PUBLIC HEALTH ASSESSMENT

EIGHTEEN MILE CREEK CORRIDOR and DOWNSTREAM REACHES

CITY OF LOCKPORT
NIAGARA COUNTY, NEW YORK

August 1, 2014 **EPA Facility ID: NYN000206456**

Prepared by:

New York State Department of Health
Center for Environmental Health
Under a Cooperative Agreement with
The U.S. Department of Health & Human Services
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia

TABLE OF CONTENTS

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, New York and Olcott, New York could affect their health.

A congressional mandate requires that a public health assessment be conducted for all sites being proposed for the federal National Priorities List (NPL). This public health assessment fulfills the mandate for the Eighteen Mile Creek Site.

The agencies have information that some residents use Eighteen Mile Creek for recreation, and that people catch and eat fish from the creek. There are access points and places where people have been observed fishing on Eighteen Mile Creek. Available environmental sampling data include chemical contamination data for sediments, surface water, soil and fill materials in and along the creek.

Land use surrounding Eighteen Mile Creek includes a mix of agricultural, recreational, residential, and active and abandoned commercial and industrial properties. This includes disposal areas, mill races, mill ponds, and several bridges, culverts and dams. The portion of the creek in the City of Lockport that flows through and out of the abandoned commercial and industrial properties, as well as a small residential neighborhood, is referred to as the “corridor”. Elevated levels of polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and heavy metals, particularly lead, are documented 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 exhibited elevated contamination levels resulting from sediment deposition during high water events.

The Eighteen Mile Creek Corridor Site was subdivided by the New York State Department of Environmental Conservation (DEC) into six Operable Units (OUs). The United States Environmental Protection Agency (EPA) has not subdivided the site in the same manner.

Eighteen Mile Creek receives waters from both the New York State Barge Canal (the Erie Canal) and urban upland watershed sources that add to contaminant loadings from waste disposal areas along its shoreline and from sediments in the creek. Additionally, the Lockport Waste Water Treatment Plant discharges to the creek downstream of the corridor area.

CONCLUSION 1

Eating fish taken from Eighteen Mile Creek could harm people's health if people don't follow DOH's fish consumption advisories.

BASIS FOR DECISION

Contaminated creek sediments have impacted Eighteen Mile Creek, including edible fish species. Fish in Eighteen Mile Creek are contaminated with PCBs at levels that could harm people's health if the fish are consumed. DOH has a "Don't eat ANY fish" fish advisory for all people for the full length of Eighteen Mile Creek (including waters above and below Burt Dam) (http://www.health.ny.gov/environmental/outdoors/fish/health_advisories/).

CONCLUSION 2

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

The highest level of total PCBs in creek sediments is estimated to pose a moderate risk for noncancer health effects. Also, elevated lead levels could result in increased exposure of children and adults to lead through incidental ingestion if the sediments are contacted when people wade and/or use the creek recreationally. Exposure to elevated levels of lead before or after birth can impair the normal development in children of the central nervous system, and can lead to learning or behavioral problems. The highest levels of PCBs in the downstream sediments are estimated to pose a low increased risk for cancer and noncancer health effects.

BASIS FOR DECISION

Sediment samples collected from Eighteen Mile Creek indicate that levels of PCBs and heavy metals exceed New York State soil cleanup objectives (SCOs). 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. Corridor area property owners 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 resident's back yards on Water Street (DEC OU 6) could harm people's health. However, 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.

BASIS FOR DECISION

Soil and sediment samples collected from the backyards of homes along Eighteen Mile Creek on Water Street contained levels of PCBs and lead, arsenic, and chromium that exceed New York State SCOs for residential uses. Repeated, long-term exposure to arsenic and chromium in the past 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.

CONCLUSION 4

The DOH and ATSDR conclude that contact with surface soil while trespassing onto the Flintkote plant property, called DEC OU 2, and long-term exposure to surface soil at Upson Park (DEC OU 4), could harm people's health. Parts of the property also constitute a physical hazard.

BASIS FOR DECISION

The additive estimated cancer risk for carcinogenic PAHs at DEC OU 2 (the Flintkote Plant site) is about 9 in 100,000 (low) or 0.9 in 10,000, which is just below the cancer risk level above which actions to reduce exposure are typically taken (1 in 10,000), 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 (DEC OU 4) 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 we consider this margin of exposure too small to adequately protect human health.

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, especially if, after periods of high water flow, new sediment is deposited or existing sediment is scoured. Since the greatest exposure to these hazards is by contacting sediments or soils at the creek bank, or accessing shoreline fill areas, people should avoid any activity that would result in

contacting these areas of contamination. People should wash their hands after contacting sediments, 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 are walking in the shoreline areas, they should remove their shoes upon entering their home to reduce the potential for tracking sediment into their homes.

People should not eat any fish taken from Eighteen Mile Creek; they should follow the DOH consumption advisory “Don't eat ANY fish”.

Also, DOH and ATSDR recommend that EPA maintain access restrictions to DEC OU 2 and evaluate the extent of PCB 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.

1. The DOH will evaluate historic records of blood lead levels in children who resided in homes on Water Street where backyard soil contains elevated level of lead, to evaluate whether additional, immediate exposure reduction measures are needed or whether additional health education about exposure reduction is needed.

2. Additional investigations are planned by the EPA. The ATSDR and DOH will work with the DEC and EPA on any of these plans for further evaluation of the nature and extent of contamination in the Eighteen Mile Creek corridor and possible contributions to it. ATSDR and DOH will evaluate these EPA 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.

3. DEC required remedial measures for the Eighteen Mile Creek Corridor (DEC OUs 1-6) in two separate Records of Decision. The ATSDR and DOH will work with the DEC and EPA as they determine whether additional measures (e.g., investigations, sampling, or remedial measures) 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 at (716) 551-4410. If you have questions about this 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 is to evaluate human exposure pathways and health risks for contaminants related to the Eighteen Mile Creek National Priorities List (NPL) site in Niagara County, New York. A congressional mandate requires that a public health assessment be conducted for all sites being proposed for the federal National Priorities List (NPL). This public health assessment fulfills the mandate for the Eighteen Mile Creek. Eighteen Mile Creek was proposed to the NPL on September 5, 2011 and it was added 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, 2012a). 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, 2012a).

BACKGROUND

A. Site Description and History:

Description

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 per second of water 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, Figures 1 and 2). 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-lying 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 two branches (East and West). 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 3).

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 ranged 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

The Eighteen Mile Creek Corridor Area was subdivided into six Operable Units (DEC OUs) by the DEC, as shown on Appendix A, Figure 2. An operable unit 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 Operable Units at the Eighteen Mile Creek Corridor Site are defined as follows:

DEC OU 1: Eighteen Mile Creek and Millrace: This operable unit consists of approximately 4,000 linear feet of contaminated creek and millrace sediment from the New York State Barge Canal to Harwood Street, and has been impacted by fill material eroding into the creek from DEC OU 2 thru DEC OU 5, and by direct discharges to the creek from the various facilities that operated at these operable units.

DEC OU 2: Former Flintkote Property: This operable unit, approximately 6.0 acres in size, consists of the former Flintkote property located at 198 and 300 Mill Street. 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 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 (DEC OU 6) on Water Street and is referred to as the Water Street Section. The disposal history of the Flintkote Company is largely unknown, although aerial photographs suggest that disposal of fill on the island was taking place by 1938. It has also been reported that ash resulting from the burning of municipal garbage was dumped on the Flintkote property. The fill material on the 198 Mill Street Parcel and Island is consistent with such a source.

DEC OU 3: Former United Paperboard Property: This operable unit is about 4.8 acres and 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 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 (DEC OU 6). The United Paperboard Company property operated in the late 1880's and early 1890's as a lumber company, and as a paper company from the late 1890's until at least 1948. The history of the property after that time is unknown. The portion of the property near the Clinton Street/Mill Street intersection is currently occupied by Duraline Abrasives. The disposal history of the United Paperboard Company property is unknown, although ash similar to that at the Former Flintkote Property is observed directly 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.

DEC OU 4: Upson Park: This operable unit is about 5.9 acres and consists of the Upson Park property on Clinton Street that operated in the mid 1880's as 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 directly at the surface along the creek. Upson Park is a public park along the Erie Canal that is used for walking, picnicking and other passive leisure activities.

DEC OU 5: White Transportation Property: This operable unit is about 2.6 acres and consists of four adjoining parcels forming the former White Transportation property at 30 thru 40 Mill Street. It was used to store tractor-trailer trucks and other equipment

associated with trucking from 1948 until the late 1990's when operations ceased. Prior to 1948, the property operated as 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 directly 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 from the property. Miscellaneous debris remains scattered throughout the property.

DEC OU 6: Water Street Residential Properties: This operable unit is approximately 2.25 acres in size and consists of nine adjoining residential and vacant properties located at 97 thru 143 Water Street. The properties are bounded to the north by the Water Street Section of the Former Flintkote Plant Site (DEC OU 2), to the east by Eighteen Mile Creek, to the south by Olcott Street, and to the west by Water Street. This OU has been impacted by fill material eroding onto the properties from the Water Street Section of DEC OU 2, and by the deposition of contaminated creek sediments during flooding events.

Downstream Creek Waters to Olcott Harbor:

Once past the corridor area, Eighteen Mile Creek flows through agricultural lands and several small hamlets on its way 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 3). EPA and DEC have divided Eighteen Mile Creek into a series of “reaches”, starting with the northern most reach designated “Reach 1” and the corridor “Reach 10”. For this document, we evaluated Reaches 1-9 together.

Site Cleanup Plans

In two separate Records of Decision (RODs), DEC determined several remedial measures to address contamination and potential exposures to contaminated fill and sediments. For DEC OU1, the Eighteen Mile Creek and Millrace, sediments and the creek bank, the ROD specified excavation, with restoration and long-term monitoring. For DEC OU2, the Former Flintkote Property, the ROD specified sediments excavation and containment. For DEC OU3, the Former United Paperboard Property, the ROD specified hazardous waste would be removed with bank stabilization and long-term monitoring. For DEC OU4, Upson Park, the ROD specified hazardous waste be

removed, with bank stabilization and long-term monitoring. For DEC OU5, White Transportation, the ROD specified hazardous waste be removed, with bank stabilization and long-term monitoring. For DEC OU6, the Water Street Residential Properties, the remedy calls for targeted excavation with bank stabilization and long-term monitoring.

While the nature and extent of the contamination varies at each of the Operable Units, 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.

The DEC referred the site to the EPA for inclusion in the NPL in 2011. EPA is reviewing the DEC's RODs, along with all site data, to make further decisions.

B. Site Visit

The DOH, ATSDR, DEC, Niagara County Health Department and the EPA made a visit on October 25, 2011 to 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. DOH staff members Matt Forcucci, Bettsy Prohonic, and Don Miles, along with staff from ATSDR, EPA, DEC and the Niagara County Department of Health visited the site. The group discussed and observed access to and recreational use of the creek. At the time of the visit, no active recreational use was observed.

C. Demographics

The DOH estimated, from the 2010 Census, that approximately 12,460 people live within a 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 Bureau of the Census 2011), the area had a higher percentage of the population living below the poverty level and the median household income is lower than that of Niagara County or New York State excluding NYC. Comparisons are provided in Table A.

Table A. Demographics for the population one mile around the Eighteen Mile Creek corridor, Niagara County, and New York State excluding New York City. Data are from the 2010 US Census and the 2006-2010 American Community Survey.

Census Demographics Estimates	1 mile around Eighteen Mile Creek	Niagara County	New York State (excluding New York City)
Total Population[1]	12460	216,469	11,202,933
Percent Male	48.9%	48.5%	49.0%
Percent Female	51.1%	51.5%	51.0%
Age Distribution[1] (%)			
<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%
>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%
Percent Hispanic	3.9%	2.2%	9.6%
Percent 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%

[1] US Census Bureau. Census 2010 Summary File 1 - New York State/ prepared by the U.S. Census Bureau, 2011.
[2] US Census Bureau 2006-2010 American Community Survey 5-Year Estimates Summary File Tracts and Block Groups. 2011
* 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 existing environmental and exposure information to complete an assessment of health risks presented by exposure to chemical contaminants in

Eighteen Mile Creek. We have 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") (http://www.health.ny.gov/environmental/outdoors/fish/health_advisories/). We do not have information about how much fish people catch and eat from the creek, although anecdotal evidence suggests that eating fish from the creek is common.

A. Environmental Contamination

Environmental contamination data for Eighteen Mile Creek, from the contaminated areas of DEC OU 1-6 and downstream to its outlet into Lake Ontario, were collected over several years. These data have recently been summarized by EPA in their March 2012 *Remedial Investigation Report* (CH2M Hill et al., 2012). For this public health assessment, we evaluate 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 DEC OU 2 did not contain detectable levels of PCBs (detection limit 0.050 micrograms per liter (mcg/L)); three samples downstream of DEC OU 2 contained the commercial PCB mixture Aroclor 1248 at levels ranging from 0.084 mcg/L to 0.33 mcg/L. PCBs are synthetic chemicals which were sold in mixtures under the trade name "Aroclor." These levels of PCBs in the surface water are below the New York State public drinking water standard and the federal Maximum Contaminant Level for PCBs of 0.5 mcg/L. The drinking water standard is based on a water ingestion rate much greater than would result from recreational use of Eighteen Mile Creek, and, therefore, the levels of PCBs detected in the surface water samples do not constitute a public health concern and exposure to Creek water will not be discussed further. The creek is not known to be used for drinking water.

Surface Soil

Surface soil sampling data are available for DEC OUs 2, 3, 4, 5 and 6 (DEC OU 1 was not included since the creek and associated water bodies have no exposed soil associated with them) (Table B). We used summary data from the RODs for the DEC OUs to evaluate the contamination. Data for these RODs came from remedial investigation reports. Data for DEC OU 2 were taken from the ROD for DEC OU 2 (DEC, 2006a). Data for DEC OUs 3, 4, and 5 were taken from the ROD for those DEC OUs (DEC, 2010). Data for DEC OU 6 are from the DEC Remedial Investigation for the Eighteen Mile Creek Corridor site (DEC, 2006b). PCB Aroclor-specific data for DEC OU 3 and 4 came from the Eighteen Mile Creek Corridor site Remedial Investigation Report (DEC 2006a).

Surface Sediments

Data for chemical contamination of Eighteen Mile Creek surface sediments downstream from DEC OUs 1-6 were taken from EPA's Hazardous Ranking System (HRS) documentation for Eighteen Mile Creek (EPA, 2011a; Ecology and Environment Engineering PC, 2009; CH2M Hill et al., 2011) (Table B).

Table B. Ranges of Surface Soil and Sediment Sampling Results for Eighteen Mile Creek.
(All values in milligrams per kilogram soil (mg/kg_s).

Sampling Location	Total PCBs*	Aroclor 1242	Aroclor 1248	Aroclor 1254	Aroclor 1260	Lead
Surface Soil						
DEC OU 1	N/A	N/A	N/A	N/A	N/A	N/A
DEC OU 2	ND – 4.6	ND	ND	ND – 4.6	ND	58 - 7610
DEC OU 3	ND - 4.3	NA	NA	NA	NA	4.5 - 3600
DEC OU 4	ND -23	NA	NA	NA	NA	19 - 3480
DEC OU 5	ND -0.67	NA	NA	NA	NA	9.7 - 3750
DEC OU 6	ND -27	NA	NA	NA	NA	30 - 4630
Sediments						
DEC OU 1 (ROD)	ND - 201	NA	NA	NA	NA	11.3 – 25,400
DEC OU 1 - 6	N/A	ND - 46	ND- 180	ND - 57	ND	NA
Downstream	N/A	ND	0.38 - 41	0.52 - 55	8.3 - 42	NA

*Total PCBs and Aroclor data came from different data sets, so they do not always match.
ND: not detected
N/A: not applicable
NA – not available

B. 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) 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, according to the Niagara County Department of Health, there are no permitted bathing or swimming facilities situated along the boundaries of the Creek. 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, but, as noted above, contaminant levels did not exceed comparison values for drinking water, which are derived assuming a person drinks 2 liters of water per day (which is significantly more water than one would ingest while swimming).

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

Residents along Water Street who have backyards that are contaminated with site-related contamination are likely contacting 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.

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.

C. Public Health Implications Adult and Child's Health Considerations

Contact with contaminated surface soils on some residential properties and at some locations at Upson Park (DEC OU 4) 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 (DEC OUs 2, 3, and 5, respectively) 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 creek related chemical contaminants in sediments. An evaluation and characterization of the health risks for exposure to residential surface soil and sediment contaminants by incidental ingestion and dermal absorption is presented below.

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

In general, we first compared the highest detected level of contaminants in surface soil and creek sediments to the corresponding New York State residential or restricted residential SCOs (DEC/DOH, 2006c) and ATSDR comparison values for soil (ATSDR, 2012). SCOs are soil concentrations that are contaminant-specific remedial goals based on current, intended or reasonably anticipated future land use. 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. The health based SCOs are set at a soil concentration at which cancer and noncancer health effects are unlikely to occur (i.e., a cancer risk level of one in one million for carcinogens, or at a hazard quotient of one for noncancer effects). If a health based SCO is calculated to be lower than the contaminant's rural soil background concentration (i.e., typical levels of the contaminant in soil), the final SCO is set at the rural soil background concentration.

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

We selected residential soil contaminants for further evaluation if the highest detected levels exceeded their New York State residential SCOs or ATSDR comparison values. We selected soil contaminants from DEC OUs 2, 3, 4 and 5 and creek sediment contaminants for further evaluation if the highest detected levels exceeded their New York State restricted residential SCOs or ATSDR comparison values. We then evaluated the selected contaminants and characterized the risks with site-specific health based comparison values (Appendix B, Tables 2,4,5 and 8). Exposure assumptions for these site-specific comparison values are briefly described below:

For residential soil contaminants, we evaluated the risk for getting 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 dermal contact 31 weeks each year (five days per week for the first 17 years of life, 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, we assume a child is exposed by incidental ingestion and skin contact five days a week during the non-winter months. For residential properties, we assume 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, we evaluated the risks for soil ingestion and dermal contact only. Additional details on the exposure parameters used for residential properties are found in the footnotes to Table 2, Appendix B. Sample calculations of exposure doses, cancer risk and noncancer hazard quotients are found in Table 1, Appendix C.

For properties involving trespassing (i.e., DEC OUs 2, 3, and 5 [Flintkote, United Paperboard and White Transportation properties]), we assume 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 we also assume 11 years of exposure (representing ages 10 to 21, or the ages of people who might be reasonably anticipated to trespass on the properties). We 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 the footnotes to Table 4, Appendix B. Calculation of exposure doses for nonresidential properties are found in Table 2 of Appendix C.

For a park or a recreational area, such as Upson Park (DEC OU4), we 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 for Upson Park are found in the footnotes to Table 5, Appendix B. Sample calculation of comparison values for Upson Park are found in Appendix D.

For estimating health risks in creek sediments, we 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). We evaluate the noncancer risks for a 3 year old child, and for cancer risks, we assume a person is exposed for 15 years from age 3 to 18. Additional details on the exposure parameters used for our evaluation of creek sediments are found in the footnotes to Table 8, Appendix B. Calculation of exposure doses for creek sediments are found in Table 3 of Appendix C.

<u>Residential Properties (DEC OU 6)</u>

Chemicals Selected for Further Evaluation

As shown in Table 1, Appendix B, the levels of polychlorinated biphenyls (PCBs), arsenic, chromium, lead and zinc on the residential properties exceed their residential

SCOs or ATSDR comparison values. We therefore selected these contaminants for further evaluation.

We further evaluated the residential soil contaminants using site-specific health based comparison values. PCBs, arsenic and chromium exceeded their health based comparison values, and we therefore characterized the health risks for these contaminants and for lead. A general discussion of the health effects of these contaminants is presented below.

Health Effects of PCBs (ATSDR 2011; EPA 2013)

Polychlorinated biphenyls are synthetic chemicals that were sold in mixtures in the United States under the trade name “Aroclor.” There are no known natural sources of PCBs. PCBs are either oily liquids or solids that are colorless to light yellow. Some PCBs can exist as a vapor in air. PCBs have no known smell or taste.

PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they don't burn easily and are good insulators. The manufacture of PCBs was stopped in the United States in 1977 because of evidence they build up in the environment and can cause harmful health effects. Products made before 1977 that may contain PCBs include old fluorescent lighting fixtures and electrical devices containing PCB capacitors, and old microscope and hydraulic oils.

PCBs cause liver cancer in laboratory animals exposed to high levels for their lifetimes. In addition to the animal studies, a number of epidemiological studies of workers exposed to PCBs have found an increased risk for liver cancers and malignant melanoma. The EPA has classifies PCBs as probably human carcinogens based on the presence of cancer in the same target organ (liver) following exposures to PCBs both in animals and in humans and the finding of liver cancers and malignant melanomas across several human studies. The Department of Health and Human Services has concluded that PCBs may reasonably be anticipated to be carcinogens, and the International Agency for Research on Cancer has determined that PCBs are carcinogenic to humans.

Numerous studies in laboratory animals (including nonhuman primates) show that PCBs can cause several types of noncancer toxicity, including effects on the immune system, the reproductive system, the nervous system and the endocrine system. The effects on the immune system include decreases in thymus gland size and reductions in immune response and resistance to disease in PCB-exposed animals. Concerning the reproductive system, PCB exposure has been found to reduce the birth weight, conception rates and live birth rates of monkeys and other species, and to reduce sperm counts in rats. The nervous system effects include deficits in neurological development (e.g., visual recognition, short-term memory and learning) in newborn monkeys and PCBs can cause endocrine system effects such as the reduction of thyroid hormone in laboratory animals.

Epidemiology studies of people exposed to PCBs report associations between PCB exposure and many of the noncancer health effects documented in animals, including increased vulnerability to certain types of infections, decreased birth weights and gestational age, learning deficits and changes in thyroid hormone levels. Epidemiology studies have also reported associations between PCB exposure and an increase risk for diabetes. Although the role of other factors in causing these associations is not completely understood, these human studies, taken together with the studies in animals support the conclusion that PCBs may cause these effects in humans.

A variety of other non-cancer effects of PCBs have been reported in animals and humans, including dermal and ocular effects in monkeys and humans, and liver toxicity in rodents. Elevations in blood pressure, serum triglyceride, and serum cholesterol have also been reported with increasing serum levels of PCBs in humans.

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, 2005). The primary health effects associated with exposure to chromium are cancer and noncancer effects on the digestive and lymphatic systems, blood and liver (ATSDR, 2012b). 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 Lead

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

People can be exposed to lead 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, 2007). 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 should 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. Recent scientific research, however, has clearly shown that blood lead levels below 10 mcg/dL can cause serious harmful effects in children. Recently, CDC indicated that, based on reviewing extensive blood-lead health information, the blood lead reference level should be lowered to 5 mcg/dL, which is the 97.5 percentile blood lead level in U.S. children based on the latest National Health and Nutrition Examination Survey (CDC, 2012). CDC plans to use this reference value to identify high-risk childhood populations and geographic areas most in need of primary prevention.

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, 2008). 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, 2008).

Risk Characterization for Residential Properties

The primary health effects associated with exposure to PCBs are cancer and noncancer effects on the immune system (ATSDR, 2001). Surface soil samples from the creek bank showed elevated levels of total PCBs (27 milligrams per kilogram soil (mg/kg_s) and 17.4 mg/kg_s) at two of the eight residential yards sampled. Exposure to PCBs in soils at the creek bank via homegrown fruits and vegetables is unlikely because the bank is wooded, shady, subject to periodic flooding, and unsuitable for gardening. Therefore for these samples, we evaluated the risks for soil ingestion and dermal contact only. Repeated, long-term exposure to soil PCB levels of 27 mg/kg_s and 17.4 mg/kg_s 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 health-based comparison value (which corresponds to the reference dose[1]), 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 we consider too small to adequately protect human health). Repeated and long-term (30 years) exposure to soil PCB levels of 27 mg/kg_s or 17.4 mg/kg_s at the creek bank locations is estimated to pose a low increased risk for getting cancer (Appendix E), which means the estimated increased risk is between one in one million and one in ten 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_s. 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 only be associated with creek bank soils.

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 and lymphatic systems, blood and liver (ATSDR, 2012b). The levels of arsenic and chromium exceed their residential SCOs (16 mg/kg_s and 22 mg/kg_s) at four of the eight residential properties sampled. The levels also exceeded health-based comparison values. The levels above the SCOs at these properties ranged from 23.1 mg/kg_s to 66.5 mg/kg_s for arsenic and 25.6 mg/kg_s to 164 mg/kg_s for chromium. The following table (Table A) characterizes the estimated cancer and noncancer health risks for long-term, repeated exposure to the highest and average soil levels of arsenic and chromium in individual yards where the levels exceeded the residential SCOs. Risk characterization for both the highest and average contaminant levels are presented since there is uncertainty about which measure is more representative of a person's exposure on these properties. An example of cancer and noncancer risk calculations is presented in Appendix B.

[1]The noncancer comparison value is a soil concentration that will result in a contaminant exposure equal to the contaminant's reference dose if a person is exposed according to the assumptions for residential exposure. The reference dose is a level of exposure to a contaminant that is unlikely to result in adverse noncancer health effects assuming a person is exposed for up to a lifetime.

Table A. Cancer and Noncancer Risk Descriptors for Highest and Average Levels of Arsenic and Chromium in Soil at Individual Residential Properties Along the Eighteen Mile Creek.

Highest Level in Soil (mg/kg_s)	Cancer Risk Descriptor	Noncancer Risk Descriptor	Average Level in Soil (mg/kg_s)	Cancer Risk Descriptor	Noncancer Risk Descriptor
Arsenic					
23.1	moderate	low	14.2	moderate	low
26.4	moderate	low	15.9	moderate	low
30.4	moderate	low	29.9	moderate	low
66.5	moderate	moderate	30.7	moderate	low
Chromium[a]					
25.6	moderate	minimal	--[b]	--[b]	--[b]
39.1	moderate	minimal	26.8	moderate	minimal
114.5	moderate	low	48.2	moderate	low
164.0	moderate	low	58.0	moderate	low

[a]Environmental sampling results are for total chromium. Risk characterization assumes all the chromium is in the hexavalent form.

[b]Only one sample available.

mg/kg_s: milligrams per kilogram of soil

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. At all eight properties, lead was detected in residential surface soil above its residential SCO (400 mg/kg_s), and ranged from 29.8 mg/kg_s to 4630 mg/kg_s. The presence of lead at these properties could result in increased exposure of children to lead through incidental ingestion of soil during typical residential activities. Based on the soil lead levels and likely nature of expected exposures, the soil levels would be the primary contributor to potential increases in blood lead levels, which, when added to other background lead exposures, could be considerable in light of the recent Centers for Disease Control and Prevention revision of the blood lead reference value to 5 micrograms per deciliter of blood (mcg/dL) (CDC, 2012). Whether the exposures in soil would actually increase blood lead levels of a child depends primarily on the actual lead level in the soil the child contacts, the bioavailability of lead, the frequency and duration of exposure, and how much soil the child ingests.

<u>DEC Operable Units 2, 3, 4 and 5</u>

Chemicals Selected for Further Evaluation

We screened the levels of surface soil contaminants at the Flintkote Plant site, the former United Paperboard property, Upson Park and the White Transportation property (DEC OUs 2, 3, 4, and 5, respectively) against their restricted residential SCOs or ATSDR comparison values. As shown in Table 3, Appendix B, carcinogenic PAHs, PCBs, arsenic, barium, cadmium, chromium, lead, mercury, nickel, and zinc exceed their restricted residential SCO or ATSDR comparison value at some or all of these OUs. Antimony, for which an SCO is not available, exceeded its EPA Regional Screening Level for Soil (EPA, 2012b).

We further evaluated these chemicals using health based cancer and noncancer comparison values (Table 4, Appendix B). These health based comparison values are contaminant concentrations in soil that pose very low or minimal health risks to a child or adolescent that trespasses the properties and is exposed to soil contaminants by incidental ingestion and skin contact. Of the chemicals detected in surface soil in DEC OUs 2, 3 and 5 above their restricted residential SCOs, the levels of carcinogenic PAHs (benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene), arsenic and chromium exceeded their cancer comparison values on at least one property (see Table 4, Appendix B). We therefore characterized the cancer risks for these chemicals. Of the chemicals detected in surface soil above their restricted residential SCOs at Upson Park, which is a recreational area, the levels of carcinogenic PAHs (benz(a)anthracene, benzo(a)pyrene and benzo(b)fluoranthene), PCBs, arsenic and chromium exceeded their cancer comparison values and the levels of PCBs, arsenic and chromium exceeded their noncancer comparison values. We therefore characterized the risks for cancer and noncancer health effects for these chemicals at Upson Park.

The health effects of PCBs, arsenic, and chromium have previously been discussed. A general discussion of the health effects of PAHs 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, 1994) and a human carcinogen by other agencies (WHO, 2012).

Risk Characterization for DEC Operable Units 2, 3, and 5 (Flintkote, United Paperboard and White Transportation Properties)

The highest levels of carcinogenic PAHs, arsenic and chromium in surface soil at DEC OUs 2, 3 and 5, and the highest levels of carcinogenic PAHs at DEC OUs 3 and 5 are estimated to pose a low increased risk of getting cancer if someone trespassed 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 (i.e., the increased lifetime cancer risk is between one in one million and one in ten thousand). However, the additive estimated cancer risk for carcinogenic PAHs at DEC OU 2 (the Flintkote Plant site) is about 9 in 100,000, which is just below the cancer risk level above which actions to reduce exposure are typically taken (1 in 10,000). The risk for noncancer effects for the highest detected levels of the other contaminants detected at DEC OUs 2, 3, and 5 is minimal. Table 6, Appendix B lists the cancer and noncancer risk descriptors for the individual chemicals at each DEC OU.

Risk Characterization for Upson Park (DEC OU4)

Based on the available soil sampling information, the highest level of 23 mg/kg_s of total PCBs at Upson Park (DEC OU 4) may be localized to a specific area and therefore may not be representative of potential exposures for the entire park. Repeated, long-term exposure to the highest level of total PCBs (23 mg/kg_s) is estimated to pose a moderate risk for noncancer health effects, because the estimated exposure more than 8 times higher than the noncancer health comparison value (which corresponds to the reference dose), and is only 29 times lower than the lowest exposure that caused immune toxicity in laboratory animals (EPA, 1996a), which is a margin of exposure we consider too small to adequately protect human health. The noncancer risk posed by the highest levels of arsenic (63 mg/kg_s) is low. Repeated, long-term (30 years) exposure to the highest level of carcinogenic PAHs, total PCBs, arsenic and chromium in surface soil is estimated to pose a low increased risk for getting cancer (the estimated increased cancer risk is between one in one million and one in ten thousand). Table 7, Appendix B lists the cancer and noncancer risk descriptors for the individual chemicals at Upson Park.

Lead at Operable Units 2, 3, 4 and 5

Surface soil samples at the Flintkote Plant site (DEC OU 2), Former United Paperboard property (DEC OU 3), Upson Park (DEC OU 4) and the White Transportation Property (DEC OU 5) contained lead levels above the restricted residential SCO of 400 mg/kg_s. The highest levels of lead were 7,610 mg/kg_s, 3,600 mg/kg_s, 3,480 mg/kg_s and 3,750 mg/kg_s, respectively. As with the residential properties, the presence of lead at these properties could result in increased exposure of children and adults to lead through incidental ingestion of soil. The contribution of lead exposure from soil could be significant given the recent revision of the Centers for Disease Control and Prevention's blood lead reference value to 5 mcg/dL of blood (CDC, 2012). As with the residential

properties, whether or not the exposures in soil would actually increase blood lead levels depends primarily on the actual soil lead levels in the location they are contacted, the frequency and duration of exposure, and how much soil is ingested.

Corridor Creek Sediments

Chemicals Selected for Further Evaluation (DEC OU 2 through 6)

Sampling results of shallow creek sediments where the creek runs through the residential and nonresidential properties that make up DEC OUs 2 through 6 (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$_s$ and 34 mg/kg$_s$). For total PCBs, 30 shallow sediment sampling results ranged from 0.058 mg/kg$_s$ to 201 mg/kg$_s$. The average of detected PCB levels was 10.9 mg/kg$_s$, with 26 of 30 results below 10 mg/kg$_s$ and half below 1 mg/kg$_s$. Sixteen shallow sediment samples provided information on the levels of specific Aroclors (commercial mixtures of PCBs). Aroclor 1242 ranged from 1.2 mg/kg$_s$ to 46 mg/kg$_s$, Aroclor 1254 ranged from 0.73 mg/kg$_s$ to 180 mg/kg$_s$, and Aroclor 1260 ranged from 0.67 to 57 mg/kg$_s$. The average of the detected levels was 24.7 mg/kg$_s$, 22.8 mg/kg$_s$ and 8.0 mg/kg$_s$ 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$_s$ for each) and their ATSDR comparison values (0.096 mg/kg$_s$ and 0.35 mg/kg$_s$ for benzo(a)pyrene and total PCBs, respectively), and we therefore evaluated the health risks for exposure by incidental ingestion and skin contact.

We further evaluated these chemicals in creek sediments using health based cancer and noncancer comparison values (Table 8, Appendix B). The sediment levels of benzo(a)pyrene exceeded its cancer comparison value, and the PCBs levels exceeded both the cancer and noncancer comparison value. Therefore we characterized the health risks for these contaminants in sediments.

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

Risk Characterization for Corridor Creek Sediments

The highest levels of benzo(a)pyrene and total PCBs in the corridor creek sediments are estimated to pose a low increased risk of getting cancer (the risk is between one in one million and one in ten thousand). Exposure to the highest level of total PCBs in creek sediments (201 mg/kg$_s$) is estimated to pose a moderate risk for noncancer health effects because the estimated exposure is 10 times higher than the health comparison value (which corresponds to the reference dose) and is only 25 times lower than the lowest exposure that caused immune toxicity in laboratory animals (EPA, 1996a), which is a margin of exposure we consider too small to adequately protect human health. The noncancer risks for benzo(a)pyrene are estimated to be minimal. The sampling data (summarized in the previous paragraph) suggest that the PCB levels in shallow sediment are variable and that high levels of PCBs 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 risks posed by the presence of the PCBs in shallow sediments may be lower than those estimated here due to the conservative nature of our evaluation. Table 9, Appendix B lists the cancer and noncancer risk descriptors for the individual chemicals corridor creek sediments.

Lead in Corridor Creek Sediments

Samples of the shallow creek sediments through the corridor contain lead ranging from 11.3 to 25,400 mg/kg$_s$. Numerous creek sediment samples contained lead above the restricted residential SCO of 400 mg/kg$_s$. 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 when people wade and/or use the creek recreationally. The contribution of elevated lead in sediment could be significant given the recent revision of the Centers for Disease Control and Prevention's blood lead reference value to 5 mcg/dL (CDC, 2012). Whether or not the exposures would actually increase blood lead levels depends primarily on the actual sediment lead levels in the location they are contacted, the frequency and duration of exposure, and how much sediment is ingested.

Downstream Creek Sediments

PCB sampling results of shallow creek sediments at 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 sedments 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$_s$, 55 mg/kg$_s$, and 42 mg/kg$_s$, respectively. The levels in the downstream sediments exceed the restricted residential SCO and ATSDR comparison value for PCBs (1 mg/kg$_s$ and 0.35 mg/kg$_s$, respectively), and we therefore further evaluated these levels with the health based comparison values derived under the same assumptions as were used to evaluate the corridor sediments (Table 8, Appendix B). The PCB sediment levels exceeded both the cancer and noncancer comparison value, and therefore we characterized the health risks for this contaminant in sediments.

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. Table 9, Appendix B lists the cancer and noncancer risk descriptors for the individual chemicals downstream creek sediments.

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.

We considered the possibility that children may be more sensitive to the health effects of environmental contaminants when we evaluated the surface soil sampling results for chromium at the residential properties, and the PAH benzo(a)pyrene at the Flintkote Plant site (DEC OU 2), the Former United Paperboard property (DEC OU 3), Upson Park (DEC OU 4) and the White Transportation Property (DEC OU 5). 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 chemicals 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). Therefore, children may be more sensitive than adults to the carcinogenic effects of hexavelant chromium and benzo(a)pyrene. To account for this possible greater sensitivity, we followed the EPA guidance (EPA, 2005; 2006) and increased our estimated cancer risk calculations for these contaminants by a factor of ten for children exposed during the first two years of life, and by a factor of three for children exposed from ages three through age 15. In addition, for all contaminants, we 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 alone. Antagonism takes place when the combined effect 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, both of 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 our 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, we will assume 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.

D. Health Outcome Data Evaluation.

The DOH evaluated historic records from 1994-2011 of blood lead levels in children who resided in homes on Water Street where backyard soil may have 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 ug/dl. Additional evaluation of health outcomes would be difficult due to lack of exposure information, availability of relevant health data and 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 Operational Units that comprise the Corridor site, and other waste sites in Lockport, we 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 people don't follow DOH's fish consumption advisories. Contaminated creek sediments have impacted Eighteen Mile Creek, including edible fish species. Fish in Eighteen Mile Creek are contaminated with PCBs at levels that could harm people's health if the fish are consumed. DOH has a "Don't eat ANY fish" fish advisory for the full length of Eighteen Mile Creek (including waters above and below Burt Dam) (http://www.health.ny.gov/environmental/outdoors/fish/health_advisories/).

DOH and ATSDR conclude that contact with Eighteen Mile Creek sediments and contaminated fill materials could harm people's health (see Appendix D). Sediment samples collected from Eighteen Mile Creek indicate that levels of PCBs and heavy metals exceed New York State SCOs. 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. Corridor area property owners have attempted to secure contaminated areas from trespassers and recreational fishermen, but there is evidence of trespass.

The highest level of total PCBs in creek sediments in the corridor is estimated to pose a moderate risk for noncancer health effects. Also, elevated lead levels could result in increased exposure of children and adults to lead through incidental ingestion if the sediments are contacted when people wade and/or use the creek recreationally. In children, exposure to elevated levels of lead before or after birth can impair the normal development of the central nervous system, and can lead to learning or behavioral problems. The highest levels of PCBs in the downstream sediments are estimated to pose a low increased risk for cancer and noncancer health effects.

The DOH and ATSDR conclude that contact with sediment and soil in resident's backyards on Water Street in the past could harm people's health. Soil and sediment samples collected from the backyards of homes along Eighteen Mile Creek on Water Street contain levels of PCBs, lead, arsenic and chromium that exceed New York State SCOs for residential uses. However, 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.

The DOH and ATSDR conclude that contact with surface soil while trespassing onto the Flintkote plant property (DEC OU 2), and long-term exposure to surface soil at Upson Park (DEC OU 4) could harm people's health.

The additive estimated cancer risk for carcinogenic PAHs at the Flintkote Plant site is about 9 in 100,000 or 0.9 in 10,000 (low), which is just below the cancer risk level above which actions to reduce exposure are typically taken (1 in 10,000), 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 actual cancer risk may be lower

since it is unlikely that an individual would trespass 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, which is a margin of exposure we consider too small to adequately protect human health.

RECOMMENDATIONS

For those people using or living along the Eighteen Mile Creek Corridor and downstream, the DOH and ATSDR recommend that measures be taken 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, especially if, after periods of high water flow, new sediment is deposited or existing sediment is scoured. Since the greatest exposure to these soil and sediment hazards is by contacting sediments or soils at the creek bank, or accessing shoreline fill areas, people should avoid any activity that would result in contacting these areas of contamination. People should wash their hands after contacting sediments, 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. Children's hands, feet and toys should be washed after playing or digging in the soil. If people are walking in the shoreline areas, they should remove their shoes upon entering their home to reduce the potential for tracking sediment into their homes.

Also, DOH and ATSDR recommend that EPA maintain access restrictions to DEC OU 2 and evaluate the extent of PCB contamination of surface soil at Upson Park. Based on the evaluation at Upson Park, additional exposure reduction measures may be warranted.

People should not eat any fish taken from Eighteen Mile Creek; they should follow the DOH consumption advisories "Don't eat ANY fish" (http://www.health.ny.gov/environmental/outdoors/fish/health_advisories/).

PUBLIC HEALTH ACTION PLAN

Actions completed:

The DOH provided exposure-reduction advice in letters written to homeowners of Water Street 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.

Actions Planned:

The DOH will evaluate historic records of blood lead levels in children who reside or resided in homes on Water Street where backyard soil contains elevated level of lead, to evaluate whether additional, immediate exposure reduction measures, or additional health education about exposure reduction are needed for current residents.

The DOH and ATSDR will work with the DEC and EPA on plans for further evaluation of the nature and extent of contamination in the Eighteen Mile Creek corridor and possible contributions to it. DOH and ATSDR will evaluate these EPA data as they become available to us to determine whether additional actions are needed to reduce people's exposure to contamination in the creek.

DEC determined remedial measures for DEC OU 1 to OU 6 in two separate RODS (DEC, 2006a; 2010). The DOH and ATSDR will work with the DEC and EPA as they determine whether additional measures (e.g. investigations, sampling, or remedial measures) are needed in the corridor and whether measures will be needed in the downstream portion of Eighteen Mile Creek.

The 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 affected communities.

REFERENCES

Ahlborn GJ, Nelson GM, Grindstaff RD, et al. 2009. Impact of life stage and duration of exposure on arsenic-induced proliferative lesions and neoplasia in C3H mice. Toxicology. 262: 106-113.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Atlanta, GA: US Department of Health and Human Services, Public Health Service.

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. ToxFAQs™ for Polychlorinated Biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service,

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures. Atlanta, GA: Division of Toxicology, Agency for Toxic Substances and Disease Registry, Public Health Service, US Department of Health and Human Services. Available on-line at: http://www.atsdr.cdc.gov/interactionprofiles/index.asp.

ATSDR (Agency for Toxic Substances and Disease Registry). 2007a. Toxicological Profile for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

ATSDR (Agency for Toxic Substances and Disease Registry). 2007b. Toxicological Profile for Lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

ATSDR (Agency for Toxic Substances and Disease Registry). 2008. Toxicological Profile for Chromium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

ATSDR (Agency for Toxic Substances and Disease Registry). 2011. Addendum to the Toxicological Profile for Polychlorinated Biphenyls. Accessed (March 20, 2014) on-line at http://www.atsdr.cdc.gov/toxprofiles/pcbs_addendum.pdf.

ATSDR (Agency for Toxic Substances and Disease Registry). 2012. Soil Comparison Values from ATSDR's Sequoia Database. August, 2012.

CDC (Centers for Disease Control and Prevention). 2012. Lead. Accessed (7/17/2012) on-line at http://www.cdc.gov/nceh/lead/.

CH2M Hill (CH2M Hill, Ecology and Environment, Inc., Environmental Design International, Inc. and Teska Associates Inc) et al. 2011. Data Summary Report Site

Characterization of the Eighteenmile Creek Area of Concern. Prepared for U.S. Environmental Protection Agency. March.

CH2M Hill (CH2M Hill, Ecology and Environment, Inc., Environmental Design International, Inc., Teska Associates, Inc., Critigen, LLC) et al. 2012. Remedial Investigation Report, Eighteenmile Creek Area of Concern, Niagara County, New York, March.

DEC (New York State Department of Environmental Conservation) 2006a. Record of Decision Eighteenmile Creek Corridor Site, Operable Unit Nos. 2 FlintKote State Superfund Project, Lockport, Niagara County, New York, Site Number 932121. Division of Environmental Remediation. March 2006. Available on-line at: http://www.dec.ny.gov/docs/regions_pdf/flintrod.pdf

DEC (New York State Department of Environmental Conservation) 2006b. Remedial Investigation Report Eighteenmile Creek Corridor. Region 9 Division of Environmental Remediation. September.

DEC/DOH (New York State Department of Health/New York State Department of Environmental Conservation). 2006c. New York State Brownfield Cleanup Program. Development of Soil Cleanup Objectives. Technical Support Document. September 2006. Available on-line at http://www.dec.ny.gov/chemical/34189.html.

DEC (New York State Department of Environmental Conservation) 2008. Eighteen Mile Creek Site No. 932123 Sampling Report. October.

DEC (New York State Department of Environmental Conservation) 2010. Record of Decision Eighteenmile Creek Corridor Site, Operable Unit Nos. 1, 3, 4, 5 and 6, State Superfund Project, Lockport, Niagara County, New York, Site Number 932121. Division of Environmental Remediation. March. Available on-line at: http://www.dec.ny.gov/docs/regions_pdf/18milerod.pdf

Ecology and Environment Engineering P.C. 2009. Final Supplemental Remedial Investigation Report for the Eighteenmile Creek Corridor Site. Prepared for New York State Department of Environmental Conservation. July. Available on-line at: http://www.dec.ny.gov/docs/regions_pdf/18msuppri.pdf

EPA (United States Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A) Interim Final. Accessed (9/5/2012) on line at http://www.epa.gov/oswer/riskassessment/ragsa/index.htm).

EPA (United States Environmental Protection Agency). 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-

93/089. Available on line at:
http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=49732.

EPA (United States Environmental Protection Agency). 1994. Integrated Risk Information System. Benzo(a)pyrene (BaP) (CASRN 50-32-8). Washington, DC: Office of Research and Development, National Center for Environmental Assessment. Accessed (1/11/12) on-line at http://www.epa.gov/iris/subst/0136.htm.

EPA (United States Environmental Protection Agency). 1996a. Integrated Risk Information System. Aroclor 1254 (CASRN 11097-69-1). Washington, DC: Office of Research and Development, National Center for Environmental Assessment. Accessed (9/30/2013) on-line at http://www.epa.gov/iris/subst/0389.htm.

EPA (United States Environmental Protection Agency). 1996b. Integrated Risk Information System. Polychlorinated biphenyls (PCBs) (CASRN 1336-36-3). Washington, DC: Office of Research and Development, National Center for Environmental Assessment. Accessed (1/11/2012) on-line at:
http://www.epa.gov/iris/subst/0294.htm.

EPA (United States Environmental Protection Agency). 1998. Integrated Risk Information System. Arsenic, inorganic (CASRN 7440-38-2). Washington, DC: Office of Research and Development, National Center for Environmental Assessment. Accessed (1/11/12) on-line at http://www.epa.gov/iris/subst/0278.htm.

EPA (United States Environmental Protection Agency). 2000. Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures. Risk Assessment Forum Technical Panel. EPA/630/R-00/002. Washington, DC: Risk Assessment Forum.

EPA (United States Environmental Protection Agency). 2004. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part E) Interim Final. Accessed (9/5/2012) on line at
http://www.epa.gov/oswer/riskassessment/ragse/pdf/introduction.pdf.

EPA (United States Environmental Protection Agency). 2005. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Risk Assessment Forum. EPA/630/R-03/003F. Available on line at:
http://www.epa.gov/ttn/atw/childrens_supplement_final.pdf

EPA (United States Environmental Protection Agency). 2006. Implementation of the cancer guidelines and accompanying supplemental guidance - Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing risk assessments that include carcinogens described in the Supplemental Guidance as having a mutagenic mode of action. Memo (June 14, 2006) from William H. Farland to Science Policy Council.

EPA (United States Environmental Protection Agency). 2009. An evaluation of the mode of action framework for mutagenic carcinogens: Chromium (VI): SOT. Accessed (12/11/12) on-line at http://cfpub.epa.gov/si/si_public_record_Report.cfm?dirEntryId=203947&CFID=112726327&CFTOKEN=80019090&jsessionid=4e30a1d75a385d1d84b2166a55311eb74573.

EPA (United States Environmental Protection Agency) 2011a. HRS Documentation Record Eighteenmile Creek. September. Available on-line at: http://www.epa.gov/superfund/sites/docrec/pdoc1851.pdf

EPA (United States Environmental Protection Agency). 2011b. Exposure Factors Handbook. National Center for Environmental Assessment. Office of Research and Development EPA/600/R-09/052F Accessed (9/5/2012) on line at http://www.epa.gov/ncea/efh/report.html.

EPA (United States Environmental Protection Agency) 2012a. Eighteen Mile Creek Area of Concern. Available on-line at: http://epa.gov/glnpo/aoc/eighteenmile.html

EPA (United States Environmental Protection Agency). 2012b. Regional Screening Level (RSL) Resident Soil Table April 2012. Accessed (9/13/12) on-line at http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/Generic_Tables/pdf/ressoil_sl_table_run_MAY2012.pdf.

EPA (United States Environmental Protection Agency). 2012c. Region 3 Technical Guidance Manual. Assessing Dermal Contact with Soil; Existing Guidance. Accessed (9/5/2012) on-line at: http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm

EPA (United States Environmental Protection Agency). 2013. Polychlorinated Biphenyls (PCBs). Health Effects of PCBs. Accessed (March 21, 2014) on-line at http://www.epa.gov/wastes/hazard/tsd/pcbs/pubs/effects.htm.

Marshall G, Ferreccio C, Yuan Y, et al. 2007. Fifty-year study of lung and bladder cancer mortality in Chile related to arsenic in drinking water. J Natl Cancer Inst. 99: 920-928.

NRC (National Research Council). 2001. Arsenic in Drinking Water; 2001 Update. Washington, DC: National Academy Press.

NTP (National Toxicology Program). 2005. Report on Carcinogens, Eleventh Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service.

NTP (National Toxicology Program). 2008. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Sodium Dichromate Dihydrate (CAS No. 7789-12-0) in

F344/N Rats and B6C3F1 Mice (Drinking Water Studies). July 2008. Research Triangle Park: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication No. 08-5887.

Smith AH, Marshall G, Yuan Y, et al. 2006. Increased mortality from lung cancer and bronchiectasis in young adults after exposure to arsenic *in utero* and in early childhood. Environ Health Perspect. 114: 1293-6.
Tokar EJ, Diwan BA, Ward JM, et al. 2011. Carcinogenic effects of "whole-life" exposure to inorganic arsenic in CD1 mice. Toxicol Sci. 119: 73-83.

Waalkes MP, Ward JM, Liu, J, and Diwan BA. 2003. Transplacental carcinogenicity of inorganic arsenic in the drinking water: Induction of hepatic, ovarian, pulmonary and adrenal tumors in mice. Toxicol. Appl. Pharmacol. 86:7–17.

Waalkes MP, Liu J, Ward JM, et al. 2006. Urogenital carcinogenesis in female CD1 mice induced by in utero arsenic exposure is exacerbated by postnatal diethylstilbestrol treatment. Cancer Res. 66:1337–1345.

Waalkes MP, Liu J, and Diwan BA. 2007. Transplacental arsenic carcinogenesis in mice. Toxicol Appl Pharmacol. 222: 271-280.

Waalkes MP, Liu J, Germolec DR, et al. 2009. Arsenic exposure in utero exacerbates skin cancer response in adulthood with contemporaneous distortion of tumor stem cell dynamics. Cancer Res. 68: 8278-8285.

WHO (World Health Organization). 2012. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. International Agency for Research on Cancer. Accessed (5/29/12) on-line at: http://monographs.iarc.fr/ENG/Classification/.

AGENCY INFORMATION

New York State Department of Health Authors

Matt Forcucci
Public Health Specialist
Bureau of Environmental Exposure Investigation

Don Miles
Public Health Specialist
Bureau of Environmental Exposure Investigation

Thomas Johnson, Ph.D., Stephen J. Shost, Ph.D., M.P.H and Andrea Candara, M.S
Research Scientists
Bureau of Toxic Substance Assessment

Steve Forand
Research Scientist
Bureau of Environmental and Occupational Epidemiology

ATSDR Technical Project Officer

Teresa C. Foster, MPH
Environmental Health Scientist
Division of Community Health Investigation

ATSDR Regional Representatives

Leah Graziano
Regional Director - Region 2
Division of Community Health Investigation

Elena Vaouli
Lieutenant Commander, US Public Health Service
Regional Representative, Region 2

APPENDIX A

Figures

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

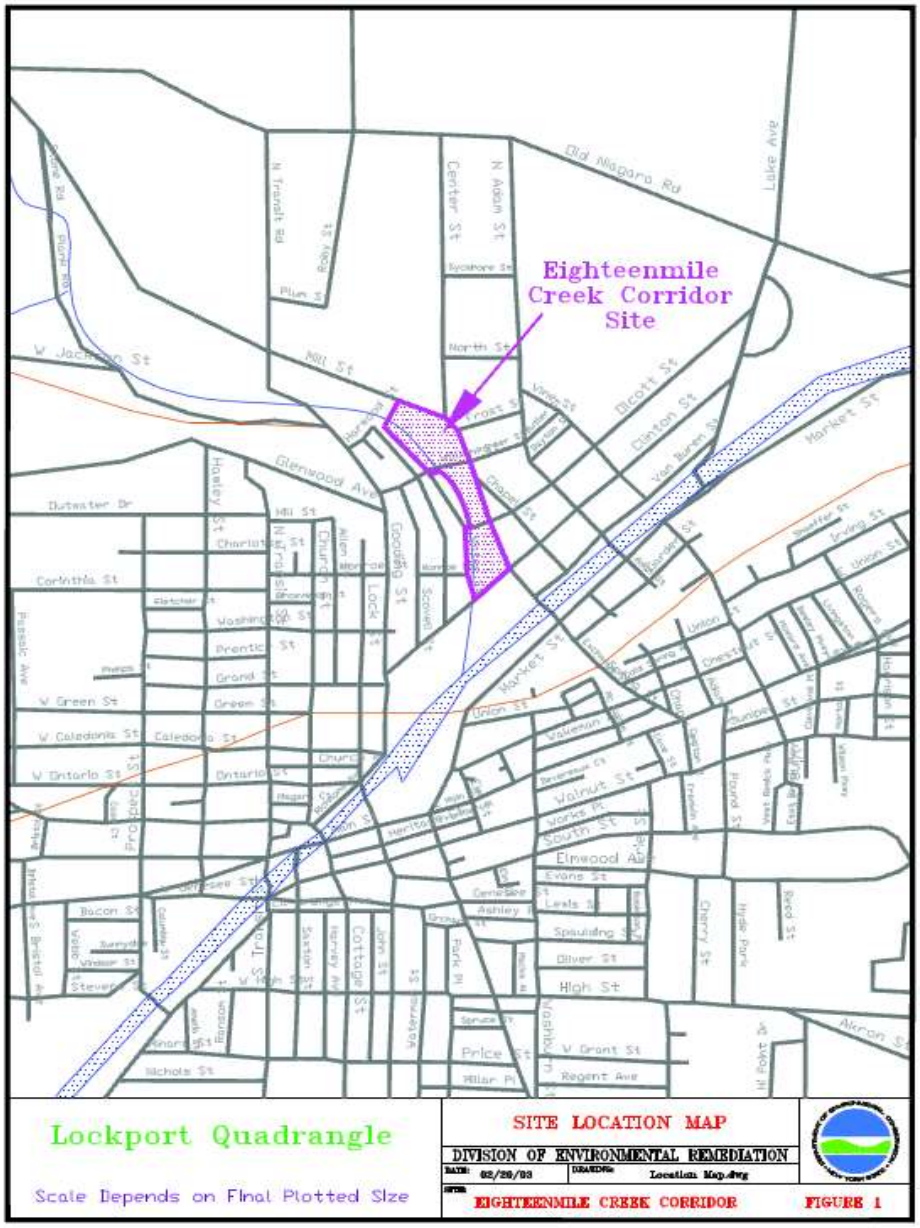


Figure 2. Detail map of the Eighteen Mile Creek Corridor Operable Units, Lockport, New York

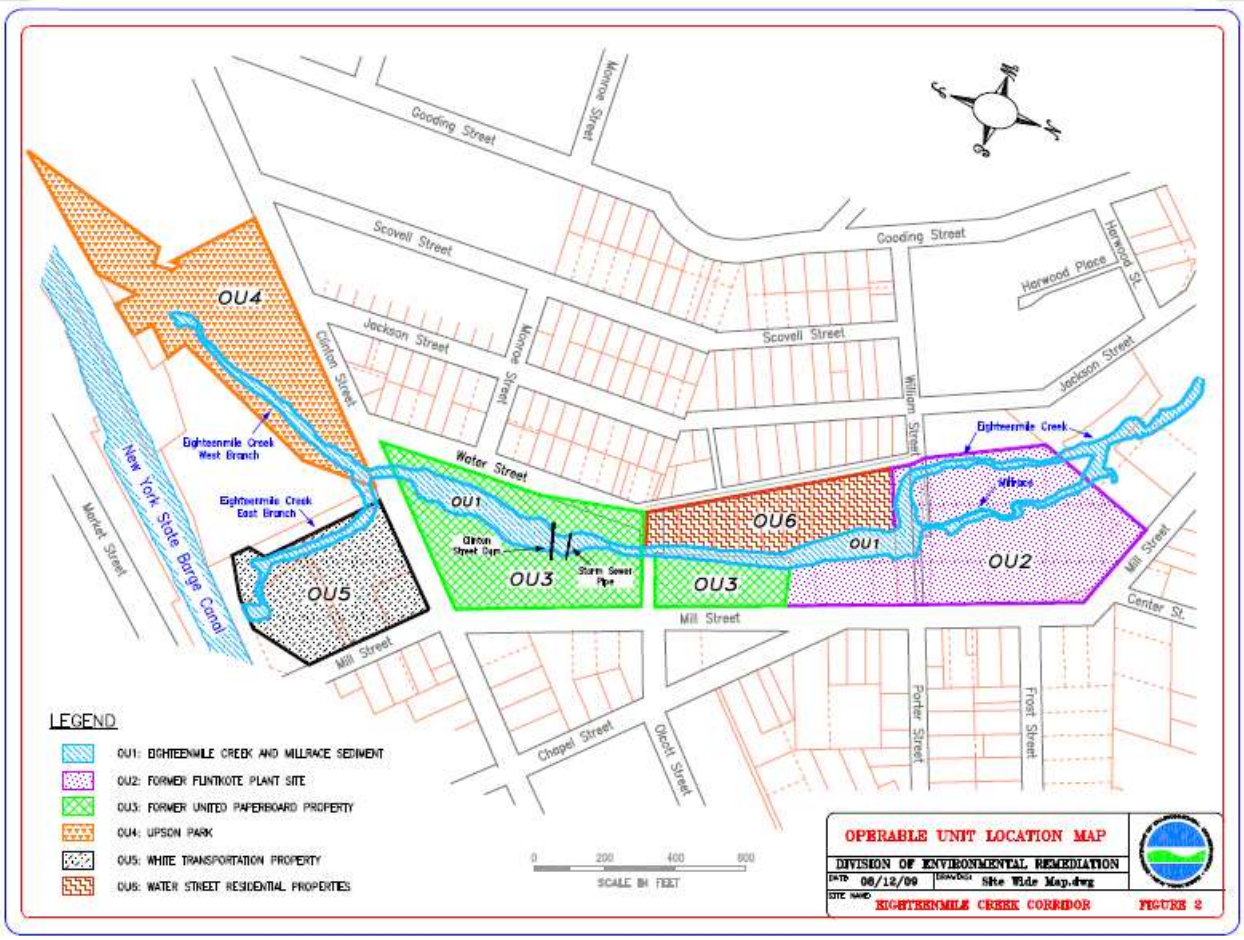
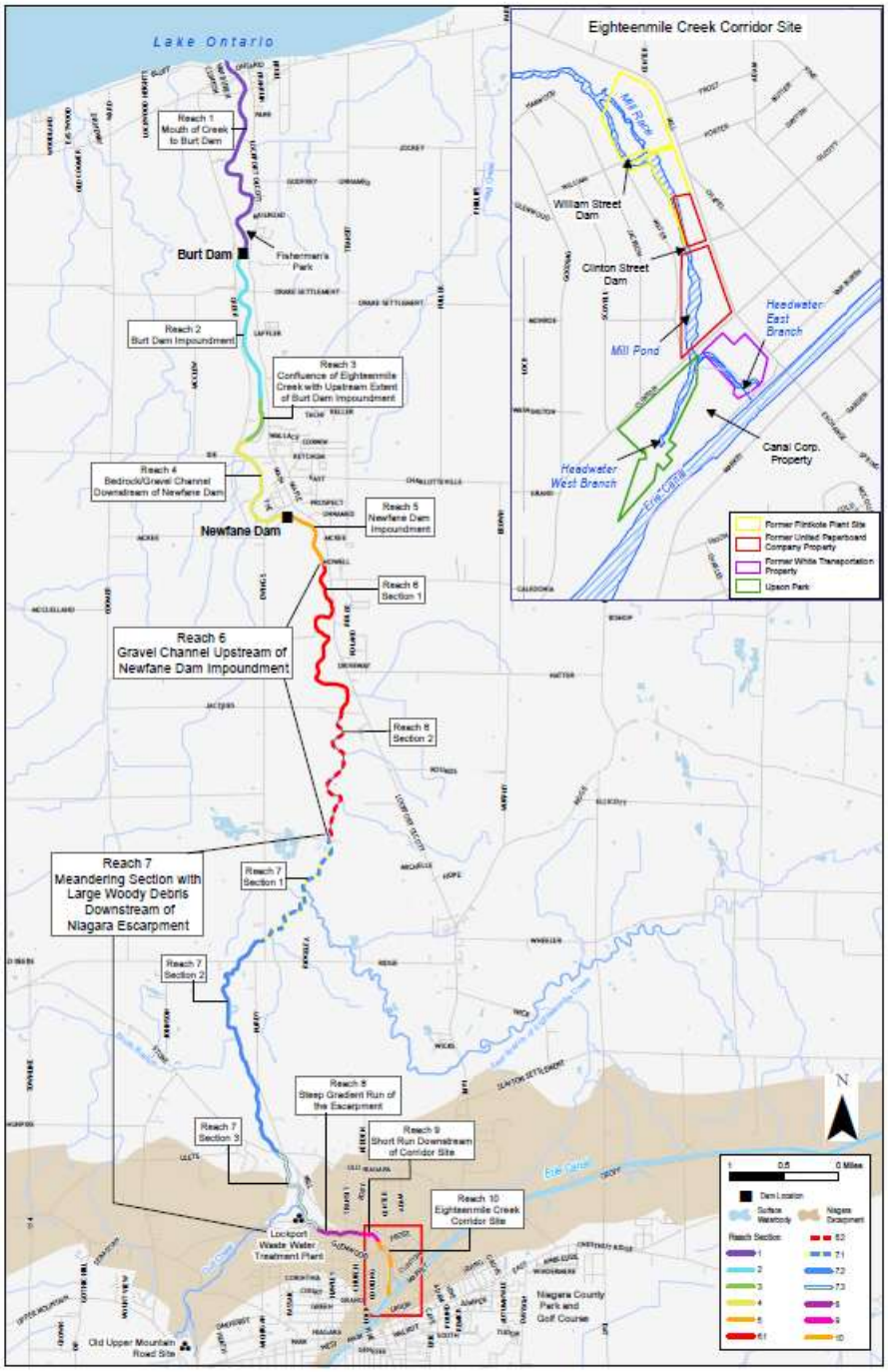


Figure 3. Overview map of the Eighteen Mile Creek from the New York State Barge Canal (Erie Canal) in Lockport to Lake Ontario



APPENDIX B

Tables

Table 1. Contaminants Detected Above Residential Soil Cleanup Objectives at Residential Properties Along the Eighteen Mile Creek.

(all values in milligrams per kilogram soil (mg/kg_s))

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

[1]DEC/DOH, 2006c

[2]ATSDR, 2012

Table 2. Contaminant Levels in Residential Surface Soil and Health Comparison Values Based on Ingestion and Dermal Exposure for Eighteen Mile Creek Contaminants Selected for Further Evaluation*

(All values in milligrams per kilogram of soil (mg/kg_s))

Contaminant	Contaminant Level	Comparison Value			
		Cancer[1]	Basis	Noncancer[2]	Basis
Aroclor 1248	14	0.67	EPA CPF[3]	2.7	ATSDR MRL[4]
Aroclor 1254	13	0.67	EPA CPF[3]	2.7	ATSDR MRL[4]
Aroclor 1260	8	0.67	EPA CPF[3]	2.7	ATSDR MRL[4]
Total PCBs	27	0.67	EPA CPF	2.7	ATSDR MRL[4]
arsenic	66.5	0.29	EPA CPF	13	EPA RfD
chromium	114.5	0.88	CA EPA CPF	44	ATSDR MRL
zinc	2390	--	--	13,300	EPA RfD

*Contaminants exceeded soil cleanup objectives or ATSDR generic comparison values in initial screening (see Table 1).

[1]Cancer comparison values are based on the dose corresponding to a one in one million risk level and calculated for a person who contacts soil for 31 weeks each year (5 days per week for the first 17 years of life and 2 days per week for the next 13 years) and is exposed by incidental ingestion and dermal absorption. For the first year of life, we assumed a child ingests 30 mg_s/day outdoor soil and 15 mg_s/day indoor dust originating as outdoor soil (EPA 2011b); for ages 1 through 5 years, we assumed the child ingests 80 mg_s/day outdoor soil and 40 mg_s/day indoor dust originating as outdoor soil, for a total of 120 mg_s/day soil (DEC/DOH, 2006); for ages 6 through 30 years, we assumed an outdoor soil ingestion rate of 100 mg_s/d (EPA 1989). Body weights are those recommended by the EPA Exposure Factors Handbook (2011b). For the first year of life, we assumed soil contact of a child's face, forearms, hands, lower legs and feet (DEC/DOH, 2006). Beyond the first year of life, surface area recommendations are those recommended by the EPA (2004). Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c). Comparison value = [1 mg/kg_s x 0.000001/cancer potency factor $(mg/kg/day)^{-1}$]/total dose. Total dose is the sum of oral and dermal doses. A sample calculation of oral and dermal doses for residential exposure is found in Table 1, Appendix C. A factor of 5 is used to account for exposure via homegrown fruits and vegetables (DEC/DOH, 2006) except for Aroclors and PCBs, because the elevated PCB levels were located on the creek bank where gardening is unlikely.

[2]Noncancer comparison values are based on the reference dose or similar value and calculated for a 11.4 kg child who contacts sediments 5 days per week, 31 weeks per year. A soil to skin adherence factor of 0.2 mg_s/cm^2-day, a daily soil ingestion rate of 120 mg_s/day, and a surface area value for upper and lower extremities of 2800 cm^2 are assumed (DEC/DOH, 2006). Calculation: Comparison value = 1 mg/kg_s x reference dose (mg/kg/day)/total dose. Total dose is the sum of oral and dermal doses. Oral dose = 1 mg/kg_s x 120 mg_s/day x 1/11.4 kg x 10^{-6} kg_s/mg_s x 5 days/7 days x 31 weeks/52 weeks. Dermal dose = [1 mg/kg_s x 2800 cm^2 x 0.2 mg_s/cm^2-day x dermal absorption factor x 10^{-6} kg_s/mg_s x 5 days/7 days x 31 weeks/52 weeks]/11.4 kg. Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c). A factor of 5 is used to account for exposure via homegrown fruits and vegetables (DEC/DOH, 2006) except for Aroclors and PCBs, because the elevated Aroclor/PCB levels were located on the creek bank where gardening is unlikely.

[3]Based on upper-bound cancer potency factor for high risk and persistence (EPA, 1996b).

[4]The ATSDR value is used to evaluate unspecified mixtures of polychlorinated biphenyls (PCBs) and is based on Aroclor 1254.

ATSDR MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level
CA EPA CPF: California Environmental Protection Agency Cancer Potency Factor
EPA CPF: United States Environmental Protection Agency Integrated Risk Information System
EPA RfD: United States Environmental Protection Agency Integrated Risk Information System Reference Dose

Table 3. Contaminants Detected Above Restricted Residential Soil Cleanup Objectives at Operable Units 2, 3, 4 and 5 along the Eighteen Mile Creek.

(all values in milligrams per kilogram soil (mg/kg_s))

Contaminant	Highest Detected Level	NYS Restricted Residential Soil Cleanup Objective[1]	ATSDR Comparison Value[2]
Operable Unit 2 (Flinkote Plant Site)			
benz(a)anthracene	110	1	---
benzo(a)pyrene	20	1	0.096
benzo(b)fluoranthene	160	1	---
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	31*	20
arsenic	59.6	16	0.47
barium	2440	400	10,000
chromium	186	110	50
lead	7610	400	---
mercury	10.8	0.81	15
nickel	549	310	1000
zinc	21,900	10,000	15,000
Operable Unit 3 (Former United Paperboard Company)			
benz(a)anthracene	26	1	---
benzo(a)pyrene	20	1	0.096
benzo(b)fluoranthene	26	1	---
benzo(k)fluoranthene	7.3	3.9	---
chrysene	23	3.9	---
indeno(1,2,3-cd)pyrene	11	0.5	---
Total PCBs	4.3	1	0.35
arsenic	66	16	0.47
lead	3600	400	---

Operable Unit 4 (Upson Park)			
benz(a)anthracene	4.4	1	---
benzo(a)pyrene	2.3	1	0.096
benzo(b)fluoranthene	3.5	1	---
indeno(1,2,3-cd)pyrene	1.3	0.5	---
Total PCBs	23	1	0.35
arsenic	63.2	16	0.47
barium	2360	400	10,000
cadmium	27.4	4.3	5
chromium	162	110	50
lead	3480	400	---
mercury	10.8	0.81	15
Operable Unit 5 (White Transportation Property)			
benz(a)anthracene	1.2	1	---
benzo(a)pyrene	1.1	1	0.096
benzo(b)fluoranthene	2	1	---
indeno(1,2,3-cd)pyrene	0.51	0.5	---
arsenic	30.3	16	0.47
cadmium	8.3	4.3	5
chromium	411	110	50
lead	3750	400	--

[1]DEC/DOH, 2006c

[2]ATSDR, 2012

*No SCO available. Value is US EPA Regional Screening Level for soil (EPA, 2012b).

Table 4. Contaminant Levels in Surface Soil at Operable Units 2, 3 and 5 and Health Comparison Values Based on Ingestion and Dermal Exposure for Eighteen Mile Creek Contaminants Selected for Further Evaluation*

(All values in milligrams per kilogram of soil (mg/kg_s))

Contaminant	Contaminant Level**	Comparison Value			
		Cancer[1]	Basis	Noncancer[2]	Basis
benz(a)anthracene	110	7.5	DEC CPF[3]	2490	CA EPA RfD[4]
benzo(a)pyrene	20	0.8	DEC CPF	2490	CA EPA RfD
benzo(b)fluoranthene	160	7.5	DEC CPF[3]	2490	CA EPA RfD[4]
benzo(k)fluoranthene	200	75	DEC CPF[3]	2490	CA EPA RfD[4]
chrysene	92	75	DEC CPF[3]	2490	CA EPA RfD[4]
dibenz(a,h)anthracene	16	0.8	DEC CPF[3]	2490	CA EPA RfD[4]
indeno(1,2,3-c,d)pyrene	11	7.5	DEC CPF[3]	2490	CA EPA RfD[4]
Aroclor 1254	4.6	8.3	EPA CPF[5]	29	ATSDR MRL[6]
Total PCBs	4.3	8.3	EPA CPF[5]	29	ATSDR MRL[6]
antimony	149	--	--	856	EPA RfD
arsenic	66	15	EPA CPF	596	EPA RfD
barium	2440	--	--	428,000	EPA RfD
cadmium	8.3	--	--	1550	DEC RfD
chromium	411	50	CA EPA CPF	2140	ATSDR MRL
mercury	10.8	--	--	342	CA EPA RfD
nickel	549	--	--	42,800	EPA RfD
zinc	21,900	--	--	642,000	EPA RfD

*Contaminants exceeded soil cleanup objectives or ATSDR generic comparison values in initial screening (see Table 3).

**The highest detected contaminant level from Operable Units 2, 3 and 5 are shown.

[1]Cancer comparison values are based on the dose corresponding to a one in one million risk level and calculated for a person who contacts soil for 2 days per week for 6 months each year for 11 years (ages 10 to 21), and is exposed by incidental ingestion and dermal absorption. We assumed an outdoor soil ingestion rate of 100 mg_s/d (EPA 1989). Body weights are those recommended by the EPA Exposure Factors Handbook (2011b) and surface area recommendations are those recommended by the EPA (2004). Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c). Comparison value = [1 mg/kg_s x 0.000001/cancer potency factor $(mg/kg/day)^{-1}$]/total dose. Total dose is the sum of oral and dermal doses. A sample calculation of oral and dermal doses for nonresidential soil exposure is found in Table 2, Appendix C.

[2]Noncancer comparison values are based on the reference dose or similar value and calculated for a 31.8 kg person who contacts sediments 2 days per week, 6 months per year. A soil to skin adherence factor of 0.07 mg_s/cm^2-day, a daily soil ingestion rate of 100 mg_s/day, and a surface area value for upper and lower extremities of 5700 cm^2 are assumed (EPA 2004, 2011b). Calculation: Comparison value = 1 mg/kg_s x reference dose (mg/kg/day)/total dose. Total dose is the sum of oral and dermal doses. Oral dose = 1 mg/kg_s x 100 mg_s/day x 1/31.8 kg x 10^{-6} kg_s/mg_s x 2 days/7 days x 6 month/12 months. Dermal dose = [1 mg/kg_s x 5700 cm^2 x 0.07 mg_s/cm^2-day x dermal absorption factor x 10^{-6} kg_s/mg_s x 2 days/7 days x 6 months/12 months]/31.8 kg. Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c).

[3]Relative potency factors were applied to the carcinogenic PAHs as described in DEC/DOH (2006).

[4]The reference dose for benzo(a)pyrene is used as a surrogate in the absence of a chemical-specific value.

[5]Based on upper-bound cancer potency factor for high risk and persistence (EPA, 1996b).

[6]The ATSDR value is used to evaluate unspecified mixtures of polychlorinated biphenyls (PCBs) and is based on Aroclor 1254.

ATSDR MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level
CA EPA CPF: California Environmental Protection Agency Cancer Potency Factor
CA EPA RfD: California Environmental Protection Agency Reference Dose
DEC CPF: New York State Department of Environmental Conservation Cancer Potency Factor
DEC RfD: New York State Department of Environmental Conservation Reference Dose
EPA CPF: United States Environmental Protection Agency Integrated Risk Information System
EPA RfD: United States Environmental Protection Agency Integrated Risk Information System Reference Dose

Table 5. Contaminant Levels in Surface Soil at Upson Park (DEC Operable Unit 4) and Health Comparison Values Based on Ingestion and Dermal Exposure for Eighteen Mile Creek Contaminants Selected for Further Evaluation*

(All values in milligrams per kilogram of soil (mg/kg_s))

Contaminant	Contaminant Level**	Comparison Value			
		Cancer[1]	Basis	Noncancer[2]	Basis
benz(a)anthracene	4.4	1.4	DEC CPF[3]	236	CA EPA RfD[4]
benzo(a)pyrene	2.3	0.14	DEC CPF	236	CA EPA RfD
benzo(b)fluoranthene	3.5	1.4	DEC CPF[3]	236	CA EPA RfD[4]
indeno(1,2,3-c,d)pyrene	1.3	1.4	DEC CPF[3]	236	CA EPA RfD[4]
Total PCBs	23	0.67	EPA CPF[5]	2.7	ATSDR MRL[6]
arsenic	63.2	1.3	EPA CPF	59	EPA RfD
barium	2360	--	--	42,600	EPA RfD
cadmium	27.4	--	--	155	DEC RfD
chromium	162	4.3	CA EPA CPF	213	ATSDR MRL
mercury	10.8	--	--	34	CA EPA RfD
zinc	6564	--	--	63,900	EPA RfD

*Contaminants exceeded soil cleanup objectives or ATSDR generic comparison values in initial screening (see Table 3).

**The highest detected contaminant level from Operable Unit 4 is shown.

[1]Cancer comparison values are based on the dose corresponding to a one in one million risk level and calculated for a person who contacts soil for 31 weeks each year (5 days per week for the first 17 years of life and 2 days per week for the next 13 years) and is exposed by incidental ingestion and dermal absorption. For the first year of life, we assumed a child ingests 30 mg_s/d outdoor soil and 15 mg_s/d indoor dust originating as outdoor soil (EPA 2011b); for ages 1 through 5 years, we assumed the child ingests 80 mg_s/d outdoor soil and 40 mg_s/d indoor dust originating as outdoor soil, for a total of 120 mg_s/d soil (DEC/DOH, 2006); for ages 6 through 30 years, we assumed an outdoor soil ingestion rate of 100 mg_s/d (EPA 1989). Body weights are those recommended by the EPA Exposure Factors Handbook (2011b). For the first year of life, we assumed soil contact of a child's face, forearms, hands, lower legs and feet (DEC/DOH, 2006). Beyond the first year of life, surface area recommendations are those recommended by the EPA (2004). Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c). Comparison value = [1 mg/kg_s x 0.000001/cancer potency factor $(mg/kg/day)^{-1}$]/total dose. Total dose is the sum of oral and dermal doses. A sample calculation of oral and dermal doses for residential exposure is found in Table 1, Appendix C.

[2]Noncancer comparison values are based on the reference dose or similar value and calculated for a 11.4 kg child who contacts sediments 5 days per week, 31 weeks per year. A soil to skin adherence factor of 0.2 mg/cm^2-day, a daily soil ingestion rate of 120 mg_s/day, and a surface area value for upper and lower extremities of 2800 cm^2 are assumed (DEC/DOH, 2006). Calculation: Comparison value = 1 mg/kg_s x reference dose (mg/kg/day)/total dose. Total dose is the sum of oral and dermal doses. Oral dose = 1 mg/kg_s x 120 mg_s/day x 1/11.4 kg x 10^{-6} kg_s/mg_s x 5 days/7 days x 31 weeks/52 weeks. Dermal dose = [1 mg_s/kg_s x 2800 cm^2 x 0.2 mg_s/cm^2-day x dermal absorption factor x 10^{-6} kg_s/mg_s x 5 days/7 days x 31 weeks/52 weeks]/11.4 kg. Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c).

[3]Relative potency factors were applied to the carcinogenic PAHs as described in DEC/DOH (2006).

[4]The reference dose for benzo(a)pyrene is used as a surrogate in the absence of a chemical-specific value.

[5]Based on upper-bound cancer potency factor for high risk and persistence (EPA, 1996b).

[6]The ATSDR value is used to evaluate unspecified mixtures of polychlorinated biphenyls (PCBs) and is based on Aroclor 1254.

ATSDR MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level
CA EPA CPF: California Environmental Protection Agency Cancer Potency Factor
DEC CPF: New York State Department of Environmental Conservation Cancer Potency Factor
DEC RfD: New York State Department of Environmental Conservation Reference Dose

EPA CPF: United States Environmental Protection Agency Integrated Risk Information System
EPA RfD: United States Environmental Protection Agency Integrated Risk Information System Reference Dose

Table 6. Cancer and Noncancer Risk Descriptors for Surface Soil Contaminants at Operable Units 2, 3 and 5 along the Eighteen Mile Creek.

Contaminant	Highest Detected Level (mg/kg_s)	Cancer Risk Qualitative Descriptor	Noncancer Risk Qualitative Descriptor
Flintkote Property (Operable Unit 2)			
benz[a]anthracene	110	low*	minimal
benzo[a]pyrene	20	low*	minimal
benzo[b]fluoranthene	160	low*	minimal
benzo[k]fluoranthene	200	low*	minimal
chrysene	92	low*	minimal
dibenz[a,h]anthracene	16	low*	minimal
arsenic	59.6	low	minimal
chromium	186	low	minimal
Former United Paperboard Property (Operable Unit 3)			
benz[a]anthracene	26	low	minimal
benzo[a]pyrene	20	low	minimal
benzo[b]fluoranthene	26	low	minimal
indeno[1,2,3-cd]pyrene	11	low	minimal
arsenic	66	low	minimal
chromium	73.7	low	minimal
White Transportation Property (Operable Unit 5)			
benzo[a]pyrene	1.1	low	minimal
arsenic	30.3	low	minimal
chromium	411	low	minimal

*The additive cancer risk estimate for carcinogenic PAHs is 9 in 100,000 (0.9 in 10,000), which is just below the cancer risk level above which actions to reduce exposure are typically taken (1 in 10,000).

mg/kg_s = milligrams per kilogram soil

Table 7. Cancer and Noncancer Risk Descriptors for Surface Soil Contaminants at Upson Park (Operable Unit 4) along the Eighteen Mile Creek.

Contaminant	Highest Detected Level (mg/kg_s)	Cancer Risk Qualitative Descriptor	Noncancer Risk Qualitative Descriptor
benz(a)anthracene	4.4	low	minimal
benzo(a)pyrene	2.3	low	minimal
benzo(b)fluoranthene	3.5	low	minimal
Total PCBs	23	low	moderate
arsenic	63.2	low	low
chromium	162	low	minimal

mg/kg_s = milligram per kilogram soil

Table 8. Contaminant Levels in Creek Sediments and Health Comparison Values Based on Ingestion and Dermal Exposure for Eighteen Mile Creek Contaminants Selected for Further Evaluation*

(All values in milligrams per kilogram of soil (mg/kg_s))

Contaminant	Contaminant Level**	Comparison Value			
		Cancer[1]	Basis	Noncancer[2]	Basis
benzo(a)pyrene	34	1.1	DEC CPF	1760	CA EPA RfD
Aroclor 1242	46	5.1	EPA CPF[3]	20	ATSDR MRL[4]
Aroclor 1248	180	5.1	EPA CPF[3]	20	ATSDR MRL[4]
Aroclor 1254	57	5.1	EPA CPF[3]	20	ATSDR MRL
Aroclor 1260	42	5.1	EPA CPF[3]	20	ATSDR MRL[4]
Total PCBs	201	5.1	EPA CPF[3]	20	ATSDR MRL[4]

*Contaminants exceeded soil cleanup objectives or ATSDR generic comparison values in initial screening [see section entitled "Corridor Creek Sediments/Chemicals Selected for Further Evaluation (DEC OU 2 through 6)].

**The highest detected contaminant level from corridor and downstream creek sediments is shown.

[1]Cancer comparison values are based on the dose corresponding to a one in one million risk level and calculated for a person who contacts sediment 2 days per week for 4 months each year and is exposed by incidental ingestion and dermal absorption from age 3 to 18. For ages 3 through 5 years, we assumed the child ingests 120 mg_s/d sediment (DEC/DOH, 2006); for ages 6 through 18 years, we assumed a sediment ingestion rate of 100 mg_s/d (EPA 1989). Body weights are those recommended by the EPA Exposure Factors Handbook (2011b). We Surface area recommendations are those recommended by the EPA (2004). Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c). Comparison value = [1 mg/kg_s x 0.000001/cancer potency factor $(mg/kg/day)^{-1}$]/total dose. Total dose is the sum of oral and dermal doses. A sample calculation of oral and dermal doses for sediment exposure is found in Table 3, Appendix C.

[2]Noncancer comparison values are based on the reference dose or similar value and calculated for a 18.6 kg child who contacts sediments 2 days per week for 4 months each year. A soil to skin adherence factor of 0.2 mg_s/cm^2-day, a daily soil ingestion rate of 120 mg_s/day, and a surface area value for upper and lower extremities of 2800 cm^2 are assumed (DEC/DOH, 2006). Calculation: Comparison value = 1 mg/kg_s x reference dose (mg/kg/day)/total dose. Total dose is the sum of oral and dermal doses. Oral dose = 1 mg/kg_s x 120 mg_s/day x 1/18.6 kg x 10^{-6} kg_s/mg_s x 2 days/7 days x 17 weeks/52 weeks. Dermal dose = [1 mg/kg_s x 2800 cm^2 x 0.2 mg_s/cm^2-day x dermal absorption factor x 10^{-6} kg_s/mg_s x 2 days/7 days x 17 weeks/52 weeks]/18.6 kg. Dermal absorption factors are from Chapter 3 (Exhibit 3-4) of EPA (2004) or from EPA (2012c).

[3]Based on upper-bound cancer potency factor for high risk and persistence (EPA, 1996b).

[4]The ATSDR value is used to evaluate unspecified mixtures of polychlorinated biphenyls (PCBs) and is based on Aroclor 1254.

ATSDR MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level
CA EPA RfD: California Environmental Protection Agency Reference Dose
DEC CPF: New York State Department of Environmental Conservation Cancer Potency Factor
EPA CPF: United States Environmental Protection Agency Integrated Risk Information System

Table 9. Cancer and Noncancer Risk Descriptors for Corridor and Downstream Sediment Contaminants in the Eighteen Mile Creek.

Contaminant	Highest Detected Level (mg/kg_s)	Cancer Risk Qualitative Descriptor	Noncancer Risk Qualitative Descriptor
Corridor (runs through Operable Units 2 through 6)			
benzo[a]pyrene	34	low	minimal
Aroclor 1242	46	low	low
Aroclor 1248	180	low	moderate
Aroclor 1254	57	low	low
Total PCBs	201	low	moderate
Downstream Reaches			
Aroclor 1248	41	low	low
Aroclor 1254	55	low	low
Aroclor 1260	42	low	low

mg/kg_s = milligram per kilogram soil

Appendix C

Sample Spreadsheets for Calculation of Oral and Dermal Doses

Table 1. Calculation of Contaminant Oral and Dermal Doses from a Soil Concentration of 1 mg/kg$_s$ for Cancer Risk Evaluation of Residential Soil Contaminant Exposure

PCB Dose from Soil Ingestion

Yr	Range	*C* (mg/kg)	*IR* (mg/d)	*CF* (kg/mg)	*BW* (kg)	d/wk	wk/y	*E* *(d/d)*	365 d/y Ing Dose (mg/kg/d)	*E*-weighted Ing Dose (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**
									Avg PCB Dose	**1.68E-06** **mg/kg/d**

Yr	Range	C (mg/kg)	IR (mg/d)	CF (kg/mg)	BW (kg)	d/wk	wk/y	*E* *(d/d)*	365 d/y Ing Dose (mg/kg/d)	*E*-weighted Ing Dose (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**

PCB Dose from Dermal Absorption

Yr	Range	*C* (ppm)	*SA* (cm2)	*AF* (mg/cm2-d)	DABS	CF (kg/mg)	*BW* (kg)	*EF1* d/wk	*EF2* wk/y	*E* (d/d)	365 d/y Derm Dose (mg/kg/d)	*E*-weighted Derm Dose (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**

Avg PCB Dose **1.13E-06** **mg/kg/d**

Yr	Range	*C* (ppm)	*SA* (cm2)	*AF* (mg/cm2-d)	DABS	CF (kg/mg)	*BW* (kg)	*EF1* d/wk	*EF2* wk/y	*E* (d/d)	365 d/y Derm Dose (mg/kg/d)	*E*-weighted Derm Dose (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**
28	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**
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**
30	29 to <30	1	5,700	0.07	0.140	1.0E-06	80.0	0.286	0.596	0.170	6.983E-07	**1.189E-07**

Avg PCB Dose **1.232E-07** **mg/kg/d**

Age Period	ED/Lifetime	Avg PCB Dose	Dose Weight (mg/kg/day over 70 years)
0 to 17	0.2429	1.13E-06	2.74E-07
18 to 30	0.1857	1.23E-07	2.29E-08
30 years			**2.97E-07**

Sample PCB Cancer Risk Calculation (using results from above residential spreadsheet)

Total Dose from 1 mg/kg_s

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/kg_s 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/kg_s PCBs

Cancer Risk = Total Dose x Cancer Potency Factor = 2.01E-5 mg/kg/day x 2.0/mg/kg/day = 4.0E-5 (low)

Sample PCB Noncancer Hazard Quotient Calculation

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 cm^2 x 0.2 mg_s/cm^2-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, 2006c). 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 of 1 mg/kg for Cancer Risk Evaluation of Nonresidential Soil Contaminant Exposure

Benzo(a)pyrene Dose from Soil Ingestion

Yr	Range	C (mg/kg)	IR (mg/d)	CF (kg/mg)	BW (kg)	d/wk	wk/y	E (d/d)	365 d/y Ing Dose (mg/kg/d)	*E*-weighted Ing Dose (mg/kg/d)	Age-Dependent Adjustment Factors*	Adjusted Time-Weighted Dose** (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	
											Avg B(a)P Dose	5.56E-07	mg/kg/day

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

Benzo(a)pyrene Dose from Dermal Absorption

Yr	Range	C (ppm)	SA (cm2)	AF (mg/cm2-d)	DABS	CF (kg/mg)	BW (kg)	EF1 d/wk	EF2 wk/y	E (d/d)	365 d/y Derm Dose (mg/kg/d)	*E*-weighted Derm Dose (mg/kg/d)	Age-Dependent Adjustment Factors*	Adjusted Time-Weighted Dose** (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	
													Avg B(a)P Dose	**2.88E-07**	mg/kg/day

Age Period	ED/Lifetime	Adjusted Dose (mg/kg/day over 70 years)*
11 years (10 -21 years of age)	0.1571	**4.53E-08**

*Age-Dependent Adjustment Factors (ADAFs) are recommended by US EPA (2005) for chemicals with a mutagenic mode-of-action (MOA) for carcinogenicity.

**Adjusted Time-Weighted Dose is the E-weighted Dose multiplied by the Age Dependent Adjustment Factor.

Table 3. Calculation of Contaminant Oral and Dermal Doses from a Sediment Concentration of 1 mg/kg for Cancer Risk Evaluation of Sediment Contaminant Exposure

PCB Dose From Soil Ingestion

Yr	Range	*C* (mg/kg)	*IR* (mg/d)	*CF* (kg/mg)	*BW* (kg)	d/wk	wk/y	*E* *(d/d)*	365 d/y Ing Dose (mg/kg/d)	*E*-weighted Ing Dose (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**	
									Avg PCB Dose	**2.84E-07**	mg/kg/day

Age Period	ED/Lifetime	Avg Dose	Dose (mg/kg/day over 70 years)
15 years (3 to 18 years of age)	0.2143	2.84E-07	**6.08E-08**

PCB Dose from Dermal Absorption

Yr	Range	*C* (ppm)	*SA* (cm2)	*AF* (mg/cm2-d)	DABS	CF (kg/mg)	*BW* (kg)	*EF1* d/wk	*EF2* wk/y	*E* (d/d)	365 d/y Derm Dose (mg/kg/d)	*E*-weighted Derm Dose (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**	
											Avg PCB Dose	**1.74E-07**	**mg/kg/day**

Age Period	ED/Lifetime	avg Dose	Dose (mg/kg/day over 70 years)
15 years (3 to 18 years of age)	0.2143	1.74E-07	**3.72E-08**

APPENDIX D

Sample Calculations for Comparison Values

Noncancer Comparison Value

Contaminant: Chromium
Location: Upson Park (see Table 5, Footnote 2 for complete description of exposure assumptions).

Calculation of Oral Dose at Contaminant Concentration of 1 mg/kg$_s$:

1 mg/kg$_s$ x 120 mg$_s$/day x 1/11.4 kg x 10^{-6} kg$_s$/mg$_s$ x 5 days/7 days x 31 weeks/52 weeks =

4.48E-6 mg/kg/day

Calculation of Dermal Dose at Contaminant Concentration of 1 mg/kg$_s$:

[1 mg/kg$_s$ x 2800 cm^2 x 0.2 mg$_s$/cm^2-day x 0.01 x 10^{-6} kg$_s$/mg$_s$ x 5 days/7 days x 31 weeks/52 weeks]/11.4 kg =

2.09E-7 mg/kg/day

Calculation of Total Dose at Contaminant Concentration of 1 mg/kg$_s$:

4.48E-6 mg/kg/day + 2.09E-7 mg/kg/day = 4.69E-6 mg/kg/day

Calculation of Comparison value:

[1 mg/kg$_s$ x 1E-3 mg/kg/day]/4.69E-6 mg/kg/day = 213 mg/kg$_s$

Cancer Comparison Value

Contaminant: PCBs
Location: Upson Park (see Table 5, Footnote 1 for complete description of exposure assumptions).
Note: The calculation of the total dose from oral and dermal exposure considers the entire exposure period (infant to adult), and that body weights, exposure frequency, body surface area and ingestion rates change over the period of time the exposure is evaluated (i.e., the first 30 years of life). Table 1, Appendix C shows the calculation of the oral and dermal dose from a soil concentration of 1 mg/kg$_s$ for each year of the exposure using assumptions specific to that year.

Calculation of Total Dose at Contaminant Concentration of 1 mg/kg$_s$ (from Table 1, Appendix C):

4.48E-7 mg/kg/day + 2.97E-7 mg/kg/day = 7.45E-7 mg/kg/day

Calculation of Comparison value:

[(1 mg/kg$_s$ x 0.000001)/ 2 (mg/kg/day)$^{-1}$]/ 7.45E-7 mg/kg/day = 0.67 mg/kg$_s$

APPENDIX E

DOH Procedure for Evaluating Cancer Risks
for Contaminants of Concern

To evaluate the cancer risks from contaminants at the Eighteen Mile Creek site, the DOH used site-specific information on exposure levels for the contaminants of concern and applied cancer potency factors derived for the contaminants by authoritative health agencies such as the EPA or, in some cases, by the DOH. The cancer potency factor is a numerical estimate of the carcinogenic strength (potency) of a chemical. The DOH then used the following qualitative ranking of cancer risk estimates (developed by the DOH), to rank the risk from very low to very high. For example, if the qualitative descriptor was low, then the excess lifetime cancer risk from that exposure is in the range of greater than one per million to less than one per ten thousand. Other qualitative descriptors are listed below.

Qualitative Descriptors for Excess Lifetime Cancer Risk

Excess Lifetime Cancer Risk	Qualitative Descriptor
Equal to or less than one per million	Very low
Greater than one per million to less than one per ten thousand	Low
One per ten thousand to less than one per thousand	Moderate
One per thousand to less than one per ten	High
Equal to or greater than one per ten	Very high

An estimated increased excess lifetime cancer risk is not a specific estimate of expected cancers. Rather, it is a plausible estimate of the probability that a person may develop cancer sometime in his or her lifetime following exposure to that contaminant.

There is insufficient knowledge about how chemicals cause cancer to decide if there exists a level of exposure to a cancer-causing chemical below which there is no risk of getting cancer, namely, a threshold level. Therefore, every exposure (no matter how low) to a cancer-causing chemical is assumed to have some increased risk. As exposure to a carcinogen decreases, the chance of developing cancer decreases, but each exposure is accompanied by some increased risk.

There is general consensus among the scientific and regulatory communities on what level of estimated excess cancer risk is acceptable. An increased lifetime cancer risk of one in one million or less is generally not considered a significant public health concern.

APPENDIX F

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.