currently occupied by Duraline Abrasives. The OU-2 investigation will address contamination at the former United Paper Board property.

Upson Park

Upson Park is about 5.9 acres in size and is located on Clinton Street. In the mid-1880s the Upson Park property was used by a canal boat building company. By 1892 the canal boat company was no longer in operation, but a pulp mill and pulp company operated on the property. The pulp mill operated until sometime between 1919 and 1928, while the pulp company operated until at least 1928. The pulp company was in ruins by 1948. The history of the property after that time is unknown. The property is bounded to the north by Clinton Street, to the east by the White Transportation property and property owned by New York State, to the south by the New York State Barge Canal and property owned by New York State, and to the west by wooded, vacant land. The disposal history of the Upson Park property is also unknown, although ash similar to that at other properties within the Eighteen Mile Creek Corridor site is observed at the surface along the creek. Upson Park is a public park along the Erie Canal used for walking, picnicking and other passive leisure activities. The OU-2 investigation will address contamination at Upson Park.

White Transportation Property

The White Transportation property is about 2.6 acres in size and consists of four adjoining parcels at 30 through 40 Mill Street. The property was used by White Transportation to store tractor-trailer trucks and other equipment associated with trucking from 1948 until the late 1990s, when operations ceased. Prior to 1948, the property was used by the New York Cotton Batting Company, the James O. Ring Company, the Niagara Paper Mills, the D.C. Graham Box Factory, the L. Huston Cold Storage Facility, the Lockport Leather Board Company, and the Simon William Brewery. The property is bounded to the north by Clinton Street, to the east by Mill Street, to the south by the New York State Barge Canal and property owned by New York State, and to the west by Upson Park and property owned by New York State. The disposal history of the White Transportation property is unknown, although slag material is observed at the surface. When White Transportation closed, tractor-trailers were located throughout the property, many of which contained drums and miscellaneous debris. An open drum containing a petroleum product was observed along Eighteen Mile Creek during the site reconnaissance conducted as part of the Supplemental Remedial Investigation. The trailers and related drums have been removed, but miscellaneous debris remains scattered throughout the property. The OU-2 investigation will address contamination at the White Transportation property.

Operable Unit 3 – The Creek North of the Corridor to Lake Ontario

Once past the corridor area, the Eighteen Mile Creek flows through agricultural lands and several small hamlets to Lake Ontario at Olcott, New York. Access to the creek through this stretch is difficult due to either shoreline growth or bank steepness (up to 35 feet in elevation drop), although fishing access locations have been developed and enhanced by DEC at the Burt Dam (Appendix A, Figure 2). EPA and DEC have divided the Eighteen Mile Creek into a series of "reaches", starting with the northern most reach designated "Reach 1" and the corridor "Reach 10". OU-3 will address contaminated sediment from the corridor to the creek's discharge to Lake Ontario.

NYSDEC Actions

In two separate RODs issued in 2006 and 2010, the DEC determined that several remedial measures were needed to address contamination and potential exposures to contaminated fill and sediments associated with the Eighteen Mile Creek Corridor site. The 2010 ROD addressed the Eighteen Mile Creek and millrace, the United Paper Board property, Upson Park, the White Transportation property and the Water Street properties. The 2006 ROD addressed the former Flintkote Property and specified sediments for excavation and containment. For the former United Paperboard property, Upson Park and the White Transportation property, the ROD specified that hazardous waste would be removed with bank stabilization and long-term monitoring. For the

Water Street residential properties, the remedy called for targeted excavation with bank stabilization and long-term monitoring.

While the nature and extent of the contamination varies at each of the properties, the DEC drafted the RODs to address both short term and long-term exposure issues related to elevated PCBs, PAHs, lead and other metals that are found in creek sediments and fill materials. DEC referred the site to the EPA for inclusion on the NPL in 2011. The remedies called for in DEC's RODs have not been implemented.

Site Visit

On October 25, 2011, staff from the New York State Department of Health (DOH), the Agency for Toxic Substances and Disease Registry (ATSDR), DEC, Niagara County Health Department and the EPA visited the areas surrounding the Eighteen Mile Creek Corridor to evaluate inactive hazardous waste sites situated along the creek and to visually assess the impact of these sites on the creek. The group discussed and observed access to and recreational use of the creek. At the time of the visit, the group observed no active recreational activity on or near the creek.

Demographics

The DOH estimated from the 2010 Census (US Census Bureau 2011a) that approximately 12,460 people live within one mile of Eighteen Mile Creek. There were about 2,800 females of reproductive age (ages 15-44 years) living within one mile of the site. Based on the 2006-2010 American Community Survey (US Census Bureau 2011b), the area had a higher percentage of the population living below the poverty level, and a lower median household income than those of Niagara County or New York State excluding NYC. Table A shows these comparisons.

2010 Census Demographics	Area Within One Mile of Eighteen Mile Creek Corridor Site	Niagara County	New York State Excluding New York City
Population ¹			
Total	12,460	216,469	11,202,933
Male	48.9%	48.5%	49.0%
Female	51.1%	51.5%	51.0%
Age Distribution ¹			
Less than 6 years	8.3%	6.4%	6.9%
6-19 years	18.9%	18.0%	19.0%
20-64 years	61.6%	59.7%	59.6%
Greater than 64 years	11.3%	15.9%	14.5%
Race/Ethnic Distribution ¹			
White	84.6%	88.5%	81.6%
Black or African American	9.2%	6.9%	8.8%
American Indian and Alaska Native	<1%	1.1%	<1%
Asian	<1%	<1%	3.4%
Native Hawaiian and Other Pacific Islander	0.0%	0.0%	0.0%
Some Other Race	<1%	<1%	3.4%
Two or More Races	4.2%	2.2%	2.3%
Hispanic	3.9%	2.2%	9.6%
Minority*	17.6%	12.7%	23.4%
Economic Description ²			
Median household income	\$32,463.17	\$45,964	\$59,994
Percent below poverty level	25.0%	12.8%	10.5%

Table A. Demographics for the Community Living Near the Eighteen Mile CreekCorridor Site (Niagara County) Compared with County and State Data.

¹US Bureau of the Census (2011a).

 ^2US Bureau of the Census (2011b).

*Minorities include Hispanics, Blacks or African Americans, American Indians and Alaska Natives, Asians, Native Hawaiians and Other Pacific Islanders, individuals of some other race, and individuals of two or more races.

DISCUSSION

DOH and ATSDR used available environmental and exposure information to complete an assessment of health risks posed by exposure to chemical contaminants at the Eighteen Mile Creek Corridor site. The assessment is based on the environmental contamination data for creek sediments, surface soil and surface water. Historic sampling data of fish taken from Eighteen Mile Creek are not presented here, but were used as the basis for the current fish consumption advisory issued by DOH for the creek ("Eat None" [DOH 2014]). Information about how much fish people catch and eat from the creek is not available, although anecdotal evidence suggests that eating fish from the creek is common.

Environmental Contamination

Environmental contamination data for Eighteen Mile Creek, from the contaminated areas of the creek corridor and downstream to its outlet into Lake Ontario, were collected over several years. Additional data have recently been summarized by EPA in their March 2012 Remedial Investigation Report (CH2M Hill et al. 2012). For this public health assessment, DOH evaluated surface water, surface soil and fill, and surface sediment data because these are the media that people are most likely to contact. Nevertheless, deeper soil and sediment could become available for contact if disturbed by human activity or flood erosion.

Surface Water

DEC collected surface water samples in 2008 (DEC 2008). Two samples upstream of the Flintkote property did not contain detectable levels of PCBs (detection limit 0.050 micrograms per liter [mcg/L]), and three samples downstream of the property contained the commercial PCB mixture Aroclor 1248 at levels ranging from 0.084 mcg/L to 0.33 mcg/L. These levels of PCBs in the surface water are below the New York State public drinking water standard and the federal Maximum Contaminant Level drinking water standard for PCBs of 0.5 mcg/L. The level of Aroclor 1248 in the three surface water samples exceeds the ATSDR Cancer Risk Evaluation Guide (CREG) of 0.018 mcg/L for PCBs (ATSDR, 2013a).

Surface Soil

DOH used summary data from DECs RODs for the OUs to evaluate soil contamination. Data for these RODs came from remedial investigation reports. Data for the former Flintkote property were taken from DEC's 2006 ROD (DEC 2006a). Data for the United Paperboard property, Upson Park and the White Transportation property were taken from DEC's 2010 ROD (DEC 2010). Data for the Water Street properties are from the DEC Remedial Investigation for the Eighteen Mile Creek

Corridor site (DEC 2006b). PCB Aroclor-specific data for the former United Paperboard property and Upson Park also came from the Remedial Investigation Report (DEC 2006b). Table B shows surface soil results for PCBs and lead.

Surface Sediments

Data for chemical contamination of Eighteen Mile Creek surface sediments within the corridor and downstream from DEC OUs were taken from EPA's Hazardous Ranking System (HRS) documentation for Eighteen Mile Creek National Priorities Listing (EPA 2011a; Ecology and Environment Engineering PC 2009; CH2M Hill et al. 2011) (Table B).

Table B. Ranges of Surface Soil and Sediment Sampling Resultsfor PCBs and Lead at Eighteen Mile Creek.

(all values in milligrams per kilogram soil or sediment)

Sampling Location	Total PCBs*	Aroclor 1242	Aroclor 1248	Aroclor 1254	Aroclor 1260	Lead
Surface Soil						
Creek in Creek Corridor	N/A	N/A	N/A	N/A	N/A	N/A
Former Flintkote Property	ND – 4.6	ND	ND	ND – 4.6	ND	58 - 7610
Former United Paper Board Property	ND - 4.3	NA	NA	NA	NA	4.5 - 3600
Upson Park	ND - 23	NA	NA	NA	NA	19 - 3480
White Transportation Property	ND - 0.67	NA	NA	NA	NA	9.7 - 3750
Water Street Properties	ND - 27	NA	NA	NA	NA	30 - 4630
Sediments						
Creek in Creek Corridor	ND - 201	NA	NA	NA	NA	60 - 25,400
Creek Corridor Properties	N/A	ND - 46	ND- 180	ND - 57	ND	NA
Downstream of Creek Corridor	N/A	ND	0.38 - 41	0.52 - 55	8.3 - 42	NA

*Total PCBs and Aroclor data came from different data sets, therefore the sum of the Aroclor results will not necessarily equal the total PCB value.

ND = not detected; N/A = not applicable; NA = not available.

Pathways Analysis

This section of the public health assessment identifies completed exposure pathways associated with past, present and future uses of the creek. An exposure pathway describes how an individual could be exposed to contaminants in the creek and the surrounding area. An exposure pathway is comprised of five elements:

- (1) A contaminant source,
- (2) Environmental media and transport mechanisms,
- (3) A point of exposure,
- (4) A route of exposure, and,
- (5) A receptor population.

The source of contamination is the place where contaminant releases to the environment occurs (any waste disposal area or point of discharge). In the case of Eighteen Mile Creek, the original source is unknown. Environmental media and transport mechanisms carry contaminants from the source area to points where human exposures may occur. The exposure point is a location where actual or potential human contact with a contaminated medium (soil, air, water, biota such as fish) may occur. The route of exposure is the manner in which a contaminant actually enters or contacts the body (ingestion, inhalation, and dermal absorption). The receptors are the people who are exposed or may potentially become exposed to contaminants at a point of exposure. A completed exposure exists when all five elements of an exposure pathway are documented. A potential exposure pathway exists when any one of the five elements comprising an exposure pathway is not met or not known to have been met.

Completed Exposure Pathways

People use the creek and its surrounding sediments for wading, fishing, and boating. This occurs in both the corridor areas and the downstream portion of the creek. Some people may also be swimming in some locations, however, there are no permitted bathing or swimming facilities situated along the boundaries of the Creek according to the Niagara County Department of Health. People are likely exposed, to a varying extent, to contaminated sediments during these recreational activities. People who eat fish taken from the creek are being exposed to site-related contaminants contained in the fish. Exposure to contaminants in creek water is also a completed exposure pathway.

People trespassing onto the former industrial and commercial properties and people using Upson Park may be exposed to surface soil contamination on those properties.

Residents along Water Street who have backyards that are contaminated with site related contamination from flooding and deposition of sediment may have likely been in contact with contaminants during normal backyard use. Residents in the past have

been advised to avoid contaminated areas in their backyards and keep the areas covered with grass or vegetation. However, we do not know whether residents have heeded this advice. As mentioned, EPA placed clean soil on the properties as an interim remedial measure to reduce potential exposures. In 2014, EPA acquired the privately owned properties on Water Street and relocated the residents to comparable housing.

Eliminated Exposure Pathways

No use of contaminated groundwater is occurring and no private or public wells are likely to be impacted. The affected area in Lockport is served by public water.

Public Health Implications and Adult/Child Health Considerations

Contact with contaminated surface soils on some residential properties and at some locations at Upson Park along the Eighteen Mile Creek could result in increased exposure to site related contaminants. Trespassing on the Flintkote Plant site, the former United Paperboard property and the White Transportation property could also result in contact with surface soil contaminants. Recreational use of the Eighteen Mile Creek during activities such as swimming, boating and fishing could also result in exposure to chemical contaminants in sediments and surface water. An evaluation and characterization of the health risks for exposure to contaminants in surface soil, sediment and surface water is presented below.

People who eat fish taken from Eighteen Mile Creek are likely exposed to contaminants contained in these fish. However, we do not have information about which and how much fish people catch and eat from the creek, we therefore cannot evaluate these exposures.

DOH compared the highest detected level of contaminants in surface soil and creek sediments to the corresponding New York State residential or restricted residential soil cleanup objectives (SCOs) (DEC/DOH 2006) and ATSDR comparison values for soil (ATSDR 2013b). SCOs are soil concentrations that are contaminant-specific remedial goals based on current, intended or reasonably anticipated future land use, and are set at a soil concentration at which cancer and noncancer health effects are unlikely to occur¹. The restricted residential and residential health-based SCOs are based on the assumption that people living at a property are exposed through ingestion of contaminated soil, indoor dust and inhalation of soil particles in air. The residential SCOs also assume exposure through homegrown fruits and vegetables, while the restricted residential SCOs do not. ATSDR comparison values for soil are chemical-

¹ New York State health-based SCOs are set at a cancer risk level of one in one million for carcinogens, and at a hazard quotient of one for noncancer effects. If the health-based SCO is lower than the contaminant's rural soil background level, the rural soil background level is used as the final SCO.

specific soil concentrations that are used by ATSDR health assessors and others to identify environmental contaminants at hazardous waste sites that require further evaluation. They incorporate generic assumptions of daily exposure to the chemical and a standard amount of soil that someone may likely take into their body each day.

DOH selected residential soil contaminants for a more site-specific evaluation if the highest detected levels exceeded their New York State residential SCOs or ATSDR comparison values. Likewise, using data from DEC's investigation of the creek corridor properties (e.g., Flintkote, United Paperboard and White Transportation), DOH selected soil contaminants and creek sediment contaminants for a more site-specific evaluation if the highest detected levels exceeded their New York State restricted residential SCOs or ATSDR comparison values. Contaminants at Upson Park were also selected based on comparison with the restricted residential SCO and ATSDR soil comparison values. Exposure assumptions for these site-specific evaluations are briefly described below.

For residential soil contaminants, DOH evaluated the risk for cancer assuming that for the first 30 years of life (which approximates the 95th percentile value for residential occupancy [EPA 2011b]), a person is exposed to soil by ingestion and skin contact 31 weeks each year (five days per week for the first 17 years, and two days per week for the next 13 years) during non-winter months (i.e., to account for the portion of the year when the ground is not frozen or snow covered). For noncancer health endpoints, DOH assumed a toddler is exposed by incidental ingestion and skin contact five days a week during the non-winter months. For residential properties, DOH assumed that additional exposure to soil contaminants could occur through ingestion of homegrown fruits and vegetables. However, for soils in the creek bank, exposure via homegrown fruits and vegetables is unlikely because the bank is wooded, shady, subject to periodic flooding, and unsuitable for gardening. Therefore, for creek bank soils, DOH evaluated the risks for soil ingestion and dermal contact only. Additional details on the exposure parameters used to evaluate risks at residential properties are found in Appendix C, Table 1.

For properties involving trespassing (Flintkote, United Paperboard and White Transportation properties), DOH assumed an adolescent is exposed by soil ingestion and skin contact two days per week and six months per year (to account for the portion of the year the ground is not frozen or snow covered). To evaluate the cancer risk for these properties DOH assumed 11 years of exposure (representing ages 10 to 21, or the ages of people who might reasonably be anticipated to trespass on the properties). DOH evaluated the noncancer risks for a 10 year old child. DOH assumed shorter exposure duration and less exposure frequency than for residential areas because people are not living on the properties and are unlikely to trespass as adults. Additional details on the exposure parameters used for these nonresidential properties are found in Appendix C, Table 2. For a park or a recreational area, such as Upson Park, DOH evaluated the contaminant levels in soil using the same assumptions used for the residential properties, excluding exposure via homegrown fruits and vegetables. Additional details on the exposure parameters used to evaluate soil contaminants at Upson Park are found in Appendix C, Table 3.

For estimating health risks in creek sediments, DOH assumed that a person is exposed to sediments by ingestion and dermal contact two days per week for four months (mid-May to mid-September) each year (since it is unlikely a person would swim or wade in the creek during the non-summer months). DOH evaluated the noncancer risks for a 3 year old child, and for cancer risks, DOH assumed a person is exposed for 15 years from age 3 to 18. Additional details on the exposure parameters used to evaluate risk for creek sediment contaminants are found in Appendix C, Table 4.

The cancer risk is estimated by multiplying the exposure from soil or sediment by the contaminant's cancer potency factor, which is a numerical estimate of the carcinogenic strength (potency) of a chemical. To evaluate noncancer risks, the estimated exposures are compared to the contaminant's reference dose, which is a lifetime exposure to the contaminant that is expected to be without appreciable risk for noncancer health effects. Cancer potency factors and reference doses used in this evaluation are found at the end of Appendix C.

Residential Properties on Water Street

Chemicals Selected for Further Evaluation

As shown in Table C, the residential soil levels of PCBs, arsenic, chromium, lead and zinc exceeded their residential SCOs or ATSDR comparison values. DOH therefore selected these contaminants for further evaluation using a site-specific assessment.

Table C. Contaminants Detected Above Residential Soil CleanupObjectives at Residential Properties along the Eighteen Mile Creek.

Contaminant	Highest Level Detected in Soil at Residential Properties	NYS Residential Soil Cleanup Objective ¹	ATSDR Comparison Value ²
PCBs	27	1	0.35
arsenic	66.5	16ª	0.47
chromium	164	22 ^b	50
lead	4630	400	
zinc	2390	2200	15,000

(all values in milligrams per kilogram soil)

¹DEC/DOH (2006c)

²ATSDR (2013b)

^aBased on New York State rural soil background concentration.

^bBased on chromium (VI).

NYS = New York State; ATSDR = Agency for Toxic Substances and Disease Registry; PCBs = polychlorinated biphenyls.

A general discussion of the health effects of the residential soil contaminants selected for further evaluation is presented below.

Health Effects of PCBs (ATSDR 2014; EPA 2013)

PCBs are a group of man-made chemicals (also known by the trade name Aroclor) that have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment. The manufacture of PCBs was stopped in the United States in 1977 because of evidence that they build up in the environment and can cause harmful health effects.

The most commonly observed health effects in people exposed 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.

Animals that ate food containing large amounts of PCBs 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.

A 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 has concluded that PCBs may reasonably be anticipated to be carcinogens (NTP 2014). EPA classifies PCBs as probable human carcinogens, and the International Agency for Research on Cancer classifies PCBs as carcinogenic to humans (Group 1) (EPA 1996a; IARC 2014).

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 that evaluated motor skills and short-term memory. Other studies suggest that the immune system was affected in children born to and nursed by mothers exposed to increased levels of PCBs.

Health Effects of Arsenic

Arsenic is a metal found in ores of copper, lead and other minerals, and in soil, groundwater and surface water. Arsenic compounds are used in wood preservatives and have been used in commercial pesticides.

The EPA classifies arsenic as a human carcinogen based on convincing evidence from a large number of scientific studies that show an increased risk for skin, lung and bladder cancer among people who have been exposed to high levels of arsenic in drinking water (EPA 1998; ATSDR 2007a; NRC 2001; NTP 2014).

In addition, recent evidence from studies of people and animals suggests that the very young may be more sensitive to the carcinogenic effects of arsenic than adults (Ahlborn et al. 2009; Marshall et al. 2007; Smith et al. 2006; Tokar et al. 2011; Waalkes et al. 2003, 2006, 2007, 2009). Arsenic also causes noncancer health effects such as stomach irritation, and effects on the nervous system, heart, blood vessels and skin (ATSDR 2007a).

Health Effects of Chromium

Chromium is a common element in rocks, soil, water, plants, and animals. It gets into surface or groundwater after dissolving from rocks and soil. Chromium is used to manufacture steel, to electroplate metal, and in the textile, tanning, and leather industries. Chromium is found in the environment in two principal forms: chromium (III) and chromium (VI). Chromium (III) compounds are the most common chromium compounds in the environment. Chromium (VI) compounds are less common in the environment and are typically associated with an industrial source. Depending on the conditions, each form of chromium can be converted into the other form in the environment.

Chromium (VI) is the more toxic form of chromium. There is strong evidence from human studies in many countries that occupational exposures to chromium (VI) in air can cause lung cancer (ATSDR 2012a). There is weaker evidence from studies in China that long-term exposure to chromium (VI) in drinking water can cause stomach cancer. Chromium (VI) causes cancer in laboratory animals exposed almost daily to high levels in air (lung cancer) or drinking water (mouth and intestinal cancers) over their lifetimes (NTP 2008). Adverse noncancer gastrointestinal tract effects (oral ulcers, stomach or abdominal pain, diarrhea) also are associated with long-term human exposures to oral doses of chromium (VI). In laboratory animals, repeated exposures to high oral doses of chromium (VI) has caused blood, liver, and kidney damage in adult animals, and can adversely affect the developing fetus and the male and female reproductive organs (ATSDR 2012a).

Health Effects of Lead

Lead is a naturally occurring metal that is resistant to corrosion. It can form alloys with other metals used in pipes, automotive batteries, weights, shot and ammunition, cable covers, and radiation shields.

Exposure to lead can occur by ingesting lead in paint chips or dust, by breathing in lead dust, by ingesting lead in soil or sediments, and by drinking water that contains lead. Lead can be harmful to health when it builds up in the body. Young children are at greater risk of health effects from lead than older children and adults because they are smaller, their bodies are still developing, and they have a greater ability to absorb lead into their bodies once it is ingested. The developing fetus is also sensitive to the health effects of lead.

Health risks from exposure to lead are evaluated using blood lead levels. Numerous scientific studies show that elevated blood lead levels in children (before or after birth) cause or are associated with adverse effects on the developing nervous system. These include reductions in several measures of cognitive ability, which are an indicator of a child's ability to learn (ATSDR 2007b). There is no evidence from these studies that a threshold (i.e., a level of exposure below which health effects do not occur) exists for lead, and therefore blood lead levels ought to be lowered to the greatest extent practical. Until recently, the U.S. Centers for Disease Control and Prevention (CDC) had established a level of concern of 10 micrograms lead per deciliter of blood (mcg/dL) to identify high-risk children in need of direct public-health interventions to reduce blood lead levels. After reviewing recent scientific research, CDC lowered the blood lead reference level to 5 mcg/dL (CDC 2014). CDC uses this reference value (which is the 97.5 percentile blood lead level in U.S. children based on the latest National Health and Nutrition Examination Survey) to identify high-risk childhood populations and geographic areas most in need of primary prevention.

Health Effects of Zinc

Zinc is one of the most common metals in the earth's crust and is found in air, soil, water and food. It is used to galvanize steel and iron and to make alloys and dry cell batteries. Zinc is an essential nutrient necessary for maintaining good health. However, exposure to too much zinc can cause adverse health effects. Ingesting large amounts of zinc can cause stomach cramps, nausea and vomiting in humans (ATSDR 2005). Longer-term human exposure to high levels of zinc can cause effects on the blood (e.g., anemia). Exposure to large amounts of zinc causes adverse effects on the blood and damages the pancreas and kidneys of laboratory animals.

Risk Characterization for Residential Surface Soil

The primary health effects associated with exposure to PCBs are cancer and noncancer effects on the immune system (EPA 1996a,b). Surface soil samples from the creek bank showed elevated levels of total PCBs (27 milligrams per kilogram soil (mg/kg) and 17.4 mg/kg) at two of the eight residential yards sampled (property designations A and B). Exposure to PCBs in soils at the creek bank of these properties via homegrown fruits and vegetables was unlikely because the bank is wooded, shady, subject to periodic flooding, and unsuitable for gardening. Therefore, for these samples, DOH evaluated the risks for soil ingestion and dermal contact only. Repeated, long-term past exposure to soil PCB levels of 27 mg/kg and 17.4 mg/kg at the creek bank locations (the highest levels found at residential properties) is estimated to pose a moderate risk for noncancer health effects, because the estimated exposures are about 6 to 10 times higher than the reference dose, and are only 25 to 39 times lower than the lowest PCB exposures that caused immune toxicity in laboratory animals (a margin of exposure that DOH considers too small to adequately protect human health). Stated another way, the estimated past exposure of residents to PCBs in surface soil at these properties approaches exposure levels that are known to cause PCB-related health effects.

Repeated and long-term (30 years) past exposure to soil PCB levels of 27 mg/kg or 17.4 mg/kg at the creek bank locations is estimated to pose a low increased risk for getting cancer (three in one hundred thousand to four in one hundred thousand).

Ten additional soil samples from these properties in areas other than the creek bank show lower total PCB levels, ranging from less than detection limits to 0.46 mg/kg. This suggests that the elevated PCB levels along the creek bank may not be representative of levels over the entire properties. The elevated health risks from PCB exposures may be associated only with creek bank soils. Health risks from exposure to PCBs at the remaining six residential properties are minimal or low.

Arsenic is a known human carcinogen (EPA 1998) and causes noncancer effects on the blood vessels and skin (ATSDR 2007a). The primary health effects associated with exposure to chromium are cancer and noncancer effects on the digestive systems,

blood and liver (ATSDR 2007a). The levels of arsenic exceed its residential SCO or ATSDR comparison value at four of the eight residential properties sampled (property designations A, B, C and G), while the levels of chromium exceed its residential SCO or ATSDR comparison value at five of the eight residential properties sampled (property designations A, B, F, G and H). The levels above the SCOs at these properties ranged from 23.1 mg/kg to 66.5 mg/kg for arsenic and 25.6 mg/kg to 164 mg/kg for chromium (assumed to be chromium [VI]). Past exposure to the highest levels of arsenic and chromium in surface soil at these properties are estimated to pose a moderate increased risk of getting cancer. The estimated increased cancer risks range from three in ten thousand to one in one thousand for arsenic and from one in ten thousand to eight in ten thousand for chromium. Past exposure to the highest level of arsenic found at Property B (66.5 mg/kg) also is estimated to poses a moderate risk for noncancer health effects because the estimated exposure is over five times higher than the reference dose. The noncancer risks for arsenic and chromium at the other properties are either minimal or low. The risk for health effects for past exposure to zinc is minimal at all the residential properties because the estimated exposures are lower than the reference dose. Actions taken by EPA in the fall of 2013 (installing a clean soil cover over contaminated soil) now prevents residents from contacting contaminated soil in the back yards. In addition, EPA is working to relocate these individuals to prevent potential future exposures.

Sample calculations and exposure parameters used to evaluate the health risks of exposure to contaminants in residential surface soil are found in Appendix C, Table 1. A summary of the evaluation of residential surface soil contamination is presented in Table D.

Property Designation	Contaminant	Highest Level in Surface Soil (mg/kg)	Cancer Risk	Hazard Quotient ¹
	arsenic	23.1	3 in 10,000	1.8
۸	chromium ²	164	8 in 10,000	4.1
~	zinc	2390		0.2
	PCBs	27	4 in 100,000	10.0
	arsenic	66.5	1 in 1,000	5.1
В	chromium ²	39.1	2 in 10,000	1.0
	PCBs	17.4	3 in 100,000	6.4
С	arsenic	26.4	4 in 10,000	2.0
F	chromium ²	25.6	1 in 10,000	0.6
0	arsenic	30.4	5 in 10,000	2.3
G	chromium ²	30.6	2 in 10,000	0.8
Н	chromium ²	114.5	6 in 10,000	2.9

Table D: Cancer and Noncancer Risk Characterization for Contaminants in Residential Surface Soil

¹The hazard quotient is the ratio of the estimated exposure to the contaminant's reference dose. ²Evaluated as chromium (VI).

Lead on Residential Properties

Scientific studies show that elevated blood lead levels in children (before or after birth) cause or are associated with adverse effects on the developing nervous system. These include reductions in several measures of cognitive ability, which are an indicator of a child's ability to learn (ATSDR 2007b). There is no evidence from these studies that a threshold (i.e., a level of exposure below which health effects do not occur) exists for lead. Lead ranged from 29.8 mg/kg to 4,630 mg/kg in surface soil at the eight residential properties, and exceeded its residential SCO (400 mg/kg) at six of them.

To estimate the contribution of environmental lead to children's blood lead levels, EPA developed the Integrated Exposure Uptake Biokinetic (IEUBK) Model. The IEUBK model estimates the percentage of children six months to seven years of age that exceed a specified blood lead level at certain soil lead concentrations. The EPA recommends that the lead concentration in soil should not result in a 5% probability of exceeding a specified blood lead concentration (EPA 1994a,b; 2007). The CDC blood lead reference level is 5 mcg/dL (CDC 2014), and is the 97.5 percentile blood lead level in US children based on the latest National Health and Nutrition Examination Survey.

DOH ran the IEUBK model using EPA's default parameters for lead in air and water, dietary intake, mother's blood lead level and soil ingestion. The following default parameters were used in the current version of the IEUBK model for lead in children:

- Lead in air: 0.1 mcg/m³
- Lead in drinking water: 4 mcg/L
- Soil/dust ingestion rate: 0.085 to 0.135 g/day
- Drinking water intake: 0.2 to 0.59 L/day
- Dietary lead intake: 1.95 to 2.26 mcg/day
- Maternal blood lead concentration at childbirth: 1 mcg/dL
- Geometric standard deviation: 1.6 mcg/dL
- Age interval: 6 to 84 months

The IEUBK model results are shown in Table E. Based on the modeled results for blood lead levels, past exposure to the lead levels in residential surface soil posed a risk for lead-related health effects for children 6 months to seven years (84 months) of age.

Table E: IEUBK Child Lead Model Results* for Residential Surface Soil.

Property Designation	Lead Concentration in Surface Soil (mg/kg)	Modeled Geometric Mean Blood Lead Level (mcg/dL)	Modeled Estimate of Percent Exceeding 5 mcg/dL
А	4,250	24.7	99.97
В	4,630	26.0	99.98
С	1,110	9.8	92.39
D	438	4.8	46.84
E	342	4.0	31.77
F	344	4.0	32.10
G	1,420	11.8	96.57
Н	3,680	22.6	99.93

*The Child Lead Model was run using the indicated soil concentration and EPA default values for the remaining parameters. The output values are for a modeled population of exposed children aged 6-84 months.

IEUBK: Integrated Exposure Uptake Biokinetic; mg/kg: milligrams per kilogram; mcg/dL: micrograms per deciliter.

Nonresidential Properties

Chemicals Selected for Further Evaluation

DOH screened the levels of surface soil contaminants at the Flintkote Plant site, the former United Paperboard property, the White Transportation property and Upson Park against their restricted residential SCOs or ATSDR comparison values. As shown in Appendix B, Table 1, carcinogenic PAHs, PCBs, antimony, arsenic, barium, cadmium, chromium, lead, mercury, nickel, and zinc exceed their restricted residential SCO or ATSDR comparison value at some or all of these properties. DOH therefore selected these contaminants for further evaluation using a site-specific assessment.

The health effects of PCBs, arsenic, lead and chromium have previously been discussed. A general discussion of the health effects of PAHs and cadmium is presented below.

Health Effects of PAHs

PAHs are a group of over 100 chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat (ATSDR 1995). They can also be found in substances, some natural, such as crude oil, coal, coal tar pitch, creosote, and tar used for roofing. There are potentially a large number of PAHs, but attention has been focused on only some of the

PAHs. Of particular concern as environmental contaminants are seven PAHs (benzo[a]pyrene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) that are known to be carcinogenic in animals.

Occupational exposure to complex mixtures containing PAHs (e.g., during coal gasification, coke production, coal-tar distillation, paving and roofing, aluminum production, and chimney sweeping) increases the risk of cancer in humans. Benzo[a]pyrene is considered a probable human carcinogen by the EPA (EPA 1994c) and a human carcinogen by other agencies (WHO 2012).

Health Effects of Cadmium

Cadmium is a silvery-white metal that occurs in nature in many different compounds. It is also found naturally in soil, food and in tobacco smoke. It has many uses in industry and consumer products, mainly in the production of batteries, pigments, plastics and metal coatings (galvanized pipes, for example).

Cadmium has been associated with an increased risk of lung cancer in industrial workers who breathed elevated levels of the metal over long periods of time in workplace air (ATSDR 2012b). There is some evidence that cadmium causes cancer in rats exposed to high levels in their drinking water over their lifetime. Some people exposed to large amounts of cadmium suffered kidney and bone damage. Exposure to high levels of cadmium damages the kidneys, blood, liver, heart and the immune and nervous systems of laboratory animals. High exposure also damages the unborn offspring of laboratory animals exposed during pregnancy.

Risk Characterization for the Flintkote, United Paperboard and White Transportation Properties

Exposure to complex mixtures of PAHs increases the risk for cancer in humans. Surface soil samples from the nonresidential properties show elevated levels of carcinogenic PAHs (up to 200 mg/kg) as well certain metal contaminants, primarily arsenic (up to 66 mg/kg) and chromium (up to 411 mg/kg). DOH estimates that the highest level of total carcinogenic PAHs in surface soil at the Flintkote property could pose a moderate risk for getting cancer (about one in ten thousand) to people who may trespass on the property on a repeated basis over several years (i.e., two days per week, six months per year for 11 years). The estimated risk for getting cancer from exposure while trespassing to the highest levels of PAHs at the United Paperboard and White Transportation properties, as well as to the highest levels of arsenic, chromium, cadmium and PCBs on the three nonresidential properties is very low or low (ranging from two in one hundred million to three in one hundred thousand). The risk for noncancer health effects at the three nonresidential properties is minimal. Sample calculations and exposure parameters used in our evaluation of the health risks of exposure to contaminants in nonresidential surface soil are found in Appendix C, Table 2. A summary of the estimated risks for health effects for contaminants in surface soil at the Flintkote, United Paperboard and White Transportation properties is found in Table F.

Table F: Cancer and Noncancer Risk Characterizationfor Contaminants in Nonresidential Surface Soil at theFlintkote, United Paperboard and White Transportation Properties

Orminant	Highest Level in		Userand Outstingth			
Contaminant Surface Soil (mg/kg) Cancer KISK ^a Hazard Quotient ^o						
	110	2 in 100 000	0.04			
benzelalaurana	110	2 III 100,000	0.04			
benzo[b]fluoronthono	20	3 III 100,000	0.01			
benzo[k]fluoranthene	160	2 III 100,000	0.06			
benzo[k]iluoranthene	200	3 IN 1,000,000	0.08			
dihanala hlanthraaana	92		0.04			
	16	2 11 100,000	0.01			
Arocior 1254	4.6	6 IN 10,000,000	0.16			
antimony	149		0.17			
arsenic	59.6	9 in 1,000,000	0.10			
barium	2440		0.01			
chromium ^c	186	9 in 1,000,000	0.10			
mercury	10.8		0.03			
nickel	549		0.01			
zinc	21,900		0.03			
Former United Paperboar	d Property					
benz[a]anthracene	26	4 in 1,000,000	0.01			
benzo[a]pyrene	20	3 in 100,000	0.01			
benzo[b]fluoranthene	26	4 in 1,000,000	0.01			
benzo[k]fluoranthene	7.3	1 in 10,000,000	0.003			
chrysene	23	3 in 10,000,000	0.01			
indeno[1,2,3-cd]pyrene	11	2 in 1,000,000	0.004			
PCBs	4.3	5 in 10,000,000	0.15			
arsenic	66	1 in 100,000	0.11			
White Transportation Property						
benz[a]anthracene	1.2	2 in 10,000,000	0.0005			
benzo[a]pyrene	1.1	2 in 1,000,000	0.0004			
benzo[b]fluoranthene	2.0	3 in 10,000,000	0.0008			
chrysene	1.2	2 in 100,000,000	0.0005			
indeno[1,2,3-cd]pyrene	0.51	7 in 100,000,000	0.0002			
PCBs	0.67	8 in 100,000,000	0.02			
arsenic	30.3	4 in 1,000,000	0.05			
cadmium	8.3	2 in 100,000,000	0.04			
chromium ^c	411	2 in 100,000	0.21			

^aThe total estimated cancer risks for carcinogenic PAHs are 1 in 10,000, 4 in 100,000 and 2 in 1,000,000 for the Flintkote, United Paperboard and White Transportation properties, respectively.

^bThe hazard quotient is the ratio of the estimated exposure to the contaminant's reference dose. ^cEvaluated as chromium (VI).

mg/kg = milligrams per kilogram of soil; PCBs = polychlorinated biphenyls.
Risk Characterization for Upson Park

Based on the available soil sampling information, the highest level of 23 mg/kg of total PCBs at Upson Park may be localized to a specific area and therefore may not be representative of potential exposures for the entire park. Assuming a residential scenario, repeated, long-term exposure to the highest level of total PCBs (23 mg/kg) is estimated to pose a moderate risk for noncancer health effects, because the estimated exposure more than 8 times higher than the reference dose, and is only 29 times lower than the lowest exposure that caused immune toxicity in laboratory animals (EPA, 1996b). DOH considers this margin of exposure too small to adequately protect human health. Stated another way, the estimated exposure of residents to the highest level of PCBs in surface soil at Upson Park approaches exposure levels that are known to cause PCB-related health effects. Repeated, long-term exposure to the highest levels of total carcinogenic PAHs, arsenic and chromium (evaluated as chromium (VI) in surface soil at Upson Park is estimated to pose a moderate risk of getting cancer (about one in ten thousand to two in ten thousand). The highest level of PCBs in surface soil at Upson Park is estimated to pose a low increased risk for getting cancer. The risk for noncancer health effects from exposure to the other contaminants in surface soil at Upson Park is low or minimal. Sample calculations and exposure parameters used in our evaluation of the health risks of exposure to contaminants at Upson Park are found in Appendix C, Table 3. A summary of the estimated risks for health effects for contaminants in surface soil at Upson Park is found in Table G.

Contaminant	Highest Level in Surface Soil (mg/kg)	Cancer Risk ^a	Hazard Quotient ^b
benz[a]anthracene	4.4	2 in 100,000	0.019
benzo[a]pyrene	2.3	8 in 100,000	0.010
benzo[b]fluoranthene	3.5	1 in 100,000	0.015
benzo[k]fluoranthene	1	4 in 10,000,000	0.004
chrysene	3.6	1 in 1,000,000	0.015
indeno[1,2,3-cd]pyrene	1.3	5 in 1,000,000	0.006
PCBs	23	3 in 100,000	8.5
arsenic	63.2	2 in 10,000	1.1
barium	2360		0.06
cadmium	27.4	8 in 10,000,000	1.2
chromium ^c	162	2 in 10,000	0.84
mercury	10.8		0.32

Table G. Cancer and Noncancer Risk Characterizationfor Contaminants in Surface Soil at Upson Park

^aThe total estimated cancer risk for carcinogenic PAHs is 1 in 10,000.

^bThe hazard quotient is the ratio of the estimated exposure to the contaminant's reference dose.

°Evaluated as chromium (VI).

mg/kg = milligrams per kilogram of soil; PCBs = polychlorinated biphenyls.

Lead at Upson Park and the Flintkote, United Paperboard Properties and White Transportation Properties

Surface soil samples at Upson Park and the Flintkote, United Paperboard and White Transportation properties contained lead levels above the restricted residential SCO of 400 mg/kg. The highest level of lead at each of these properties was 3,480 mg/kg, 7,610 mg/kg, 3,600 mg/kg, and 3,750 mg/kg, respectively. The presence of lead at these properties could result in increased exposure to lead through incidental ingestion of soil.

As with the residential properties, DOH used EPA's IEUBK model to estimate the contribution of environmental lead to children's blood lead levels. Upson Park is an active recreational property, and therefore DOH used the same model parameters for lead in air and water, dietary intake, mother's blood lead level and soil ingestion as were used for the residential properties (see previous section entitled "Lead on Residential Properties"). For the Flintkote, United Paperboard and White Transportation properties, DOH ran the model while leaving out the contribution of lead in soil and dust in the first three years of life to be more consistent with the age a child that might reasonably access the site, and setting the combined soil and dust intakes for the 3-4, 4-5, 5-6 and 6-7 year age groups to 0.027, 0.020, 0.018 and 0.017 grams per day, respectively, to reflect 20% of the generic residential exposure frequency.

The IEUBK model results for Upson Park and the Flintkote, United Paperboard and White Transportation properties are shown in Table H. Based on the modeled results for blood lead levels, exposure to the lead levels at these locations pose a risk to children for lead-related health effects.

Property	Lead Concentration in Surface Soil (mg/kg)	Modeled Geometric Mean Blood Lead Level (mcg/dL)	Modeled Estimate of Percent Exceeding 5 mcg/dL							
Upson Park	3,480	14.3	98.75							
Flintkote	7,610	6.3	68.43							
United Paperboard	3,600	3.5	22.49							
White Transportation	3,750	3.6	24.50							

Table H: IEUBK Child Lead	
odel Results* for Nonresidential Surf	ace Soil

NЛ

*For Upson Park, the Child Lead Model was run using the indicated soil concentration and EPA default values for the remaining parameters (see previous section entitled "Lead on Residential Properties." For the Flintkote, United Paperboard and White Transportation properties, DOH evaluated the 3-4, 4-5, 5-6 and 6-7 year old age groups by setting the soil/dust ingestion rates to 0.027, 0.020, 0.018 and 0.017 grams per day, respectively to reflect 20% of the generic residential exposure frequency

IEUBK: Integrated Exposure Uptake Biokinetic; mg/kg: milligrams per kilogram; mcg/dL: micrograms per deciliter.

Creek Corridor Sediments

Chemicals Selected for Further Evaluation at the Corridor Properties

Samples that were collected and analyzed to characterize the nature and extent of siterelated contamination in creek sediments where the creek runs through the residential and nonresidential properties that make up the corridor showed elevated levels of benzo[a]pyrene and PCBs. There were only two sampling results for benzo[a]pyrene (7.1 mg/kg and 34 mg/kg). For total PCBs, 30 shallow sediment (0 to 2 inches) sampling results ranged from 0.058 mg/kg to 201 mg/kg. The average of detected PCB levels was 10.9 mg/kg, with 26 of 30 results below 10 mg/kg and half below 1 mg/kg. Sixteen shallow sediment samples provided information on the levels of specific Aroclors (commercial mixtures of PCBs). Aroclor 1242 ranged from 1.2 mg/kg to 46 mg/kg, Aroclor 1254 ranged from 0.73 mg/kg to 180 mg/kg, and Aroclor 1260 ranged from 0.67 to 57 mg/kg. The average of the detected levels was 24.7 mg/kg, 22.8 mg/kg and 8.0 mg/kg for Aroclors 1242, 1254 and 1260, respectively. The levels of benzo[a]pyrene and total PCBs in the shallow creek sediments exceed the restricted residential SCO for these contaminants (1 mg/kg for each) and their ATSDR comparison values (0.096 mg/kg and 0.35 mg/kg for benzo[a]pyrene and total PCBs, respectively [ATSDR 2013b]). DOH therefore selected these contaminants for further evaluation using a site-specific assessment.

Risk Characterization for Creek Corridor Sediments

The health effects of PAHs and PCBs have previously been discussed.

People could be exposed to contaminants in shallow corridor sediments by ingestion and dermal contact during recreational use of the creek. The highest levels of benzo[a]pyrene in the creek corridor sediments are estimated to pose a moderate increased risk for getting cancer risk (about one in ten thousand), while the highest levels of total PCBs pose a low increased risk of getting cancer (about four in one hundred thousand).

Exposure to the highest level of total PCBs in creek sediments (201 mg/kg) is estimated to pose a moderate risk for noncancer health effects because the estimated exposure is 10 times higher than the reference dose and is only 25 times lower than the lowest exposure that caused immune toxicity in laboratory animals (EPA, 1996a). DOH considers this margin of exposure too small to adequately protect human health. Stated another way, the estimated exposure to PCBs in shallow sediments in the creek corridor approaches exposure levels that are known to cause PCB-related health effects. The noncancer health risk for exposure to benzo[a]pyrene in sediments is estimated to be minimal. Sample calculations and exposure parameters used in our evaluation of the health risks of exposure to contaminants in creek corridor sediments

are found in Appendix C, Table 4. A summary of the estimated risks for health effects for contaminants in creek corridor sediments is found in Table I.

The sampling data (summarized in the previous section) suggest that the benzo(a)pyrene and PCB levels in shallow sediment are variable and that high levels of these contaminants may be limited to specific areas of the creek. People may not access and have contact with sediments at these locations only. In addition, the samples with the higher levels of contaminants are generally located in areas that are inaccessible due to vegetation and steep embankments, making significant long term exposure to the contaminants unlikely.

Contaminant	Highest Level in Sediment (mg/kg)	Cancer Risk	Hazard Quotient ^a
benzo[a]pyrene	34	1 in 10,000	0.02
Aroclor 1242	180	4 in 100,000	9.0
Aroclor 1248	57	1 in 100,000	2.8
Aroclor 1254	46	9 in 1,000,000	2.3
Total PCBs	201	4 in 100,000	10.0

Table I. Cancer and Noncancer Risk Characterizationfor Contaminants in Creek Corridor Sediments

^aThe hazard quotient is the ratio of the estimated exposure to the contaminant's reference dose. mg/kg = milligrams per kilogram of soil; PCBs = polychlorinated biphenyls.

Lead in Creek Corridor Sediments

Samples that were collected and analyzed to characterize the nature and extent of lead contamination in creek sediments in inaccessible portions of the corridor contain lead ranging from 60 mg/kg to 25,400 mg/kg and averaging 1,406 mg/kg. Ten out of 32 creek sediment samples contained lead above the restricted residential SCO of 400 mg/kg. The presence of elevated lead levels could result in increased exposure of children and adults to lead through incidental ingestion if the sediments are contacted and ingested. However, according to field staff, these sediment samples were taken at locations of the creek corridor that are inaccessible due to vegetation and steep embankments, which make significant long-term exposure is unlikely.

Downstream Creek Sediments

PCB sampling results of shallow creek sediments in the downstream reaches between the corridor and the Burt Dam showed elevated levels of Aroclors (commercial mixtures of PCBs). In general, the sampling showed higher levels in deeper sediments in areas closer to the corridor, and lower levels in sediments further downstream. In sediment samples up to one foot in depth, the highest detected levels of Aroclor 1248, Aroclor 1254 and Aroclor 1260 were 41 mg/kg, 55 mg/kg, and 42 mg/kg, respectively. The levels in the downstream sediments exceed the restricted residential SCO and ATSDR comparison value for PCBs (1 mg/kg and 0.35 mg/kg, respectively), and DOH therefore

further evaluated PCB levels in several of the downstream reaches of the creek with a site specific assessment.

Risk Characterization for Downstream Creek Sediments

The highest levels of Aroclors in the downstream sediments are estimated to pose a low increased risk for cancer and noncancer health effects during recreational uses of the creek. Sample calculations and exposure parameters used in our evaluation of the health risks of exposure to contaminants in downstream creek sediments are found in Appendix C, Table 4. A summary of the estimated risks for health effects for contaminants in downstream creek sediments is found in Table J.

Highest Level in Sediments (mg/kg)	Cancer Risk	Hazard Quotient ^a								
1.9	4 in 10,000,000	0.09								
19	4 in 1,000,000	0.9								
8.3	2 in 1,000,000	0.4								
0.88	2 in 10,000,000	0.04								
25	5 in 1,000,000	1.2								
41	8 in 1,000,000	2.0								
55	1 in 100,000	2.7								
42	8 in 1,000,000	2.1								
	Highest Level in Sediments (mg/kg) 1.9 19 8.3 0.88 25 41 55 42	Highest Level in Sediments (mg/kg) Cancer Risk 1.9 4 in 10,000,000 19 4 in 1,000,000 8.3 2 in 1,000,000 0.88 2 in 10,000,000 25 5 in 1,000,000 41 8 in 1,000,000 41 8 in 1,000,000 42 8 in 1,000,000								

Table J. Cancer and Noncancer Risk Characterizationfor Contaminants in Downstream Creek Sediments

^aThe hazard quotient is the ratio of the estimated exposure to the contaminant's reference dose. mg/kg = milligrams per kilogram of soil.

Surface Water

Three surface water samples taken downstream of the Flintkote property contained the commercial PCB mixture Aroclor 1248 at levels ranging from 0.084 mcg/L to 0.33 mcg/L. These levels are below the New York State Part 5 Drinking Water Standard of 0.5 mcg/L (DOH 2011), but over the ATSDR Cancer Risk Evaluation Guide (CREG) of 0.018 mcg/L for PCBs (ATSDR, 2013a). The CREG is the PCB water concentration associated with an increased lifetime cancer risk of one in one million, and assumes a person drinks two liters of water containing this PCB concentration every day for a lifetime. The CREG is also about three times lower than the reporting limit (0.05 mcg/L) for EPA method 508. Since the CREG is exceeded, DOH further evaluated at the request of ATSDR the risk associated with incidental ingestion of surface water during recreational activities.

Long-term (30 years) incidental ingestion exposure to PCBs in surface water at 0.33 mcg/L while swimming or during other recreational uses of the river is estimated to pose

a very low risk of getting cancer (less than one in one million) and a minimal risk for noncancer health effects. Sample calculations and exposure parameters used in our evaluation of the health risks of exposure to PCBs in surface water are found in Appendix C.

Child Health Considerations

ATSDR and DOH consider children when evaluating exposure pathways and potential health effects for environmental contaminants. Children are of special concern because their behavior patterns, play activities, and physiology can result in more exposure than adults. Children sometimes differ from adults in their sensitivity to the effects of chemicals, but this depends on the chemical, and whether or not there is a difference can also change as the child gets older.

DOH considered the possibility that children may be more sensitive to the health effects of environmental contaminants when evaluating the surface soil sampling results for arsenic, chromium and the PAH benzo[a]pyrene. Hexavalent chromium and benzo[a]pyrene are identified by the EPA as chemicals that cause cancer by causing permanent changes in DNA (EPA 2005, 2006, 2009). Such contaminants are considered to pose a higher risk for cancer if exposure occurs early in life compared to the risk from exposure during adulthood (EPA, 2005). Recent studies of people and animals suggest that the very young may be more sensitive to the carcinogenic effects of arsenic than adults (Ahlborn et al. 2009; Marshall et al. 2007; Smith et al. 2006; Tokar et al. 2011; Waalkes et al. 2003, 2006, 2007, 2009). To account for the possible greater sensitivity to these contaminants, DOH followed the EPA guidance (EPA 2005, 2006) and included in our cancer risk calculations an additional factor of ten for children exposed during the first two years of life, and an additional factor of three for children exposed from ages three through age 15. In addition, for all contaminants, DOH evaluated the exposures during the child portion of life, during which more soil (relative to body weight) is ingested compared to adults.

Chemical Interactions

Most hazardous waste sites contain multiple chemical contaminants. Therefore, the possibility for interactions among the chemicals detected in and around the Eighteen Mile Creek was considered when evaluating the potential health risks. The three types of interactions that can take place among chemicals are additivity, synergy and antagonism. Additivity means that the combined effect of the chemicals of a mixture acting together is equal to the sum of the effects of the chemicals acting alone. Synergy takes place when the combined effect of the chemicals acting together is greater than the sum of the effects of the chemicals acting takes place when the combined effect of the chemicals acting together is greater than the sum of the effects of the chemicals acting alone. Antagonism takes place when the combined effect of the chemicals acting together is greater than the sum of the chemicals acting together is less than the sum of the effects of the chemicals acting alone.

The primary contaminants at and around the Eighteen Mile Creek are PAHs and PCBs, which are mixtures containing many individual chemicals. Historically, different approaches have been developed to evaluate the toxicity of different types of mixtures. Generally, one of three types of toxicological data is used to evaluate the toxicity of a mixture: data on the individual components of the mixture, data on the mixture itself, or data on similar mixtures.

Mixtures of PAHs are typically evaluated using data on the individual chemicals of the mixture. Additive interactions among the chemicals in a mixture are most likely to occur when the chemicals cause the same effect on the same body organ in the same manner (ATSDR 2004; EPA 2000). The carcinogenic PAHs are considered to cause cancer at some of the same organs by a common mode of action (EPA 1993). Therefore, in this assessment, we assumed the cancer risks for exposure to carcinogenic PAHs to be additive. Assuming additive interactions means that the cancer risk associated with exposure to mixtures of carcinogenic PAHs would be higher than the cancer risk from exposure to any individual PAH in the mixture.

Much of the available toxicological information for PCBs is based on the specific commercial mixtures (Aroclors) of many PCB congeners. Aroclors are the majority type of PCBs released into the environment, but over the years, the composition these Aroclors has been changed by natural forces. Thus, environmental mixtures of PCBs are typically evaluated by using on data on similar mixtures (that is, Aroclors). Although we do not know whether the adverse health effects from exposure to Aroclor mixtures are based on additive or other types of interactions among the PCB congeners, what we do know is the potency of Aroclors to cause specific health effects. Therefore, in our assessment of the health effects from exposure to PCBs, DOH assumed that the mixtures of PCBs in the environment (whether based on summing concentrations of individual PCB congeners or Aroclors) will have a toxic potency similar to that of a selected Aroclor.

Health Outcome Data Evaluation

The DOH evaluated historic records from 1994 to 2011 of blood lead levels in children who resided in homes on Water Street where backyard soil contained elevated level of lead. Although there was a small number of children tested, none of the tests revealed a blood lead level higher than 5 mcg/dL. Additional evaluation of health outcomes would be difficult due to the lack of exposure information and relevant health data, and the small size of the population potentially exposed.

COMMUNITY HEALTH CONCERNS

During public meetings held by DEC to discuss Proposed Remedial Action Plans for the Flintkote site, the OUs that comprise the corridor site, and other waste sites in Lockport, DOH received general expressions of concerns from people living within the Eighteen Mile Creek Corridor about the possible health concerns from children playing in the creek and adults fishing in the creek and consuming the fish. Potential health risks from contact with contaminated creek sediments and soil are discussed in this document. The fish consumption advisory issued by DOH for Eighteen Mile Creek ("Eat None") is also discussed in this document. DOH and ATSDR plan further community outreach activities in the future, to gather and address community concerns for this site.

CONCLUSIONS

Eating fish taken from Eighteen Mile Creek could harm people's health if they do not follow DOH's fish consumption advisories. Contaminated creek sediments have impacted Eighteen Mile Creek, including edible fish species. If consumed, fish in Eighteen Mile Creek contain PCBs at levels that could harm people's health. DOH has a "Don't eat <u>ANY</u> fish" fish advisory for the full length of Eighteen Mile Creek (including waters above and below Burt Dam [DOH 2014]).

DOH and ATSDR conclude that prolonged contact with Eighteen Mile Creek sediments could harm people's health (see Appendix D). The highest level of total PCBs in creek sediments is estimated to pose a moderate risk for noncancer health effects and the highest level of benzo[a]pyrene poses a moderate risk for getting cancer based on exposure to the sediments through incidental ingestion and dermal contact if the sediment are contacted during recreational activities such as wading into the creek.

There are several accessible areas where contact with sediments and fill is possible, through fishing, wading and other recreational activities. Accessible areas include the mill pond areas where the water is deeper and sediment contamination is the highest, and locations downstream of the corridor specifically developed for fishing access. Property owners along the creek corridor have attempted to secure contaminated areas from trespassers and recreational fishermen, but there is evidence of trespass. Long term significant exposure to the surface sediments with the highest levels of contamination is unlikely because the locations are inaccessible due to steep creek embankments and vegetation.

The DOH and ATSDR conclude that past contact with sediment and soil in residential backyards on Water Street (EPA OU-1) could harm people's health. Repeated, long-term past exposure to arsenic and chromium in surface soil and sediment is estimated to pose a moderate risk for cancer, and repeated long-term exposure to PCBs at the creek bank locations (where the highest levels were found at the residential properties)

is estimated to pose a moderate risk for noncancer health effects. In addition, past exposure to elevated lead levels in soil and sediment in several residential yards areas could result in increased exposure of children and adults to lead through incidental ingestion. Based on results using the EPA Integrated Exposure Uptake Biokinetic Model, this increased exposure could result in increases in blood lead levels. However, actions taken by EPA in the fall of 2013 (installing a clean soil cover over contaminated soil) followed by the relocation of residents in 2014 prevents future residents or trespassers from contacting contaminated soil in the backyards on Water Street.

The DOH and ATSDR conclude that prolonged contact with surface soil while trespassing onto the former Flintkote plant property and long-term exposure to surface soil at Upson Park could harm people's health. Parts of the Flintkote property also constitute a physical hazard. Exposure to PAHs in surface soil at the Flintkote Plant property poses a moderate increased risk of getting cancer, assuming someone trespassed onto the property for two days per week, six months per year for 11 years, and was exposed to the soil contaminants by incidental ingestion and skin contact. With fencing around the property to restrict access, the cancer risk may be lower since it is unlikely that an individual would trespass onto the property with the same frequency and duration assumed by the exposure scenario.

Based on the available sampling information, the highest level of total PCBs at Upson Park may be localized to a specific area and therefore may not be representative of potential exposures for the entire park. However, repeated, long-term exposure to the highest level of total PCBs in surface soil at Upson Park is estimated to pose a moderate risk for noncancer health effects. The estimated exposure is only 29 times lower than the lowest exposure that caused immune toxicity in laboratory animals, and DOH considers this margin of exposure too small to adequately protect human health.

Exposure to elevated lead levels in soil at the Flintkote property and at Upson Park could result in increased exposure of children and adults to lead through incidental ingestion. Based on results using the EPA Integrated Exposure Uptake Biokinetic Model, this increased exposure could result in increases in blood lead levels.

EPA will be conducting additional sampling in Upson Park to further evaluate the nature and extent of contamination. Results of this analysis will be documented in the EPA's OU2 RI/FS that includes a human health risk assessment under current and future conditions. Decisions regarding remediation will also be documented in a Proposed Plan and a ROD.

RECOMMENDATIONS

Do not eat any fish taken from Eighteen Mile Creek. Follow the DOH consumption advisory "Don't eat <u>ANY</u> fish" for the entire Eighteen Mile Creek (DOH 2014).

For those people using or living along the Eighteen Mile Creek Corridor and downstream, the DOH and ATSDR recommend measures to reduce exposure to contaminated soils and sediments. People recreating in or living around the creek can reduce the risk of exposure to chemical contaminants by avoiding the creek sediments and uncontrolled fill areas along the corridor, particularly after periods of high water flow, when new sediment may be deposited or existing sediment may be scoured. Since the greatest exposure to the contaminants is by contacting sediments or soils at the creek bank, or accessing shoreline fill areas, avoid any activity that would result in contacting these areas of contamination. In cases where people have contacted sediments, washing hands would reduce exposure, especially before eating. If people get sediments on more than just their hands and arms, it may also be helpful to take a shower to wash off the creek mud. Wash children's hands and feet after playing or digging in the soil, and also wash toys used in these play activities. If people walk the shoreline areas, removing shoes upon entering their homes would reduce the potential for tracking sediment inside.

Also, DOH and ATSDR recommend that EPA maintain access restrictions to the former Flintkote property and evaluate the extent of PCB and lead contamination of surface soil at Upson Park. Based on the evaluation at Upson Park, additional exposure reduction measures may be warranted.

PUBLIC HEALTH ACTION PLAN

Past and Current Actions

The DOH provided exposure-reduction advice in letters written to Water Street homeowners after soil in their yards were sampled by DEC in 2002. The EPA installed a clean soil cover over contaminated soil in the fall of 2013 to prevent residents from contacting contaminated soil in the back yards of Water Street. Six privately owned properties on Water Street were acquired by the federal government in 2014 and EPA has since relocated residents to comparable housing.

Actions Planned

DOH and ATSDR will work with DEC and EPA on plans for further evaluation of the nature and extent of contamination in the Eighteen Mile Creek corridor as well as possible contamination sources. DOH and ATSDR will evaluate EPA data as they

become available to us, to help determine whether additional actions are needed to reduce people's exposure to contamination in the creek.

DOH and ATSDR will work with DEC and EPA as they determine whether additional investigations, sampling, or remedial measures are needed in the corridor, and whether exposure reduction measures will be needed in the downstream portion of Eighteen Mile Creek.

DOH will work with agency partners and community stakeholders to identify opportunities to engage community members in discussions about possible health concerns related to Eighteen Mile Creek. DOH will develop and/or provide information as needed in response to any health concerns raised by the community.

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APPENDIX A Figures

Figure 1. Overview map of the Eighteen Mile Creek Corridor, Lockport, New York.





Figure 2. Overview map of the Eighteen Mile Creek from the New York State Barge Canal (Erie Canal) in Lockport to Lake Ontario



Figure 3. Operable Unit Boundaries. EPA OU1 and OU2 Eighteen Mile Creek Corridor

APPENDIX B Screening of Contaminants at Nonresidential Properties

Table 1. Contaminants Detected Above New York State RestrictedResidential Soil Cleanup Objectives and ATSDR Comparison Valuesin Nonresidential Surface Soil along the Eighteen Mile Creek.

	Highost	NYS Restricted	ATSOP									
Contaminant	Detected Level	Cleanup Objective ¹	Comparison Value ²									
Flinkote Plant Site Property												
benz[a]anthracene	110	1 ^a										
benzo[a]pyrene	20	1 ^a	0.096									
benzo[b]fluoranthene	160	1 ^a										
benzo[k]fluoranthene	200	3.9										
chrysene	92	3.9										
dibenz[a,h]anthracene	16	0.33										
Aroclor 1254	4.6	1	1									
antimony	149		20									
arsenic	59.6	16ª	0.47									
barium	2,440	400	10,000									
chromium	186	110 ^b	50									
lead	7,610	400										
mercury	10.8	0.81	15									
nickel	549	310	1,000									
zinc	21,900	10,000	15,000									
Former United Paperb	oard Company Prope	erty										
benz[a]anthracene	26	1 ^a										
benzo[a]pyrene	20	1 ^a	0.096									
benzo[b]fluoranthene	26	1 ^a										
benzo[k]fluoranthene	7.3	3.9										
chrysene	23	3.9										
indeno[1,2,3- cd]pyrene	11	0.5										
PCBs	4.3	1	0.35									
arsenic	66	16ª	0.47									
lead	3,600	400										

(all values in milligrams per kilogram soil)

White Transportation Property											
benz[a]anthracene	1.2	1 ^a									
benzo[a]pyrene	1.1	1 ^a	0.096								
benzo[b]fluoranthene	2	1 ^a									
indeno[1,2,3- cd]pyrene	0.51	0.5ª									
arsenic	30.3	16 ^a	0.47								
cadmium	8.3	4.3	5								
chromium	411	110 ^b	50								
lead	3,750	400									
Upson Park											
benz[a]anthracene	4.4	1 ^a									
benzo[a]pyrene	2.3	1 ^a	0.096								
benzo[b]fluoranthene	3.5	1 ^a									
indeno(1,2,3- cd)pyrene	1.3	0.5									
PCBs	23	1	0.35								
arsenic	63.2	16 ^a	0.47								
barium	2,360	400	10,000								
cadmium	27.4	4.3	5								
chromium	162	110 ^b	50								
lead	3,480	400									
mercury	10.8	0.81	15								

¹DEC/DOH, 2006

²ATSDR, 2013b

^aBased on New York State rural soil background concentration. ^bBased on chromium (VI).

APPENDIX C Exposure Parameters and Sample Dose and Risk Calculations

Table 1. Calculation of Contaminant Oral and Dermal Doses from a Soil Concentration of 1 mg/kgs for Evaluation of Residential Soil Contaminant Exposure

P	СВ	Dose	from	Soil	Ingestion	

									365 d/y	E-weighted
		С	IR	CF	BW			E	Ing Dose	Ing Dose
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)
1	0 to <1	1	45	1.E-06	7.8	0.714	0.596	0.426	5.77E-06	2.46E-06
2	1 to <2	1	120	1.E-06	11.4	0.714	0.596	0.426	1.05E-05	4.48E-06
3	2 to <3	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06
4	3 to <4	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06
5	4 to <5	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06
6	5 to <6	1	100	1.E-06	18.6	0.714	0.596	0.426	5.38E-06	2.29E-06
7	6 to <7	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06
8	7 to <8	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06
9	8 to <9	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06
10	9 to <10	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06
11	10 to <11	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06
12	11 to <12	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07
13	12 to <13	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07
14	13 to <14	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07
15	14 to <15	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07
16	15 to < 16	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07
17	16 to <17	1	100	1.E-06	71.6	0.714	0.596	0.426	1.40E-06	5.95E-07

		с	IR	CF	вw			E	365 d/y Ing Dose	<i>E</i> -weighted Ing Dose
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)
18	17 to <18	1	100	1.E-06	71.6	0.286	0.596	0.170	1.40E-06	2.38E-07
19	18 to <19	1	100	1.E-06	71.6	0.286	0.596	0.170	1.40E-06	2.38E-07
20	19 to <20	1	100	1.E-06	71.6	0.286	0.596	0.170	1.40E-06	2.38E-07
21	20 to <21	1	100	1.E-06	71.6	0.286	0.596	0.170	1.40E-06	2.38E-07
22	21 to <22	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
23	22 to <23	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
24	23 to <24	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
25	24 to <25	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
26	25 to <26	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
27	26 to <27	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
28	27 to <28	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
29	28 to <29	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07
30	29 to <30	1	100	1.E-06	80.0	0.286	0.596	0.170	1.25E-06	2.13E-07

Avg PCB Dose 2.21E-07 mg/kg/d

Age Period	ED/Lifetime	Avg PCB Dose	Dose Weight (mg/kg/day over 70 years)
0 to 17	0.2429	1.68E-06	4.07E-07
18 to 30	0.1857	2.21E-07	4.10E-08
30 years			4.48E-07

Avg PCB Dose 1.68E-06 mg/kg/d

PCB Dermal Dose from Soil

DI	RMAL E	XPOS	URE										
											365 d/y	E-weighted	
		с	SA	AF		CF	BW	EF1	EF2	Ε	Derm Dose	Derm Dose	
Yr	Range	(ppm)	(cm2)	(mg/cm2-d)	DABS	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	
1	0 to <1	1	1,900	0.20	0.140	1.0E-06	7.8	0.714	0.596	0.426	6.821E-06	2.904E-06	
2	1 to <2	1	2,800	0.20	0.140	1.0E-06	11.4	0.714	0.596	0.426	6.877E-06	2.928E-06	
3	2 to <3	1	2,800	0.20	0.140	1.0E-06	18.6	0.714	0.596	0.426	4.215E-06	1.795E-06	
4	3 to <4	1	2,800	0.20	0.140	1.0E-06	18.6	0.714	0.596	0.426	4.215E-06	1.795E-06	
5	4 to <5	1	2,800	0.20	0.140	1.0E-06	18.6	0.714	0.596	0.426	4.215E-06	1.795E-06	
6	5 to <6	1	2,800	0.20	0.140	1.0E-06	18.6	0.714	0.596	0.426	4.215E-06	1.795E-06	
7	6 to <7	1	5,700	0.07	0.140	1.0E-06	31.8	0.714	0.596	0.426	1.757E-06	7.480E-07	
8	7 to <8	1	5,700	0.07	0.140	1.0E-06	31.8	0.714	0.596	0.426	1.757E-06	7.480E-07	
9	8 to <9	1	5,700	0.07	0.140	1.0E-06	31.8	0.714	0.596	0.426	1.757E-06	7.480E-07	
10	9 to <10	1	5,700	0.07	0.140	1.0E-06	31.8	0.714	0.596	0.426	1.757E-06	7.480E-07	
11	10 to <11	1	5,700	0.07	0.140	1.0E-06	31.8	0.714	0.596	0.426	1.757E-06	7.480E-07	
12	11 to <12	1	5,700	0.07	0.140	1.0E-06	56.8	0.714	0.596	0.426	9.835E-07	4.188E-07	
13	12 to <13	1	5,700	0.07	0.140	1.0E-06	56.8	0.714	0.596	0.426	9.835E-07	4.188E-07	
14	13 to <14	1	5,700	0.07	0.140	1.0E-06	56.8	0.714	0.596	0.426	9.835E-07	4.188E-07	
15	14 to <15	1	5,700	0.07	0.140	1.0E-06	56.8	0.714	0.596	0.426	9.835E-07	4.188E-07	
16	15 to < 16	1	5,700	0.07	0.140	1.0E-06	56.8	0.714	0.596	0.426	9.835E-07	4.188E-07	
17	16 to <17	1	5,700	0.07	0.140	1.0E-06	71.6	0.714	0.596	0.426	7.802E-07	3.322E-07	
											_		<i></i>
-											Average	1.13E-06	mg/kg/day
											365 d/y	E-weighted	
		С	SA	AF		CF	BW	EF1	EF2	Ε	Derm Dose	Derm Dose	
Yr	Range	(ppm)	(cm2)	(mg/cm2-d)	DABS	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	
18	17 to <18	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.596	0.170	7.802E-07	1.329E-07	
19	18 to <19	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.596	0.170	7.802E-07	1.329E-07	
20	19 to <20	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.596	0.170	7.802E-07	1.329E-07	
21	20 to <21	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.596	0.170	7.802E-07	1.329E-07	
22	21 to <22	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
23	22 to <23	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
24	23 to <24	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
25	24 to <25	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
26	25 to <26	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
27	26 to <27	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
	27 to <28	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
28	27 10 20												
28 29	28 to <29	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	1.189E-07	
28 29 30	28 to <29 29 to <30	1 1	5,700 5,700	0.07 0.07	0.140 0.140	1.0E-06 1.0E-06	80.0 80.0	0.286 0.286	0.596 0.596	0.170 0.170	6.983E-07 6.983E-07	1.189E-07 1.189E-07	

Average 1.23

1.232E-07 mg/kg/day

Dose Weight			
(mg/kg/day over 70 years)	Avg Dose	ED/Lifetime	ge Period
2.74E-07	1.13E-06	0.2429	Young
2.29E-08	1.23E-07	0.1857	Old
2.97E-07			years

Sample PCB Cancer Risk Calculation for Residential Soil (using results from above residential spreadsheets)

Total Dose from 1 mg/kgs

Total Dose = Oral Dose + Dermal Dose = 4.48E-7 mg/kg/day + 2.97E-7 mg/kg/day = 7.45E-7 mg/kg/day*

Total Dose from 27 mg/kgs PCBs

Total Dose = (7.45E-7 mg/kg/day x 27 mg/kg_s)/ 1 mg/kg_s = 2.01E-5 mg/kg/day

Cancer Risk from 27 mg/kgs PCBs

Cancer Risk = Total Dose x Cancer Potency Factor = 2.01E-5 mg/kg/day x 2.0/mg/kg/day = 4E-5 (low)

Sample PCB Noncancer Hazard Quotient Calculation for Residential Soil

 $Oral \ Dose = 27 \ mg/kg_{s} \ x \ 120 \ mg_{s}/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_{s}/mg_{s} \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.21E-4 \ mg/kg/day^{*}$

Dermal dose = [27 mg/kg_s x 2800 cm² x 0.2 mg_s/cm²-d x 0.14 x 1E-6 kg_s/mg_s x 5 d/7 d x 31 wks/52 wks]/11.4 kg = 7.91E-5 mg/kg/d

Total Dose = 1.21E-4 mg/kg/day + 7.91E-5 mg/kg/day = 2.00E-4 mg/kg/day

Hazard Quotient = Total Dose/Reference Dose = 2.00E-4 mg/kg/day / 2E-5 mg/kg/day = 10 (moderate)

*A factor of 5 is applied to the oral dose to account for exposure via homegrown fruits and vegetables (DEC/DOH, 2006). This factor was not used in the calculation for PCBs because the creek bank location of the samples is an unlikely site for a garden.

Table 2. Calculation of Contaminant Oral and Dermal Doses from a Soil Concentration of1 mg/kg for Evaluation of Nonresidential Soil Contaminant Exposure

									365 d/y	E-weighted	Age-Dependent	Adjusted Time-
		С	IR	CF	BW			Ε	Ing Dose	Ing Dose	Adjustment	Weighted Dose**
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	Factors*	(mg/kg/d)
1	10 to <11	1	100	1.E-06	31.8	0.286	0.500	0.143	3.14E-06	4.49E-07	3	1.35E-06
2	11 to <12	1	100	1.E-06	56.8	0.286	0.500	0.143	1.76E-06	2.52E-07	3	7.55E-07
3	12 to <13	1	100	1.E-06	56.8	0.286	0.500	0.143	1.76E-06	2.52E-07	3	7.55E-07
4	13 to <14	1	100	1.E-06	56.8	0.286	0.500	0.143	1.76E-06	2.52E-07	3	7.55E-07
5	14 to <15	1	100	1.E-06	56.8	0.286	0.500	0.143	1.76E-06	2.52E-07	3	7.55E-07
6	15 to < 16	1	100	1.E-06	56.8	0.286	0.500	0.143	1.76E-06	2.52E-07	3	7.55E-07
7	16 to <17	1	100	1.E-06	71.6	0.286	0.500	0.143	1.40E-06	2.00E-07	1	2.00E-07
8	17 to <18	1	100	1.E-06	71.6	0.286	0.500	0.143	1.40E-06	2.00E-07	1	2.00E-07
9	18 to <19	1	100	1.E-06	71.6	0.286	0.500	0.143	1.40E-06	2.00E-07	1	2.00E-07
10	19 to <20	1	100	1.E-06	71.6	0.286	0.500	0.143	1.40E-06	2.00E-07	1	2.00E-07
11	20 to <21	1	100	1.E-06	71.6	0.286	0.500	0.143	1.40E-06	2.00E-07	1	2.00E-07
											AVERAGE =	5.56E-07

Benzo[a]pyrene Dose from Soil Ingestion

Age Period	ED/Lifetime	Average Dose	Adjusted Dose (mg/kg/day over 70 years)*
11 years (10 -			
21 years of			
age)	0.1571	5.56E-07	8.74E-08

Benzo[a]pyrene Dermal Dose from Soil

		с	SA	AF		CF	BW	EF1	EF2	E	365 d/y Derm Dose	<i>E</i> -weighted Derm Dose	Age-Dependent Adjustment	Adjusted Time- Weighted Dose
Yr	Range	(ppm)	(cm2)	(mg/cm2-d)	DABS	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	Factor	(mg/kg/d)
11	10 to <11	1	5,700	0.07	0.130	1.0E-06	31.8	0.286	0.500	0.143	1.631E-06	2.330E-07	3	6.99E-07
12	11 to <12	1	5,700	0.07	0.130	1.0E-06	56.8	0.286	0.500	0.143	9.132E-07	1.305E-07	3	3.91E-07
13	12 to <13	1	5,700	0.07	0.130	1.0E-06	56.8	0.286	0.500	0.143	9.132E-07	1.305E-07	3	3.91E-07
14	13 to <14	1	5,700	0.07	0.130	1.0E-06	56.8	0.286	0.500	0.143	9.132E-07	1.305E-07	3	3.91E-07
15	14 to <15	1	5,700	0.07	0.130	1.0E-06	56.8	0.286	0.500	0.143	9.132E-07	1.305E-07	3	3.91E-07
16	15 to < 16	1	5,700	0.07	0.130	1.0E-06	56.8	0.286	0.500	0.143	9.132E-07	1.305E-07	3	3.91E-07
17	16 to <17	1	5,700	0.07	0.130	1.0E-06	71.6	0.286	0.500	0.143	7.244E-07	1.035E-07	1	1.03E-07
18	17 to <18	1	5,700	0.07	0.130	1.0E-06	71.6	0.286	0.500	0.143	7.244E-07	1.035E-07	1	1.03E-07
19	18 to <19	1	5,700	0.07	0.130	1.0E-06	71.6	0.286	0.500	0.143	7.244E-07	1.035E-07	1	1.03E-07
20	19 to <20	1	5,700	0.07	0.130	1.0E-06	71.6	0.286	0.500	0.143	7.244E-07	1.035E-07	1	1.03E-07
21	20 to <21	1	5,700	0.07	0.130	1.0E-06	71.6	0.286	0.500	0.143	7.244E-07	1.035E-07	1	1.03E-07
													AVERAGE =	2.88E-07

Age Period	ED/Lifetime	Average Dose	Adjusted Dose (mg/kg/day over 70 years)*
11 years (10 - 21 years of			
age)	0.1571	2.88E-07	4.53E-08
<u>Sample Benzo[a]pyrene Cancer Risk Calculation for Nonresidential Soil</u> (using results from above nonresidential spreadsheets)

Total Dose from 1 mg/kgs

Total Dose = Oral Dose + Dermal Dose = 8.74E-8 mg/kg/day + 4.53E-8 mg/kg/day = 1.33E-7 mg/kg/day

Total Dose from 20 mg/kgs Benzo[a]pyrene

Total Dose = (1.33E-7 mg/kg/day x 20 mg/kg)/ 1 mg/kg = 2.65E-6 mg/kg/day

Cancer Risk from 20 mg/kgs Benzo[a]pyrene

Cancer Risk = Total Dose x Cancer Potency Factor = 2.65E-6 mg/kg/day x 11/mg/kg/day = 3E-5 (low)

Sample Benzo[a]pyrene Noncancer Hazard Quotient Calculation for Nonresidential Soil

Dermal dose = [20 mg/kg_s x 5700 cm² x 0.07 mg_s/cm²-d x 0.13 x 1E-6 kg_s/mg_s x 2 d/7 d x 26 wks/52 wks]/31.8 kg = 4.66E-6 mg/kg/d

Total Dose = 8.98E-6 mg/kg/day + 4.66E-6 mg/kg/day = 1.36E-5 mg/kg/day

Hazard Quotient = Total Dose/Reference Dose = 1.36E-5 mg/kg/day / 1.7E-3 mg/kg/day = 0.008 (rounded to 0.01, minimal)

Table 3. Calculation of Contaminant Oral and Dermal Doses from a Soil Concentration of 1 mg/kg for Evaluation of Soil Contaminant Exposure at Upson Park

									365 d/y	E-weighted		E-weighted
		С	IR	CF	BW			Ε	Ing Dose	Ing Dose		Ing Dose X ADAF
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	ADAF	(mg/kg/d)
1	0 to <1	1	45	1.E-06	7.8	0.714	0.596	0.426	5.77E-06	2.46E-06	10	2.46E-05
2	1 to <2	1	120	1.E-06	11.4	0.714	0.596	0.426	1.05E-05	4.48E-06	10	4.48E-05
3	2 to <3	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06	3	8.24E-06
4	3 to <4	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06	3	8.24E-06
5	4 to <5	1	120	1.E-06	18.6	0.714	0.596	0.426	6.45E-06	2.75E-06	3	8.24E-06
6	5 to <6	1	100	1.E-06	18.6	0.714	0.596	0.426	5.38E-06	2.29E-06	3	6.87E-06
7	6 to <7	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06	3	4.02E-06
8	7 to <8	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06	3	4.02E-06
9	8 to <9	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06	3	4.02E-06
10	9 to <10	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06	3	4.02E-06
11	10 to <11	1	100	1.E-06	31.8	0.714	0.596	0.426	3.14E-06	1.34E-06	3	4.02E-06
12	11 to <12	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07	3	2.25E-06
13	12 to <13	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07	3	2.25E-06
14	13 to <14	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07	3	2.25E-06
15	14 to <15	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07	3	2.25E-06
16	15 to < 16	1	100	1.E-06	56.8	0.714	0.596	0.426	1.76E-06	7.50E-07	3	2.25E-06
17	16 to <17	1	100	1.E-06	71.6	0.714	0.596	0.426	1.40E-06	5.95E-07	1	5.95E-07

Chromium Dose from Soil Ingestion

					-		_	365 d/y	E-weighted		E-weighted	
		C	ік	CF	BW		E	Ing Dose	Ing Dose		Ing Dose X ADAF	
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk wk/	y (d/d)	(mg/kg/d)	(mg/kg/d)	ADAF	(mg/kg/d)	
18	17 to <18	1	100	1.E-06	71.6	0.286 0.59	6 0.170	1.40E-06	2.38E-07	1	2.38E-07	
19	18 to <19	1	100	1.E-06	71.6	0.286 0.59	6 0.170	1.40E-06	2.38E-07	1	2.38E-07	
20	19 to <20	1	100	1.E-06	71.6	0.286 0.59	6 0.170	1.40E-06	2.38E-07	1	2.38E-07	
21	20 to <21	1	100	1.E-06	71.6	0.286 0.59	6 0.170	1.40E-06	2.38E-07	1	2.38E-07	
22	21 to <22	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
23	22 to <23	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
24	23 to <24	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
25	24 to <25	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
26	25 to <26	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
27	26 to <27	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
28	27 to <28	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
29	28 to <29	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	
30	29 to <30	1	100	1.E-06	80.0	0.286 0.59	6 0.170	1.25E-06	2.13E-07	1	2.13E-07	

		Average	2.21E-07	mg/kg/day
			Dose Weight (mg/kg/day	
Age Period	ED/Lifetime	avg Dose	over 70 years)	
Young	0.2429	7.82E-06	1.90E-06	
Old	0.1857	2.21E-07	4.10E-08	
30 years			1.94E-06	

Average 7.82E-06 mg/kg/day

Chromium Dermal Dose from Soil

										365 d/y	E-weighted		E-weighted
	с	SA	AF		CF	BW	EF1	EF2	Е	Derm Dose	Derm Dose		Derm Dose X ADAF
Yr Range	(ppm)	(cm2)	mg/cm2-d	DABS	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	ADAF	(mg/kg/d)
1 0 to <1	1	1,900	0.20	0.010	1.0E-06	7.8	0.714	0.596	0.426	4.872E-07	2.075E-07	10	2.075E-06
2 1 to <2	1	2,800	0.20	0.010	1.0E-06	11.4	0.714	0.596	0.426	4.912E-07	2.092E-07	10	2.092E-06
3 2 to <3	1	2,800	0.20	0.010	1.0E-06	18.6	0.714	0.596	0.426	3.011E-07	1.282E-07	3	3.846E-07
4 3 to <4	1	2,800	0.20	0.010	1.0E-06	18.6	0.714	0.596	0.426	3.011E-07	1.282E-07	3	3.846E-07
5 4 to <5	1	2,800	0.20	0.010	1.0E-06	18.6	0.714	0.596	0.426	3.011E-07	1.282E-07	3	3.846E-07
6 5 to <6	1	2,800	0.20	0.010	1.0E-06	18.6	0.714	0.596	0.426	3.011E-07	1.282E-07	3	3.846E-07
7 6 to <7	1	5,700	0.07	0.010	1.0E-06	31.8	0.714	0.596	0.426	1.255E-07	5.343E-08	3	1.603E-07
8 7 to <8	1	5,700	0.07	0.010	1.0E-06	31.8	0.714	0.596	0.426	1.255E-07	5.343E-08	3	1.603E-07
9 8 to <9	1	5,700	0.07	0.010	1.0E-06	31.8	0.714	0.596	0.426	1.255E-07	5.343E-08	3	1.603E-07
10 9 to <10	1	5,700	0.07	0.010	1.0E-06	31.8	0.714	0.596	0.426	1.255E-07	5.343E-08	3	1.603E-07
11 10 to <11	1	5,700	0.07	0.010	1.0E-06	31.8	0.714	0.596	0.426	1.255E-07	5.343E-08	3	1.603E-07
12 11 to <12	1	5,700	0.07	0.010	1.0E-06	56.8	0.714	0.596	0.426	7.025E-08	2.991E-08	3	8.974E-08
13 12 to <13	1	5,700	0.07	0.010	1.0E-06	56.8	0.714	0.596	0.426	7.025E-08	2.991E-08	3	8.974E-08
14 13 to <14	1	5,700	0.07	0.010	1.0E-06	56.8	0.714	0.596	0.426	7.025E-08	2.991E-08	3	8.974E-08
15 14 to <15	1	5,700	0.07	0.010	1.0E-06	56.8	0.714	0.596	0.426	7.025E-08	2.991E-08	3	8.974E-08
16 15 to < 16	1	5,700	0.07	0.010	1.0E-06	56.8	0.714	0.596	0.426	7.025E-08	2.991E-08	3	8.974E-08
17 16 to <17	1	5,700	0.07	0.010	1.0E-06	71.6	0.714	0.596	0.426	5.573E-08	2.373E-08	1	2.373E-08
1 1													
												Average	4.11E-07
												Average	4.11E-07
										365 d/y	<i>E</i> -weighted	Average	4.11E-07 <i>E</i> -weighted
	с	SA	AF		CF	BW	EF1	EF2	Ε	365 d/y Derm Dose	<i>E</i> -weighted Derm Dose	Average	4.11E-07 <i>E</i> -weighted Derm Dose X ADAF
Yr Range	C (ppm)	<i>SA</i> (cm2)	AF mg/cm2-d	DABS	CF (kg/mg)	BW (kg)	<i>EF1</i> d/wk	EF2 wk/y	<i>E</i> (d/d)	365 d/y Derm Dose (mg/kg/d)	<i>E</i> -weighted Derm Dose (mg/kg/d)	Average ADAF	4.11E-07 <i>E</i> -weighted Derm Dose X ADAF (mg/kg/d)
Yr Range 18 17 to <18	С (ррт) 1	SA (cm2) 5,700	AF mg/cm2-d 0.07	DABS 0.010	CF (kg/mg) 1.0E-06	<i>BW</i> (kg) 71.6	EF1 d/wk 0.286	EF2 wk/y 0.596	<i>E</i> (d/d) 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08	<i>E</i> -weighted Derm Dose (mg/kg/d) 9.492E-09	Average ADAF 1	4.11E-07 E -weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09
Yr Range 18 17 to <18	С (ррт) 1 1	SA (cm2) 5,700 5,700	<i>AF</i> mg/cm2-d 0.07 0.07	DABS 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6	<i>EF1</i> d/wk 0.286 0.286	EF2 wk/y 0.596 0.596	<i>E</i> (d/d) 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08	<i>E</i> -weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09	Average ADAF 1 1	4.11E-07 <i>E</i> -weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09
Yr Range 18 17 to <18	С (ppm) 1 1 1	<i>SA</i> (cm2) 5,700 5,700 5,700 5,700	<i>AF</i> mg/cm2-d 0.07 0.07 0.07	DABS 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6 71.6	EF1 d/wk 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09	Average ADAF 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09
Yr Range 18 17 to <18	С (ppm) 1 1 1 1	5 4 (cm2) 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07	DABS 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6 71.6 71.6 71.6	<i>EF1</i> d/wk 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 9.492E-09	Average ADAF 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1	<i>SA</i> (cm2) 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07	DABS 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6 71.6 71.6 80.0	<i>EF1</i> d/wk 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 9.492E-09 8.495E-09	Average ADAF 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1	<i>SA</i> (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07	DABS 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	BW (kg) 71.6 71.6 71.6 71.6 80.0 80.0	<i>EF1</i> d/wk 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596	<i>E</i> (d/d) 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1	4.11E-07 <i>E</i> -weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1 1 1	SA (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700	<i>AF</i> mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	BW (kg) 71.6 71.6 71.6 71.6 80.0 80.0 80.0	<i>EF1</i> <i>d/wk</i> 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1 1 1	5A (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700	<i>AF</i> mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07 0.0	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6 71.6 80.0 80.0 80.0 80.0	EF1 d/wk 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1 1 1 1	5A (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07 0.0	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	<i>BW</i> (kg) 71.6 71.6 71.6 80.0 80.0 80.0 80.0 80.0	EF1 d/wk 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596	<i>E</i> (<i>d/d</i>) 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09	Average <u>ADAF</u> 1 1 1 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1 1 1 1 1 1	SA (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07 0.0	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	BW (kg) 71.6 71.6 71.6 71.6 80.0 80.0 80.0 80.0 80.0 80.0	EF1 d/wk 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	С (ррт) 1 1 1 1 1 1 1 1 1 1 1 1 1	5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07 0.0	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	BW (kg) 71.6 71.6 71.6 71.6 80.0 80.0 80.0 80.0 80.0 80.0 80.0	EF1 d/wk 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09
Yr Range 18 17 to <18	C (ppm) 1 1 1 1 1 1 1 1 1 1 1 1 1 1	<i>SA</i> (cm2) 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700 5,700	AF mg/cm2-d 0.07 0.07 0.07 0.07 0.07 0.07 0.07 0.0	DABS 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010 0.010	CF (kg/mg) 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06 1.0E-06	BW (kg) 71.6 71.6 71.6 71.6 80.0 80.0 80.0 80.0 80.0 80.0 80.0 80	EF1 d/wk 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286 0.286	EF2 wk/y 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596 0.596	E (d/d) 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170 0.170	365 d/y Derm Dose (mg/kg/d) 5.573E-08 5.573E-08 5.573E-08 5.573E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08 4.988E-08	E-weighted Derm Dose (mg/kg/d) 9.492E-09 9.492E-09 9.492E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09 8.495E-09	Average ADAF 1 1 1 1 1 1 1 1 1 1 1 1 1	4.11E-07 E-weighted Derm Dose X ADAF (mg/kg/d) 9.49E-09 9.49E-09 9.49E-09 9.49E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09 8.50E-09

Average 8

8.80E-09 mg/kg/day

Age Period	ED/Lifetime	Average Dose (mg/kg/day)	Dose Weight (mg/kg/day over 70 years)
Young	0.2429	4.11E-07	9.97E-08
Old	0.1857	8.80E-09	1.63E-09
30 years			1.01E-07

<u>Sample Chromium Cancer Risk Calculation for Upson Park Soil</u> (using results from above spreadsheets)

Total Dose from 1 mg/kgs

Total Dose = Oral Dose + Dermal Dose = 1.94E-6 mg/kg/day + 1.01E-7 mg/kg/day = 2.04E-6 mg/kg/day

Total Dose from 162 mg/kgs Chromium

Total Dose = (2.04E-6 mg/kg/day x 162 mg/kg)/ 1 mg/kg = 3.30E-4 mg/kg/day

Cancer Risk from 162 mg/kgs Chromium

Cancer Risk = Total Dose x Cancer Potency Factor = 3.30E-4 mg/kg/day x 0.5/mg/kg/day = 1.6E-4 (moderate)

Sample PCB Noncancer Hazard Quotient Calculation for Upson Park Soil

 $Oral \ Dose = 23 \ mg/kg_s \ x \ 120 \ mg_s/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1E-6 \ kg_s/mg_s \ x \ 5 \ d/7 \ d \ x \ 31 \ wks/52 \ wks = 1.03E-4 \ mg/kg/day \ x \ 1/11.4 \ kg \ x \ 1/11.4 \ x \ 1/11.4 \ kg \ x \ 1/11.4 \ x \ 1/11.4 \ x \ 1/11.4 \ kg \ x \ 1/11.4 \ kg \ x \ 1/11.4 \ x \ 1/$

Dermal dose = [23 mg/kg_s x 2800 cm² x 0.2 mg_s/cm²-d x 0.14 x 1E-6 kg_s/mg_s x 5 d/7 d x 31 wks/52 wks]/11.4 kg = 6.74E-5 mg/kg/d

Total Dose = 1.03E-4 mg/kg/day + 6.74E-5 mg/kg/day = 1.70E-4 mg/kg/day

Hazard Quotient = Total Dose/Reference Dose = 1.70E-4 mg/kg/day / 2E-5 mg/kg/day = 8.52 (moderate)

Table 4. Calculation of Contaminant Oral and Dermal Doses from a Sediment Concentration of 1 mg/kg for Evaluation of Creek Sediment Contaminant Exposure

PCB Do	se from S	ediment	Ingestic	on						
									365 d/y	E-weighted
		С	IR	CF	BW			E	Ing Dose	Ing Dose
Yr	Range	(mg/kg)	(mg/d)	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	_ (mg/kg/d)	(mg/kg/d)
1	3 to <4	1	120	1.E-06	18.6	0.286	0.327	0.093	6.45E-06	6.03E-07
2	4 to <5	1	120	1.E-06	18.6	0.286	0.327	0.093	6.45E-06	6.03E-07
3	5 to <6	1	100	1.E-06	18.6	0.286	0.327	0.093	5.38E-06	5.02E-07
4	6 to <7	1	100	1.E-06	31.8	0.286	0.327	0.093	3.14E-06	2.94E-07
5	7 to <8	1	100	1.E-06	31.8	0.286	0.327	0.093	3.14E-06	2.94E-07
6	8 to <9	1	100	1.E-06	31.8	0.286	0.327	0.093	3.14E-06	2.94E-07
7	9 to <10	1	100	1.E-06	31.8	0.286	0.327	0.093	3.14E-06	2.94E-07
8	10 to <11	1	100	1.E-06	31.8	0.286	0.327	0.093	3.14E-06	2.94E-07
9	11 to <12	1	100	1.E-06	56.8	0.286	0.327	0.093	1.76E-06	1.64E-07
10	12 to <13	1	100	1.E-06	56.8	0.286	0.327	0.093	1.76E-06	1.64E-07
11	13 to <14	1	100	1.E-06	56.8	0.286	0.327	0.093	1.76E-06	1.64E-07
12	14 to <15	1	100	1.E-06	56.8	0.286	0.327	0.093	1.76E-06	1.64E-07
13	15 to < 16	1	100	1.E-06	56.8	0.286	0.327	0.093	1.76E-06	1.64E-07
14	16 to <17	1	100	1.E-06	71.6	0.286	0.327	0.093	1.40E-06	1.30E-07
15	17 to <18	1	100	1.E-06	71.6	0.286	0.327	0.093	1.40E-06	1.30E-07
									Average	2.84E-07

			Dose Weight
		Average Dose	(mg/kg/day over
Age Period	ED/Lifetime	(mg/kg/day)	70 years)
15 years	0.2143	2.84E-07	6.08E-08

Ρ	CB Der	mal Dos	e from S	edimen	t									
												365 d/y	E-weighted	
			С	SA	AF		CF	BW	EF1	EF2	Ε	Derm Dose	Derm Dose	
	Yr	Range	(ppm)	(cm2)	(mg/cm2-d)	DABS	(kg/mg)	(kg)	d/wk	wk/y	(d/d)	(mg/kg/d)	(mg/kg/d)	
	1	3 to <4	1	2,800	0.20	0.140	1.0E-06	18.6	0.286	0.327	0.093	4.215E-06	3.937E-07	
	2	4 to <5	1	2,800	0.20	0.140	1.0E-06	18.6	0.286	0.327	0.093	4.215E-06	3.937E-07	
	3	5 to <6	1	2,800	0.20	0.140	1.0E-06	18.6	0.286	0.327	0.093	4.215E-06	3.937E-07	
	4	6 to <7	1	5,700	0.07	0.140	1.0E-06	31.8	0.286	0.327	0.093	1.757E-06	1.641E-07	
	5	7 to <8	1	5,700	0.07	0.140	1.0E-06	31.8	0.286	0.327	0.093	1.757E-06	1.641E-07	
	6	8 to <9	1	5,700	0.07	0.140	1.0E-06	31.8	0.286	0.327	0.093	1.757E-06	1.641E-07	
	7	9 to <10	1	5,700	0.07	0.140	1.0E-06	31.8	0.286	0.327	0.093	1.757E-06	1.641E-07	
	8	10 to <11	1	5,700	0.07	0.140	1.0E-06	31.8	0.286	0.327	0.093	1.757E-06	1.641E-07	
	9	11 to <12	1	5,700	0.07	0.140	1.0E-06	56.8	0.286	0.327	0.093	9.835E-07	9.186E-08	
	10	12 to <13	1	5,700	0.07	0.140	1.0E-06	56.8	0.286	0.327	0.093	9.835E-07	9.186E-08	
	11	13 to <14	1	5,700	0.07	0.140	1.0E-06	56.8	0.286	0.327	0.093	9.835E-07	9.186E-08	
	12	14 to <15	1	5,700	0.07	0.140	1.0E-06	56.8	0.286	0.327	0.093	9.835E-07	9.186E-08	
	13	15 to < 16	1	5,700	0.07	0.140	1.0E-06	56.8	0.286	0.327	0.093	9.835E-07	9.186E-08	
	14	16 to <17	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.327	0.093	7.802E-07	7.287E-08	
	15	17 to <18	1	5,700	0.07	0.140	1.0E-06	71.6	0.286	0.327	0.093	7.802E-07	7.287E-08	
												Average	1.74E-07	mg/kg/day
														_
												Dose	Dose Weight	
												(mg/kg/day	(mg/kg/day	
										Age Period	ED/Lifetime)	over 70 years)	
										15 Years	0.2143	1.74E-07	3.72E-08	

Sample PCB Cancer Risk Calculation for Sediments (using results from above spreadsheets)

Total Dose from 1 mg/kgs

Total Dose = Oral Dose + Dermal Dose = 6.08E-8 mg/kg/day + 3.72E-8 mg/kg/day = 9.80E-8 mg/kg/day

Total Dose from 201 mg/kg_s PCBs

Total Dose = (9.80E-8 mg/kg/day x 201 mg/kg)/ 1 mg/kg = 1.97E-5 mg/kg/day

Cancer Risk from 201 mg/kg_s PCBs

Cancer Risk = Total Dose x Cancer Potency Factor = 1.97E-5 mg/kg/day x 2.0/mg/kg/day = 4E-5 (low)

Sample PCB Noncancer Hazard Quotient Calculation for Sediments

Oral Dose = 201 mg/kgs x 120 mgs/day x 1/18.6 kg x 1E-6 kgs/mgs x 2 d/7 d x 17 wks/52 wks = 1.21E-4 mg/kg/day

Dermal dose = [201 mg/kg_s x 2800 cm² x 0.2 mg_s/cm²-d x 0.14 x 1E-6 kg_s/mg_s x 2 d/7 d x 17 wks/52 wks]/18.6 kg = 7.91E-5 mg/kg/d

Total Dose = 1.21E-4 mg/kg/day + 7.91E-5 mg/kg/day = 2.00E-4 mg/kg/day

Hazard Quotient = Total Dose/Reference Dose = 2.00E-4 mg/kg/day / 2E-5 mg/kg/day = 10 (moderate)

Calculation of Doses and Risks from Incidental Ingestion of Surface Water

Surface Water

1) PCB Cancer Risk Calculation

0.00033 mg/L x 0.053 L/day x 1/70 kg x 2 d/7 d x 17 w/52 w x 30 y/70 y = 1.00E-8 mg/kg/day

Cancer Risk = Dose x Cancer Potency Factor = 1.00E-8 mg/kg/day x 2.0/mg/kg/day = 2 in 100,000,000 (very low)

2) PCB Noncancer Risk Calculation

0.00033 mg/L x 0.09 L/day x 1/18.6 kg x 2 d/7 d x 17 w/52 w = 1.49E-7 mg/kg/day

Hazard Quotient = Dose/Reference Dose = 1.49E-7 mg/kg/day / 2E-5 mg/kg/day = 0.007 (minimal)

Notes on Sample Calculations

Soil ingestion rates are from EPA (2011) and DEC/DOH (2006). Body weights are those recommended by EPA (2011b). Dermal absorption factors and surface area recommendations are found in EPA (2004) (Chapter 3 [Exhibit 3-4]) and EPA (2012). Incidental ingestion rates for surface water are those recommended by ATSDR based on Dufour et al. (2006).

Toxicity Values

Toxicity values used to evaluate risks for contaminants at the Eighteen Mile Creek site are summarized in the following table.

	Cancer Potency			
	Factor	_	Reference Dose	_
Contaminant	(mg/kg/day)⁻¹	Source	(mg/kg/day)	Source
benz[a]anthracene	1.1 ^(a)		0.0017 ^(b)	
benzo[a]pyrene	11	DEC (2010)	0.0017	CA EPA PHG
benzo[b]fluoranthene	1.1 ^(a)		0.0017 ^(b)	
benzo[k]fluoranthene	0.11 ^(a)		0.0017 ^(b)	
chrysene	0.11 ^(a)		0.0017 ^(b)	
dibenz[a,h]anthracene	11 ^(a)		0.0017 ^(b)	
indeno[1,2,3-cd]pyrene	1.1 ^(a)		0.0017 ^(b)	
PCBs	2	US EPA IRIS	0.00002 ^(c)	
Aroclor 1242	2	US EPA IRIS	0.00002 ^(c)	
Aroclor 1248	2	US EPA IRIS	0.00002 ^(c)	
Aroclor 1254	2	US EPA IRIS	0.00002	US EPA IRIS
Aroclor 1260	2	US EPA IRIS	0.00002 ^(c)	
antimony	na		0.0004	US EPA IRIS
arsenic	1.5	US EPA IRIS	0.0003	US EPA IRIS
barium	na		0.2	US EPA IRIS
cadmium	0.067	NYS HHFS	0.0001	ATSDR)
chromium ^d	0.5	CA EPA PHG	0.0009	ATSDR MRL
mercury	na		0.00016	CA EPA PHG
nickel	na		0.02	US EPA IRIS
zinc	na		0.3	US EPA IRIS

Toxicity Values Used to Evaluate Risks for Contaminants at the Eighteen Mile Creek Site

^aRelative potencies applied as described in DEC/DOH (2006).

^bThe CA EPA reference dose for benzo[a]pyrene was used to evaluate noncancer risks for the other carcinogenic PAHs.

^oThe EPA reference dose for Aroclor 1254 was used to evaluate noncancer risks for PCBs and Aroclors 1242, 1248 and 1260. ^dEvaluated as chromium (VI).

Sources:

ATSDR MRL (Agency for Toxic Substances and Disease Registry Minimal Risk Levels). Accessed (November 30, 2011) on-line at http://www.atsdr.cdc.gov/mrls/index.asp, with supporting documentation at http://www.atsdr.cdc.gov/toxprofiles/index.asp.

DEC (New York State Department of Environmental Conservation). 2010. Draft Human Health Fact Sheet. Ambient Water Quality Value for Protection of Human Health and Sources of Potable Water. Benzo[a]pyrene (BaP) and Six Polynuclear Aromatic Hydrocarbons (PAHs). Albany, NY: Division of Water.

CA EPA PHG (California Environmental Protection Agency, Office of Environmental Health Hazard Assessment). Public Health Goals for Chemicals. Accessed (November 3, 2011) on-line at http://www.oehha.ca.gov/water/phg/allphgs.html.

NYS HHFS (New York State Human Health Fact Sheet). Ambient Water Quality Value for Protection of Human Health and Source of Potable Water.

US EPA IRIS (United States Environmental Protection Agency, Integrated Risk Information System). Accessed (11/21/2011) on-line at http://www.epa.gov/iris/.

APPENDIX D Conclusion Categories and Hazard Statements

ATSDR has five distinct descriptive conclusion categories that convey the overall public health conclusion about a site or release, or some specific pathway by which the public may encounter site-related contamination. These defined categories help ensure a consistent approach in drawing conclusions across sites and assist the public health agencies in determining the type of follow-up actions that might be warranted. The conclusions are based on the information available to the author(s) at the time they are written.

1. Short-term Exposure, Acute Hazard "ATSDR concludes that...could harm people's health."

This category is used for sites where short-term exposures (e.g. < 1 yr) to hazardous substances or conditions could result in adverse health effects that require rapid public health intervention.

2. Long-term Exposure, Chronic Hazard "ATSDR concludes that...could harm people's health."

This category is used for sites that pose a public health hazard due to the existence of long-term exposures (e.g. > 1 yr) to hazardous substance or conditions that could result in adverse health effects.

3. Lack of Data or Information "ATSDR cannot currently conclude whether...could harm people's health."

This category is used for sites in which data are insufficient with regard to extent of exposure and/or toxicologic properties at estimated exposure levels to support a public health decision.

4. Exposure, No Harm Expected "ATSDR concludes that ... is not expected to harm people's health."

This category is used for sites where human exposure to contaminated media may be occurring, may have occurred in the past and/or may occur in the future, but the exposure is not expected to cause any adverse health effects.

5. No Exposure, No Harm Expected "ATSDR concludes that ...will not harm people's health."

This category is used for sites that, because of the absence of exposure, are not expected to cause any adverse health effects.

Greetings,

You are receiving a document from the Agency for Toxic Substances and Disease Registry (ATSDR). We are very interested in your opinions about the document you received. We ask that you please take a moment now to complete the following ten question survey. You can access the survey by clicking on the link below.

Completing the survey should take less than 5 minutes of your time. If possible, please provide your responses within the next two weeks. All information that you provide will remain confidential.

The responses to the survey will help ATSDR determine if we are providing useful and meaningful information to you. ATSDR greatly appreciates your assistance as it is vital to our ability to provide optimal public health information.

https://www.surveymonkey.com/r/ATSDRDocumentSatisfaction

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