# Site Characterization Report Addendum No. 1 Baseline Health Risk Assessment and Baseline Environmental Risk Assessment

Cumberland Bay Sludge Bed - Wilcox Dock Work Assignment No. D002520-32

Prepared for:



SUPERFUND STANDBY PROGRAM
New York State
Department of Environmental Conservation
50 Wolf Road
Albany, New York 12233

Prepared by:

Rust Environment & Infrastructure
12 Metro Park Road
Albany, New York 12205

## Site Characterization Report Addendum No. 1 Baseline Health Risk Assessment and Baseline Environmental Risk Assessment

Cumberland Bay Sludge Bed - Wilcox Dock

Cumberland Bay
Work Assignment No. D002520-32

Prepared for:

State Superfund Standby Program

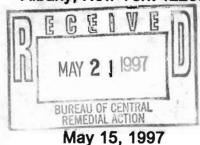
New York State

Department of Environmental Conservation
50 Wolf Road

Albany, New York 12233-7010

Submitted By:

Rust Environment & Infrastructure
12 Metro Park Road
Albany, New York 12205



#### TABLE OF CONTENTS

Char	oter			Page				
1.0	INTR	ODUCT	TON					
1.0	1.1		1-1					
	1.2		•					
	1.2	Ditte 11	istory	1-2				
2.0	SITE	INVEST	NS 2-1					
	2.1	Site In	Investigations Prior to the SC					
	2.2	Site C	haracterization Report 2-					
	2.3	Supple	emental Site Investigation 2-3					
3.0	BASI	EI INE L	IFAI TH	RISK ASSESSMENT				
3.0	3.1							
	3.1	3.1.1	w 3-1					
		3.1.2		3-1				
		3.1.2		and Organization of the HRA				
	3.2			eation				
	3.3			yay Analysis				
	3.5	3.3.1		erization of the Exposure Setting				
		3.3.2		l Human Exposure Pathways				
		3.3.2	3.3.2.1	Air				
			3.3.2.1	Sludge Bed				
			3.3.2.3	Wood Chips and Other Debris on Beaches				
			3.3.2.4					
			3.3.2.4	Surface Water				
				Fish Ingestion				
		222	3.3.2.6					
		3.3.3	cation of Exposure					
			3.3.3.1	Recreational Exposure to Surface Water and Sludge 3-7 3.3.3.1.1 Surface Water Ingestion				
				3.3.3.1.1 Surface Water Ingestion				
				3.3.3.1.3 Sludge Ingestion				
			3.3.3.2	Recreational Ingestion of Fish from Cumberland Bay 3-14				
	3.4	Tovio	sment					
	3.4	3.4.1	•	Toxicity Information				
	. 1	3.4.1	3.4.1.1	Polychlorinated Biphenyls				
		3.4.2		Information Summary				
	3.5			zation				
	3.3	3.5.1		cinogenic Risks 3-19				
		3.5.2		genic Risks				
		3.5.3		in Sludge Bed Area Near Wilcox Dock				
			_					
		3.5.4		ing in Beach Areas North of the Sludge Bed				
		3.5.5	Recreati	ional Use of Beach Areas North of the Sludge Bed 3-21				

		3.5.6 Recreational Ingestion of Cumberland Bay Fish						
		3.5.7 Uncertainties in the Health Risk Assessment						
	3.6	Summary and Conclusions						
4.0	FISE	AND WILDLIFE IMPACT ANALYSIS						
	4.1	eline Assessment						
	7.1							
		4.1.2 Wetlands Within One-Half and Two Miles of the Site 4-4						
		4.1.3 Streams and Related Surface Water Bodies Within One-Half Mile and						
		Two Miles of the Site						
	4.2	Resource Characterization Within One-Half and Two Miles of the Site 4-5						
		4.2.1 Endangered, Threatened or Special Concern Fish and Wildlife or						
		Plant Species or Significant Habitats						
		4.2.2 Fish and Wildlife Species Potentially Using Habitats Within a						
		One-Half Mile Radius of the Site						
		4.2.3 General Habitat Quality Within One-Half Mile of the Site 4-7						
	4.3	Applicable Fish and Wildlife Regulatory Criteria						
	4.4	Contaminant-Specific Impact Analysis 4-9						
		4.4.1 Pathway Analysis						
		4.4.2 Criteria-Specific Analysis 4-9						
		4.4.3 Toxicity Analysis						
		4.4.3.1 Mink Toxicity Analysis						
		4.4.3.1 Osprey Toxicity Analysis						
	4.5							
	4.3	Summary and Conclusions 4-16						
5.0	/ELOPMENT OF REMEDIAL ACTION OBJECTIVES 5-1							
		LIST OF TABLES						
Table								
3-1	1 1	Inclusion/Exclusion Analysis of Potential Exposure Pathways/Routes for the Sludge						
	]	Bed-Wilcox Dock Health Risk Assessment						
3-2		Summary of PCBs Concentrations Detected Near Wilcox Dock						
3-3		Exposure From Surface Water and Sludge Contact While Wading in Sludge Bed Area						
٠.		ear Wilcox Dock: Area Resident Exposure - Adolescent						
3-4		•						
3-4		Exposure From Surface Water and Sludge Contact While Wading in Sludge Bed Area Near Wilcox Dock: Area Resident Exposure - Adult Exposure From Sludge and Surface Water Contact While Wading in Sludge Bed Area						
3-								
		Near Wilcox Dock: Area Resident Exposure - Summary						
3-0	6 I	Exposure From Surface Water and Sludge While Swimming in Beach Area North of						
	6	Sludge Bed - Young Child						
3-1		Exposure From Surface Water and Sludge While Swimming in Beach Area North of						
		Sludge Bed - Adolescent						

- 3-8 Exposure From Surface Water and Sludge While Swimming in Beach Area North of Sludge Bed Adult
- 3-9 Potential Exposure From Swimming in Beach Area North of Sludge Bed Summary
- 3-10 Exposure To Sand and Wood Debris During Recreational Use of the Beach Area North of Sludge Bed Young Child
- 3-11 Exposure To Sand and Wood Debris During Recreational Use of the Beach Area North of Sludge Bed - Adolescent
- 3-12 Exposure To Sand and Wood Debris During Recreational Use of the Beach Area North of Sludge Bed - Adult
- 3-13 Potential Exposure From Sand and Wood Chip Debris During Recreational Use of the Beach Area North of Sludge Bed Summary
- 3-14 Exposure From Recreational Ingestion of Fish From Cumberland Bay
- 3-15 Exposure From Recreational Ingestion of Fish From Cumberland Bay Summary
- 3-16 Noncarcinogenic and Carcinogenic Toxicity Values For Total PCBs
- 3-17 EPA Weight-of-Evidence Classification System for Carcinogenicity
- 3-18 Potential Carcinogenic and Noncarcinogenic Health Risks From Wading in Sludge Bed Area Near Wilcox Dock: Area Resident
- 3-19 Potential Carcinogenic and Noncarcinogenic Health Risks From Swimming in Beach Area North of Sludge Bed
- 3-20 Potential Carcinogenic and Noncarcinogenic Health Risks From Recreational Use of the Beach Area North of Sludge Bed
- 3-21 Potential Carcinogenic and Noncarcinogenic Health Risks From Recreational Ingestion of Fish From Cumberland Bay
- 3-22 Summary of Potential Health Risks By Exposure Scenario
- 4-1 Dominant Vegetation in Natural Areas Within One-Half Mile of the Site
- 4-2 Mammal/Amphibian/Reptile Species that Could Potentially Utilize Habitats Within One-Half Mile of the Site
- 4-3 Fish Species that Could Potentially Utilize Habitats Within One-Half Mile of the Site
- 4-4 Bird Species that Could Potentially Utilize Habitats Within One-Half Mile of the Site
- 4-5 PCB Concentrations in Sludge Bed 0 25 Centimeters

#### LIST OF FIGURES

#### Figure

- 1 Site Plan
- 2 Land Use/Cover Type Map
- 3 NYSDEC Regulated Wetlands Locations

#### **APPENDICES**

#### **Appendix**

A Toxicity Profile for Polychlorinated Biphenyls

#### 1.0 INTRODUCTION

A qualitative baseline health risk assessment (HRA) and a Fish and Wildlife Impact Analysis (FWIA) have been prepared for the New York State Department of Environmental Conservation (NYSDEC) as part of the Site Characterization of the Cumberland Bay Sludge Bed Site (Site), NYS Site Number 5-10-017. The work is being performed under Work Assignment D002520-32 of the State Superfund Contract between the NYSDEC and Rust Environment & Infrastructure (Rust) and it's subconsultant, TAMS Consultants, Inc. (TAMS).

Under this work assignment a Site Characterization (SC) Report was prepared in November 1995 to determine the nature and extent of contamination in the sludge bed and surrounding areas. A Feasibility Study (FS) Report was also prepared to identify and evaluate remedial alternatives for the site. This report constitutes Addendum No. 1 to the SC Report and includes a baseline HRA and a FWIA which have been prepared to evaluate the potential risks posed under current site conditions and to verify the need for Site remedial measures.

The baseline HRA and FWIA are limited to the existing environmental conditions found at the Site. The HRA considers the current commercial/recreational and potential future uses of the Site.

Section 2 of this report summarizes the results of the SC report and other previous investigations which are pertinent to the evaluation of risk at the Site. The baseline HRA is included as Section 3 and the FWIA is included as Section 4. The results of these evaluations are used to assess remedial action objectives and the need for remedial measures at the Site. This assessment is summarized in Section 5.

#### 1.1 Site Description

The Site is located in the northwest corner of Cumberland Bay in Lake Champlain, east of the City of Plattsburgh, Clinton County, New York. The Site is bordered to the south by Wilcox Dock (also referred to as the New York State Department of Transportation Barge Terminal) and to the west by the shoreline. The Site extends to the north to the approximate location of the Chamber of Commerce Building and to the east approximately 750 feet offshore. The present site definition includes all underwater areas within and along the northwestern portion of Cumberland Bay in Lake Champlain that contain accumulations of contaminated sludge. A Site location map is included as Figure 1.

Cumberland Bay is a small, somewhat rectangular part of the west side of Lake Champlain. Depths in the Bay can exceed 50 feet but water depths in the vicinity of the Site do not exceed 17 feet and are generally under 10 feet. The City of Plattsburgh is located on the west side of the Bay, where the Saranac River and Scomotion Creek flow into the Bay. The north shoreline of Cumberland Bay is occupied by the Plattsburgh Municipal Beach, a NYS Office of Parks and Recreation campground, and numerous motels and restaurants. On the east side, Cumberland Head, a large peninsula, extends into the Bay.

The Wilcox Dock is an engineered structure, 200 feet wide by 400 feet long and is presently controlled by the New York State Canal Corporation under the jurisdiction of the New York State Thruway Authority (NYSTA). Historically, land deeded to Willard G. Wilcox by the State of New York in the late 1800's was reappropriated back to the State of New York Department of Public Works in 1914. Subsequently, a barge canal terminal was envisioned, planned, designed and constructed. In the mid-1960's, as a result of a rehabilitation project conducted to preserve the dock as a Barge Canal Terminal at Plattsburgh, the south and east sides of the dock and a short portion of the north side of the dock were reinforced with sheet piling. The NYS Canal Corporation currently issues permits for the mooring of small water craft around the dock and limits access to the dock. The Georgia Pacific Corporation also controls access to a second entry to the dock area where it maintains a pump house for plant operations. Georgia Pacific presently operates a lakewater pump intake located between the southwest portion of the Wilcox Dock and the adjacent breakwater.

The sludge bed material covers most of the Site area ranging in thickness from approximately 0.25 to 10 feet. The thickest portions are located in a dredged channel adjacent to Wilcox Dock. The underlying "natural" soils consist of sand and silt.

The deepest water locations were between Wilcox Dock and the breakwater located to the south (approximately 10 to 17 feet) resulting from previous dredging activities performed by the NYSTA for the passage of barges. Similar dredging was performed along the north and northeast sides of Wilcox Dock. Water depths to the top of the sludge within the sludge bed varies between 0 feet at the shore line and approximately 7 feet during the 1995 SC investigation. The lake water elevation during the 1995 SC investigation was approximately 95.3 feet above mean sea level.

#### 1.2 Site History

Historically, land deeded to Willard G. Wilcox by the State of New York in the late 1800's was reappropriated back to the State of New York Department of Public Works in 1914, during which time a barge canal terminal was envisioned, planned, designed and constructed. Completion of construction occurred circa 1920 with the beginning of commercial traffic to the dock facility.

Industries in the area at the turn of the century and early 1900's included the Lozier Automobile Company, Saranac Pulp and Paper Company, Standard Pulp and Products Company, and Borst-Forest-Dixfiel. Several oil companies, including Colonial Beacon, Standard, Shell and Sucony Vacuum Oil Company maintained pipe lines from Wilcox Dock to storage facilities inland from the bay. In 1935 and in 1951, the NYS Division of Canals and Waterways dredged the canal access along the dock to accommodate larger fuel bearing vessels. The Diamond Match Company (1944), Vanity Fair (1955), and ultimately, the Georgia Pacific Company (1963-present) occupied property adjacent to the Site.

Records indicate that for several decades wastes from some local industries were discharged to local streams which ultimately discharge into Cumberland Bay or were directly discharged into the bay. Sawmills on the Saranac River discharged wastes into Cumberland Bay, where prevailing winds and currents in the summer dispersed the solids against the beach areas at the north end of the bay. Also,

pulp and paper mills on the shore of the bay near Dead Creek disposed of solids and organic materials (Frederic R. Harris, Inc., 1979). Untreated waste disposal ended in the early 1970's when the Plattsburgh Sewage Treatment Plant began treating wastes from the local industries. Over the years, wave action and water currents eroded the sludge bed and transported wood chips and organic debris along the shorelines and beaches to the north as well as to other areas within Cumberland Bay. For several years, the Site was considered a public nuisance, emitting unpleasant odors and hampering boating and swimming activities in the area. Environmental sampling from 1992 through 1994 confirmed the presence of polychlorinated biphenyls (PCBs), and to a lesser extent polychlorinated dibenzodioxins (dioxins) and dibenzofurans (furans) within the sludge and along the shoreline and beach areas.

At the present time, there is a health advisory in effect for several species of fish within Lake Champlain and Cumberland Bay due to elevated PCB levels in the fish; the advisory specifies, "eat no more than one meal per month". In addition, the commercial sale of yellow perch from Cumberland Bay is prohibited due to PCB concentrations in the fish, which exceed the US Food and Drug Administration (FDA) marketplace standard of 2 ppm.

#### 2.0 SITE INVESTIGATIONS

#### 2.1 Site Investigations Prior to the SC

A number of relevant investigations, including physical and analytical sampling studies, have been conducted at the Site. A brief summary of pertinent investigations is provided below.

- G.E. Myer and K. W. Loach of the SUNY at Plattsburgh prepared a report entitled *Preliminary Report of the Physical Parameters of the Plattsburgh, New York Sludge Bed*, dated March 1974. This report summarizes preliminary testing of the sludge bed depth, thickness and percent solids. It indicates that the sludge has a high water content, averaging 91.7 percent of the total mass.
- The NYSDEC has performed PCB analysis of the fish in Lake Champlain since 1979 to the present.
- A report entitled, Final Report Mudflats Removal Feasibility Study, Plattsburgh, New York was prepared by Frederic R. Harris, Inc. Consulting Engineers in July, 1979 under contract with the Economic Development Administration. The report summarizes "mudflat deposits" or sludge bed properties. Properties include: composition (wet, dry, and percent organic); chemical oxygen demand (COD); fecal coliform; volume estimates; filtration and leaching trials; and decomposition estimates. This report referenced a second report entitled Physical and Chemical Parameters of the Plattsburgh, New York Sludge Bed: A Second Report. This report estimated the total volume of sludge to be 380,000 cubic meters, of which 47% is under water, and 53% is exposed during the summer months.
- The NYSDEC Division of Water collected 14 sludge, sediment, and wood debris samples from the sludge bed and adjacent locations near Wilcox Dock for PCB analysis in the summer of 1993. In addition, six (6) core samples were collected on March 17, 1994 from the sludge bed and analyzed for PCBs, percent solids, organic content, and volume of solids. Core depths ranged from approximately 20.5 to 45.5 centimeters (8.1 to 17.9 inches) below the sludge surface. Concentrations ranged from below laboratory detection limits to 1,850 ppm.
- The NYSDOH collected a total of 29 water, sediment, sludge and wood debris samples from the beach/shoreline north of the sludge bed and the bay water during August, November, and December, 1994. Samples were analyzed for PCBs. Lake water samples ranged from below laboratory detection limits to 310 ppt for PCBs. Sediment and wood chip analytical sample concentrations ranged from below laboratory detection limits to 210 ppm.
- The NYSDEC Division of Hazardous Waste Remediation collected sludge and sediment samples from the sludge bed and bay areas south of Wilcox Dock and east across the bay on Cumberland Head shoreline on August 9 and 10, 1994. Samples were analyzed for

PCB, pesticides, metals and cyanide, dioxin, and furans. Total core recovery depths ranged from 14 to 136 cm (5.5 to 53.5 inches) below the top of the sludge/sediment surface. Concentrations of PCBs ranged from below the laboratory detection limits to 550 ppm. Dioxins ranged from below the laboratory detection limit to 330 ppt of octachlorodibenzodioxin (OCDD).

#### 2.2 Site Characterization Report

The purpose of the SC was to summarize the information gathered from a technical data review, site reconnaissance, and summarize the findings during the sludge bed and geotechnical boring sampling and investigation program performed at the Site.

A total of 56 cores were collected from the sludge bed and surrounding locations and physically logged. Cores were also collected from 10 beach locations from the Plattsburgh Chamber of Commerce Building northeast to the Dead Creek. Selected samples were analyzed for PCBs and dioxin.

#### Nature of the Sludge Bed Material

Field immunoassay test kits of the sludge bed and surrounding locations revealed that PCB concentrations which exceed 2 parts per million (ppm) are generally limited to the upper 12 inches of the sludge bed, however, 10 cores exhibited sludge thicknesses ranging from 14 to more than 22 inches below the surface. Beach samples analyzed with the test kits indicated that PCB concentrations which exceeded 2 ppm were generally limited to the upper 12 to 18 inches, however, cores located adjacent to the sludge bed exhibited sludge thicknesses as thick as 35 inches below the surface. Commercial laboratory PCB analyses indicated that PCB concentrations within the sludge ranged from approximately 1 ppm to 1,800 ppm of total PCBs. A composite sample collected from the sludge bed was analyzed by the NYSDEC laboratory and exhibited approximately 21 ppm of total PCB. The beach sample intervals ranged from below laboratory detection to 14 ppm of total PCBs. Total adjusted dioxin data (toxicity equivalent factor adjustment) ranged from 0.06 parts per trillion (ppt) to 161.9 ppt in core intervals from 30 to 36 inches and 6 to 12 inches, respectively.

The general stratigraphy within the sludge bed (based on observations made during the SC) consists of a top layer of dark (brown to black) fibrous pulp with highly organic material such as wood chips, root matter, and peat (supersaturated sludge) exhibiting a chemical-type odor. The thickness of this material ranges from essentially non-existent to a maximum of 17 inches. Directly below this initial sludge layer, a lighter-colored (grey) fibrous pulp layer is typically present. This sludge, where present, ranges in thickness from 6 to 14 inches. However, the investigation revealed that the sludge bed thickness generally ranges from 3 inches to greater than 10 feet. Further north from the center of the sludge bed, a sand with silt layer generally overlies a dark (black) organic sludge with wood chips.

Generally beneath both light and dark organic pulp sludge layers, a brown sand with silt layer exists with interlayered wood chips. This layer grades to a grey and/or brown "native" sand containing silt and, in certain areas, a layer of coarse wood chips. Cores collected at locations in the channel

adjacent to Wilcox Dock contained an extensive layer of white paper pulp sludge which appeared to exhibit medium to high plasticity properties and high PCB concentrations.

Extent of the Sludge Bed Material

The lateral extent of sludge was calculated during this investigation using the core information as well as manually probing. The sludge volume is estimated to range from approximately 90,000 to 95,000 cubic yards. The portion of the sludge bed which exhibited the greatest sludge thickness was located north and northeast, along Wilcox Dock where thicknesses exceeded approximately 10 feet.

#### 2.3 Supplemental Site Investigation

Based upon the findings of the 1995 Site Characterization Report, a supplemental site investigation was performed in 1996. The purpose of the supplemental investigation was to collect additional data from the sludge bed and the underlying natural sediments to support preparation and implementation of a remedial design. The supplemental investigation included the collection and logging of six (6) cores within and outside the sludge bed. Select samples were obtained from each core and screened for PCBs utilizing immunoassay field screening test kits. Confirmatory samples were sent to the NYSDEC Laboratory and/or to Aquatec Laboratory for PCB analysis. Analytical results indicated that the sludge bed samples contained PCB concentrations ranging from 44 to 1,800 ppm. Limited sediment sampling was also conducted at the Cumberland Bay State Park to the northeast of the Site. No significant levels of PCBs were detected at this off-site location.

#### 3.0 BASELINE HEALTH RISK ASSESSMENT

#### 3.1 Introduction

#### 3.1.1 Overview

Numerous conservative assumptions have been made throughout the preparation of this baseline HRA. The HRA is limited to the existing conditions found at the Site. The HRA considers the current and potential future uses of the Site and surrounding area. At the direction of the NYSDEC, the evaluations performed in this HRA considered the concentrations of PCBs, only, in environmental media at the Site.

#### 3.1.2 Purpose

Prepared as part of the SC for the Site, this HRA characterizes the potential for human exposure and the possibility of health effects associated with exposure to PCBs at the Site and surrounding area. As discussed previously and within, the State of New York has instituted fish advisories due to the finding of high PCB levels in Cumberland Bay fish. The HRA was performed to verify the necessity of the fish advisories, to identify other potential human exposure pathways within the area of the sludge bed, and to determine whether the Site may pose a risk to human health based on data collected as part of the SC or previous investigations performed by or on behalf of the State of New York. The HRA was also performed to fulfill the requirements of the Remedial Investigation/Feasibility Study required under the National Oil and Hazardous Substances Contingency Plan (the NCP) (EPA, 1994b).

#### 3.1.3 Scope and Organization of the HRA

This HRA is based on a myriad of data used to characterize the exposure routes, the chemical intakes and potential risks associated with the Site conditions. The following items were addressed within the HRA's scope of work:

- evaluation of Site history, chemical, hydrologic, demographic and other information;
- identification and evaluation of potential exposure pathways through a review of data collection activities, analytical protocols, current and surrounding land use, populationsat-risk and other related data;
- characterization of completed exposure pathways by the evaluation of chemical release sources, fate and transport, human exposure (contact) points and chemical intake routes;
- quantification and summarization of estimated potential chemical intakes, chemicalspecific risk-based criteria and potential toxic effects; and,
- characterization and discussion of potential chemical-specific carcinogenic and noncarcinogenic risks and their respective uncertainties.

From this review and analysis, the human exposure routes that present a potential toxic concern were then identified. Potential human exposures and health risks were fully characterized using principles and procedures consistent with the following U.S. Environmental Protection Agency (EPA) documents:

- Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Interim Final, Part A (December 1989, EPA/540/1-89/002)
- Guidance on Risk Characterization for Risk Managers and Risk Assessors.

  Memorandum. February 26, 1992
- Guidance for Data Usability in Risk Assessment, Interim Final (October 1990, EPA/540/G-90/008)
- Superfund Exposure Assessment Manual (April 1988, EPA/540/1-88/001) although a
- Exposure Factors Handbook (March 1990, EPA/600/8-89/043).

The human health risk assessment included data collection and evaluation (hazard identification), assessment of potential exposures to chemicals of concern, assessment of the toxicity of the chemicals of concern and characterization of the risks which may result from exposures.

#### 3.2 Hazard Identification

The purpose of the hazard identification is to review and evaluate available information regarding a chemical's potential to cause adverse effects. As discussed previously in Section 2.0, a number of investigations have been conducted at the Site. As per the NYSDEC, the levels of PCBs in the sludge and fine wood debris found in the shallows of the bay and adjacent to Wilcox Dock is the major concern at the Site. PCBs have also been detected in surface water, transported wood chips and organic debris along the shorelines and beaches to the north, and fish within the Cumberland Bay.

#### 3.3 Exposure Pathway Analysis

The purpose of an exposure assessment is to identify pathways through which people can be exposed under current and potential future use scenarios. The exposure assessment utilizes the current conditions at the Site and surrounding area in determining exposure scenarios and exposure concentrations. Additionally, future uses of the Site and surrounding area are also considered. Future use investigations utilized chemical concentrations which were derived from the validated SC chemical database, as well as those collected in previous investigations performed by the NYSDEC and NYSDOH. Therefore, this analysis may be considered an evaluation of the "no action alternative", in which minimal removal or treatment of the chemical constituents is undertaken. The analysis assumes that the concentrations of chemical constituents in environmental media has stabilized and will not change significantly over time.

- surface water; and
- · local fish populations.

A discussion of the reasons for their selection and an evaluation of each potential pathway is presented in the following subsections.

#### 3.3.2.1 Air

Although some studies indicate significant volatilization of PCBs from sand, PCBs are generally considered to be relatively non-volatile organics and tend to adsorb to soil with high organic content. Because the sludge material containing PCBs has a relatively high organic content, the degree of volatilization of PCBs is expected to be insignificant (i.e., PCBs will remain adsorbed to the sludge).

The results of air sampling at three locations at the Site during the SC support this conclusion. Air samples were collected over an approximate 8 hour period in accordance with NIOSH Method 5503 at three work areas (on the boat during core collection, in the core logging area and in the sample testing area) and analyzed for PCBs. All samples contained less than the laboratory detection limit of approximately 0.002 ppb. These results indicate that there was no significant volatilization of PCBs or suspended particulates containing PCBs resulting from the disturbance of sludge during the SC. No other air samples have been collected during previous investigations.

The potential exists for PCBs to become suspended in air due to wind erosion. However, the sludge bed is not expected to provide a complete potential exposure pathway since it is covered with water or contains very high water content where exposed. Wood chips and other debris (originating from the sludge bed) deposited onto the nearby beach areas/shoreline may provide a complete exposure pathway from wind blown dust, however, these impacted environmental media are generally too large in size to become suspended in air and are usually wet from wave action, etc. Based on these considerations, the air pathway is not considered a complete exposure pathway.

#### 3.3.2.2 Sludge Bed

The concentrations of PCBs in sludge bed material have been evaluated in the SC and previous studies by the NYSDEC and NYSDOH. General results are summarized in Section 1.1. The SC and State of New York investigations concluded that PCBs are present throughout the sludge bed. The SC indicated that the highest concentrations of PCBs are generally located within the upper 12 inches of the sludge bed. The sludge materials provide a point for potential human exposures. A potential route of human exposure to the sludge bed materials is through dermal contact with sludge bed materials on the lake bottom. The potential for human exposure exists due to recreational uses of Cumberland Bay in the area of the sludge bed. Because the area is used for mooring of boats and for fishing, direct contact with sludge material may occur during wading and fishing activities which could involve walking in the sludge bed. These direct contact exposures are expected to be limited due to the nature of the activities, the restricted access to Wilcox Dock and the relatively heavy aquatic vegetation directly off the shore line.

### 3.3.2.3 Wood Chips and Other Debris on Beaches

Environmental sampling performed in the SC, and by the NYSDEC and NYSDOH, have revealed the presence of wood chips and other debris (e.g., sludge) originating from the sludge bed which have been deposited immediately off-shore and onto the beaches areas to the north of the Site through wind and wave action. The known presence of these materials prompted the NYSDEC to take measures to remove the impacted materials from the beaches when possible. However, the presence of the sludge bed provides a continuous source of wood chips/debris resulting in a constant potential for contact with these materials on the beaches. Therefore, the potential for direct contact with these materials exists due to the recreation uses of the beaches to the north including walking, sun bathing, and swimming.

#### 3.3.2.4 Surface Water

Although PCBs are relatively insoluble in water and tend to bind to soil/sediments/sludge, trace amounts of PCBs have been detected in the water column at the Site by the State of New York. In the vicinity of the Site, Cumberland Bay is not used as a source of drinking water for any public or private water supply. It is possible that individuals may come into contact with Cumberland Bay surface water during recreational activities. Direct contact may occur during wading activities in the sludge bed area and while swimming in nearby beach areas.

or detected in any comple from a series and a water of your properties in place of the

#### 3.3.2.5 Fish Ingestion

Due to the presence of PCBs in Lake Champlain, fish consumption advisories have been instituted on both a lake-wide basis and locally within the vicinity of Plattsburgh (NYSDEC, 1994-95; New York Fishing Regulations Guide). Previous studies have indicated that fish and other aquatic biota bioconcentrate PCBs present in surface water and sediment. Due to their lipophilic properties, PCBs tend to bioaccumulate/biomagnify within the food chain. Fish sampling has been performed by the NYSDEC since 1979 and has demonstrated the presence of PCBs in several fish species sampled. A ban on commercial sale of yellow perch taken from Cumberland Bay was implemented by the NYSDEC since the PCB concentrations in this fish population exceeds the U.S. Food and Drug Administration's marketplace standard of 2 ppm for PCBs in commercial fish. The Site is frequently used for recreational fishing, particularly off Wilcox Dock, but also from boats or through the ice during the winter months. Recreational ingestion of fish populations from Cumberland Bay is, therefore, considered a complete exposure pathway under current and potential future use considerations.

#### 3.3.2.6 Exposure Pathway Summary

In summary, an evaluation of the SC, State of New York, and exposure assessment data indicates that the significant potential exposure points associated with the Site would be:

• direct contact with Cumberland Bay surface water and the sludge bed by nearby residents during wading activities near Wilcox Dock;

Where:

CW = Chemical Concentration In Surface Water (mg/L)

CR = Contact Rate (L/event)

EF = Exposure Frequency (events/yr)

ED = Exposure Duration (yr)

BW = Body Weight (kg)

AT = Averaging Time (dy): Pathways specific period of exposure for

noncarcinogenic effects (i.e., ED x 365 dy/yr), and 70 yr lifetime for

carcinogenic effects (i.e., 70 yr x 365 dy/yr).

#### Wading Near Wilcox Dock/Sludge Bed

It is unlikely that individuals would swim directly over the sludge bed on a routine basis due to restricted accessability to Wilcox Dock, knowledge of the sludge bed presence, and the aquatic vegetation over the sludge bed. Therefore, it was assumed that contact with surface water directly over the sludge bed would occur only through wading activities. In evaluating this exposure scenario, it was assumed that the quantity of surface water ingested (CR) during wading activities would be limited to approximately one mouthful, or 0.01 L. Potential receptors were assumed to wade in the sludge bed area 2 times/week from May through October (26 weeks), resulting in an exposure frequency (EF) of 52 events/year. It was assumed that wading activities would be limited to adolescent children (aged 7-16 years) and adults (>16 years), with body weights (BW) of 40 kg and 70 kg, respectively. In accordance with EPA guidance (EPA, 1991; 1989a) area residents that use Cumberland Bay for recreational purposes were assumed to live in the same home for 30 years. This represents the 90th percentile for time spent at one residence. Therefore, the exposure durations (ED) for the adolescent and adult were 10 years and 20 years, respectively (30 year total exposure period). The surface water concentration (CW) for this scenario was assumed to be the detected concentration (i.e., 0.00031 mg/L of Aroclor 1242) from the surface water sample collected by the NYSDOH (August 1994) directly over the sludge bed.

#### Swimming In Public Beach Areas

Surface water sampling performed by the NYSDOH has revealed the presence of PCBs in surface water collected near beach areas located to the north of the sludge bed. Therefore, it was assumed that individuals may come in contact with this water during swimming activities in these beach areas. As discussed above, area residents that use Cumberland Bay for recreational purposes were assumed to live in the same home for 30 years (EPA, 1991; 1989). To represent exposures over this 30 year exposure period, separate ingestion rates were calculated for young children (age 1-6 years), adolescents (age 7-16 years), and adults. In evaluating this exposure scenario, the EPA (1989a) has estimated that the quantity of surface water ingested (CR) by adults during swimming activities is 0.05 L. It was assumed that a young child (1-6 years) and adolescent (7-16 years) would have ingestion rates of 0.01 L and 0.02 L (CR) during swimming events. The exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. It was assumed that nearby residents may swim in nearby beach areas 2 times/week from June through September (17 weeks), resulting in an exposure frequency

(EF) of 34 events/year. The surface water concentration (CW) for this scenario was assumed to be the detected concentration (i.e., 0.00021 mg/L of Aroclor 1260) from the sample collected by the NYSDOH (August 1994) from the public beach area to the north of the sludge bed.

#### 3.3.3.1.2 Dermal Surface Water Absorption

The dermal absorption of PCBs in surface water was estimated by the following formula from the EPA guidelines (EPA, 1989a):

$$Dermal Dose (mg/kg/day) = \frac{CW * SA * PC * ET * EF * ED * CF}{BW * AT}$$

Where:

CW = Chemical Concentration In Surface Water (mg/L)

SA = Skin Contact Area (cm<sup>2</sup>)

PC = Chemical Dermal Permeability Constant (cm/hr)

ET = Exposure Time (hr/event)

EF = Exposure Frequency (events/yr)

ED = Exposure Duration (yr)

CF = Volumetric Water Conversion Factor = 1 L/1000 cm<sup>3</sup>

BW = Body Weight (kg)

AT = Averaging Time (dy): Pathways specific period of exposure for noncarcinogenic effects (i.e., ED x 365 dy/yr), and 70 yr lifetime for carcinogenic effects (i.e., 70 yr x 365 dy/yr).

The estimation of doses resulting from skin contact with water requires the use of chemical-specific skin permeability constants (PC), which are provided in units of cm/hr. These skin penetration rates are not only a function of chemical property data but also of biological properties (e.g., skin thickness). For PCBs, the PC was derived from the following equation (Brown and Rossi, 1989):

$$PC = 0.1[K_{ow}^{0.75}/(120 + K_{ow}^{0.75})]$$

Where:

 $K_{ow}$  = Octanol/water partition coefficient for the chemical

Doses calculated using PCs represent absorbed doses, however, essentially no health criteria exist based on absorbed doses. Therefore, these skin absorbed doses need to be adjusted for comparison to the existing health criteria, e.g. oral Reference Doses (RfD) or Cancer Potency Factors (CPF). This adjustment was accomplished by modifying the PC to account for the oral absorption of PCBs in order to normalize the dermal dose to the oral dose for risk characterization. This adjustment was made using the following equation:

$$Adjusted PC = \frac{PC}{Oral Absorption of Analyte}$$

Based on a calculated  $K_{ow}$  for PCBs of 9.98 x  $10^{-2}$  cm/hr, and assuming an oral absorption efficiency of 100% for PCBs in aqueous media (ATSDR, 1992), a adjusted PC of 9.98 x  $10^{-2}$  cm/hr may be calculated. Therefore, for the purposes of this HRA the normalized (adjusted) PC for PCBs was assumed to be 9.98 x  $10^{-2}$  cm/hr.

#### Wading Near Wilcox Dock/Sludge Bed

As discussed previously, nearby residents were assumed to wade in the sludge bed area 2 times/week from May through October (26 weeks), resulting in an exposure frequency (EF) of 52 events/year. Individuals were assumed to wade in the sludge bed for 0.5 hr/event (exposure time - ET). It was assumed that wading activities would be limited to adolescents (aged 7-16 years) and adults (>16 years), with body weights (BW) of 40 kg and 70 kg, respectively. The exposure durations (ED) for the adolescent and adult were 10 years and 20 years, respectively, over the 30 year total exposure period. The skin contact area (SA) was assumed to include the hands, forearms, legs below the knees and feet. These body parts comprise approximately 27 percent of the total adult body surface area of males (19,400 cm²) (EPA, 1989b), or 5,170 cm². If a similar body surface area ratio is used for a adolescent (age 7-16 years), the skin surface area available for contact with surface water is assumed to be 3,600 cm² (total surface area for adolescent males of 13,300 cm² - EPA, 1989b). The PC was assumed to be 9.98 x 10-2 cm/hr, as discussed above. The surface water concentration (CW) for this scenario was assumed to be the detected concentration (i.e., 0.00031 mg/L of Aroclor 1242) from the sample collected by the NYSDOH (August 1994) from directly over the sludge bed.

#### Swimming In Public Beach Areas

As discussed previously, area residents that use Cumberland Bay for recreational purposes were assumed to live in the same home for 30 years (EPA, 1989a; 1991). To represent exposures over this 30 year exposure period, separate contact rates were calculated for young children (age 1-6 years), adolescents (age 7-16 years), and adults during the one (1) hour (EPA, 1992b) swimming events (ET). The exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. It was assumed that nearby residents may swim in nearby beach areas 2 times/week from June through September (17 weeks), resulting in an exposure frequency (EF) of 34 events/year. The body contact area (SA) was assumed to be the entire body or 7,280 cm², 13,200 cm² and 19,400 cm² for the young child, adolescent and adult, respectively (EPA, 1989b). The PC was assumed to be 9.98 x 10-2 cm/hr, as discussed above. The surface water concentration (CW) for this scenario was assumed to be the detected concentration (i.e., 0.00021 mg/L of Aroclor 1260) from the sample collected by the NYSDOH (August 1994) from the public beach area to the north.

#### 3.3.3.1.3 Sludge Ingestion

The following equation was used to estimate ingestion of PCBs present in sludge bed material and wood chips/debris (EPA, 1989a):

$$Ingested\ Dose\ =\ \frac{CS*IR*CF*FI*EF*ED*ABS}{BW*AT}$$

Where:

CS = Chemical Concentration In Soil/Sediment (mg/kg)

IR = Ingestion Rate (mg/dy)

CF = Conversion Factor (1 x 10<sup>-6</sup> kg/mg)

FI = Fraction Ingested from Contaminated Source

EF = Exposure Frequency (dy/yr) ED = Exposure Duration (yr)

ABS = Absorption Factor (Percentage of chemical desorbed from

soil matrix and absorbed via ingestion)

BW = Body Weight (kg)

AT = Averaging Time (dy): Pathways specific period of exposure for noncarcinogenic effects (i.e., ED x 365 dy/yr), and 70 yr lifetime for carcinogenic effects (i.e., 70 yr x 365 dy/yr).

#### Wading Near Wilcox Dock/Sludge Bed

As discussed previously, nearby residents were assumed to wade in the sludge bed area 2 times/week from May through October (26 weeks), resulting in an exposure frequency (EF) of 52 events/year. It was assumed that wading activities would be limited to adolescents (aged 7-16 years) and adults (>16 years), with body weights (BW) of 40 kg and 70 kg, respectively. An incidental ingestion rate of 100 mg/day was assumed (EPA, 1989a; EPA, 1991). A conservative approach assumed that 100% of the soil ingested (FI) was from the sludge bed material. The exposure durations (ED) for the adolescent and adult were 10 years and 20 years, respectively, over the 30 year total exposure period. The bioavailability of chemicals adsorbed onto ingested soils will affect the amount of contaminant exposure. Therefore, when evaluating the ingestion of contaminated soils, a relative absorption factor should be used to account for the differing bioavailability between the contaminant in the soil matrix and the contaminant in an experimentally administered medium such as solvent or food. In accordance with the EPA (1989c), the relative absorption factor (ABS) for PCBs was assumed to be 30% (0.3). The sediment concentration (CS) for this scenario was assumed to be the 95% UCL concentration of sludge data collected during the SC, and by the NYSDEC and NYSDOH.

#### Swimming In Public Beach Areas

To represent potential exposures, separate ingestion rates were calculated for young children (age 1-6 years), adolescents (age 7-16 years), and adults during the swimming events. The ingestion rate (IR) was assumed to be 200 mg/day for the young child, and 100 mg/day for adolescents and adults (EPA, 1989a; EPA, 1991). It was assumed that 50% of the impacted material ingested (FI) was from sludge deposits off-shore of the beaches (the remainder was from recreational beach activities - see below). The exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. It was assumed that nearby residents may swim in nearby beach areas 2 times/week from June through September (17 weeks), resulting in an exposure frequency (EF) of 34 events/year. In accordance with the EPA (1989c), the relative absorption factor (ABS) for PCBs was assumed to be 30% (0.3). Because only one sediment sample has been collected from areas located off-shore of the beach areas to the north of the sludge bed, it was assumed that the off-shore sediments would have PCB concentrations similar to those observed in the sand/beach cores/wood chip debris collected from the beach areas to the north of the sludge bed. Therefore, the sediment concentration (CS) for this scenario was assumed to be the 95% UCL concentration of the one (1) off-shore sediment sample and the sand/beach cores/wood chip debris collected by the NYSDEC and NYSDOH, and during the SC, from the beach areas to the north of the sludge bed (i.e., Route 9 beach and the Plattsburgh public beach).

#### Playing/Sun-Bathing On Public Beach Areas

Environmental sampling performed in the SC, and by the NYSDEC and NYSDOH have revealed that wood chips and other debris (e.g., sludge) originating from the sludge bed have been deposited immediately off-shore and onto the public beaches north of the Site. Therefore, the potential exists for individuals to come in contact with these materials while playing or sun-bathing on the beach areas. As discussed previously, area residents that use Cumberland Bay for recreational purposes were assumed to live in the same home for 30 years (EPA, 1989a; 19891). To represent exposures over this 30 year exposure period, separate ingestion rates were calculated for young children (age 1-6 years), adolescent (age 7-16 years), and adults. The ingestion rate (IR) was assumed to be 200 mg/day for the young child, and 100 mg/day for adolescents and adults (EPA, 1989a; EPA, 1991). It was assumed that 50% of the soil ingested (FI) was from impacted soil/sand/wood debris (the remainder was from swimming activities - see above). It was assumed that nearby residents may play on nearby beach areas 2 times/week from June through September (17 weeks), resulting in an exposure frequency (EF) of 34 events/year. The exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. In accordance with the EPA (1989c), the relative absorption factor (ABS) for PCBs was assumed to be 30% (0.3). The soil concentration (CS) for this scenario was assumed to be the 95% UCL concentration of sand/beach cores/wood chip debris collected by the NYSDEC and NYSDOH, and during the SC, from the beach areas to the north of the sludge bed (i.e., Route 9 beach and the public beach).

#### 3.3.3.1.4 Dermal Sludge Absorption

Dermal absorption of PCBs present in sludge bed material, beach cores and wood chips/debris was estimated using the following equation (EPA, 1989a):

$$Dermal Dose = \frac{CS * CF * SA * AF * ABS * EF * ED}{BW * AT}$$

Where:

CS = Chemical Concentration In Soil/Sediment (mg/kg)

 $CF = \text{Conversion Factor } (1 \times 10^{-6} \text{ kg/mg})$ 

SA = Skin Contact Area (cm<sup>2</sup>/event)

AF = Adherence Factor (mg/cm<sup>2</sup>)

ABS = Absorption Factor (Percentage of chemical desorbed from

soil matrix and absorbed through skin)

EF = Exposure Frequency (events/yr)

ED = Exposure Duration (yr)

BW = Body Weight (kg)

AT = Averaging Time (dy): Pathways specific period of exposure for

noncarcinogenic effects (i.e., ED x 365 dy/yr), and 70 yr lifetime for

carcinogenic effects (i.e., 70 yr x 365 dy/yr).

#### Wading Near Wilcox Dock/Sludge Bed

As discussed previously, it was assumed that wading activities over the sludge bed would be limited to adolescents (aged 7-16 years) and adults (>16 years), with body weights (BW) of 40 kg and 70 kg, respectively. Nearby residents were assumed to wade in the sludge bed area 2 times/week from May through October (26 weeks), resulting in an exposure frequency (EF) of 52 events/year. The skin contact area (SA) was assumed to include the hands, forearms, legs below the knees and feet, or 3,600 cm² for adolescents and 5,170 cm² for adults (refer to Section 3.3.3.1.2). The adherence factor (AF) used was the upper limit of that reported by the EPA, or 1.0 mg/cm² (EPA, 1992a). The fraction of PCBs desorbed from the soil and absorbed through the skin (ABS) was assumed to be 5% (0.05) (EPA, 1989c). The sediment concentration (CS) for this scenario was assumed to be the 95% UCL concentration of sludge data collected during the SC, and by the NYSDEC and NYSDOH.

#### Swimming In Public Beach Areas

As discussed in Section 3.3.3.1.1, it was assumed that individuals may swim in Cumberland Bay beach areas to the north of the sludge bed 34 times/year (EF) over a 30 year period. To represent potential exposures, separate contact rates were calculated for young children (age 1-6 years), adolescents (age 7-16 years), and adults during the swimming events. It was assumed that

individuals would only come in direct dermal contact with impacted sediments while walking into the water to swim. As such, the body contact area was assumed to be limited to the feet. The feet comprise approximately 7% of the entire body surface area of 7,280 cm², 13,200 cm² and 19,400 cm² for the young child, adolescent and adult, respectively (EPA, 1989b). Therefore, the dermal contact area was assumed to be 510 cm², 924 cm² and 1,358 cm² for the young child, adolescent and adult, respectively. An adherence factor (AF) of 1.0 mg/cm² was assumed (EPA, 1992a). The fraction of PCBs desorbed from the soil and absorbed through the skin (ABS) was assumed to be 5% (0.05) (EPA, 1989c). As discussed previously, the sediment concentration (CS) for this scenario was assumed to be the 95% UCL concentration of the one (1) off-shore sediment sample and the sand/beach cores/wood chip debris collected by the NYSDEC and NYSDOH, and during the SC, from the beach areas to the north of the sludge bed (i.e., Route 9 beach and the Plattsburgh public beach).

#### Playing/Sun-Bathing On Public Beach Areas

As discussed previously, the potential exists for individuals to come in contact with wood chips and other debris originating from the sludge bed while playing or sun-bathing on the beach areas to the north of the sludge bed. Separate contact rates were calculated for young children (age 1-6 years), adolescents (age 7-16 years), and adults during the potential 30 year exposure period. A conservative approach assumed that the entire body was the contact area (SA) for these exposures, or 7,280 cm², 13,200 cm² and 19,400 cm² for the young child, adolescent and adult, respectively (EPA, 1989b). An adherence factor (AF) of 1.0 mg/cm² was assumed (EPA, 1992a). The fraction of PCBs desorbed from the soil and absorbed through the skin (ABS) was assumed to be 5% (0.05) (EPA, 1989c). It was assumed that nearby residents may play on nearby beach areas 2 times/week from June through September (17 weeks), resulting in an exposure frequency (EF) of 34 events/year. The exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. As discussed previously, the soil concentration (CS) for this scenario was assumed to be the 95% UCL concentration of sand/beach cores/wood chip debris collected by the NYSDEC and NYSDOH, and during the SC, from the beach areas to the north of the sludge bed (i.e., Route 9 beach and the Plattsburgh public beach).

The oral and dermal exposure estimates to PCBs in surface water and sludge material while wading over the sludge bed are presented in Table 3-3 (adolescent) and Table 3-4 (adult), and are summarized in Table 3-5. The oral and dermal exposure estimates to PCBs in surface water and sludge during swimming activities in public beach areas north of the sludge bed are presented in Table 3-6 (young child), Table 3-7 (adolescent) and Table 3-8 (adult), and are summarized in Table 3-9. The oral and dermal exposure estimates to PCBs in beach sand and wood chips/debris (originating from the sludge bed) during recreational activities (e.g., sun-bathing and playing) on public beach areas north of the Site are presented in Table 3-10 (young child), Table 3-11 (adolescent) and Table 3-12 (adult), and are summarized in Table 3-13.

#### 3.3.3.2 Recreational Ingestion of Fish from Cumberland Bay

The sampling of Cumberland Bay fish by the NYSDEC since 1979 has revealed the presence of PCBs in fish populations exceeding the U.S. Food and Drug Administrations marketplace standard

of 2 ppm of PCBs in commercial fish. As a result, fish consumption advisories and a commercial ban on yellow perch from Cumberland Bay has been instituted by the NYSDEC. However, because Cumberland Bay and the area around Wilcox Dock are used for recreational fishing, the potential exists for consumption of fish from Cumberland Bay during recreational fishing activities. Human exposure estimates for this fish ingestion scenario were calculated using the following equation (EPA, 1989a):

$$Ingested\ Dose = \frac{CF * IR * FI * EF * ED}{BW * AT}$$

Where:

CF = Chemical Concentration In Fish (mg/kg)

IR = Ingestion Rate (mg/dy)

FI = Fraction Ingested from Contaminated Source

EF = Exposure Frequency (kg/meal)

ED = Exposure Duration (yr)

BW = Body Weight (kg)

AT = Averaging Time (dy): Pathways specific period of exposure for noncarcinogenic effects (i.e., ED x 365 dy/yr), and 70 yr lifetime for carcinogenic effects (i.e., 70 yr x 365 dy/yr).

In evaluating this exposure scenario, separate fish ingestion rates were estimated for a young child (1-6 years), adolescent (7-16 years), and adult, with body weights (BW) of 16 kg, 40 kg and 70 kg, respectively. The EPA (EPA, 1994a) recommends an ingestion rate (IR) for young children of 0.085 kg/meal and an IR for adults of 0.227 kg/meal (EPA, 1994a) for recreational fish ingestion. The IR for adolescents was estimated to be 0.129 kg/meal based on a body weight adjustment for a 70 kg adult versus a 40 kg adolescent (i.e., 40 kg/70 kg = 0.57; 0.227 kg/meal x 0.57 = 0.129 kg/meal) (EPA, 1994a). It was further assumed that individuals would ingest fish at an exposure frequency (EF) of 48 meals/year (EPA, 1989a). A conservative approach assumed that 100% (FI = 1.0) of the fish ingested were taken from Cumberland Bay. As discussed previously, the exposure durations (ED) for the young child, adolescent and adult over the 30 year total period of exposure were 6 years, 10 years, and 14 years, respectively. The concentration of PCBs in fish (CF) was the 95% UCL concentration from 20 yellow perch samples collected by the NYSDEC (December 1994) from Cumberland Bay.

Estimated recreational fish intake exposures for the young child, adolescent and adult are presented in Table 3-14, and are summarized in Table 3-15.

#### 3.4 Toxicity Assessment

The purpose of the toxicity assessment is to review and evaluate available information regarding the potential of chemicals to cause adverse effects. The assessment considers molecular structure and dose (exposure level, time and frequency of exposure, absorption, etc.). As the level of exposure increases, the likelihood or probability of an adverse effect (response) occurring also increases. This

dose-response relationship is used in the risk assessment to derive the exposure level at which no observable adverse effect occurs (NOAEL). Reactions to exposures can be short-term and "acute" in character. These usually involve exposures to high levels of a substance over brief periods of time. Reactions can also be "chronic", generally involving repeated exposure to lower levels of a substance over prolonged periods. Using estimated exposure levels (Section 3.3) and chemical-specific dose response parameters, the goal is to establish quantitative exposure levels that can be correlated with the likelihood of an adverse outcome or effect, i.e., an assessment of risk.

For the purposes of this toxicity assessment, a toxicological profile for PCBs been developed using computerized data searches. Evaluation and incorporation of the data into this report followed the review hierarchy referenced in applicable EPA guidance documents (EPA, 1989a). Unless otherwise indicated, RfDs and CPFs for PCBs were selected from the following references in the order presented:

- Integrated Risk Information System (IRIS, 1997);
- Health Effects Assessment Summary Table (HEAST, 1995);
- EPA Criteria Documents;
- Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profiles;
- · Relevant New York State Regulations and Guidelines; and
- Regulations and Guidelines Established by Other States (e.g., Massachusetts).

Within the toxicological profile there are two broad categories discussed: noncarcinogenicity and carcinogenicity. Noncarcinogenic effects can include either acute or chronic effects. Such toxic endpoints might include impairment of organ(s), system dysfunction, (e.g., respiratory, neurological and immune), reproductive interference (including sterility and birth defects), metabolic changes, etc. These effects can be either reversible or irreversible. Noncarcinogenic effects generally occur only after a "threshold" dose has been exceeded. A RfD is derived from the no observed adverse effect level (NOAEL) or lowest observed adverse effect level (LOAEL) of the experimental toxicology data. The RfD represents the threshold dose below which noncarcinogenic effects are not expected to occur in exposed populations. Effects in sensitive populations are taken into account by the use of uncertainty factors, which are multiplicative adjustments that reduce the allowable dose level 10-fold to account for sensitive or more susceptible members of an exposed population. If necessary, additional uncertainty factors of 10 may also be used to: (1) extrapolate from a LOAEL to a NOAEL, (2) provide a margin of safety in comparing animal studies to human responses, and (3) adjust data obtained in subchronic or acute studies to evaluate risks associated with chronic exposures. As a result, the RfD values used in risk assessments are often 100 to 1,000 times lower than the lowest dose found to be associated with the least severe adverse effect for a given chemical. The RfD is used as a benchmark for comparison with the estimated exposure dose for the noncarcinogenic effects of PCBs.

Carcinogenic effects refer to the demonstrated or suspected capacity of a chemical to induce cancers. A chemical's carcinogenic potential is ranked by the EPA based on a "weight-of-evidence" classification (Table 3-17). CPFs are calculated based upon available scientific evidence of the dose-response relationship between the cancer induced and the level(s) of chemical administered that caused the carcinogenic response. Specifically, the 95% UCL of the dose-response curve is used in

multi-stage dose-response models to calculate CPFs. The CPFs are then used to estimate the probability that exposure to the chemical will result in a carcinogenic response. Because the link between cancer and chemicals/physical agents (e.g., radiation) is unknown, federal (EPA, Occupational Safety and Health Administration, and the Consumer Product Safety Commission) and state regulatory agencies have adopted a policy that assumes that no threshold dose exists for carcinogens. Under this policy, the agencies assume that the levels of exposure below which cancer will not appear cannot be defined. These assumptions lead to conservative estimates of carcinogenic risks for some chemicals. The CPFs are used in this quantitative HRA estimate the probability of carcinogenic effects occurring due to the estimated exposures to PCBs.

#### 3.4.1 General Toxicity Information

A complete toxicological profile for PCBs was developed in accordance with EPA guidance (EPA, 1989a) and SARA Section 110. The complete toxicological profile is presented in Appendix A. For convenience, an abbreviated summary of this profile is presented in this section.

#### 3.4.1.1 Polychlorinated Biphenyls

PCBs are absorbed through the gastrointestinal tract and distributed throughout the body. Oral absorption efficiencies of 75% to almost 100% have been observed in animal studies. The highest concentrations are found in adipose (fat) tissue.

PCB exposure is associated with a wide array of adverse health effects in humans and experimental animals. The health effects of PCBs are still under investigation and currently there is not sufficient information on the specific congeners to develop congener-specific quantitative estimates of health risk. Due to the lack of congener-specific information, the EPA Office of Water recommends, as an interim measure, that total PCB concentrations be reported as the sum of Aroclors.

The EPA (IRIS, 1997) has developed an oral RfD of 2 x 10<sup>-5</sup> mg/kg/day for Aroclor 1254 based on ocular effects and distorted growth of finger and tow nails at the lowest dose tested in monkeys of 0.005 mg/kg/day. Significant repression of immune responses were also observed in sheep at the lowest dose tested. On the basis of these effects, a lowest observed adverse effect level (LOAEL) was established. The EPA applied uncertainty factors of 10 to account for sensitive individuals, 3 for extrapolation from monkeys to humans, and 3 for extrapolation from a subchronic to a chronic RfD. A partial factor was also applied for use of a minimal LOAEL since the changes in the periocular tissues and nail bed seen at the 0.005 mg/kg/day were not considered to be of marked severity. The total uncertainty factor applied was 300.

PCBs have been classified by the EPA as Group B2 carcinogens (IRIS, 1997) based on sufficient animal carcinogenicity data and insufficient human carcinogenicity data. A 1996 study found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to Aroclor 1260. These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. The cancer potency of PCB mixtures is determined using a tiered approach that depends on the information available. The

following tier descriptions discuss all potential environmental exposure routes considered by the EPA (IRIS, 1997) when evaluating risks to PCBs:

#### High Risk and Persistence

Upper-bound slope factor:

2.0 (mg/kg/day)-1

Criteria for Use: - Food chain exposure

- Sediment or soil ingestion - Dust or aerosol inhalation

- Dermal exposure, if an absorption factor has been applied

- Early life exposure (all pathways and mixtures)

#### Low Risk and Persistence

Upper-bound slope factor:

0.4 (mg/kg/day)-1

Criteria for Use: - Ingestion of water-soluble congeners

- Inhalation of evaporated congeners

- Dermal exposure, if no absorption factor has been applied

#### Lowest Risk and Persistence

Upper-bound slope factor:

0.07 (mg/kg/day)-1

Criteria for Use:

Congener or isomer analyses verify that congeners with more than 4

chlorines comprise less than ½% of total PCBs

#### 3.4.2 **Toxicity Information Summary**

The noncarcinogenic and carcinogenic toxicity values reviewed for this human health evaluation are summarized in Table 3-16. For the purposes of this human health evaluation, the chronic oral RfD was used for calculating noncarcinogenic risks for PCBs. Cancer slope factors have been established for PCBs using a tiered approach based on relative risk and persistence via specific exposure pathways. Only the upper-bound slope factors were used for calculating carcinogenic risks PCBs. The toxicity profile for PCBs is located in Appendix A.

#### 3.5 Risk Characterization

Risk characterization is the final step in the HRA process. The purpose of this section is to estimate and characterize the potential for non-cancer adverse toxic effects and potential cancer risks. Risks are characterized for specific exposure pathways and for exposed hypothetical reference populations and to exhibit maximum chemical sensitivities. Reasonable maximum exposures are defined for all exposure pathways. The reasonable maximum estimate is a plausible estimate of risk for those at the upper end of the risk distribution. Conceptually, the reasonable maximum estimate provides risk estimates above the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest risk (i.e., the reasonable maximum risk). The noncarcinogenic and carcinogenic risks that were characterized were dependent upon numerous assumptions made throughout each stage of the HRA process. The presentation of these risks considered the attendant scientific uncertainties and limitations of the available data.

Caution should be exercised in attempting to draw conclusions regarding any actual or perceived risks to individuals that may or may not exist within a potentially exposed population. The risk estimates are population-based and cannot be interpreted or used to evaluate an individual's personal risks. The quantitative risk estimates that may be attributable to chemical exposures are often based on limited experimental toxicology or epidemiological data and, therefore, must rely on numerous scientific and professional judgments of toxicologists or other health professionals. Nonetheless, the risk assessment process does provide a systematic and consistent basis for evaluating the potential risks that may exist and identifying exposure pathways of possible concern. This information can then be factored into the selection of the most cost-effective and feasible remedial risk management alternative that is protective of the public health and the environment.

#### 3.5.1 Noncarcinogenic Risks

The potential for non-cancer adverse effects associated with chronic exposure to noncarcinogens is characterized using the ratio of Chronic Daily Intake (CDI) to the corresponding RfD. The CDI was estimated from the pollutant pathway analysis and estimates of exposure route concentrations associated with those pathways.

Toxic risks are assessed on a substance-by-substance basis (Hazard Quotients - HQs), as well as on a cumulative basis (Hazard Indices -- HIs). This ratio of CDIs to RfDs provides an indication of the relationship between the estimated daily dose and the lowest dose that has been associated with the potential for adverse effects in the most sensitive population. It DOES NOT represent a numerical estimate of either the probability or severity of chronic non-cancer effects. The numerical value for the CDI/RfD ratios is often referred to as a HQ and may be thought of more accurately as a margin of safety at values below 1.0. If the HQ exceeds 1.0, this should be interpreted as an indication that adverse effects could be of concern, especially for sensitive populations. It does not unequivocally indicate adverse effects will occur. However, a HI of less than one, particularly if it represents the aggregated (sum) HQs of many of the chemicals of concern, in general provides a degree of assurance that adverse impacts will not occur. This is due to the conservativeness inherent in the way that RfD values are derived and in the way that risks are characterized.

In order to assess the potential for adverse noncarcinogenic effects associated with multiple chemical exposures, an additive approach is used. The sum of all the HQs for the selected chemical exposure is considered the HI. The HI is calculated as follows:

Hazard Index (HI) = 
$$\sum HQ_i = \sum_{I}(CDI/RfD)_I = \sum_{I}(CDI_i/RfD_i)$$

where subscript (I) is the CDI and RfD for each chemical in the exposure pathway. The potential for adverse noncarcinogenic effects associated with a reference individual being exposed to the

chemicals of concern by more than one pathway is calculated by summing the doses across all pathways, and calculating HQs and HIs as described. This approach is consistent with the EPA guidance referenced earlier. The toxicologic parameters used in the noncarcinogenic risk assessment are derived mainly from peer-reviewed government sources (EPA, 1989a).

A noncarcinogenic HQ was calculated for PCBs in each exposure pathway using reasonable maximum exposure estimates. Only the chronic RfD was used in these calculations. These assumptions were felt to provide a reasonable estimate of the range of population risk distributions.

#### 3.5.2 Carcinogenic Risks

Cancer risks are often quantified as occurrences of new cancer cases per million people exposed under the circumstances defined. This is equivalent to a per capita risk expressed as the odds per million that a hypothetical exposed person will develop cancer as a result of his or her reasonable worst-case exposure at a site. In order to evaluate potential carcinogenic health risks, a lifetime average daily dose (LADD) is first estimated from the pollutant pathway analysis and estimates of exposure route concentrations involving those pathways. The incremental cancer risk due to exposure to a particular chemical is calculated from the LADD and the cancer potency factor (CPF) for that chemical as follows:

Incremental Lifetime Cancer Risk = LADD (mg/kg/dy) x CPF (mg/kg/dy)-1

The LADD used to assess risks refers to the daily lifetime average dose received through a given exposure pathway (for pathway specific risks), or to the total dose of a given chemical received by a hypothetical reference individual from a number of exposure pathways.

In calculating cancer risks from multiple chemical exposures, the risk associated with each individual exposure are summed to give an estimate of total cancer risks:

Excess Lifetime Cancer Risk =  $\sum_{i}(LADD_{i} \times CPF_{i})$ 

where subscript (I) refers to the LADDs and the CPFs for each of the chemical of concern. This approach does not explicitly take into account the potential for either positive (synergistic) or negative (antagonistic) chemical interactions. It is assumed the conservatism inherent in the estimation of the CPF values, combined with the conservatism employed in exposure and dose assessment modeling, exceed the greatest multiplicative synergistic effect expected in any population. Finally, antagonistic or interference interactions between chemicals are at least as likely, on biochemical grounds, as synergistic ones. Taking all of the above into account, it is probable that the risk assessment methods used here will result in conservative estimates of cancer risks due to multiple chemical exposures.

The values used for the CPFs were taken or derived from EPA sources such as the IRIS or EPA Health Assessment documents. These CPFs are considered to be conservative since they represent the 95% upper-bound confidence interval of the cancer slope parameter derived from animal or human dose-response curves. Cancer risks was calculated for PCBs in each exposure pathway using

median and reasonable maximum exposure estimates. These assumptions were felt to provide a reasonable estimate of the range of population cancer risk distributions.

#### 3.5.3 Wading in Sludge Bed Area Near Wilcox Dock

The potential noncarcinogenic hazards posed by the ingestion and dermal absorption of PCBs in surface water and sludge bed material during wading activities in the sludge bed area near Wilcox Dock are presented in Table 3-18. The HI for oral and dermal exposures to PCBs in surface water and sludge due to wading activities was 5.3. This value is above unity (i.e., 1.0) indicating that noncarcinogenic effects may occur from this potential exposure.

The potential carcinogenic risks posed by the ingestion and dermal absorption of PCBs in surface water and sludge bed material during wading activities in the sludge bed area near Wilcox Dock are also presented in Table 3-18. The excess cancer risk was estimated to be 7.8 x 10<sup>-5</sup>. The potential total excess cancer risk for this scenario is within the risk range often used by EPA (10<sup>-6</sup> to 10<sup>-4</sup>) in setting cleanup goals under Superfund (EPA, 1994b).

#### 3.5.4 Swimming in Beach Areas North of the Sludge Bed

The potential noncarcinogenic hazards and carcinogenic risks posed by the incidental ingestion and dermal absorption of off-shore sludge deposits and surface water while swimming in Route 9 and public beach areas located to the north of the sludge bed are presented in Table 3-19. The HI of 1.1 x 10<sup>-1</sup> is below unity indicating that noncarcinogenic effects are unlikely to occur from this potential exposure. The estimated potential excess cancer risk of 7.6 x 10<sup>-7</sup> for this scenario is below the lower end of the risk range often used by EPA (10<sup>-6</sup> to 10<sup>-4</sup>) in setting cleanup goals under Superfund (EPA, 1994b).

#### 3.5.5 Recreational Use of Beach Areas North of the Sludge Bed

The potential noncarcinogenic hazards and carcinogenic risks posed by the incidental ingestion and dermal absorption of sand and wood chips/debris during recreational use (e.g., sun-bathing and playing) of the Route 9 and public beach areas located to the north of the sludge bed are presented in Table 3-20. The HI of 5.7 x 10<sup>-1</sup> is below unity indicating that noncarcinogenic effects are unlikely to occur from this potential exposure. The estimated potential excess cancer risk of 6.8 x 10<sup>-6</sup> for this scenario is near the lower end of the risk range often used by EPA (10<sup>-6</sup> to 10<sup>-4</sup>) in setting cleanup goals under Superfund (EPA, 1994b).

#### 3.5.6 Recreational Ingestion of Cumberland Bay Fish

The potential noncarcinogenic hazards and carcinogenic risks posed by the ingestion of fish from Cumberland Bay are presented in Table 3-21. The HI of  $2.9 \times 10^{+2}$  is well above unity indicating that noncarcinogenic effects may occur from this potential exposure. The estimated potential excess cancer risk of  $3.4 \times 10^{-3}$  for this scenario is above the upper end of the risk range often used by EPA ( $10^{-6}$  to  $10^{-4}$ ) in setting cleanup goals under Superfund (EPA, 1994b).

#### 3.5.7 Uncertainties in the Health Risk Assessment

Scientific uncertainties are associated with site chemical characterization, estimates of exposures, dose-response relationships and risks presented in any human health evaluation. It is generally accepted in the scientific community that the degree of uncertainty associated with quantitative risk assessments cannot be stated in absolute terms.

The uncertainties in this report are unavoidable in that they depend, to a greater or lesser extent, upon many technical judgments and imperfect mathematical models of the physical, chemical and biological processes involved.

An uncertainty present in any risk assessment is that the exposure point concentrations utilized in the analysis are assumed to be representative of the entire Site as a whole. Use of the 95% UCL of the arithmetic mean concentrations for the environmental and biota sampling that was conducted help to ensure that the analysis provides a realistic estimate of the range of potential human exposures. It is also assumed in the risk analysis process that the exposure point concentrations have not changed since sampling was conducted and will not change over time; i.e., that they remain constant.

The toxicity data for the HRA were obtained from observed toxic effects in humans or from laboratory animal experiments. The HRA involves the extrapolation and inference of adverse health effects under the expected conditions of human exposure. These health effects may have been based on adverse effects that occurred under conditions of exposure, and may have included different dose levels, different exposure routes, different populations, different species, etc. Thus, there exists many uncertainties concerning the extrapolation of the experimental conditions of exposure to those assumed to occur in this HRA.

The risk estimates are derived from independent analyses of exposure and dose/risk characterization. The parameter values chosen for use in these analyses were the reasonable maximum estimates (at or above the 90% upper-bound confidence interval) of their actual expected values. It is unlikely that actual risks calculated for any pathway will exceed those predicted from this high end (reasonable maximum) estimate. Inasmuch as most of the risk estimates discussed were developed using these assumptions and conservatively-derived parameter values, the resulting calculated risks are extremely unlikely to underestimate any actual risks involved.

#### 3.6 Summary and Conclusions

This HRA includes an evaluation of levels of PCBs detected in sludge bed, surface water, off-shore sludge deposists, beach/sand cores and wood/debris at the Cumberland Bay Wilcox Dock Site. This HRA is limited to the existing conditions found at the Site and nearby beach areas that may be due to Site influences.

Cumberland Bay is located on the west side of Lake Champlain, directly east of the City of Plattsburgh, New York. The water depths at the Site do not exceed 17 feet and are generally less than 10 feet. The Saranac River flows into the bay south of the Site and the Dead Creek flows into

the bay from the opposite side, north and east of the Site. The north shoreline of Cumberland Bay is occupied by the Plattsburgh Chamber of Commerce Building, the Plattsburgh Municipal Beach, a campground operated by the Office of Parks and Recreation, and numerous hotels and restaurants. To the east, Cumberland Head, a large peninsula, extends into the Bay. Wilcox Dock is located on the northwest shoreline of the bay and extends out into the bay approximately 400 feet. A breakwater owned and operated by the New York State Throughway Authority (NYSTA) is found just south of the dock, and creates a calm inlet area.

The Site and immediately surrounding area is currently used primarily for recreational activities including boating, fishing and swimming. Recreational fishing is common in this area although a fish advisory has been issued by the NYSDEC limiting fish consumption. Commercial fishing has been banned. It is expected that the Site and surrounding area will continue to be used for these purposes in the future. Therefore, this HRA evaluated the potential exposure of nearby residents and area visitors to PCBs present in environmental media and biota at the Site and immediate surrounding area (i.e., beach areas to the north of the sludge). The evaluation of potential future exposures assumes that the levels of chemical contaminants will not change over time.

Environmental data collected in the SC, and by the NYSDEC and NYSDOH revealed that the sludge bed, off-shore deposits of sludge, surface water, and beach sand and wood chips/debris (from beach areas to the north) contain detectable levels of PCBs. It is expected that the recreational activities directly over the sludge bed would be limited to wading activities because of restricted access to Wilcox Dock and the relatively heavy aquatic vegetation immediately off the shoreline. Therefore, potential limited exposure of nearby residents and area visitors (current and future) to the surface water and sludge bed material during wading activities were evaluated in this HRA. Because the Route 9 and public beach areas to the north are used for recreational purposes, swimming and recreational beach activities (e.g., sun-bathing and playing) were also evaluated. In addition, recreational fishing in Cumberland Bay was also evaluated in this HRA.

The toxicity assessment of PCBs included a review of the most up-to-date regulatory and toxicology computerized databases and information. This information was reviewed and then summarized in a toxicology profile. This information was then used to characterize the toxic hazards and risks.

Exposures to PCBs were quantitated using actual environmental analytical data. These environmental data were validated when possible and then used to estimate potential noncarcinogenic and carcinogenic risks following EPA risk assessment guidelines.

The potential health risks posed by each of the exposure pathways identified in this HRA are summarized in Table 3-22. Based on the exposure pathways presented in this baseline HRA, there are two current and potential future exposure scenarios that may pose a potential long-term health concern. These exposure pathways are:

- direct contact with sludge bed and surface water while wading in the sludge bed near Wilcox Dock; and
- · recreational ingesting of Cumberland Bay fish.

Each of these exposure pathways may pose a risk of chronic systemic effects and risk of developing cancer due to the presence of PCBs.

#### LITERATURE CITED IN SECTION 3.0 (HRA)

- Barsotti, D.A., and J.P. Van Miller. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. Toxicology, 30(1): 31-44.
- EPA. 1988. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Final document, prepared for Office of Drinking Water. Cincinnati, OH, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, ECAO-CIN-414, i.p., April 1988;
- EPA. 1985. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Environmental Criteria and Assessment Office, ECAO-CIN-414, EPA-600/X-84-198, April 1985.
- EPA. 1984. Health Effects Assessment for Polychlorinated Biphenyls. Final Draft. Cincinnati, Environmental Criteria and Assessment Office, ECAO-CIN-HO04, EPA-540/1-86/004, 55 pp., September 1984.
- Gosselin, R.E., et al. 1984. Clinical Toxicology of Commercial Products, 5th Edition. Baltimore, Williams & Wilkins.
- Hazardous Substances Data Bank (HSDB). 1997. *Polychlorinated Biphenyls*. National Library of Medicine, National Toxicology Information System, Bethesda, MD.
- HEAST. 1995. Health Effects Assessment Summary Tables, FY-1995 Annual. Office of Research and Development, Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, DC. OERR 9200.6-303 (95-1). May 1995.
- Integrated Risk Information System (IRIS) Database. 1997. Polychlorinated Biphenyls, Aroclor 1016, Aroclor 1248, Aroclor 1254, and Aroclor 1260. Environmental Protection Agency, Washington, DC. March 19, 1997.
- Ito, N., et al. 1978. Enhancing effect of chemicals on production of hyperplastic liver nodules induced by N-2-fluorenylacetamide in hepatectomized rats. Gann. 69:143-144, 1978;
- Kimbrough, R.D. and Linder, R.E. 1974. Induction of adenofibrosis and hepatomas of the liver in BALB/cj mice by polychlorinated biphenyls (Aroclor 1254). J. Natl. Cancer Inst. 53(2): 547-552.
- Kimbrough, R.D., et al. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl (Aroclor 1260). J. Natl. Cancer Inst. 55(6): 1453-1459.

- Kimura, N.T., Kanematsu, T. and Baba, T. 1976. Polychlorinated biphenyl(s) as a promoter in experimental hepatocarcinogenesis in rats. Z. Krebsforsch. 87: 257-266.
- Kitigawa, T. and Sugano, H. Enhancement of azo-dye hepatocarcinogenesis with dietary phenobarbital in rats. Gann. 68: 255-256.
- Klaassen, C.D., M.O. Amdur, and J. Doull (eds.). 1991. Casarett and Doull's Toxicology-The Basic Science of Poisons, Fourth Edition. New York, Macmillan. 1991.
- Nagasaki, H., et al. 1972. Hepatocarcinogenicity of polychlorinated biphenyls in mice. Gann. 63: 805.
- NCI. 1978. Bioassay of Aroclor 1254 for possible carcinogenicity. National Cancer Institute, DHEW Publication Number (NIH) 78-838.
- Nishizumi, M. 1976. Enhancement of diethylnitrosamine hepatocarcinogenesis in rats by exposure to polychlorinated biphenyls or phenobarbital. Cancer Lett. 2: 11-6.
- Norback, D. H. and Weltman, R.H. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 60: 97-105.
- Pantaleoni, G.C., et al. 1988. Effects of maternal exposure to polychlorinated biphenyls (PCBs) on  $F_1$  generation behavior in the rat. Fund. and Appl. Toxicol. 11: 440-449.
- Peraino, C., Fry, R.J.M. and Staffeldt, E. 1971. Reduction and enhancement by phenobarbital of hepatocarcinogenesis induced in the rat by 2-acetylaminofluorene. Cancer Res. 31: 1506-1512.
- Peraino, C., et al. 1974. Comparative enhancing effects of phenobarbital, amobarbital, diphenylhydantoin, and dichlorodiphenyl-trichloroethane of 2-acetylaminofluorene-induced hepatic tumorigenesis in the rat. Cancer Res. 35: 2884-2890.
- Reynolds, S.H., et al. 1987. Activated oncogenes in B6C3F<sub>1</sub> mouse liver tumors: implications for risk assessment. Science 237: 1309-1316.
- Rogan, W.J., et al. 1988. Cogenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan. Science 241: 334-341.
- Taylor, P.R., Stelma, J.M. and Lawrence, C.E. 1989. The relation of polychlorinated biphenyls to birth weight and gestational age in the offspring of occupationally exposed mothers. Amer. J. Epidemiol. 129(2): 395-406.
- Weisburger, J.H., et al. 1975. Modification of dimethylnitrosamine liver carcinogenesis with phenobarbital but not with immunosuppression. J. Natl. Cancer Inst. 54: 1185-1188.

**SECTION 3.0** 

**TABLES** 

TABLE 3-1

## INCLUSION/EXLUSION ANALYSIS OF POTENTIAL EXPOSURE PATHWAYS/ROUTES FOR THE SLUDGE BED-WILCOX DOCK HEALTH RISK ASSESSMENT

#### CUMBERLAND BAY PLATTSBURGH, NEW YORK

Potentially-Exposed Populations	Exposure Medium	Exposure Pathway/Route	Exposure Point	Pathway Selected?	Rationale
Current Nearby Resident/ Visitor	Air	Inhalation of volatiles/dusts	On-site contact/ Off-site transport	No	PCBs are relatively non-volatile and tend to bind to soils. with high organic content. Sludge bed and beach debris are generally covered with water or are wet due to wave action, thereby, precluding suspension in air. Air sampling results during SC activities were non-detect for PCBs.
	Sludge Bed	Incidental ingestion; Dermal contact	On-site contact	Yes	Area is used for boat mooring and fishing. Potential for wading during these activities. Swimming not expected due to restricted access and aquatic vegetation.
	Beach Sand and Wood Chips/Debris	Incidental ingestion; Dermal contact	On-site contact	Yes	Recreational use of area. Potential for direct contact during swimming, sun-bathing, and playing on beach areas to the north.
	Surface Water	Incidental ingestion; Dermal contact	On-site contact	Yes	Recreational use of area. Potential for direct contact during swimming, sun-bathing, and playing on beach areas to the north.
	Fish	Recreational ingestion	On-site	Yes	Area used for recreational fishing. Commercial ban and fish consumption advisories established. However, recreational fish ingestion may occur.

TABLE 3-2
SUMMARY OF PCB CONCENTRATIONS DETECTED NEAR WILCOX DOCK<sup>1</sup>

	Minimum	Maximum			Frequency
MALA Signal	Detected	Detected	Average	95%	of
Parameter	Concentration	Concentration	Concentration	UCL	Detection
Exposure Scenario -	Wading in Sludge l	Bed Area Near	Wilcox Dock		
(Sludge Bed Materia	THE RESERVE AND THE PARTY OF TH				
Aroclor - 1221	BRDL	BRDL			0/87
Aroclor - 1242	0.023	1850			69/87
Aroclor - 1248	BRDL	BRDL			0/87
Aroclor - 1254	0.170	0.170			1/87
Aroclor - 1260	0.130	4.1			2/87
Total PCBs	0.023	1850	85.186	141.096	70/87
Exposure Scenario -	Swimming in Beacl	h Areas North o	f Sludge Bed		
(Beach Cores/Wood	Debris & Off-Shore	e Sludge: 0-1 foo	ot depth)		
Aroclor - 1221	BRDL	BRDL	The state of the s		0/13
Aroclor - 1242	0.002	14			10/13
Aroclor - 1248	0.022	0.022			1/13
Aroclor - 1254	0.0014	0.0014			1/13
Aroclor - 1260	0.48	0.48			1/13
Total PCBs	0.002	14.48	1.768	4.192	11/13
Exposure Scenario -	- Recreational Use of	f Beach Areas N	orth of Sludge	Bed	
	Debris: 0-1 foot de		and the contract of		
Aroclor - 1221	BRDL	BRDL			0/11
Aroclor - 1242	0.002	14			10/11
Aroclor - 1248	BRDL	BRDL			0/11
Aroclor - 1254	0.0014	0.0014			1/11
Aroclor - 1260	0.48	0.48			1/11
Total PCBs	0.002	14.48	2.085	4.980	10/11
Exposure Scenario	- Recreational Inges	tion of Cumber	land Bay Fish		
(Yellow Perch Sam)					
Aroclor - 1221	BRDL	BRDL			0/21
Aroclor - 1242	BRDL	BRDL			0/21
Aroclor - 1248	0.790	18.000			21/21
Aroclor - 1254	0.340	8.880			12/21
Aroclor - 1260	BRDL	BRDL			0/21
		18.000	5.730	8.213	21/21

<sup>1 -</sup> All concentrations expressed in mg/kg (parts per million or ppm).

BRDL = Below Reportable Detection Limit

Average Concentration = Arithmetic average concentration, assuming a value of on-half the detection limit for non-detected results.

95% UCL = 95 percent upper confidence limit on the arithmetic average concentration.

TABLE 3-3

EXPOSURE FROM SURFACE WATER CONTACT WHILE WADING IN SLUDGE BED AREA NEAR WILCOX DOCK: AREA RESIDENT EXPOSURE - ADOLESCENT

thway: Ingestion of surface water -	adolescent (7-1	6 yrs)		111111111111111111111111111111111111111	1 1			7777	To Fair He William		
CW Surface Water Conc. (mg/L)	IR Ingestion Rate (L)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
0.00031	0.01	1	1	52	10	1	40	25550	3650	1.58E-09	1.10E-08
thway: Dermal absorption of surfa	ce water - adole	scent (7-16 yr)	15 W.		-						1.4.6.4
CW Surface Water Conc. (mg/L)	SA Surface Area (sq cm)	CF Conversion Factor	PC Permeability Constant (mg/sq cm/hr)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ET Exposure Time (hours/day)	BW Body Weight (kg)	AT Averaging Time	AT Averaging Time	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
0.00031	3600	0.001	9.98E-02	52	10	0.5	40	25550	3650	2.83E-08	1.98E-07
	CW Surface Water Conc. (mg/L)  0.00031  thway: Dermal absorption of surface Water Conc. (mg/L)	CW IR Surface Water Ingestion Conc. Rate (mg/L) (L)  0.00031 0.01  thway: Dermal absorption of surface water - adole  CW SA Surface Water Surface Conc. Area (mg/L) (sq cm)	Surface Water Conversion Conc. Rate Conversion (mg/L) (L) Factor  0.00031 0.01 1  thway: Dermal absorption of surface water - adolescent (7-16 yr)  CW SA CF Surface Water Surface Conc. Area Conversion (mg/L) (sq cm) Factor	CW IR CF FI Surface Water Ingestion Conc. Rate Conversion Fraction (mg/L) (L) Factor Ingested  0.00031 0.01 1 1  thway: Dermal absorption of surface water - adolescent (7-16 yr)  CW SA CF PC Surface Water Surface Permeability Conc. Area Conversion Constant (mg/L) (sq cm) Factor (mg/sq cm/hr)	CW IR CF FI EF Surface Water Ingestion Conc. Rate Conversion Fraction Frequency (mg/L) (L) Factor Ingested (days/year)  0.00031 0.01 1 1 52  thway: Dermal absorption of surface water - adolescent (7-16 yr)  CW SA CF PC EF Surface Water Surface Permeability Exposure Conc. Area Conversion Constant Frequency (mg/L) (sq cm) Factor (mg/sq cm/hr) (days/year)	CW IR CF FI ESposure Exposure Conc. Rate Conversion Fraction Ingested (days/year)  0.00031 0.01 1 1 52 10  CW SA CF PC EF ED Surface Water Surface Conversion Constant (mg/L) (sq cm) Factor (mg/sq cm/hr)  CW SA CF PC EF ED Exposure Exposure Exposure Conc. Area Conversion Constant Frequency (days/year) (years)	CW IR CF FI EF ED ABS Surface Water Ingestion	CW IR CF FI EF ED ABS BW Surface Water Ingestion	Cancer CW IR CF FI EF ED ABS BW AT Surface Water Ingestion Conc. Rate Conversion Fraction Ingested (days/year)  UNIT OF THE CONVERSION FRACTOR (May Ingested Conversion Ingested (days/year)  UNIT OF THE CONVERSION FRACTOR (May Ingested Conversion Ingested Conversion Ingested (days/year)  UNIT OF THE CONVERSION INGESTINATION INTERPRETATION INTE	Cancer Non-Cancer CW IR CF FI EF ED ABS BW AT AT Surface Water Ingestion Conc. Rate Conversion Fraction Frequency Duration Absorption Weight Time Time (mg/L) (L) Factor Ingested (days/year) (years) Factor (kg) (days) (days)  0.00031 0.01 1 1 52 10 1 40 25550 3650  thway: Dermal absorption of surface water - adolescent (7-16 yr)  CANCER Non-Cancer (kg) (days) (days)  Cancer Non-Cancer CW SA CF PC EF ED ET BW AT AT Surface Water Surface Permeability Exposure Exposure Exposure Body Averaging Averaging Conc. Area Conversion Constant Frequency Duration Time Weight Time Time (mg/L) (sq cm) Factor (mg/sq cm/hr) (days/year) (years) (hours/day) (kg) (days) (days)	CW IR CF FI EF ED ABS BW AT AT LADD Surface Water Ingestion

#### EXPOSURE FROM SLUDGE CONTACT WHILE WADING IN SLUDGE BED AREA NEAR WILCOX DOCK: AREA RESIDENT EXPOSURE - ADOLESCENT

Hypothetical Exposure Pat	thway: Ingestion of sludge - adole	scent (7-16 yrs)							1.00			
Analyte	CS Sludge Conc. (mg/kg)	IR Ingestion Rate (mg/day)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	141.096	100	0.000001	1	52	10	0.3	40	25550	3650	2.15E-06	1.51E-05
Hypothetical Exposure Pat	thway: Dermal absorption of slud	ge - adoloescent (	7-16 yrs)	Table 1			100	Y STATE	Cancer	Non-Cancer	17	
Analyte	CS Sludge Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	AT Averaging Time (days)	AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	141.096	3600	0.000001	1.0	52	10	0.05	40	25550	3650	1.29E-05	9.05E-05

TABLE 3-4

EXPOSURE FROM SURFACE WATER CONTACT WHILE WADING IN SLUDGE BED AREA NEAR WILCOX DOCK: AREA RESIDENT EXPOSURE - ADULT

Hypothetical Exposure P	athway: Ingestion of surface water -	adult	THE RESERVE	- Y-49-10	TO VIEW OF THE PARTY OF THE PAR				1. 1860.7	No a Strain	PERM	
Analyte	CW Surface Water Conc. (mg/L)	IR Ingestion Rate (L)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00031	0.01	1	1	52	20	1	70	25550	7300	1.80E-09	6.31E-09
Hypothetical Exposure P	athway: Dermal absorption of surfa	ce water - adult			Series :					- 100	7 10 10	
Analyte	CW Surface Water Conc. (mg/L)	SA Surface Area (sq cm)	CF Conversion Factor	PC Permeability Constant (mg/sq cm/hr)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ET Exposure Time (hours/day)	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00031	5170	0.001	9.98E-02	52	20	0.5	70	25550	7300	4.65E-08	1.63E-07

#### EXPOSURE FROM SLUDGE CONTACT WHILE WADING IN SLUDGE BED AREA NEAR WILCOX DOCK: AREA RESIDENT EXPOSURE - ADULT

Hypothetical Exposure Pati	way: Ingestion of sludge - adult			5 T. T. T.								
Analyte	CS Sludge Cone. (mg/kg)	IR Ingestion Rate (mg/day)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	141.096	100	0.000001	1	52	20	0.3	70	25550	7300	2.46E-06	8.61E-06
Hypothetical Exposure Path	way: Dermal absorption of slud	ge - adult									1000	
Analyte	CS Sludge Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Dàily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	141.096	5170	0.000001	1.0	52	20	0.05	70	25550	7300	2.12E-05	7.42E-05

TABLE 3-5

# EXPOSURE FROM SLUDGE AND SURFACE WATER CONTACT WHILE WADING IN SLUDGE BED AREA NEAR WILCOX DOCK: AREA RESIDENT EXPOSURE - SUMMARY

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day)	Total Chronic Daily Intake <sup>1</sup> (mg/kg/day)
PCBs - Sludge Bed	3.87E-05	1.06E-04
PCBs - Surface Water	7.82E-08	2.09E-07

<sup>1 -</sup> CDI for adolecent was utilized since this represents the most sensitive receptor under this exposure scenario

TABLE 3-6

EXPOSURE TO SURFACE WATER WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - YOUNG CHILD

Hypothetical Exposure P	athway: Ingestion of surface water -	young child (1-	6 yrs)			377						30.
Analyte	CW Surface Water Conc. (mg/L)	CR Contact Rate (L/hour)	CF Conversion Factor	ET Exposure Time (hours/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00021	0.01	1	1	34	6	1	16	25550	2190	1.05E-09	1.22E-08
Hypothetical Exposure P	athway: Dermal absorption of surfa	ce water young	child (1-6 yrs)								S. Park	770
Analyte	CW Surface Water Conc. (mg/L)	SA Surface Area (sq cm)	CF Conversion Factor	PC Permeability Constant (sq cm/hr)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ET Exposure Time (hours/day)	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00021	7280	0.001	9.98E-02	34	6	-1	16	25550	2190	7.61E-08	8.88E-07

#### EXPOSURE TO SLUDGE WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - YOUNG CHILD

way: Ingestion of sludge - your	ng child (1-6 yrs)										
CS Sludge Conc. (mg/kg)	IR Ingestion Rate (mg/day)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
4.192	200	0.000001	0.5	34	6	0.3	16	25550	2190	6.28E-08	7.32E-07
way: Dermal absorption of sluc	dge - young child	(1-6 yrs)							9,71		
CS Sludge Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
4.192	510	0.000001	1.0	34	6	0.05	16	25550	2190	5.33E-08	6.22E-07
	CS Sludge Conc. (mg/kg)  4.192  way: Dermal absorption of sludge CS Sludge Conc. (mg/kg)	Sludge Ingestion Conc. Rate (mg/kg) (mg/day)  4.192 200  way: Dermal absorption of sludge - young child  CS SA Sludge Surface Conc. Area (mg/kg) (sq cm)	CS IR CF Sludge Ingestion Conc. Rate Conversion (mg/kg) (mg/day) Factor  4.192 200 0.000001  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF Sludge Surface Conc. Area Conversion (mg/kg) (sq cm) Factor	CS IR CF FI Sludge Ingestion Conc. Rate Conversion Fraction (mg/kg) (mg/day) Factor Ingested  4.192 200 0.000001 0.5  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF Sludge Surface Adherence Conc. Area Conversion Factor (mg/kg) (sq cm) Factor (mg/sq cm/day)	CS IR CF FI Exposure Conc. Rate Conversion Fraction Frequency (mg/kg) (mg/day) Factor Ingested (days/year)  4.192 200 0.000001 0.5 34  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF EF Sludge Surface Adherence Exposure Conc. Area Conversion Factor Frequency (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year)	CS IR CF FI Exposure Exposure Conc. Rate Conversion Ingested (days/year) (years)  4.192 200 0.000001 0.5 34 6  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF EF ED Sludge Surface Adherence Exposure Exposure Conc. Area Conversion Factor Frequency (days/year) (years)	CS IR CF FI EF ED ABS Sludge Ingestion Conc. Rate Conversion Fraction Ingested (days/year)  4.192 200 0.000001 0.5 34 6 0.3  CS SA CF AF EF ED ABS Sludge Surface Conversion Factor Frequency (days/year)  CS SA CF AF EF ED ABS Sludge Surface Adherence Exposure Exposure Feative Prequency (days/year)  CS SA CF AF EF ED ABS Sludge Surface Adherence Exposure Exposure Relative Prequency Duration Absorption (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor	CS IR CF FI EF ED ABS BW Sludge Ingestion Conc. Rate Conversion Fraction Ingested (days/year)  (mg/kg) (mg/day) Factor Ingested (days/year)  CS SA CF AF EF ED ABS BW Sludge Surface Adherence Exposure Exposure Relative Body  CS SA CF AF EF ED ABS BW Sludge Surface Conversion Factor Frequency (days/year)  CS SA CF AF EF ED ABS BW Sludge Surface Exposure Exposure Relative Body Conc. Area Conversion Factor Frequency Duration Absorption Weight (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg)	CS IR CF FI EF ED ABS BW AT Sludge Ingestion Conc. Rate Conversion (mg/kg) (mg/day) Factor Ingested (days/year) (years) Factor (kg) (days)  4.192 200 0.000001 0.5 34 6 0.3 16 25550  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF EF ED ABS BW AT Sludge Surface Adherence Exposure Exposure Relative Body Averaging Conc. Area Conversion Factor Frequency Duration Absorption Weight Time (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg) (days)	CS IR CF FI EF ED ABS BW AT AT SIudge Ingestion Conc. Rate Conversion Fraction Ingested (days/year) (years) Factor (kg) (days) (days) (days)  4.192 200 0.000001 0.5 34 6 0.3 16 25550 2190  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF EF ED ABS BW AT AT SIudge Surface Adherence Exposure Exposure Relative Body Averaging Averaging (days/year) (years) Factor Relative Body Averaging Averaging Conc. Area Conversion Factor Frequency Duration Absorption Weight Time Time (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg) (days) (days) (days)	CS IR CF FI EF ED ABS BW AT AT LADD  Sludge Ingestion Fraction Fraction Frequency Duration Absorption Weight Time Daily Dose (mg/kg) (mg/day) Factor Ingested (days/year) (years) Factor (kg) (days) (days) (days) (mg/kg/day)  4.192 200 0.000001 0.5 34 6 0.3 16 25550 2190 6.28E-08  way: Dermal absorption of sludge - young child (1-6 yrs)  CS SA CF AF EF ED ABS BW AT AT AT LADD  Sludge Surface Adherence Exposure Exposure Relative Body Averaging Averaging Lifetime Avg.  Cancer Non-Cancer  Cancer Non-Cancer

TABLE 3-7

EXPOSURE TO SURFACE WATER WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - ADOLESCENT

Hypothetical Exposure Pat	hway: Ingestion of surface water -	adolescent (7-1	6 yrs)			100		-37	1.10.12.2	411000000		
Analyte	CW Surface Water Conc. (mg/L)	CR Contact Rate (L/hour)	CF Conversion Factor	ET Exposure Time (hours/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00021	0.02	1	1	34	10	1	40	25550	3650	1.40E-09	9.78E-09
Hypothetical Exposure Pat	hway: Dermal absorption of surface	ce water - adole	scent (7-16 yrs)									7500
Analyte	CW Surface Water . Conc. (mg/L)	SA Surface Area (sq cm)	CF Conversion Factor	PC Permeability Constant (sq cm/hr)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ET Exposure Time (hours/day)	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	0.00021	13200	0.001	9.98E-02	34	10	1	40	25550	3650	9.20E-08	6.44E-07

#### EXPOSURE TO SLUDGE WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - ADOLESCENT

Hypothetical Exposure Pati	hway: Ingestion of sludge - adole	escent (7-16 yrs)		]	2.0				Cancer	Non-Cancer		
Analyte	CS Sludge Conc. (mg/kg)	IR Ingestion Rate (mg/day)	Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	AT Averaging Time (days)	AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.192	100	0.000001	0.5	34	10	0.3	40	25550	3650	2.09E-08	1.46E-07
Hypothetical Exposure Pati	hway: Dermal absorption of sluc	dge - adolescent (7	-16 yrs)			4 75		E - 1800				-
Analyte	CS Sludge Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.192	924	0.000001	1.0	34	10	0.05	40	25550	3650	6.44E-08	4.51E-07

TABLE 3-8

EXPOSURE TO SURFACE WATER WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - ADULT

thway: Ingestion of surface water -	adult							1081			
CW Surface Water Conc. (mg/L)	CR Contact Rate (L/hour)	CF Conversion Factor	ET Exposure Time (hours/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	AT Averaging Time (days)	AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
0.00021	0.05	1	1	34	14	1	70	25550	5110	2.79E-09	1.40E-08
thway: Dermal absorption of surfa	ce water - adult					120.0	THE RE				
CW Surface Water Conc. (mg/L)	SA Surface Area (sq cm)	CF Conversion Factor	PC Permeability Constant (sq cm/hr)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ET Exposure Time (hours/day)	BW Body Weight (kg)	AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
0.00021	19400	0.001	9.98E-02	34	14	1	70	25550	5110	1.08E-07	5.41E-07
	CW Surface Water Conc. (mg/L)  0.00021  thway: Dermal absorption of surface CW Surface Water Conc. (mg/L)	Surface Water Contact Conc. Rate (mg/L) (L/hour)  0.00021 0.05  thway: Dermal absorption of surface water - adult  CW SA Surface Water Surface Conc. Area (mg/L) (sq cm)	CW CR CF Surface Water Contact Conc. Rate Conversion (mg/L) (L/hour) Factor  0.00021 0.05 1  thway: Dermal absorption of surface water - adult  CW SA CF Surface Water Surface Conc. Area Conversion (mg/L) (sq cm) Factor	CW CR CF ET  Surface Water Contact Exposure  Conc. Rate Conversion Time (mg/L) (L/hour) Factor (hours/day)  0.00021 0.05 1 1  thway: Dermal absorption of surface water - adult  CW SA CF PC  Surface Water Surface Permeability Conc. Area Conversion Constant (mg/L) (sq cm) Factor (sq cm/hr)	CW CR CF ET EF Surface Water Contact Exposure Conc. Rate Conversion Time Frequency (mg/L) (L/hour) Factor (hours/day) (days/year)  0.00021 0.05 1 1 34  thway: Dermal absorption of surface water - adult  CW SA CF PC EF Surface Water Surface Permeability Exposure Conc. Area Conversion Constant Frequency (mg/L) (sq cm) Factor (sq cm/hr) (days/year)	CW CR CF ET EF ED  Surface Water Contact Conce.  Rate Conversion Factor (hours/day)  0.00021  0.05  1  1  34  14  Thway: Dermal absorption of surface water - adult  CW SA CF PC EF ED  Surface Water Surface Conversion Constant Frequency (mg/L)  (mg/L) (sq cm) Factor (aq cm/hr)  (sq cm/hr)  Exposure Exposure Exposure Exposure Exposure Conce. Area Conversion Constant Frequency (days/year)  ED  CONTACT PC  EF ED  ED  ED  EF ED  EXPOSURE  EXPOSURE	CW CR CF ET EF ED ABS Surface Water Contact Conc. Rate Conversion Time Frequency (days/year)  (mg/L) (L/hour) Factor  0.00021  0.05  1  1  34  14  1  thway: Dermal absorption of surface water - adult  CW SA CF PC EF ED ET Surface Water Surface Conc. Area Conversion Constant Frequency (mg/L) (sq cm)  Factor  Exposure Exposure (years)  EXPOSURE EF ED ET EXPOSURE EXPOSURE EXPOSURE EXPOSURE EXPOSURE EXPOSURE EXPOSURE (mg/L) (sq cm)  Factor  Constant Frequency Duration Time (mg/L) (sq cm)  Factor (sq cm/hr) (days/year) (years) (hours/day)	CW CR CF ET EF ED ABS BW Surface Water Contact Exposure Exposure Exposure Relative Body (mg/L) (L/hour) Factor (hours/day) (days/year) (years) Factor (kg)  0.00021 0.05 1 1 34 14 1 70  thway: Dermal absorption of surface water - adult  CW SA CF PC EF ED ET BW Surface Water Surface Permeability Exposure Exposure Exposure Exposure Body Conc. Area Conversion Constant Frequency Duration Time Weight (mg/L) (sq cm) Factor (sq cm/hr) (days/year) (years) (hours/day) (kg)	CW CR CF ET EF ED ABS BW AT Surface Water Contact Exposure Exposure Relative Body Averaging Conc. Rate Conversion Time Frequency Duration Absorption Weight Time (mg/L) (L/hour) Factor (hours/day) (days/year) (years) Factor (kg) (days)  0.00021 0.05 1 1 34 14 1 70 25550  thway: Dermal absorption of surface water - adult  CW SA CF PC EF ED ET BW AT Surface Water Surface Permeability Exposure Exposure Exposure Body Averaging Conc. Area Conversion Constant Frequency Duration Time Weight Time (mg/L) (sq cm) Factor (sq cm/hr) (days/year) (years) (hours/day) (kg) (days)	CW CR CF ET EF ED ABS BW AT AT Surface Water Contact Exposure Exposure Exposure Exposure Relative Body Averaging Averaging Conc. Rate Conversion Time Frequency Duration Absorption Weight Time Time (mg/L) (L/hour) Factor (hours/day) (days/year) (years) Factor (kg) (days) (days) (days)  0.00021 0.05 1 1 34 14 1 70 25550 5110  thway: Dermal absorption of surface water - adult  Cancer Non-Cancer CW SA CF PC EF ED ET BW AT AT Surface Water Surface Permeability Exposure Exposure Exposure Body Averaging Averaging Conc. Area Conversion Constant Frequency Duration Time Weight Time Time (mg/L) (sq cm) Factor (sq cm/hr) (days/year) (years) (hours/day) (kg) (days) (days)	CW CR CF ET EF ED ABS BW AT AT LADD Surface Water Contact Conc. Rate Conversion Time Frequency (days/year)  O.00021 0.05 1 1 34 14 1 70 25550 5110 2.79E-09  thway: Dermal absorption of surface water - adult  CW SA CF PC EF ED ET BW AT AT LADD Surface Water Surface Permeability Exposure Exposure Exposure Exposure Body Averaging Averaging Lifetime Avg.  Cancer Non-Cancer

#### EXPOSURE TO SLUDGE WHILE SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - ADULT

Hypothetical Exposure Path	way: Ingestion of sludge - adul	t			40.0	750		1	7.4			
Analyte	CS Sludge Conc. (mg/kg)	IR Ingestion Rate (mg/day)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.192	100	0.000001	0.5	34	14	0.3	70	25550	5110	1.67E-08	8.37E-08
Hypothetical Exposure Path	way: Dermal absorption of slu	dge - adult								N. C.	6.10.5	
Analyte	CS Sludge Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.192	1358	0.000001	1.0	34	14	0.05	70	25550	5110	7.58E-08	3.79E-07

TABLE 3-9

### POTENTIAL EXPOSURE FROM SWIMMING IN BEACH AREA NORTH OF SLUDGE BED - SUMMARY (mg/kg/day)

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day)	Total Chronic Daily Intake <sup>1</sup> (mg/kg/day)
PCBs - Sludge	2.94E-07	1.35E-06
PCBs - Surface Water	2.81E-07	9.00E-07

<sup>(1)</sup> CDI for a young child was utilized since this represents the most sensitive individual under this exposure scenario.

TABLE 3-10

EXPOSURE TO SAND AND WOOD DEBRIS DURING RECREATIONAL USE OF THE BEACH AREA NORTH OF SLUDGE BED - YOUNG CHILD

Hypothetical Exposure Pat	hway: Ingestion of sand/wood de	ebris - young child	(1-6 yrs)		V.	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)		La Contraction	3.50	CDI Chronic Daily Intake (mg/kg/day)
Analyte	CS Debris Conc. (mg/kg)	IR Ingestion Rate (mg/day)	CF Conversion Factor	FI Fraction Ingested	EF Exposure Frequency (days/year)				Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	
PCBs	4.980	200	0.000001	0.5	34	6	0.3	16	25550	2190	7.46E-08	8.70E-07
Hypothetical Exposure Pat	hway: Dermal absorption of san	d/wood debris - ye	oung child (1-6 yr	1)			100		-		_	
Analyte	CS Debris Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.980	7280	0.000001	1.0	34	6	0.05	16	25550	2190	9.05E-07	1.06E-05

TABLE 3-11

EXPOSURE TO SAND AND WOOD DEBRIS DURING RECREATIONAL USE OF THE BEACH AREA NORTH OF SLUDGE BED - ADOLESCENT

Hypothetical Exposure Pat	CS IR CF  Debris Ingestion  Conc. Rate Conversion  (mg/kg) (mg/day) Factor	(7-16 yrs)	10.7	100			100	0.370	1 10 10 10 10	Carlo Sec.		
Analyte		FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)		
PCBs	4.980	100	0.000001	0.5	34	10	0.3	40	25550	3650	2.49E-08	1.74E-07
Hypothetical Exposure Pat	thway: Dermal absorption of san	d/wood debris - a	dolescent (7-16 yrs	)	E		-					
Analyte	CS Debria Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	4.980	13200	0.000001	1.0	34	10	0.05	40	25550	3650	1.09E-06	7.65E-06

TABLE 3-12

EXPOSURE TO SAND AND WOOD DEBRIS DURING RECREATIONAL USE OF THE BEACH AREA NORTH OF SLUDGE BED - ADULT

way: Ingestion of sand/wood de	ebris - adult										
CS IR CF Debris Ingestion alyte Conc. Rate Conversion (mg/kg) (mg/day) Factor	Conversion	FI Fraction Ingested	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	AT Averaging Time (days)	AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)	
4.980	100	0.000001	0.5	34	14	0.3	70	25550	5110	1.99E-08	9.94È-08
way: Dermal absorption of san	d/wood-debris - a	dult									
CS Debris Conc. (mg/kg)	SA Surface Area (sq cm)	CF Conversion Factor	AF Adherence Factor (mg/sq cm/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
4.980	19400	0.000001	1.0	34	14	0.05	70	25550	5110	1.29E-06	6.43E-06
	CS Debris Conc. (mg/kg)  4.980  way: Dermal absorption of san  CS Debris Conc. (mg/kg)	Debris Ingestion Conc. Rate (mg/kg) (mg/day)  4.980 100  way: Dermal absorption of sand/wood debris - ac  CS SA Debris Surface Conc. Area (mg/kg) (sq cm)	CS IR CF Debris Ingestion Conc. Rate Conversion (mg/kg) (mg/day) Factor  4.980 100 0.000001  way: Dermal absorption of sand/wood-debris - adult  CS SA CF Debris Surface Conc. Area Conversion (mg/kg) (sq cm) Factor	CS IR CF FI Debris Ingestion Conc. Rate Conversion Fraction (mg/kg) (mg/day) Factor Ingested  4.980 100 0.000001 0.5  way: Dermal absorption of sand/wood debris - adult  CS SA CF AF Debris Surface Adherence Conc. Area Conversion Factor (mg/kg) (sq cm) Factor (mg/sq cm/day)	CS IR CF FI Exposure Conc. Rate Conversion Fraction Frequency (mg/kg) (mg/day) Factor Ingested (daya/year)  4.980 100 0.000001 0.5 34  way: Dermal absorption of sand/wood debris - adult  CS SA CF AF EF Debris Surface Adherence Exposure Conc. Area Conversion Factor Frequency (mg/kg) (sq cm) Factor (mg/sq cm/day) (daya/year)	CS IR CF FI Exposure Exposure Conc. Rate Conversion Ingested (days/year) (years)  4.980 100 0.000001 0.5 34 14  CS SA CF AF EF ED Debris Surface Conversion Factor Frequency (days/year) (years)	CS IR CF FI EF ED ABS Debris Ingestion Conc. Rate Conversion Fraction Ingested (days/year)  4.980 100 0.000001 0.5 34 14 0.3  CS SA CF AF EF ED ABS Debris Surface Adherence Conc. Area Conversion Factor (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year)  CS SA CF AF EF ED ABS Debris Surface Adherence Exposure Exposure Relative Frequency Duration Absorption (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor	CS IR CF FI EF ED ABS BW Debris Ingestion Conc. Rate Conversion Fraction Ingested (days/year)  (mg/kg) (mg/day) Factor Ingested (days/year)  4.980 100 0.000001 0.5 34 14 0.3 70  way: Dermal absorption of sand/wood debris - adult  CS SA CF AF EF ED ABS BW Debris Surface Adherence Exposure Exposure Relative Body Conc. Area Conversion Factor Frequency Duration Absorption Weight (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg)	CS IR CF FI EF ED ABS BW AT Debris Ingestion Conc. Rate Conversion (mg/kg) (mg/day) Factor Ingested (days/year) (years) Factor (kg) (days)  4.980 100 0.000001 0.5 34 14 0.3 70 25550  way: Dermal absorption of sand/wood debris - adult  CS SA CF AF EF ED ABS BW AT Debris Surface Adherence Exposure Exposure Relative Body Averaging Conc. Area Conversion Factor Frequency Duration Absorption Weight Time (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg) (days)	CS IR CF FI EF ED ABS BW AT AT AT Debris Ingestion (mg/kg) (mg/day) Factor Ingested (days/year) (years) Factor (kg) (days)	CS IR CF FI EF ED ABS BW AT AT LADD Debris Ingestion Conc. Rate Conversion Fraction Ingested (days/year) (years) Factor (kg) (days) (days) (days) (mg/kg) (days) (days) (mg/kg/day)  4.980 100 0.000001 0.5 34 14 0.3 70 25550 5110 1.99E-08  way: Dermal absorption of sand/wood debris - adult  Cancer Non-Cancer CS SA CF AF EF ED ABS BW AT AT AT LADD Debris Surface Adherence Exposure Exposure Relative Body Averaging Averaging Lifetime Avg. Conc. Area Conversion Factor Frequency Duration Absorption Weight Time Time Daily Dose (mg/kg) (sq cm) Factor (mg/sq cm/day) (days/year) (years) Factor (kg) (days) (days) (days) (mg/kg/day)

**TABLE 3-13** 

### POTENTIAL EXPOSURE FROM SAND AND WOOD CHIP DEBRIS DURING RECREATIONAL USE OF THE BEACH AREA NORTH OF SLUDGE BED - SUMMARY (mg/kg/day)

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day)	Total Chronic Dail Intake <sup>1</sup> (mg/kg/day)		
PCBs	3.40E-06	1.14E-05		

<sup>(1)</sup> CDI for a young child was utilized since this represents the most sensitive individual under this exposure scenario.

TABLE 3-14

EXPOSURE FROM RECREATIONAL INGESTION OF FISH FROM CUMBERLAND BAY

Hypothetical Exposure Pathw	ay: Ingestion of fish - young cl	hild (1-6 years)			New York	C Vallabellance	9 . 1		STORES !		
Analyte	CF Fish Conc. (mg/kg)	IR Ingestion Rate (kg/meal)	FI Fraction Ingested	EF Exposure Frequency (meals/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	8.213	0.085	1	48	6	1	16	25550	2190	4.92E-04	5.74E-03
Hypothetical Exposure Pathw	ay: Ingestion of fish - adolesce	nt (7-16 years)			477		175	100	F-15-01-		
Analyte	CF Fish Conc. (mg/kg)	IR Ingestion Rate (kg/meal)	FI Fraction Ingested	EF Exposure Frequency (meals/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	8.213	0.129	1	48	10	- 1	40	25550	3650	4.98E-04	3.48E-03
Hypothetical Exposure Pathw	ay: Ingestion of fish - adult							-		-	-
Analyte	CF Fish Conc. (mg/kg)	IR Ingestion Rate (kg/meal)	FI Fraction Ingested	EF Exposure Frequency (meals/year)	ED Exposure Duration (years)	ABS Relative Absorption Factor	BW Body Weight (kg)	Cancer AT Averaging Time (days)	Non-Cancer AT Averaging Time (days)	LADD Lifetime Avg. Daily Dose (mg/kg/day)	CDI Chronic Daily Intake (mg/kg/day)
PCBs	8.213	0.227	1	48	14	1	70	25550	5110	7.00E-04	3.50E-03

#### **TABLE 3.15**

### EXPOSURE FROM RECREATIONAL INGESTION OF FISH FROM CUMBERLAND BAY -SUMMARY

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day)	Total Chronic Dail Intake (mg/kg/day)	
PCBs	1.69E-03	5.74E-03	

<sup>(1)</sup> CDI for a young child was utilized since this represents the most sensitive individual under this exposure scenario.

**TABLE 3-16** 

# NONCARCINOGENIC AND CARCINOGENIC TOXICITY VALUES FOR TOTAL PCBS

# CUMBERLAND BAY PLATTSBURGH, NEW YORK

Effect	Toxicity Factor (mg/kg/day)	Source
Noncarcinogenic		
Chronic Reference Dose	2.0E-05	IRIS
Carcinogenic		
<b>EPA Classification</b>	B2	IRIS
High Risk and Persistence	2.0E+00	IRIS
Low Risk and Persistence	4.0E-01	IRIS
Lowest Risk and Persistence	7.0E-02	IRIS

IRIS = EPA's Integrated Risk Information System.

**TABLE 3-17** 

# EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY

GROUP	DESCRIPTION
Α .	Human carcinogen
В .	Probable human carcinogen
B1	indicates that limited human data are available
B2 .	indicates sufficient evidence in animals and inadequate or no evidence in humans
С	Possible human carcinogen
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

TABLE 3-18

POTENTIAL CARCINOGENIC AND NONCARCINOGENIC HEALTH RISKS FROM WADING IN SLUDGE BED AREA NEAR WILCOX DOCK:

AREA RESIDENT

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day) (all receptors)	Oral Cancer Potency Factor (mg/kg/day)-1	Total Excess Cancer Risk (all receptors)	Total Chronic Daily Intake (adolescent)	Oral Reference Dose (mg/kg/day)	Hazard Quotient (adolescent)
PCBs - Sludge Bed	3.9E-05	2.0E+00	7.7E-05	1.1E-04	2.0E-05	5.3E+00
PCBs - Surface Water	7.8E-08	4.0E-01	3.1E-08	2.1E-07	2.0E-05	1.0E-02
TOTAL			7.8E-05			5.3E+00

**TABLE 3-19** 

# POTENTIAL CARCINOGENIC AND NONCARCINOGENIC HEALTH RISKS FROM SWIMMING IN BEACH AREA NORTH OF SLUDGE BED

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day) (all receptors)	Oral Cancer Potency Factor (mg/kg/day)-1	Total Excess Cancer Risk (all receptors)	Total Chronic Daily Intake (young child)	Oral Reference Dose (mg/kg/day)	Hazard Quotient (young child)
PCBs - Sludge	2.9E-07	2.0E+00	5.9E-07	1.4E-06	2.0E-05	6.8E-02
PCBs - Surface Water	2.8E-07	4.0E-01	1.1E-07	9.0E-07	2.0E-05	4.5E-02
TOTAL			7.6E-07		178	1.1E-01

**TABLE 3-20** 

# POTENTIAL CARCINOGENIC AND NONCARCINOGENIC HEALTH RISKS FROM RECREATIONAL USE OF THE BEACH AREA NORTH OF SLUDGE BED

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day) (all receptors)	Oral Cancer Potency Factor (mg/kg/day)-1	Total Excess Cancer Risk (all receptors)	Total Chronic Daily Intake (young child)	Oral Reference Dose (mg/kg/day)	Hazard Quotient (young child)
PCBs	3.4E-06	2.0E+00	6.8E-06	1.1E-05	2.0E-05	5.7E-01
TOTAL			6.8E-06			5.7E-01

TABLE 3-21

POTENTIAL CARCINOGENIC AND NONCARCINOGENIC HEALTH RISKS FROM RECREATIONAL INGESTION OF FISH FROM CUMBERLAND BAY

Analyte	Total Lifetime Avg. Daily Dose (mg/kg/day) (all receptors)	Oral Cancer Potency Factor (mg/kg/day)-1	Total Excess Cancer Risk (all receptors)	Total Chronic Daily Intake (young child)	Oral Reference Dose (mg/kg/day)	Hazard Quotient (young child)
PCBs	1.7E-03	2.0E+00	3.4E-03	5.7E-03	2.0E-05	2.9E+02
TOTAL		77	3.4E-03			2.9E+02

TABLE 3-22
SUMMARY OF POTENTIAL HEALTH RISKS BY EXPOSURE SCENARIO

EXPOSURE PATHWAY	NONCARCINOGENIC HAZARD INDEX	EXCESS CANCER RISK
Wading In Sludge Bed Area Near Wilcox Dock	5.3E+00	7.8E-05
Swimming in Beach Area North of Sludge Bed	1.1E-01	7.6E-07
Recreational Use of Beach Area North of Sludge Bed	5.7E-01	6.8E-06
Cumberland Bay Recreational Fishing	2.9E+02	3.4E-03

#### 4.0 FISH AND WILDLIFE IMPACT ANALYSIS

This section presents the findings of the Fish and Wildlife Impact Analysis (FWIA) performed at the Cumberland Bay Site. The FWIA was performed following the NYSDEC FWIA procedures presented in the NYSDEC, Division of Fish and Wildlife, "Fish and Wildlife Impact Analysis for Inactive Hazardous Waste Sites" (dated June 18, 1991). The results of the Step I (Site Description) and the Step II (Contaminant-Specific Impact Analysis) sections of the FWIA are presented.

The objective of the Step I, site description, is to identify the fish and wildlife resources, land-use and habitat types that exist in the vicinity of the Site. In addition, fish and wildlife species that may utilize habitats that could potentially be impacted by site-related contaminants are identified. This information is necessary to allow identification of potential pathways of contaminant migration that could impact fish and wildlife resources.

The objective of the Step II, contaminant specific impact, is to determine the impacts, if any, of site-related contaminants on fish and wildlife resources. The pathway analysis evaluates and identifies potential contaminants of concern, sources of contaminants, potential pathways of contaminant migration and potential for fish and wildlife resources to be impacted by site-related contaminants. The criteria-specific analysis determines if reported chemical concentrations represent a potential threat to aquatic life and wildlife. The toxic effect analysis attempts to determine or predict what effects the chemicals of concern will have on fish and wildlife and on the use of fish and wildlife by humans.

The purpose of this analysis is to determine if the PCBs present in the sludge bed represent a potential threat to fish and wildlife resources. The document does not attempt to determine the exact magnitude of any impact or the what the complete effect any potential impact will have on individual animals, species, populations or ecosystems. A comprehensive evaluation of what the effect of any potential impact the sludge bed will have on fish and wildlife populations is beyond the scope of this document and could not be completed with the available data. However, sludge bed PCB concentrations are compared to published numerical criteria and an estimate of the effect of PCB concentrations reported in fish collected from Cumberland Bay on a sensitive piscivorous predator is evaluated.

#### 4.1 Baseline Assessment

This section documents the land-use and natural resources located in the vicinity of the Site. A cover type map documents the land use, and the terrestrial, palustrine and aquatic communities located within a one-half mile radius of the Site. Significant natural resources (e.g., wetlands, streams) located within a two mile radius of the site are documented. A description of the fish and wildlife resources that could potentially utilize the cover types located within a one half-mile radius of the Site is presented. The general habitat quality within a one-half mile radius of the Site is discussed.

### 4.1.1 Land Use/Major Plant Communities Within One-Half Mile of the Site

A cover type map detailing the major land use/plant communities within approximately a one-half mile radius of the Site are presented on Figure 2. The cover type map was prepared based on interpretation and evaluation of aerial photographs, topographic maps and NYSDEC wetland maps. Field checking was performed to verify the accuracy of the cover type map. The base map for the cover type map was prepared from aerial photographs taken in May 1991. The cover types within a half mile of the Site were classified using a combination of the New York Heritage Program Classification System (NHPCS, Reschke, 1990) and the U.S. Geological Survey Classification System (Anderson, 1976).

Where access was possible during the field check of the cover type map, the dominant vegetation in each cover type was identified for areas classified as terrestrial natural (TN) and palustrine (P). The areas not numbered were either not accessible or were similar in nature to the other areas. The numbers in each area correspond to the numbers and descriptions of dominant vegetation for each area as presented in Table 4-1 The cover type boundary lines are approximate and have not been surveyed. The determination of dominance was qualitative, based on visual estimates. Vegetative plots and transects were not used in determining dominance. These methods are beyond the scope of a Step I analysis.

Presently, approximately 90 percent of the Site itself is underwater and, as previously discussed, contains a sludge bed of varying thickness, overlying natural sediments. Water depths within the sludge bed area vary between 0 feet at the shore line and approximately 5 feet. Between Wilcox Dock and the breakwater located just south of the dock, the water depth increase to a maximum of approximately 17 feet. During the growing season, aquatic plants including fragrant water lily (Nymphaea odorata), extend out into the Site. This vegetated area would be classified as a deep emergent marsh community following the Ecological Communities of New York State (Reschke, March 1990) classification system.

The three principle aquatic habitats located within one half-mile of the Site are Cumberland Bay, the Saranac River and Scomotion Creek. Cumberland Bay is a small, somewhat rectangular part of the west side of Lake Champlain. Depths in the bay can exceed 50 feet but water depths in the vicinity of the Site do not exceed 17 feet and are generally under 10 feet. The City of Plattsburgh is located on the west side of the Bay, where the Saranac River and Scomotion Creek flow into the Bay. The north shoreline of Cumberland Bay is occupied by the Plattsburgh Municipal Beach, a NYS Office of Parks and Recreation campground, and numerous motels and restaurants. On the east side, Cumberland Head, a large peninsula, extends into the Bay. The Saranac River enters Cumberland Bay approximately 2,800 feet south of the Site. The waters of Lake Champlain in Cumberland Bay in the vicinity of the Site are designated as Class "B" by NYSDEC. Scomotion Creek is designated as a Class "C" stream from the mouth of the Creek at Lake Champlain to one mile upstream. The Saranac River is designated a Class "C" water from the mouth of the River to a point approximately one-quarter mile upstream. From the one-quarter mile point to the Imperial Wallpaper Mills Dam, the River is designated as a Class "C(t)" water body.

There are two wetland plant communities located within the Site boundaries along the southwestern shoreline. A zone of emergent vegetation exists within the sludge bed and along the shoreline and this area grades into a deciduous forested/shrub wetland with increasing distance from the waters edge. The emergent wetland plant community and the deciduous forest/shrub plant community are depicted on Figure 2 as P1 #1 and P2 #2, respectively. The emergent wetland is dominated by cattails along the shoreline (Typha species) and fragrant water lily out into the sludge bed. The deciduous forested overstory is dominated by red-maple (Acer rubrum), willow (Salix species) and common cottonwood (Populus deltoides) with red oiser dogwood (Cornus stolonifera) in the understory.

Using the Ecological Communities of New York State classification system, the P1 #1 emergent mash would best be classified as a gradation between a deep to shallow emergent marsh, with some shrub swamp characteristics in the upper reaches of the wetland area. The deciduous forest P2 #2 plant community would be classified as floodplain forest.

With the exception of the emergent wetland and the deciduous forested wetland described above, the remaining shore line within the Site boundary and south of Scomotion Creek is unvegetated and consists primarily of commercial buildings. Just north of Scomotion Creek, the land adjacent to Cumberland Bay consists of a mixture of deciduous forest and successional shrub field (Figure 2, TN2D/TN1 #6). This was a picnic area that has been abandoned. The dominant overstory tree species in this area is common cottonwood. Box elder (Acer negundo) is a dominant sapling.

Wilcox Dock and the breakwater located south of the dock (TN2D#3) are vegetated with common cottonwood and a variety of successional field herbaceous species including goldenrod (Solidago species).

NYSDEC wetland PB-5 is located approximately 1,500 feet north of the Site. Scomotion Creek flows through this wetland and enters Cumberland Bay approximately 1,000 feet north of the Site. Wetland PB-5 has been designated by NYSDEC as a Class I wetland. It is approximately 1,500 acres in size. There are at least two primary vegetative communities associated with this wetland, deciduous forested wetland and emergent wetland. There is also a shrub wetland complex present in wetland PB-5. NYSDEC wetland PB-5 is designated on Figure 2 as P2 #4 and P2 #5. The dominant overstory vegetation in the deciduous forested wetland plant community is red-maple, willow species, elm species and common cottonwood. The dominant species in the emergent marsh open areas is cattail species. The shrub areas are dominated by red-oiser dogwood. NYSDEC wetland PB-5 would be classified as a complex mixture of a floodplain forest, shallow emergent wetland and shrub swamp, using the NYSDEC Ecological Communities of New York classification system.

With the exception of a small isolated area of successional woodland, the land use southwest of the Site is primarily a mixture of residential, commercial and industrial. Just east of this commercial/industrial zone, on the west side of Route 9, is an area of successional field/shrub and a mixture of commercial and industrial land use.

### 4.1.2 Wetlands Within One-Half and Two Miles of the Site

As previously discussed, NYSDEC wetland PB-5 is the only NYSDEC regulated wetland located within a one-half mile radius of the Site and it is designated a Class I wetland. The NYSDEC wetland classification system is based on a numerical rating of I to IV, with Class I wetlands representing the most significant wetlands. A wetland is considered Class I if it exhibits at least one of seven characteristics detailed in the Freshwater Wetlands Maps and Classification Regulations (6NYCRR Part 664), which are summarized below:

- 1. is a classic kettlehole bog;
- 2. resident habitat of an endangered or threatened animal Species;
- 3. contains an endangered or threatened plant Species;
- 4. supports an animal species in abundance or diversity unusual for the state or the region;
- 5. provides significant flood control benefits for a substantially developed area;
- 6. adjacent or contiguous to an aquifer or reservoir used for public water supply; and,
- 7. contains four or more Class II wetland characteristics.

There are six additional NYSDEC regulated wetlands located within a two-mile radius of the Site; wetlands PB-1, PB-2, PB-3, PB-10, PB-11 and PB-12. Wetlands PB-2 and PB-12 are Class I wetlands, PB-1 is a Class II wetland and PB-3, PB-10 and PB-11 are Class III wetlands. The wetland locations are presented in Figure 3.

Only two of the seven NYSDEC regulated wetlands located within a two-mile radius of the Site are connected to Cumberland Bay. Wetland PB-5 is located along Scomotion Creek and Wetland PB-12 is located along the Saranac River.

### 4.1.3 Streams and Related Surface Water Bodies Within One-Half Mile and Two Miles of the Site

The Saranac River and Scomotion Creek are the only streams located within approximately a half-mile radius of the Site. Within a two-mile radius there are only four other streams, Kennon Brook and three unnamed streams. Kennon Brook is a tributary of Scomotion Creek. Two of the unnamed tributaries appear to drain into NYSDEC wetland PB-5, one approximately 2,000 feet north of I-87 and the second approximately 5,600 feet north of I 87. The third unnamed tributary, located on the north side of I-87, is a tributary of Scomotion Creek.

The waters of Lake Champlain in Cumberland Bay in the vicinity of the Site are designated as Class "B" by NYSDEC. Scomotion Creek is designated as a Class "C" stream from the mouth of the Creek at Lake Champlain to one mile upstream. The Saranac River is designated a Class "C" water from the mouth of the River to a point approximately one-quarter mile upstream. From the one-quarter mile point to the Imperial Wallpaper Mills Dam, the River is designated as a Class "C(t)" water body.

NYSDEC Water Quality Regulations, Title 6, Chapter X, Parts 700-705 define the best usage of Class C streams as fishing. Class C waters shall be suitable for fish propagation and survival. The

Class C water quality shall be suitable for primary and secondary contact recreation. The "t" designation indicates that the water will support the propagation and survival of trout. The best usage of Class B waters are primary and secondary contact recreation and fishing. These waters shall be suitable for fish propagation and survival.

#### 4.2 Resource Characterization Within One-Half and Two Miles of the Site

Resource characterization consists of determining the wildlife species that may potentially utilize, or have been determined to utilize, the plant communities or habitats identified in the previous sections as existing within one-half mile of the Site. Also, any known species of concern (i.e., endangered, threatened, etc.) or significant habitats that may exist within two miles of the Site are identified. Additionally, the general quality of the habitats that are located within one-half mile of the Site and their ability to provide for the needs of the species that may utilize the habitats is discussed. Areas of observed vegetative stress, leachate seeps, documented evidence of fish and/or wildlife mortality and any known population impacts related to site-related contaminants are presented.

# 4.2.1 Endangered, Threatened or Special Concern Fish and Wildlife or Plant Species or Significant Habitats

The United States Fish and Wildlife Service (USFWS), the NYSDEC Wildlife Resources Center and the NYSDEC Region 5 Office were contacted regarding the known occurrence of endangered, threatened, or special concern species or habitats located within a two-mile radius of the Site. The USFWS indicated that there are no known occurrences of federal endangered or threatened wildlife or plant species located within a two mile radius of the Site.

A review of the New York State Natural Heritage Program files by the New York State Department of Environmental Conservation, Wildlife Resources Center, indicated that there were no known species of concern located within the boundaries of the Site.

Two plant species, with a New York State status as rare, were identified within a two-mile radius of the Site. Marsh horsetail (Equisetum palustre) was identified as occurring approximately 10,000 feet south of the Site. Handsome sedge (Carex formosa) was identified as occurring approximately 11,000 feet east of the Site. Rare species are considered to have 20 to 30 extant sites or 3,000 to 5,000 individuals statewide. One species, Champlain beach grass (Ammophila champlainensis) was identified as occurring on the beach approximately 2,000 feet north northeast of the Site. This species is unprotected in New York, but is considered by the Natural Heritage Program as being especially vulnerable in New York State. The Autumnal Water-Starwort (Callitriche hermaphroditica), last observed in 1929, was believed to occur in Cumberland Bay, potentially within the vicinity of the Site. This species is unprotected in New York State. It is believed to be historically present in the State but has not been observed in the past 15 years. Considering the known locations of these species, Site activities are not expected to have an impact on them.

Personal communication with the NYSDEC Region 5 wildlife biologist, revealed that Osprey (Pandion haliaetus) have been observed nesting north of I-87 within wetland PB-5. The Osprey is

classified as a threatened species in New York State. Bald Eagles have been observed in Cumberland Bay, most likely representing migrating birds. The bald eagle is an endangered species in New York State.

### 4.2.2 Fish and Wildlife Species Potentially Using Habitats Within a One-Half Mile Radius of the Site

Mammals, amphibians and reptiles, and fish, and bird species that could potentially utilize the habitats within a one-half mile radius of the Site, for at least a portion of their life cycle, are listed in Tables 4-2, 4-3 and 4-4, respectively. All species that could potentially utilize the habitats within a one-half mile radius of the Site are not included on these lists. Also these lists are not meant to indicate that these species can always be found, or that all will be present at one time within one-half mile of the Site. These lists were prepared following a limited field evaluation of the habitats and review of available literature. These lists are not the result of a site-specific population survey. Actual population surveys are complex and time intensive and are beyond the scope of a Step I baseline evaluation.

Many wildlife species are mobile and generally require a range of habitat types to meet their life cycle requirements. In addition, many species will only use the area within one-half mile of the Site for a portion of their life requisites. Thus, all the species identified on these lists were not actually observed within a one-half mile radius of the Site.

During the field checking of the cover type map on March 23, 1997, the species listed below or sign of these species were observed on or within one-half mile of the Site.

- Black-capped Chickadee
- Cardinal
- Common Crow
- Red-tailed Hawk
- Hooded Merganser
- Beaver

- · Blue-jay
- Canada Goose
- Mallard Duck
- White-tailed Deer
- Pileated Wood-Pecker
- Ring-billed Gull

Only two of the above species, the Black-capped Chickadee and the Pileated Wood-Pecker were observed within the boundaries of the Site. Both species were observed in the deciduous forested wetland (P2 #2, Figure 2) plant community. There was evidence of recent beaver activity along Scomotion Creek. Canada Geese, Mallards, Ring-billed Gulls or Herring Gulls and Hooded Mergansers were observed using the open water areas at the mouth of the Saranac River where it enters Cumberland Bay.

The NYSDEC Region 5 biologist (personal communication) indicated that diving ducks such as Greater Scaup (Aythya marila mariloides), Lesser Scaup (Aythya affinis), Bufflehead (Bucephala albeola) Common Merganser (Mergus merganser americanus) and Hooded Merganser (Mergus cucullatus) utilize the Cumberland Bay as wintering habitat until ice-up of the bay. There are Great Blue Heron (Ardea herodias) and Ring-Billed Gulls (Larus delawarensis) located on islands south

of Cumberland Bay (Four Brothers and Valcour Island). However, both these species would utilize wetland and shallow water areas in Cumberland Bay as foraging areas.

The Saranac River, which enters Cumberland Bay approximately 2,800 feet south of the Site, represents a significant Atlantic Salmon (Salmo salar) fishery. The NYSDEC has completed construction of two fish ladders on the river. A third fish ladder is scheduled for construction which will open up approximately fourteen miles of the Saranac River to spawning Atlantic Salmon.

### 4.2.3 General Habitat Quality Within One-Half Mile of the Site

The deep and shallow emergent marsh habitats located within the boundaries of the Site represent poor quality habitats due to the presence of the PCB contaminated paper sludge. Otherwise, both these areas would represent high quality habitats. They would provide fish nursery habitat and foraging habitat for birds and mammals which feed on aquatic life.

The terrestrial natural habitats located within a one-half mile radius of the Site are generally poor to moderate quality habitats. The relatively small size of these areas and there juxtaposition relative to the developed residential, commercial and industrial areas limits the value of these areas as wildlife habitat. The one exception would be the successional field/shrub communities located adjacent to wetland PB-5, which represent an increase in the diversity of habitat types available to wildlife. The successional field/shrub communities represent potential feeding areas for wildlife which require a variety of habitats to meet their life requisites.

Wetland PB-5 represents a high quality natural habitat. The variety of habitat types, emergent wetland, shrub wetland, deciduous forested wetland and the Scomotion Creek aquatic habitat, located within the wetland represent a diversity of habitats available to wildlife. This area is also of sufficient size to represent a high quality habitat. It is a common ecological tenant that large blocks of undisturbed areas can support a greater number of species than smaller areas. This is partially related to the fact that larger areas will typically contain a wider variety of habitat types. Areas with diverse habitat types are more likely to contain the range of resources necessary to support a given species life cycle requirements. The greater number of habitat types the wider the diversity of plant communities. Animal species are ultimately dependent upon plants for survival, either directly in the case of herbivores, or indirectly with respect to animal species that use plants for shelter or feed on herbivores. As previously discussed, Osprey have been observed in the vicinity of wetland PB-5. Also signs of recent beaver and white-tailed deer activity were observed.

The Saranac River is an important natural resource. The river represents a high quality fisheries resource and is a valuable habitat for aquatic birds. The river is a significant Atlantic salmon spawning area. Aquatic birds such as Great-blue herons, gulls, diving and puddle ducks, and Canada Geese utilize the river for feeding and resting areas.

Scomotion Creek also represents an important aquatic resource. The creek and adjacent wetland habitat represent valuable fish spawning and nursery areas. The creek also represents an important feeding and resting area for semi-aquatic mammals and aquatic birds. This undeveloped adjacent wetland habitat increases the habitat value of the creek as feeding and resting habitat.

Cumberland Bay represents a viable fishery. However, the value of this fishery is impaired by the presence of PCBs in the sludge bed located in the western edge of the bay. Historically, Cumberland Bay was part of the commercial yellow perch fishery in Lake Champlain. However, due to elevated PCB concentrations detected in yellow perch from the Bay, there is currently a ban on the sale of yellow perch. There is also a recreational health advisory on consumption of fish from Cumberland Bay. The fish PCB levels also represent a potential threat to piscivorous wildlife that are sensitive to PCBs. Because of the propensity for PCBs to bioaccumulate, the PCBs in prey species represents a potential threat to predators species.

### 4.3 Applicable Fish and Wildlife Regulatory Criteria

The appropriate Site Specific Criteria (SSC) that may potentially be applicable to the Site are detailed below:

- Clean Water Act, 233 U.S.C. 1261 et seq. Sec. 404 regulates the discharge of pollutants into wetlands and other water bodies, including dredged or fill materials;
- The Freshwater Wetlands Act (Article 24 of the Environmental Conservation Law) and the Freshwater Wetlands Implementing Regulations (6NYCRR Parts 663 and 664) are designed to protect wetlands. Only wetlands that have been mapped by the State of New York are regulated;
- Executive Order 11990, Protection of Wetlands. This order recognized the value of wetlands and directed federal agencies to minimize the degradation, destruction and loss of wetlands;
- Endangered Species Act (87 Stat. 884, as amended; 16 U.S.C. 1531 et seq.);
- · Fish and Wildlife Coordination Act;
- NYSDEC, Division of Water Technical and Operational Guidance Series (1.1.1) Ambient Water Quality Standards and Guidance Values, October 1993;
- NYSDEC, Water Quality Regulations for Surface Waters and Groundwaters, 6NYCRR Parts 700-705; and
- NYSDEC, Technical Guidance for Screening Contaminated Sediments, November 1993;
   and,
- USEPA, Interim Sediment Criteria Values For Nonpolar Hydrophytic Organic Chemicals (May 1988). Also Periodic Updates.

### 4.4 Contaminant-Specific Impact Analysis

This section evaluates pathways through which wildlife could potentially be exposed to site related contaminants. This evaluation includes the identification of habitats that could potentially be impacted by site contaminants, the possible food chain contamination pathways, and impact to fish/wildlife, if any. Media contaminant concentrations are compared to published numerical criteria to determine if there is a potential for impacts to wildlife. A toxic effects analysis on representative sensitive species is performed to determine if reported media concentration pose a potential threat to wildlife populations.

### 4.4.1 Pathway Analysis

In order for fish and wildlife to be affected by chemical constituents from a site, two conditions must exist. There first must be an avenue by which fish and wildlife can be exposed to chemical constituents, referred to as a completed exposure pathway. In addition, the chemical concentrations within the completed exposure pathway must be of sufficient magnitude to cause an impact.

Potential fish and wildlife exposure pathways with respect to PCB exposure include ingestion of plants, animals or water, or direct contact with water, soil or sediments which contain PCBs. This section evaluates potential exposure pathways.

The environmental media from which samples have been collected and analyzed include, sludge/sediments from the Site, Lake Champlain water samples, and fish tissue samples.

PCBs have been detected in samples collected from the sludge bed located on the Site and in Lake Champlain water samples and in the tissue of fish from Cumberland Bay. Therefore there are completed exposure pathways. Wildlife could be exposed via direct contact/ingestion of contaminated sludge and water, and ingestion of prey species that contain PCB body burdens.

For instance, mink is a mammal that would be expected to utilize the habitats associated with wetland PB-5, located north of the Site. Wetland PB-5 is directly connected to Cumberland Bay via Scomotion Creek. Mink would have access to Fish containing PCBs via utilization of the aquatic habitats and adjacent wetland areas in the vicinity of Scomotion Creek. It has been documented that the reproductive success of mink is significantly impaired by exposure to low concentrations of PCBs in the diet. Predator bird species such as osprey and great-blue herons would be exposed to PCBs via consumption of fish. The diet of these species consists of a high proportion of fish. Both bird species would potentially consume fish directly from Cumberland Bay and from the aquatic habitats associated with Scomotion Creek and the Saranac River, which are directly connected to Cumberland Bay.

### 4.4.2 Criteria-Specific Analysis

This section compares available analytical data from media which represent potential fish and wildlife exposure pathways to available regulatory guidelines and available fish and wildlife toxicity data. The pathway analysis in the preceding section indicated that there are contaminated resources

and there are exposure pathways. Impacted resources include the shallow and deep emergent marsh plant communities located within the boundaries of the Site (sludge bed), surface water (Lake Champlain) and fish tissue (fish collected from Cumberland Bay).

Two surface water samples were collected by the NYSDOH in 1994. Both samples were taken outside the sludge bed, one along the Route 9 beach and a second off the public beach located north of the sludge bed; the reported concentrations were 0.31 ug/l and 0.21 ug/l, respectively. A third sample collected by NYSDEC, just outside and east of the sludge bed, exhibited between 0.006 and 0.008 ug/l of PCB Aroclor 1242. These concentrations exceed the NYSDEC surface water standard of 0.001 ug/l for protection of aquatic life. This standard is based on the propensity of PCBs to bioaccumulate and the protection of wildlife from the toxic effects of bioaccumulation. Available data indicate that there is a potential for fish and wildlife to be affected by dissolved PCBs in the Cumberland Bay surface water column.

Table 4-5 summarizes the available PCB sludge sample data from the sludge bed in samples that were collected in the upper 26 centimeters of the bed. The mean and standard deviation are presented. Although it is generally assumed that the top five centimeters of a sediment represent the biologically active zone, this assumption is not applicable to the sludge bed. The sludge bed is subject to significant wave action which causes "weathering" of the sludge bed and the material deeper in the bed may at some time become biologically available. Therefore, data from the top 26 centimeters is presented.

Aroclor 1242 was the primary PCB Aroclor detected. One sample exhibited low concentrations of Aroclors 1254 and 1260. A total of 59 samples were collected from 19 locations. PCBs were detected above the reporting limit in all but three samples. One-half the reporting limit of the non-detect samples was used in calculating the mean total PCB concentration.

The NYSDEC has established sediment criteria guidelines for protection of piscivorous wildlife from consuming fish or other aquatic life from a water body with PCB laden sediments. The sediment criteria is based on the USEPA, Equilibrium Partitioning approach (EqP). The fundamental assumption of the EqP is that the toxicity of a contaminant in sediment is attributable to the fraction of contaminant that dissolves in the sediment interstitial pore space and is therefore biologically available. This concentration is predicted based on the concentration of the contaminant in the sediment, the concentration of organic carbon in the sediment and the affinity of the contaminant for organic carbon.

The EqP is only applicable for sediments with organic carbon contents between 0.2 percent to 12 percent. At organic carbon concentrations lower than 0.2 percent and greater than 12 %, there are other factors which may influence bioavailability. Research indicates that uptake from food by benthic species is significant for some organic chemicals. At organic carbon contents greater than 10% to 12 % it is possible that the EqP approach may underestimate the bioavailability of an organic chemical. The sludge bed at the Site is composed primarily of wood pulp sludge and therefore has a very high organic carbon content. For the purposes of determining a PCB sediment quality criteria value for the sludge bed, an organic carbon content of 12 % was utilized, although the organic carbon content of the sludge is known to be significantly higher.

Assuming an organic carbon content of 12 % in the sludge bed, the sediment quality criteria (SQC) value would be approximately 166 µg/kg of PCBs.

 $SQC = WQC \times Kow \times C \times \% OC$ 

SQC = Sediment Quality Criteria (ug PCB/kg sediment)

WQC = NYSDEC Water Quality Criteria (0.001 ug/l)

Kow = Octanol water partition coefficient (1,380,348.3 l/kg)

% OC = Assumed percent organic carbon in sludge bed (100 g organic carbon/kg)

C = Conversion factor 1kg/1000g Organic Carbon

SQC = 0.001 ug/l x 1,380,348.3 l/kg x 1kg/1000 g OC x 100 g OC/kg

= 166 ug/kg PCBs

This value is based on the potential for PCBs to biomagnify in through the food chain and result in concentrations in fish and other aquatic prey species that would be toxic to a predator Species. Sediment concentrations significantly below the  $166~\mu g/kg$  level would not pose a risk to piscivorous wildlife. The average concentration of total PCBs in the top 26 centimeters of the sludge bed was 117,806~ug/kg which is significantly higher than the SQC derived concentration of 166~ug/kg. The PCB data from the sludge bed indicate that there is a significant potential for impacts to piscivorous predatory wildlife.

The EqP theory utilized in the NYSDEC "Technical Guidance for Screening Contaminated Sediments" assumes that all PCBs all equally toxic. However, as discussed in Section 4.4.3, research has indicated the co-planar PCB congeners that are structurally similar to 2,3,7,8-tetrachlorodibenzopdioxin (2,3,7,8-TCDD) are the most toxic. As discussed in Section 4.4.3, if it is assumed that the ratio of the co-planar PCB congeners to total PCBs is 0.0404, then the average concentration of co-planar PCBS in the top 26 centimeters of the sludge bed would be the average total PCB concentration 117,608 ug/kg x 0.040 which equals 4,751 ug/kg, which is still greater than the SQC derived sediment concentration of 166 ug/kg. The data indicates that the sludge bed samples exhibit PCB concentrations which represent a potential threat to piscivorous predatory wildlife based on the toxic effects of bioaccumulation.

### 4.4.3 Toxicity Analysis

This Section provides a discussion of the ecological toxicity of PCBs. An analysis of the toxicity of PCBs with respect to mink (Mustela vison), and the Osprey (Pandion haliaetus) is presented.

The mink is a good indicator for assessing the effects of PCBs in the aquatic environment. The mink is a semi-aquatic mammal which feeds heavily on fish and aquatic invertebrates and amphibians. Mink would also potentially utilize habitats in the vicinity of the Cumberland Bay Site. Mink reproductive success is sensitive to dietary exposures to PCBs (Eisler 1986). Mink is a species that would be expected to utilize the habitats in wetland PB-5 located along Scomotion Creek, approximately 1,000 feet north of the Site. This area is directly connected to Cumberland

Bay and it is likely that fish with PCB body burdens would be present in Scomotion Creek below the first fish barrier.

The Osprey is classified as a threatened species in New York State. The Osprey is a piscivorous raptor that feeds almost exclusively on fish. Osprey have been reported (personnel communication NYSDEC Region V wildlife biologist) nesting in the vicinity of the Site (wetland PB-5) and would be expected to forage for fish in Cumberland Bay.

#### 4.4.3.1 Mink Toxicity Analysis

PCBs are man made organic compounds which consist of a biphenyl group with 1 to 10 chlorine atoms substituted for hydrogen on the biphenyl. This combination can theoretically create 209 different compounds or congeners. Industrial formulations of PCBs were a combination of different congeners. Monsanto Company was the primary manufacture of PCBs in the United States and the different mixtures produced by Monsanto were termed Aroclors.

The toxicity of PCBs is congener specific. Some PCB congeners exhibit co-planar structures that are similar to that of 2,3,7,8-TCDD and show similar toxicities as 2,3,7,8-TCDD. Giesy (1994) reported that the reproductive toxicity of PCBs on mink were essentially related to the dioxin-like compounds. Safe (1990) reported that PCB congeners with chlorine substituted in both para, at least 2 meta and no ortho positions are the most toxic. In a study on the effect of dietary exposure of mink to carp from Saginaw Bay, Michigan, it was reported that environmentally derived PCBs are more toxic to mink than some common commercial PCB mixtures such as Aroclor 1254 (Heaton; et al., 1995). Additionally, it has been reported that the most toxic congeners are selectively accumulated from organisms at one trophic level to the next.

Analysis of fish collected from Cumberland Bay document that fish have been exposed to PCBs and that PCBs are accumulating in fish tissue. In September 1994, 49 individual fish including yellow perch, northern pike, largemouth bass, brown bullhead, black crappie, carp and rock bass were captured in Cumberland Bay in the vicinity of Wilcox Dock and were analyzed for PCBs. The average concentration of total PCBs was 2.84 mg/kg.

Limited congener specific analyses were performed on the largemouth bass and brown bullhead fish samples. As previously stated, the toxicity of PCBs is primarily related to the co-planar PCBs with para and meta substitution and no ortho substitution. The congener analyses consisted of the identification of approximately 119 of the 209 possible PCB congeners. However, the analysis only included data for one coplanar congener, 3,3,4,4-tetrachlorobiphenyl (IUCPAC No. 77) which is a co-planar PCB with para and meta substitution. The ratio of congener No.77 to total PCBs was 0.0404.

A toxicity analysis based on the concentration of congener No. 77 alone could potentially underestimate the potential risk. Additionally, dioxin has also been reported as present in the sludge bed wood pulp. Mink are extremely sensitive to dioxin. If the fish containing PCBs also contain dioxin, then the estimated impact on mink would potentially be greater than that estimated for congener 77 alone. Therefore, the toxicity analysis and the hazard quotient was calculated based on

the average fish tissue total PCB concentrations. The reported fish tissue total PCB concentrations are summarized in Table 4-6. The average total PCB concentration of the 49 fish samples was 2.84 mg/kg.

A hazard quotient was developed to determine if the levels of PCBs reported in fish collected from Cumberland Bay represent a potential threat to mink. The dose was calculated following the procedure presented in the USEPA Wildlife Exposure Factors Handbook (December 1993). The acceptable daily dose is based on a review of available literature.

The hazard quotient is determined by dividing the potential average daily dose by an acceptable daily dose, which is either a No Observable Adverse Effect Level (NOAEL) or a Lowest Observable Adverse Effect Level (LOAEL). Typically, where a NOAEL has not be determined it is estimated from a LOAEL by applying a safety factor varying between 10 to 100 fold to the LOAEL.

The PCB calculated dose was determined using the following equation:

 $APDD = C \times NIR \times F$ 

APDD = Average potential daily dose (mg/kg/day)

C = Contaminant concentration (mg/kg wet weight)

NIR = Normalized ingestion rate (g/g/day)

F = Percentage of contaminated food in diet

The normalized ingestion rate is assumed to be 0.16 g/g/day for a female adult mink (USEPA 1993). The percentage of fish in the diet of a mink utilizing stream/riverine habitats is assumed to be 29.7 percent (Heaton et al., 1995).

In a study on the effect of contaminated fish on the health of mink, Giesy et al. (1994) reported that the daily allowable dose was 16.2 ug/mink/day and that the average mink weighed 1 kg. In a related study, Heaton et. al. (1995) reported that the lowest observable adverse effect level was 0.13 mg/kg body weight/day and that the average mink weighed 1 kg. These values were based on the total concentration of Aroclors 1248, 1254 and 1260 present in fish tissue. These Aroclors would contain a greater percentage of the more toxic co-planar PCB congeners than Aroclor 1242, which was the primary Aroclor detected at the Site. Therefore, a lowest observable adverse effect level LOAEL for the PCBs detected in the fish from Cumberland Bay would most likely be higher than 0.13 mg/kg body weight/day. However, safety factors ranging from 10 to 100 are typically applied to a LOAEL to determine a NOAEL value. Eisler (1986) reported that the maximum tolerance level for mink is less than 1.54 ug PCBs/kg body weight day. This was based on a 100 fold safety factor applied to a dietary level of 0.64 mg PCBs/kg fresh weight of diet. Therefore, applying a safety factor of 10 instead of 100 to the LOAEL of 0.13 mg/kg/day reported by Heaton et. al.(1995) represents a usable estimate of the NOAEL (0.013 mg/kg/day) when using total PCB fish tissue concentrations as an estimate of the risk to mink from consumption of fish from Cumberland Bay.

The calculation of the hazard quotient using the average fish tissue total PCB concentration is presented below:

Hazard quotient = Average Potential Daily Dose (APDD)/Acceptable Daily Dose (ADD)

Mink APDD of PCBs from Ingestion of Fish =

(2.84 mg/kg average fish PCB Concentration) x (0.160 g/g/day NIR) x (0.297 percent of fish in diet)

APDD = 0.135 mg/kg/day

Hazard quotient (HQ) for Yellow Perch = 0.135 mg/kg/day/ 0.013 mg/kg/day

HQ = 10.38

A hazard quotient greater than 1.0 indicate a potential risk to mink reproductive success with respect to consumption of fish from Cumberland Bay. The hazard quotient exceeded 1.0 indicating a potential impact on mink reproductive success. This analysis was focused just on PCBs. It has been documented that dioxin compounds have been detected in sludge samples collected from the Site. There are no available dioxin fish data. Mink are extremely sensitive to dioxin and the LD -50 for 2,3,7,8-TCDD has been determined to be 4.2 ug/kg, body weight (Giesy, et. al. 1994). Since dioxin compounds are present in the sludge bed sediments, it is likely that dioxins are also present in the fish tissue from Cumberland Bay. Any dioxin in fish tissue would represent an additive impact on mink which consume fish from Cumberland Bay.

The mink toxicity analysis indicates a potential risk to mink residing in wetland PB-5 located north of the Site. This analysis was based on a number of assumptions due to the limited data available. The following assumptions were made in performing this analysis:

- The total PCB concentrations in the fish from Cumberland Bay are representative of fish tissue PCB concentrations in fish from Scomotion Creek in the vicinity of wetland PB-5.
   If fish tissue PCB concentration in fish from Scomotion Creek are lower, then the degree of risk could be overestimated.
- The average total PCB concentration is an accurate assessment of toxicity. If the average
  total PCB concentration is an over or under estimate of actual toxicity, then the degree of
  risk would be over or under estimated. An analysis of fish tissue for the coplanar
  congeners and application of the data to the risk analysis would provide a more accurate
  assessment of actual toxicity.
- There are no other environmental contaminants, such as dioxin, which would cause an additive effect. If dioxins are present in the fish tissue, then the potential impact on mink reproductive success would potentially be higher than estimated.

### 4.4.3.2 Osprey Toxicity Analysis

Research has indicated that reproductive impairment is one of the most significant impacts related to ingestion of PCB contaminated forage by piscivorous bird species. PCB egg residues have been used as a measure of the degree of potential PCB toxicity in piscivorous birds. Hoffman et.al. (1996) reported that PCB concentrations from 8 to 25 ppm (mg/kg) in eggs of terns, cormorants, doves and eagles resulted in decreased hatching success. Total weathered PCB concentrations in bald eagle eggs from Lakes Michigan and Huron were detected at levels as high as 98 mg/kg (Kubiak and Best, 1993).

Bowerman et. al. (1995) in a study of factors affecting bald eagle productivity in the Great Lakes region, used a total PCB concentration of 4 mg/kg in bald eagle eggs as a NOAEL. Bowerman et. al. (1995) developed a biomagnification factor (BMF) to estimate the magnification of PCBs from fish ingested by eagles to bald eagle eggs. The BMF was calculated from actual measurements of PCB concentrations in fish consumed by eagles and the actual concentration of PCBs in addled eagle eggs in the same region. The estimated BMF was 28.

The potential Osprey reproductive toxicity from ingestion of fish from Cumberland Bay was estimated using the NOAEL and BMF developed by Bowerman et.al. (1995). The average total PCB concentration in the 49 fish samples collected from Cumberland Bay in September 1994 was multiplied by the BMF of 28 and a frequency factor to estimate the PCB egg burden of Osprey feeding on fish caught in Cumberland Bay. Osprey in the northern part of their range (including northern New York State) migrate south in winter, therefore, a frequency exposure factor was included in the calculation of the egg PCB burden. It has been reported that osprey in the northern part of the range begin migrating south in late August and return in April (USEPA 1993). The frequency factor was calculated by dividing the number of days on the summer breeding ground (April 1 through August 31) by 365 days.

Osprey Egg PCB Burden =  $Cf \times BMF \times F$ 

Cf = Average PCB concentration in fish from Cumberland Bay (2.84 mg/kg)

BMF = Biomagnification factor of PCBs in fish to PCBs in eggs (28)

F = (152 days/year) / (365 days/year) = 0.416

Osprey Egg PCB Burden =  $2.84 \text{ mg/kg} \times 28 \times 0.416$ = 33.08 mg/kg

A hazard quotient (HQ) was calculated by dividing the estimated Osprey PCB egg burden by the NOAEL of 4 mg/kg.

HQ = Osprey Egg PCB Burden/ NOAEL

HQ = 33.08 mg/kg/4 mg/kg

HQ = 8.27

The hazard quotient of 8.27 indicates the potential for an impact on the reproductive success of Osprey feeding on fish from Cumberland Bay. This analysis assumes that there are no significant pharmacokinetics differences between eagles and osprey. The analysis also assumes that the average PCB concentration in the Cumberland Bay fish tissue samples is representative of the concentration in fish consumed by osprey that may be nesting in the vicinity of the Site.

#### 4.5 Summary and Conclusions

The baseline assessment indicated that the principle aquatic resources within one-half mile of the Site were Cumberland Bay, Scomotion Creek and the Saranac River. The principle palustrine wetland habitat within a one-half mile radius of the site is NYSDEC wetland PB-5 which is located along the Scomotion Creek. Also there is a small deciduous forested wetland and a shallow/deep emergent wetland complex located within the boundaries of the Site. There are no significant terrestrial habitats located within a one-half mile radius of the Site that could be impacted by the PCBs detected in the Site sediments.

Significant wildlife species that may utilize the habitats located within a one-half mile radius of the Site include Atlantic salmon, osprey, great blue herons and mink. Lake Champlain and the Saranac River represent a significant landlocked salmon fishery. Osprey, a threatened species in New York State has been observed nesting in wetland PB-5. Mink, a species highly sensitive to PCBs would be expected to utilize the habitats associated with wetland PB-5. Mink could potentially be exposed to PCBs from the Site via ingestion of fish which contain PCBs. Great blue herons have been observed feeding at the Site.

The pathways exposure analysis indicates that pathways exist via which wildlife could be exposed to PCBs from the Site. Fish samples collected in the vicinity of the Site exhibit PCBs, which documents that there has been a completed exposure pathway. Mink, utilizing the habitats in wetland PB-5 could be exposed to PCBs by consumption of fish containing PCBs.

The criteria specific analysis revealed that surface water in Cumberland Bay in the vicinity of the Site exhibit PCB concentrations that are elevated with respect to the NYSDEC surface water standard for protection of wildlife from bioaccumulation of PCBs. Available data indicate that there is a potential for fish and wildlife to be affected by dissolved PCBs in the Cumberland Bay surface water column.

Sludge samples from the Site exhibit PCB concentrations that are elevated with respect to the NYSDEC sediment quality criteria value for PCBs. The sediment criteria value for PCBs is based on protection of piscivorous wildlife from toxic effects of PCB bioaccumulation. Available data indicate a potential for an impact to piscivorous wildlife from the toxic effects associated with bioaccumulation of PCBs.

A toxicity analysis on the potential impact on mink reproductive success from consumption of fish flesh containing PCBs was performed. It was assumed that fish containing PCBs could move into Scomotion Creek and the adjacent wetland PB-5 from Cumberland Bay. The analysis indicates that

there is a potential for an impact on the reproductive success of mink which utilize the habitats associated with wetland PB-5.

The toxicity analysis with respect to osprey reproductive success resulted in a hazard quotient of 8.27, which indicates there is a potential for an impact on the reproductive success of osprey feeding on fish from Cumberland Bay.

This toxicity analysis was based on a number of assumptions which could impact the analysis and either lower or increase the potential for an effect on mink or osprey reproductive success. These assumptions include the following:

- The total fish tissue PCB concentrations are representative of fish tissue PCB concentrations in fish from Scomotion Creek in the vicinity of wetland PB-5. If fish tissue PCB concentration in fish from Scomotion Creek are lower, then the degree of risk could be overestimated.
- The average total PCB concentration is an accurate assessment of toxicity. If the average
  total PCB concentration is an over or under estimate of actual toxicity, then the degree of
  risk would be over or under estimated. An analysis of fish tissue for the coplanar
  congeners and application of the data to the risk analysis would provide a more accurate
  assessment of actual toxicity.
- There are no other environmental contaminants, such as dioxin, which would cause an additive effect. If dioxins are present in the fish tissue, then the potential impact on mink and osprey reproductive success would potentially be higher than estimated.
- The osprey toxicity analysis assumes that there are no significant pharmacokinetics differences between eagles and osprey.
- The average PCB concentration in the Cumberland Bay fish tissue samples is representative of the concentration in fish consumed by osprey that may be nesting in the vicinity of the Site.

#### LITERATURE CITED IN SECTION 4.0 (FWIA)

- Anderson, J. R. 1976. A Land Use and Land Cover Classification System for Use With Remote Sensor Data. Geological Survey Professional Paper 964.
- Andrle, R. F.; Carroll, F.R. 1988. The Atlas of Breeding Birds In New York State. Cornell University Press.
- Bellrose, F.C. 1978. Ducks, Geese & Swans of North America. Stackpole Books, Second Edition.
- Bowerman, W. W., et.al., 1995. A Review of Factors Affecting Productivity of Bald Eagles in The Great Lakes Region: Implications for Recovery. Environmental Health Perspectives, Volume 103 Supplement 4, May 1995.
- Giesy, J.P., et.al. 1994. Contaminants in Fishes from Great Lakes-Influenced Sections and above Dams of Three Michigan Rivers. II. Implications for Health of Mink. Archives of Environmental Contamination and Toxicology. pp. 27, 213-223.
- Heaton, S. N.; et. al. 1995. Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan.

  1. Effects on Reproduction and Survival, and the Potential Risks to Mink Populations.

  Archives of Environmental Contamination and Toxicology. pp. 28,334-343.
- Hoffman, D. J., et. al. 1996. PCBs and Dioxins in Birds Chapter 7; Environmental Contaminants in Wildlife, Society of Environmental and Environmental Toxicology, Lewis Publishers (1996).
- Kubiak, T.J., Best, D.A., 1993, Wildlife Risks Associated With Passage of Contaminated Anadromous Fish at Federal Energy Regulatory Commission Licensed Dams in Michigan. Manuscritp Report. East Lansing MI. U. S. Fish and Wildlife Service.
- NYSDEC. March 1990. New York Natural Heritage Program. Reschke, C., Ecological Communities of New York State.
- NYSDEC. October 1993. Division of Water Technical and Operational Guidance Series (1.1.1), Ambient Water Quality Standards and Guidance Values.
- NYSDEC. November 1993. Division of Fish and Wildlife, Technical Guidance for Screening Contaminated Sediments.
- NYSDEC. October 1994. Division of Fish and Wildlife, Fish and Wildlife Analysis, For Inactive Hazardous Waste Sites (FWIA).

- Safe, S. Polychorinated Biphenyls (PCBs), Dibenzo-p-Dioxins (PCDDS), Dibenzofurans (PCDFs) and Related Compounds: Environmental and Mechanistic Considerations Which Support the Development of Toxic Equivalency Factors (TEFs). Critical Reviews in Toxicology. Vol 21. Issue 1. pp. 51-88.
- United States Department of the Interior, Fish and Wildlife Service, Eisler, R. April 1993.

  Polychorinated Biphenyl Hazards to Fish, Wildlife and Invertebrates: A Synoptic Review, Contaminant Hazard Review. Report No. 7. Biological Report 85(1.7).
- USEPA, Office of Research and Development. December 1993. Wildlife Exposure Factors Handbook. Volume I of II. EPA/600/R-93/187a.
- USEPA. March 1989. *Risk Assessment Guidance for Superfund*. Volume II. Environmental Evaluation Manual. EPA/540/1-89/001.
- USEPA. March 1989. Environmental Research Laboratory. Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference. EPA/600/3-89/013.
- Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Company.
- Werner, R.G. 1980. Freshwater Fishes of New York State, A Field Guide. Syracuse University Press.

**SECTION 4.0** 

**TABLES** 

# TABLE 4-1 DOMINANT VEGETATION IN NATURAL AREAS WITHIN ONE-HALF MILE OF THE SITE NYSDEC CUMBERLAND BAY SITE 5-10-017

P1 # 1: Emergent Marsh

Cattails (Typha species), Fragrant Water Lily (Nymphaea odorata)

P2 # 2: Deciduous Forested Wetland/Shrub Wetland

Red Maple (Acer rubrum), Willow (Salix species), Common Cottonwood (Populus deltoides), Red-Oiser Dogwood (Cornus stolonifera)

TN2D # 3: Deciduous Forest

Common Cottonwood, Boxelder (Acer negundo)

P2 # 4: Deciduous Forested Wetland/Shrub Wetland

Red Maple, Willow, Red-Oiser Dogwood, Common Cottonwood, Cattail species

P2 # 5: Deciduous Forested Foodplain Wetland

American Elm (Ulmus americana) Willow, Boxelder, and Common Cottonwood

TN1/TN2D Deciduous Forest/Succesional Field

# 6:

Common Cottonwood, Boxelder

TN2D # 7: Deciduous Forest

Common Cottonwood, Boxelder, Ash species (Fraxinus species)

P2 #8 Coniferous Forested Wetland

Northern White Cedar (Thuja occidentalis), Speckled Alder (Alnus rugosa), Sensitive Fern (Onoclea sensibilis)

# Table 4-2 NYSDEC Cumberland Bay Site Mammal/Amphibian/Reptile Species That Could Potentially Utilize Habitats Within One-Half Mile of the Site

#### COMMON NAME | GENUS AND SPECIES

#### **Mammals**

Big Brown Bat Eptesicus fuscus Beaver Custor canadensis Eastern Cottontail Sylvilagus floridanus White-tailed Deer Odocoileus virginiana Ermine Mustella erminea Red Fox Vulpes vulpes Mink Mustella vison Hairy-tailed Mole Parascalops brewri Star-nosed Mole Condylura cristata Deer Mouse Peromyscus maniculatus House Mouse Mus musculus Meadow Jumping Mouse Zapus hudsonius Woodland Jumping Mouse Napaeozapus insignis Muskrat Ondatra zibethica Myotis keenii Keen's Myotis Little Brown Myotis Myotis lucifugus Didelphis virginiana Virginia Opossum Raccoon Procyon lotor Norway Rat Rattus norvegicus Northern Short-tailed Shrew Blarina brevicauda Striped Skunk Mephitis mephitis Gray Squirrel Sciurus carolinensis Meadow Vole Microtus pennsylvanicus Southern Red-backed Vole Clethrionomys gapperi Woodland Vole Microtus pinetroum Long-tailed Weasel Mustella frenata Woodchuck | Marmota monax

# Table 4-2 NYSDEC Cumberland Bay Site Mammal/Amphibian/Reptile Species That Could Potentially Utilize Habitats Within One-Half Mile of the Site

#### COMMON NAME | GENUS AND SPECIES

#### Amphibians/Reptiles

Bull Frog Rana catesbeiana Rana clamitans Green Frog Pickerel Frog Rana palustris Rana sylvatica Wood Frog Eastern Newt Notophthalmus viridescens Spring Peeper Hyla crucifer Ambystoma mulculatum Four-Toed Salamander Brown Snake Storeria dekayi Eastern Ribbon Snake Thammophis sauritus Northern Water Snake Nerodia sipedon Storeria occipitmaculata Redbelly Snake Clemmys muhlenbergi Bog Turtle Painted Turtle Chrysemys picta Snapping Turtle Chetydra serapentina Clemmys guttata Spotted Turtle

Table 4-3
NYSDEC Cumberland Bay
Fish Species That Could Potentially Utilize Habitats Within OneHalf Mile of the Site

COMMON NAME	GENUS AND SPECIES
Atlantic Salmon	Salmo salar
Banded Killfish	Fundulus diaphanus
Blacknose Shiner	Notropis heterolepis
Blacknose Dace	Rhinichthys atratulus
Bluegill	Lepomis macrochirus
Bluntnose Minnow	Pimephales notatus
Brook Trout	Salvelinus fontinalis
Brook Stickleback	Culaea inconstans
Brown Bullhead	Ictalurus nebulosus
Brown Trout	Salmo trutta
Burbot	Lota lota
Chain Pickerel	Esox niger
Common Shiner	Notropis cornutus
Creek Chub	Semotilus astromaculatus
Fantail Darter	Etheostoma flabellare
Fathead Minnow	Pimephales promelas
Golden Shiner	Notemigonus crysoleucas
Lake Trout	Salmo namaycush
Largemouth Bass	Micropterus salmoides
Longnose Dace	Rhinichtys cataractae
Pearl Dace	Semotilus margarita
Pumpkinseed	Lepomis gibbosus
Rainbow Trout	Salmo gairdneri
Rock Bass	Ambloplites rupestris
Smallmouth Bass	Micropterus dolomieui
Spottail Shiner	Notropis hudsonius
White Sucker	Catostomus commersoni
Yellow Perch	Perca flavescens

### Table 4-4 NYSDEC Cumberland Bay Bird Species

That Could Potentially Utilize Habitats Within One-Half Mile of the Site

#### **COMMON NAME GENUS AND SPECIES** Carduelis trustis American Goldfinch American Kestrel Falco sparverius American Crow Corvus brachyrhynchos American Black Duck Anas rubripes Turdus migratorius American Robin Haliaeetus leucocuphalus Bald Eagle Barn Swallow Hirudo rustica Black-capped Chickadee Parus atricapillus Cyanocitta cristata Blue Jay Blue-winged Teal Anas discors Brown-headed Cowbird Molothrus ater Bucephala albeola Bufflehead Canada Goose Branta canadensis Cedar Waxwing Bronbycila cedrorum Chimney Swift Chaetura pelagica Common Merganser Mergus merganser americanus Common Yellowthroat Geothypis trichas Common Barn Owl Tyto alba Common Nighthawk Chordeiles minor Common Grackle Quiscalus guiscula Accipiter cooperii Copper's Hawk Picoides pubescens Downy Woodpecker Eastern Phoebe Sayonis phoebe European Starling Stumus vulgaris Great Blue Heron Ardea herodias Great-horned Owl Dubo virginianus Aythya marila mariloides Greater Scaup Green-Backed Heron Butorides striatus Hooded Merganser Mergus cucullatus House Wren Troglodytes aedon House Sparrow Passer domesticus Killdeer Charadrius vociferus Lesser Scaup Aythya affinis Mallard Anas platyrhynchos Zenaida macroura Mourning Dove Cardinalis cardinalis Northern Cardinal Osprey Pandion haliaetus Pileated Wood-Pecker Dryocopus pileatus Red-tailed Hawk Buteo jamaicensis

Agelaius phoneniceus

Red-winged Blackbird

## Table 4-4

NYSDEC Cumberland Bay
Bird Species
That Could Potentially Utilize Habitats Within One-Half Mile of the Site

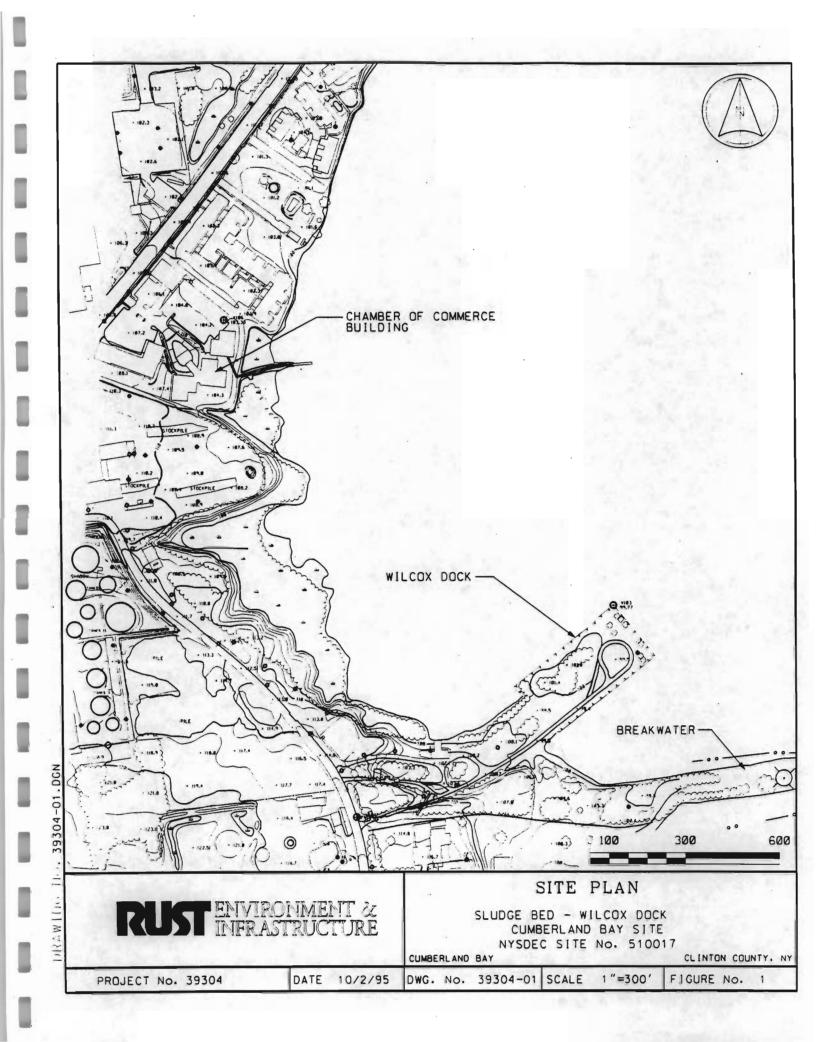
	COMMON NAME	GENUS AND SPECIES			
7	Ring-Billed Gull	Larus delawarensis			
	Rock Dove	Columba livia			
	Rough-winged Swallow	Stelgidoptery ruficollis			
	Ruby-throated Hummingbird	Archilochus colubris			
	Ruffed Grouse	Bonasa umbellus			
	Screech Owl	Otus asio			
	Song Sparrow	Melospiza melodia			
	Spotted Sandpiper	Actitis macularia			
	White-breasted Nuthatch	Sitta carolinensis			
	Wild Turkey	Meleagris gallopavo			
	Wood Duck	Axis sponsa			
	Yellow Warbler	Dendroica petechia			

TABLE 4.5
PCB Concentrations in Sludge Bed 0 - 25 Centimeters
NYSDEC Cumberland Bay Site

Analyzed by	Sample Date	Location Core ID	Core Interval (cm)	Sample Interval (cm)	1221	1242	PCB Arock 1248 (ppm)	1254	1260	Total PCBs* (ppm)
NYSDEC	03/17/94	CB1	0-25	1	0.025	30	0.175	0.175	0.175	30
				3	0.025	25	0.025	0.025	0.025	25
				5	0.025	20	0.025	0.025	0.025	20
				7	0.0875	14	0.0875	0.0875	0.0875	14
				9	0.025	13	0.025	0.025	0.025	13
				11	0.025	18	0.025	0.025	0.025	18
				13	0.025	16	0.025	0.025	0.025	16
				15	0.025	29	0.025	0.025	0.025	29
				17	0.025	27	0.025	0.025	0.025	27
				19	0.025	30	0.025	0.025	0.025	30
				21	0.025	29	0.025	0.025	0.025	29
				23	0.025	23	0.025	0.025	0.025	23
				25	0.025	22	0.025	0.025	0.025	22
NYSDEC	03/17/94	CB7a	0-26	1	0.025	96	0.025	0.025	0.025	96
				3	0.025	101	0.025	0.025	0.025	101
				5	0.025	24	0.025	0.025	0.025	24
				7	0.025	31	0.025	0.025	0.025	31
				10	0.025	34	0.025	0.025	0.025	34
				14	0.025	38	0.025	0.025	0.025	38
				18	0.025	123	0.025	0.025	0.025	123
				22	0.025	136	0.025	0.025	0.025	136
				26	0.025	182	0.025	0.025	0.025	182
NYSDEC	03/17/94	CB9	0-43.5	1	0.125	147	0.125	0.125	0.125	147
WIODEO	00/1//04	000	0 40.0	3	0.0625	63	0.0625	0.0625	0.0625	63
				5	1.25	61.6	1.25	1.25	1.25	61.6
				7	1.25	1850	1.25	1.25	1.25	1850
				10	1.25	1500	1.25	1.25	1.25	1500
					0.125	295	0.125	0.125	0.125	295
				14				1.25	1.25	
				18	1.25	368	1.25			368
				22	1.25	158	1.25	1.25	1.25	158
				26	0.00125	0.7	0.00125	0.00125	0.00125	0.7
NYSDEC	03/17/94	CB12	0-30.8	1	0.0125	13	0.0125	0.0125	0.0125	13
				3	0.00125	1.7	0.00125	0.00125	0.00125	1.7
				5	0.00125	0.00125	0.00125	0.00125	0.00125	0.00125
				7	0.00125	0.00125	0.00125	0.00125	0.00125	0.00125
				10	0.0125	0.0125	0.0125	0.0125	0.0125	0.0125
YSDEC/RECRA	08/09/94	1	0-48	0-3	0.415	0.2	0.205	0.170	0.130	0.500
				3-5	0.65	1.9	0.32	0.32	0.32	1.9
				5-25	0.415	0.079	0.205	0.205	0.205	0.079
YSDEC/RECRA	08/09/94	2	0-83.5	0-20	0.75	38	0.33	0.33	0.33	38
YSDEC/RECRA	08/09/94	3	0-89	0-15	0.355	1.7	0.175	0.175	0.175	1.7
YSDEC/RECRA	08/09/94	4	0-36	0-9	0.85	8.9	0.41	0.41	0.41	8.9
				9-11	0.8	25	0.395	0.395	0.395	25
				11-15	0.23	0.430	0.115	0.115	0.115	0.43
YSDEC/RECRA	08/09/94	5	0-81	0-18	1	2.2	0.48	0.48	0.48	2.2
YSDEC/RECRA	08/09/94	9	0-46.5	0-3	0.95	2.5	0.475	0.475	0.475	2.5
, 0020,,,20,,,	00/00/01	•	0 10.0	3-11	2.05	71	1	1	1	71
				11-16	1.95	160	0.95	0.95	0.95	160
				16-20	26.5	550	13	13	13	550
				20-23	8.5	49	4.1	4.1	4.1	49
AQUATEC	08/17/95	A-6	0-61	0-15	3.5	38	1.7	1.7	1.7	38
AQUATEC	08/17/95	. C-6	0-61	0-15	8	270	3.95	3.95	4.2	270
NYSDEC	08/17/95	C-7	10.2-40.6	10.2-25.4	0	0.73	0.00	0.00	7.2	0.073
NYSDEC	08/17/95	D-6	0- 30.5	0-15.2		11				11
AQUATEQ	08/10/95	F-7	0-61		0.235	3.6	0.115	0.115	0.6	3.6
AQUATEQ	08/10/95		0-45.7	0- 20.3		57			1.2	57
		G-6		0-15	1.65		0.8	0.8		
AQUATEQ	08/17/95	G-8	0-61	0-15	0.44	1.7	0.215	0.215	0.215	1.7
Inchcape Lab		AS-3	0-61	0-24	0.75	74	0.365	0.365	0.5	74
Inchcape Lab		AS-4	0-61	0-24	0.75	65	0.345	0.345	0.5	65
Average Concentration: Sample ndard Deviation: Minimum Concentration				1.192018 3.766634	107.4143 307.5709	0.649211 1.852367	0.645614 1.853444	0.672895 1.860169	117.8067 312.2020 0.00125	

**SECTION 4.0** 

**FIGURES** 



#### 5.0 DEVELOPMENT OF REMEDIAL ACTION OBJECTIVES

The results of the baseline HRA and FWIA verify the need to perform remedial activities at the Cumberland Bay Sludge Bed Site due to the presence of potential risks to human health and the environment created by PCBs in the Sludge Bed. These risk assessments concluded that completed exposure pathways exist for both humans and wildlife.

The two current and potential future exposure scenarios, identified in the HRA, that may pose a potential long-term human health concern are:

- direct contact with sludge bed and surface water while wading in the sludge bed near Wilcox Dock; and
- recreational ingesting of Cumberland Bay fish.

The FWIA draws the following conclusions regarding the risks to fish and wildlife:

- PCB concentrations in Cumberland Bay surface water over the sludge bed exceed the NYSDEC surface water standard developed for protection of wildlife from bioaccumulation of PCBs.
- PCB concentrations in the sludge bed exceed the NYSDEC sediment quality criteria value developed for protection of piscivorous wildlife from the toxic effects of PCB bioaccumulation.
- A potential exists for impact on the reproductive success of mink which utilize the
  habitats associated with wetland PB-5. Mink serve as an indicator of the risk to any of
  a variety of sensitive species that may live or feed in the vicinity of the Site.

The human health and environmental risks identified in the HRA and FWIA provide a basis for development of site-specific Remedial Action Objectives (RAOs). RAOs have been developed to minimize or reduce to target levels, the potential for human exposure or environmental damage due to the presence or migration of PCB impacted sediments. The RAOs for the Site are as follows:

- Mitigate the potential threat to the environment posed by the sludge bed;
- Rapidly and significantly reduce human health and environmental risks; and
- Prevent further environmental degradation resulting from this known source of contamination.

A Site remedy, capable of meeting these RAOs, would mitigate the human health and environmental risks identified in the HRA and FWIA. A post-remediation monitoring program would be necessary to evaluate the effectiveness of the remedy at reducing potential exposures of humans or wildlife to PCBs.

APPENDIX A

Toxicity Profile for Polychlorinated Biphenyls

### POLYCHLORINATED BIPHENYLS (PCBs)

#### A Toxicological Profile

#### Rust Environment & Infrastructure

This report represents a preliminary assessment of the subject chemical's potential for injury to human health and the environment and thus may not reflect all available information. Additions to, or changes in, the information contained herein may alter the assessment of the subject chemical. Therefore, any recommendations contained in, or based upon, this report are considered tentative.

#### **PCBs**

CAS No. 1336-36-3

#### **SYNONYMS**

Aroclor; Aroclor 1221; Aroclor 1232; Aroclor 1242; Aroclor 1248; Aroclor 1254; Aroclor 1260; Aroclor 1262; Aroclor 1268; Aroclor 2565; Aroclor 4465; Aroclor 5442; biphenyl, polychloro-; Chlophen; Chlorextol; chlorinated biphenyl; chlorinated diphenyl; Clophen; Fanclor, Kanechlor; PCB; PCBs; polychlorinated biphenyl; Pyranol; Pyralene; Santotherm; Sovol; Therminol FR-1; UN 2315.

#### **ORIGIN AND USES**

PCBs consist of the ten families of mono- to deca-chlorinated biphenyls; each family except deca-chlorinated biphenyl having multiple positional isomers based on the pattern of chlorine distribution on benzene ring carbon atoms. Counting all families and their isomers, the PCBs consist of 209 congeners. (HSDB, 1997).

Until banned from U.S. commerce in 1980 under the Toxic Substances Control Act (TSCA), PCBs had been sold commercially as mixtures of mono- to deca- chlorinated biphenyls, including up to 46 isomers of the penta-chlorinated compound. Until 1976, the sole U.S. producer was Monsanto, which marketed PCBs under the generic trade name Aroclor, followed by a four-digit code. The first two digits were usually 12, indicating PCBs, and the last two digits indicated the weight-percent of chlorine. For example, Aroclor 1254 denotes a mixture of PCBs containing 54 percent chlorine by weight. (HSDB, 1997)

Commercially, PCBs have been used primarily as components of capacitors and transformers. However, environmental releases were found to have resulted in widespread dissemination. PCBs have also been found to be highly persistent in the environment as a result of their tendency to strongly bind with particles and to resist physical, chemical, and biological degradation processes. (HSDB, 1997)

#### **PHARMACOKINETICS**

#### Absorption

The efficiencies with which PCBs are absorbed following exposure via the inhalation and ingestion routes have been reported to equal >50 and >90 percent, respectively (ATSDR, 1992; EPA, 1988). Absorption efficiency via the dermal route has been variously reported to equal 5% up to 59% (ATSDR, 1992; EPA, 1988). According to the U.S. EPA's 1988 Drinking Water Criteria Document:

Several dermal studies with PCB congeners or mixtures demonstrate that these compounds are readily absorbed and elicit toxic or biologic effects at dermal and distal sites. A recent study by Westar, *et al.* (1983) reported the dermal absorption in guinea pigs and monkeys of synthetic <sup>14</sup>C-labeled PCBs containing 42 and 54 chlorine (by weight).

The estimated absorption of the 42 and 54 %  $^{14}$ C mixtures was 33 and 56 % respectively, in the guinea pigs and the absorption of the 42 % mixture varied between 15 and 34 % depending on the dose (4.1  $\mu$ g/cm² or 19.3  $\mu$ g/cm²; source document, pp. III-6 to III-7).

As a first approximation, it is appropriate to assume that absorption efficiency via inhalation and ingestion are equal to 100 percent, given the indefinite upper bounds signified by ">50 and >90 percent." However, absorption by these routes is actually likely to be significantly less than 100 percent. This is particularly so in the case of PCBs which may be tightly bound to particles high in organic content, such as sediment fly ash, and may therefore be to a large degree biologically unavailable.

#### Distribution

The principal tissues and organs to which PCBs are distributed following absorption have been reported to include liver, muscle, and skin, with the highest concentrations found in adipose (fat) tissue (ATSDR, 1992; EPA, 1988).

#### Metabolism

PCBs metabolism occurs principally in the liver, involving the mixed function oxidase system of enzymes and specifically, aryl hydrocarbons hydroxylases (AHH). Metabolism proceeds by epoxidation (formation of reactive epoxides, tricyclic -C-O-C- groups), ring hydroxylation (addition of OH¹ groups to ring carbons), and oxidation of the remaining catechol (o-diphenol) nucleus (Klaassen et al., 1991). Metabolic rates of PCBs vary inversely with pattern and degree of chlorination; more highly chlorinated congeners are typically metabolized more slowly that less-chlorinated congeners. Mono-, di-, and tri-chlorinated biphenyls may be metabolized to a variety of hydroxy-, dihydroxy, and methoxy-chlorinated biphenyl compounds, as well as to excretable glucuronide conjugates. (ATSDR, 1992)

The rates of tetra-chlorinated biphenyl metabolism depend markedly on their pattern of chlorination, but produce structurally similar though not identical metabolites. Penta- and hexa- chlorinated biphenyls are metabolized to di-, tri, and tetra-chlorinated congeners, and undergo further metabolism as described above, as possibly do the higher-chlorinated congeners as well. Major metabolites are phenols and, possibly, potentially carcinogenic electrophilic arene oxide intermediates (EPA, 1985).

#### **Excretion**

PCBs are highly persistent in the human body, but excretion does occur (ATSDR, 1992; EPA, 1985). Half-times for PCB excretion in animal studies were reported as being up to 100.5 days. Some unaltered PCB was found in the feces, but PCB metabolites appear to be exclusively in the urine. Conjugates included glucuronides of hydroxylated metabolites, as well as glutathione and other sulfur-containing substances detected in the urine of mice and rats. In humans, evidence of PCB excretion has been derived from an outbreak of poisoning by PCB-contaminated rice oil in Taiwan. Calculated half-lives for the 2,4,5,2', 4'-penta- and the 2,3,4,3',4'-penta-chlorinated biphenyl isomers were 9.8 and 6.7 months, respectively, in blood, although it cannot be assumed that disappearance from blood indicates elimination from the body (see *Distribution* subsection).

#### **EXPERIMENTAL ANIMAL STUDIES**

Numerous investigations of the toxic properties of acute exposure to PCBs have been undertaken, involving mice, rats, hamster, rabbits, and monkeys. LD<sub>50</sub> values have been reported as being from 1,010 mg/kg for Aroclor 1254 to 4,250 mg/kg for Aroclor 1242 in rats following oral administration. Toxicity appears to decline with increasing chlorination. Effects in animals have included weight loss, elevation of liver weight (hepatomegaly) and fat content, depressed body temperature and appetite, thymus gland hemorrhage, kidney enlargement, splenic and lymph node regression, increased thyroid gland activity, alterations in cholesterol and fatty acid synthesis, and other effects. (ATSDR, 1992; EPA, 1985)

PCBs or their metabolites have been shown to cross the mammalian placenta, in mice and rats, as well as humans (ATSDR, 1992; EPA, 1984). Little evidence of teratogenicity exists, however, two U.S. studies evaluating exposure to PCBs assumed to have been consumed in contaminated fish have raised the possibility that PCBs may cause developmental effects (ATSDR, 1992). Due to confounding factors including exposure to DDT and other organochlorine pesticides, the adverse developmental effects cannot be attributed specifically to PCBs. Data relating to PCB teratogenicity following inhalation are unavailable. The EPA (1984) reports that teratogenicity was, however, demonstrated in a gavage study of pregnant CD-1 mice exposed during days 6-15 of gestation. At doses of ≥2 mg/kg/day, significantly elevated and dose-related incidence of cleft palate appeared, and at ≥4 mg/kg/day hydronephrosis (kidney dilation due to obstruction of urine flow) appeared. A more recent study by Pantaleoni and coworkers (1988) has revealed causation of behavioral teratogenic effects in Fischer 344 rats maternally exposed to PCBs at levels too low to affect birth size and maturation or to cause gross physical malformations. These investigators administrated 1-2 mg/kg/day of PCBs by gavage for 20 days to lactating females, and compared several behavioral capabilities of treated vs. control pups. Statistically significant behavioral deficits, such as reduced head raising during swimming, were observed in both dose groups.

Several studies evaluating the effects of PCBs on reproductive performance have been conducted. Complete reproductive failure has been induced in minks by Aroclor 1242

dietary levels of 5 mg/kg. Lengthened estrus cycles were induced by PCBs in mice exposed to 0.025 mg/day of Clophen A-60. Irregular menstrual cycles and reduced serum progesterone levels were exhibited by monkeys fed Aroclor 1248 at 2.5 mg/kg. Male rats exposed to Aroclor 1254 from birth exhibited reduced impregnation of females after 130 days of treatment, including impaired mating behavior. The author speculated that this behavioral impairment may have been mediated by insufficiency of circulating androgen levels resulting from subnormal development of accessory sex glands, as well as testicular hyperplasia in treated rats. (EPA, 1985)

In one study, conducted by Barsotti and Van Miller (1984), as little as 1.0 ppm of PCB (Aroclor 1016) in the diets of adult female rhesus monkeys was associated with a dose-related reduced birth weights of their offspring. However, a close examination of this report reveals methodological deficiencies which provide uncertainties in concluding that PCB directly caused birth weight reduction, or exerted any toxic reproductive (or fetotoxic) effect at the levels reported. First, maternal food consumption was apparently reduced by PCB in the diet. Second, the control group consisted of a distinctly different population of (feral) monkeys with respect to year of delivery to the laboratory (1973 vs. 1977), age, and acclimation to captivity, when compared with the two treatment groups. Due to these study deficiencies, a definitive conclusion cannot be made concerning whether PCBs have been shown to exert a toxic reproductive effect or a fetotoxic effect in the Barsotti and Van Miller study, although one can hypothesize that PCB reduced maternal appetite for food. In the absence of reported data about the pre-study weights of adult females in each group, it is also possible to hypothesize that the high-dose group contained somewhat smaller monkeys.

Evidence of chronic PCB effects has been principally obtained from animal experiments, almost all involving rodents (ATSDR, 1992; EPA, 1984). Dietary levels of ≥500 ppm of Aroclor 1254 eventually proved fatal to rats. Reduced appetite, as well as reduced rate of weight gain ands fina: body weight, were also exhibited in rats exposed to 500 ppm of PCB orally. At 100 ppm, exposed rats exhibited hepatomegaly (enlarged livers, in this study by factors of up to three- to four-fold). Other liver abnormalities at the same dietary exposure level included appearance of discolored nodules; proliferation of oval cells and bile duct (cholangiofibrosis); fatty liver degeneration; elevation of serum lipids and cholesterol; increased hepatic protein, RNA, and lipid; decreased DNA content; and increased microsomal total protein and cytochrome P450 content. Monkeys, which are more sensitive than rats to PCBs, exhibited liver pathology and associated alterations of serum chemistry as in other species (levels not reported). At levels as low as 2.5 ppm in the diet, monkeys also exhibited skin lesions, erythema (redness), and periorbital edema (swelling around the eyes), alopecia (baldness), acne, facial edema, and hyperpigmentation. (EPA, 1985).

Mice, rats, and rabbits have exhibited signs of PCB immunosupression, including degeneration of the thymus gland, lymphocytopenia (reduced circulating white cells in blood (IRIS, 1992; EPA, 1985). Guinea pigs injected with tetanus toxoid following dietary PCB exposure as low as 10 mg/kg exhibited dose-related reduction of hemagglutination (blood cell clumping, an indicator of immune response to an antigen). Cell-mediated immunity was also suppressed in guinea pigs. Finally, BALB/CJ mice challenged with exposure to

endotoxins or noxious organisms, such as *Salmonella typhosa* and the malaria organisms, *Plasmodium berghei* following PCB exposure (Aroclor 1242 or 1016 at 167 mg/kg) exhibited reduced survival. Suppression of immunocompetency lesions in the lungs, thymus, mesenteric lymph nodes, or spleen, although hepatocytic hyperplasia was observed in the liver. (EPA, 1985).

The majority of genotoxicity assays of PCBs have been negative. The majority of microbial assays have produced no evidence of mutagenic effects. Of the various tests on the clastogenic effect of PCBs, results have only been reported in a single study indicating clastogenic action by PCBs in dove embryos. (IRIS, 1997).

A number of studies have evaluated the carcinogenicity of PCBs to animals. Much of the animal data documents causation of liver cancer in rodents, and the significance of such data in the context of human risk is controversial for reasons relating to the observations that laboratory strains of rodents exhibit a relatively high spontaneous liver tumor incidence. However, recent research seems to have moved the weight-of-evidence toward significance. Norback and Weltman (1985) administered Aroclor 1260 (60 percent chlorine) in their diet to 140 rats for 18 months at a concentration of 100 ppm and for an additional 8 months at 50 ppm. The authors reported a statistically significant increase in the incidence of malignant liver tumors. Reynolds et al. (1987) reported that PCBs induced liver tumors in rodents which differed significantly with respect to their spectrum of activating mutations compared with tumors observed in untreated animals.

Several studies have revealed the ability of PCBs to induce liver tumors in rodents. Ito *et al.* (1978) reported that 5 of 12 mice fed a diet including 500 ppm of PCBs exhibited hepatocellular carcinoma, whereas none of the six control mice exhibited the cancer. Likewise, Kimbrough *et al.* (1975) observed a significant increase in the incidence of hepatocellular carcinoma in female rats fed a 100 ppm of Aroclor 1260 for 21 months. Male rats were not studied. Twenty-six of 184 female rats fed PCB exhibited tumors, compared with only one of 173 control rats. Additionally, 146 of the 184 treated rats exhibited neoplasms of their livers, compared with none of the controls. Kimbrough and Linder (1974) investigated PCB causation of hepatomas in mice. Nine of 22 mice fed 330 ppm of Aroclor 1254 for 11 months exhibited hepatomas, compared with no hepatomas exhibited among 100 control mice. Nagasaki (1972) reported that among 12 male mice fed Kanechlor 500 at 500 ppm, seven exhibited multiple liver tumors, whereas controls exhibited no remarkable changes in their livers.

The National Cancer Institute (NCI) conducted a long-term bioassays to evaluate the carcinogenicity of PCBs administered to rats via the diet (NCI, 1978). Aroclor 1254 was not proven carcinogenic, although adenocarcinomas of the gastrointestinal tract appeared in treated but not control animals. NCI indicated that the low historical background incidence of such lesions in its laboratory suggested that their elevated occurrence in the PCB bioassay was caused by PCB. Also, PCBs appeared to be a promoter rather than a complete carcinogen, based on evidence consisting of a high, statistically significant increase in the incidence of hepatoce'lular proliferative lesions observed in both males and females. This

result confirms the results of other investigators indicating that PCB is a promoter of hepatocarcinomas (Ito et al., 1978; Kitigawa and Sugano, 1977; Nishizumi, 1976; Peraino et al., 1971; Peraino et al., 1974; Weisburger et al., 1975). Likewise, Aroclor 1254 was a promoter of tumors i mice treated with TCDD (EPA, 1985). Kimura, et al. (1976) found PCB to be a promoter of hepatocarcinomas in rats, with the tumor incidence reaching 64 percent among rats which were first administered the known carcinogen 3'-methyl-4-dimethylaminoazobenzene. The investigators concluded that their results strongly suggest that PCBs exert a potent promoting action in experimental azo dye hepatocarcinogenesis.

The rodent data discussed previously must be interpreted in the context of uncertainty because not all chemically induced rodent liver tumors necessarily differ from spontaneous (non-chemically-induced) tumors.

#### **HUMAN STUDIES**

Acute toxic effects associated with PCBs can occur in humans under unusual, and unusually intense, exposure scenarios, such as in intentional or accidental poisoning. Although  $LD_{50}$  values for humans appear to be unknown, PCBs have been rated moderately toxic, having a probable oral  $LD_{50}$  value in the range of 0.5-5 g/kg (Gosselin, 1984).

PCBs or their metabolites have been shown to cross the mammalian placenta, in mice and rats, as well as humans (EPA, 1984). However, little evidence of teratogenicity exists. (EPA, 1985).

Although PCBs are known to have an affinity for the uterus and fetotoxic effects have been documented, little information appears to be available relating to the effects of PCBs upon the reproductive systems of adult humans or animals (EPA, 1984; EPA, 1985; Taylor et al., 1989; Rogan et al., 1988). One recent epidemiological study follows up a 1979 incident in Taiwan of mass poisoning of individuals using cooking oil contaminated with thermally degraded PCBs (Rogan et al, 1988). In 1985, 225 children were examined, including 117 born to affected women. The children born to exposed women were assumed to be exposed in utero to persistent PCB residues in maternal tissues, and were reported to differ from control children. The authors concluded that "exposed children were shorter and lighter than controls; [and] they had abnormalities of gingiva, skin, nails, teeth, and lungs more frequently than did controls". The authors acknowledged one complication of the study: "The oil in Taiwan had about 100 ppm PCBs, and about 0.1 ppm PCDFs (polychlorinated dibenzofurans). Although there has not been a human exposure to PCDFs in the absence of PCBs, it is reasonable to assume that much of the toxicity seen is due at least in part to PCDF contamination".

In another recent epidemiological study (Taylor et al., 1989), a statistically significant doseresponse relationship was observed between increased estimated serum PCB level and decreased birth weight and gestational age among women who had been occupationally exposed to PCBs (Aroclors 1254, 1242, and 1016) for at least three months in either of two capacitor manufacturing plants in upstate New York between 1946 and 1975. Serum concentrations of women exposed to PCBs via dermal contact and inhalation exhibited geometric mean serum levels of 302 ppb of total PCBs. Serum concentrations of women exposed only by inhalation exhibited a geometric mean of 61 ppb, and a reference group of women exhibited 16 ppb. Thus, the low-dose group of exposed women exhibited an incremental PCB concentration of 45 ppb (61 ppb-16 ppb).

It is difficult to relate PCB levels in blood serum measured at any point in time with chronic inhalation and dermal doses of PCBs. The work area of women exposed dermally and by inhalation was measured at levels ranging from 310 to 679 µg/m<sup>3</sup>, whereas personal air samples revealed levels of 168 µg/m<sup>3</sup>. Women exposed by inhalation only worked in areas in which air levels were measured at 27 to 260 µg/m<sup>3</sup>. Areas surrounding the plant exhibited air levels averaging 6.2 μg/m<sup>3</sup>, compared with typical urban ambient air levels in the range of 0.1 µg/m<sup>3</sup>. Assuming a mean PCB concentration in air of 40.7 µg/m<sup>3</sup>, in the inhalationexposure-only work area (based on 16 samples at 27 µg/m<sup>3</sup> and one sample at 260 µg/m<sup>3</sup>), the incremental PCB concentration above background may be estimated at 34.5 µg/m<sup>3</sup>. Assuming a breathing rate of 9.1 m<sup>3</sup>/8 hour workday yields an estimated daily intake of 314.0 μg/day (34.5 μg/m<sup>3</sup> x 9.1 m<sup>3</sup>/8 hour workday). Further assuming a body weight of 54 kg yields an estimated chronic incremental PCB dose of 5.8 μg/kg/day (314.0 mg/day ÷ 54 kg) received by the low-dose group of exposed women. The decrease in birth weight of infants born to these women was at least partially attributable to the shortening of the gestation period preceding birth. The authors concluded that "[t]he magnitude of these effects was quite small compared with those of other known determinants of gestational age and birth weight, and the biologic importance of these effects is likely to be negligible except among already low birth weight or short gestational infants".

Chronic exposure to !'CBs has been reported to cause chloracne in humans. Evidence of chronic PCB effects has been principally obtained from animal experiments, almost all involving rodents (EPA, 1984).

Only inconclusive epidemiological evidence pertains to causation of cancer in humans (EPA, 1984; EPA, 1985; IRIS, 1992). Although there are many studies the data are inadequate due to confounding exposures or a lack of exposure quantification (IRIS, 1992).

#### DOSE-RESPONSE PARAMETER ESTIMATION

The EPA estimates the dose-response parameters differently for carcinogenic and non-carcinogenic effects. Therefore, these estimates are presented separately below.

#### Noncarcinogenic

**Inhalation.** The EPA has not verified an inhalation reference dose (RfD) for PCBs (IRIS, 1997).

Oral. The EPA (IRIS, 1997) has not calculated an oral RfD for PCBs, in general. However, a chronic oral RfD has been derived for Aroclor 1254 based on monkey clinical and immunological studies (IRIS, 1997). In the studies, groups of 16 adult female rhesus monkeys ingested gelatin capsules containing Aroclor 1254 in 1:1 glycerol:corn oil vehicle daily at dosages of 0, 5, 20, 40 or 80 µg/kg/day for more than 5 years. The results of the study indicated a statistically significant increase in the total frequency of inflamed and/or prominent Meibomian glands at 0.005, 0.02 and 0.08 mg/kg/day, a decreased onset time for these effects at 0.08 mg/kg/day. Significant doserelated trends were also observed for increased total frequencies of inflamed and/or prominent Meibomian glands, decreased onset time of inflamed and/or prominent Meibomian glands, and increased incidences of eye exudate. Examination of finger and/or toe nails revealed increased incidence of certain nail changes at 0.005 mg/kg/day (nail folding) and 0.08 mg/kg/day (elevated nails), increased total frequency of certain nail changes at 0.005 mg/kg/day (nail separation), 0.04 mg/kg/day (nail folding and separation) and 0.08 mg/kg/day (nail folding, prominent beds, elevated nails). Immunologic assessment showed significant reductions in IgG and IgM antibody levels in response to injected sheep red blood cells after 23 months of exposure. A chronic oral RfD of 2 x 10<sup>-5</sup> mg/kg/day was derived by the EPA based on the lowest observed adverse effect level (LOAEL) of 0.005 mg/kg/day. The EPA applied uncertainty factors of 10 to account for sensitive individuals, 3 for extrapolation from monkeys to humans, and 3 for extrapolation from a subchronic to a chronic RfD. A partial factor was also applied for use of a minimal LOAEL since the changes in the periocular tissues and nail bed seen at the 0.005 mg/kg/day were not considered to be of marked severity. The total uncertainty factor applied was 300. These data yield inhalation, ingestion, and dermal RfDs of 0.001 ms/kg/day.

Inhala ion RfD (RfC): not available
Ingestion RfD: 2 x 10<sup>-5</sup> mg/kg/day
Dermal RfD: 2 x 10<sup>-5</sup> mg/kg/day

#### Carcinogenic

The EPA has classified PCB as a Group B2 carcinogen based on studies which found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260 (IRIS, 1997). these mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. The cancer potency of PCB mixtures is determined using a tiered approach that depends on the information available. The following tier descriptions discuss all potential environmental exposure routes considered by the EPA (IRIS, 1997) when evaluating risks to PCBs:

#### High Risk and Persistence

Upper-bound slope factor: 2.0 (mg/kg/day)<sup>-1</sup>

Criteria for Use: - Food chain exposure

- Sediment of soil ingestion - Dust or aerosol inhalation

- Dermal exposure, if an absorption factor has been applied

- Early life exposure (all pathways and mixtures)

#### Low Risk and Persistence

Upper-bound slope factor:

0.4 (mg/kg/day)-1

Criteria for Use: - Ingestion of water-soluble congeners

- Inhalation of evaporated congeners

- Dermal exposure, if no absorption factor has been applied.

#### Lowest Risk and Persistence

Upper-bound slope factor:

0.07 (mg/kg/day)-1

Criteria for Use:

Congener or isomer analyses verify that congeners with more than 4

chlorines comprise less than ½% of total PCBs

#### LITERATURE CITED

Barsotti, D.A., and J.P Van Miller. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. Toxicology, 30(1): 31-44.

EPA. 1988. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Final document, prepared for Office of Drinking Water. Cincinnati, OH, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, ECAO-CIN-414, i.p., April 1988;

EPA. 1985. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Environmental Criteria and Assessment Office, ECAO-CIN-414, EPA-600/X-84-198, April 1985.

1984. Health Effects Assessment for Polychlorinated Biphenyls. Final Draft. Cincinnati, Environmental Criteria and Assessment Office, ECAO-CIN-HO04, EPA-540/1-86/004, 55 pp., September 1984.

Gosselin, R.E., et al. 1984. Clinical Toxicology of Commercial Products, 5th Edition. Baltimore, Williams & Wilkins.

Hazardous Substances Data Bank (HSDB). 1997. *Polychlorinated Biphenyls*. National Library of Medicine, National Toxicology Information System, Bethesda, MD.

HEAST. 1995. Hea'th Effects Assessment Summary Tables, FY-1995 Annual. Office of Research and Development, Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, DC. OERR 9200.6-303 (95-1). May 1995.

Integrated Risk Information System (IRIS) Database. 1997. *Polychlorinated Biphenyls, Aroclor 1016, Aroclor 1248, Aroclor 1254, and Aroclor 1260.* Environmental Protection Agency, Washington, DC. March 19, 1997.

Ito, N., et al. 1978. Enhancing effect of chemicals on production of hyperplastic liver nodules induced by N-2-fluorenylacetamide in hepatectomized rats. Gann. 69:143-144, 1978;

Kimbrough, R.D. and Linder, R.E. 1974. *Induction of adenofibrosis and hepatomas of the liver in BALB/cj mice by polychlorinated biphenyls (Aroclor 1254)*. J. Natl. Cancer Inst. 53(2): 547-552.

Kimbrough, R.D., et al. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl (Aroclor 1260). J. Natl. Cancer Inst. 55(6): 1453-1459.

Kimura, N.T., Kanematsu, T. and Baba, T. 1976. Polychlorinated biphenyl(s) as a promoter in experimental hepopearcinogenesis in rats. Z. Krebsforsch. 87: 257-266.

Kitigawa, T. and Sugano, H. Enhancement of azo-dye hepatocarcinogenesis with dietary phenobarbital in rats. Gann. 68: 255-256.

Klaassen, C.D., M.O. Amdur, and J. Doull (eds.). 1991. Casarett and Doull's Toxicology-The Basic Science of Poisons, Fourth Edition. New York, Macmillan. 1991.

Nagasaki, H., et al. 1972. Hepatocarcinogenicity of polychlorinated biphenyls in mice. Gann. 63: 805.

NCI. 1978. Bioassay of Aroclor 1254 for possible carcinogenicity. National Cancer Institute, DHEW Publication Number (NIH) 78-838.

Nishizumi, M. 1976. Enhancement of diethylnitrosamine hepatocarcinogenesis in rats by exposure to polychlorinated biphenyls or phenobarbital. Cancer Lett. 2: 11-6.

Norback, D. H. and Weltman, R.H. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 60: 97-105.

Pantaleoni, G.C., et al. 1988. Effects of maternal exposure to polychlorinated biphenyls (PCBs) on  $F_1$  generation behavior in the rat. Fund. and Appl. Toxicol. 11: 440-449.

Peraino, C., Fry, R.J.M. and Staffeldt, E. 1971. Reduction and enhancement by phenobarbital of hep. a ocarcinogenesis induced in the rat by 2-acetylaminofluorene. Cancer Res. 31: 1506-1512.

Peraino, C., et al. 1974. Comparative enhancing effects of phenobarbital, amobarbital, diphenylhydantoin, and dichlorodiphenyl-trichloroethane of 2-acetylaminofluorene-induced hepatic tumorigenesis in the rat. Cancer Res. 35: 2884-2890.

Reynolds, S.H., et al. 1987. Activated oncogenes in  $B6C3F_1$  mouse liver tumors: implications for risk assessment. Science 237: 1309-1316.

Rogan, W.J., et al. 1988. Cogenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan. Science 241: 334-341.

Taylor, P.R., Stelma, J.M. and Lawrence, C.E. 1989. The relation of polychlorinated biphenyls to birth weight and gestational age in the offspring of occupationally exposed mothers. Amer. J. Epidemiol. 129(2): 395-406.

Weisburger, J.H., et al. 1975. Modification of dimethylnitrosamine liver carcinogenesis with phenobarbital but not with immunosuppression. J. Natl. Cancer Inst. 54: 1185-1188.



