

# **Appendix L**

## **Human Health Risk Assessment**

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### **Contents**

Human Health Risk Assessment (HHRA)

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# Contents

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<b>Contents.....</b>	<b>i</b>
<b>1    Introduction .....</b>	<b>1-1</b>
1.1    Introduction .....	1-1
1.2    Site Background .....	1-1
<b>2    Conceptual Site Model .....</b>	<b>2-1</b>
<b>3    Scope of Human Health Risk Assessment.....</b>	<b>3-1</b>
<b>4    Hazard Assessment/Identification of COPCs .....</b>	<b>4-1</b>
4.1    Data Summary.....	4-1
4.1.1    Surface Sediment .....	4-1
4.1.2    Surface Water .....	4-2
4.1.3    Ambient Air.....	4-2
4.1.4    Fish Tissue .....	4-2
4.2    Data Evaluation.....	4-3
4.3    Selection of COPCs .....	4-4
4.4    Constituents of Potential Concern.....	4-5
<b>5    Exposure Assessment.....</b>	<b>5-1</b>
5.1    Characterization of Exposure Setting.....	5-1
5.1.1    Physical Setting.....	5-1
5.1.2    Potentially Exposed Populations.....	5-1
5.2    Identification of Exposure Pathways .....	5-2
5.2.1    Contaminant Sources .....	5-2
5.2.2    Release and Transport Mechanisms .....	5-2
5.2.3    Exposure Points and Exposure Routes.....	5-3
5.2.4    Summary of Potential Exposure Pathways .....	5-3
5.3    Quantification of Exposure.....	5-3
5.3.1    Exposure Concentrations .....	5-3
5.3.2    Estimation of Chemical Intakes.....	5-5
<b>6    Toxicity Assessment.....</b>	<b>6-1</b>
6.1    Toxicity Information for Noncarcinogenic Effects .....	6-2
6.2    Toxicity Information for Carcinogenic Effects.....	6-2
6.3    Approach for Potential Mutagenic Effects .....	6-3
<b>7    Risk Characterization.....</b>	<b>7-1</b>
7.1    Methods for Estimating Risks .....	7-1
7.1.1    Noncarcinogenic Hazard Estimation.....	7-1
7.1.2    Carcinogenic Risk Estimation.....	7-1
7.1.3    Lead .....	7-2
7.2    Risk Assessment Results .....	7-3
7.2.1    Recreational Adults .....	7-4
7.2.2    Recreational Adolescents.....	7-4
7.2.3    Recreational Children .....	7-4
7.2.4    Recreational Receptors – Lifetime Carcinogenic Risks .....	7-5
7.2.5    Industrial Workers .....	7-6

7.2.6	Adult Residents .....	7-6
7.2.7	Child Residents .....	7-7
7.2.8	Child/Adult (Lifetime) Residents .....	7-7
7.2.9	Adult Anglers .....	7-8
7.2.10	Adolescent Anglers.....	7-9
7.2.11	Children of Anglers .....	7-9
7.2.12	Anglers - Lifetime Carcinogenic Risks .....	7-10
<b>8</b>	<b>Uncertainty Assessment.....</b>	<b>8-1</b>
8.1	Uncertainty in Data Evaluation and COPC Identification .....	8-1
8.2	Uncertainty Associated with Exposure Assessment .....	8-2
8.3	Uncertainty Associated with Toxicity Assessment .....	8-4
<b>9</b>	<b>Human Health Risk Summary .....</b>	<b>9-1</b>
<b>10</b>	<b>References.....</b>	<b>10-1</b>

## **Attachment**

USEPA RAGS Part D and Supporting Tables

## **Tables**

- 4-1 Summary of Data Quantitatively Used in HHRA
- 4-2 Chemicals of Potential Concern
- 5-1 WHO Dioxin-like PCB TEFs
- 5-2 Preparation/Cooking Loss Factor (PCF) for Fish
- 7-1 Summary of RME Cancer Risks and Hazard Indexes
- 7-2 Summary of CTE Cancer Risks and Hazard Indexes
- 7-3 PCBs, Mercury, and Arsenic in Canal and Reference Fish and Crab Tissue Samples
- 7-4 Summary of Total RME Cancer Risks for Recreational User and Angler
- 7-5 Summary of Total CTE Cancer Risks for Recreational User and Angler

## **Figures**

- 2-1 Conceptual Exposure Model for Human Health Risk Assessment

## **SECTION 1**

# **Introduction**

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## **1.1 Introduction**

This baseline human health risk assessment (HHRA) for the Gowanus Canal Superfund Site is Appendix L to the remedial investigation (RI) report. The baseline HHRA was conducted to assess the nature, magnitude, and probability of potential harm to public health posed by environmental media containing hazardous substances released to the Gowanus Canal by past activities along the canal—specifically, the potential human health risks associated with direct contact with sediment and surface water in the canal, with ingestion of fish and crabs caught in the canal, with direct contact of sediment and surface water that overtops the canal during significant rainfall events, and with inhalation of emissions from the canal into the ambient air near the canal. The data evaluated in the HHRA are presented in Appendix I to the RI report and discussed in Section 4 of the RI report.

## **1.2 Site Background**

The Gowanus Canal is located in Brooklyn, New York, a borough of New York City. The canal is connected to the Gowanus Bay in the Upper New York Bay and borders the neighborhoods of Red Hook, Cobble Hill, Carroll Gardens, and Park Slope. The waterfront adjacent to the canal is primarily commercial and industrial and currently consists of concrete plants, warehouses, and parking lots. There are five bridge crossings over the canal, at Union Street, Carroll Street, Third Street, Ninth Street, and Hamilton Avenue (Figure 1-1 of the RI). The Gowanus Expressway and the New York Subway also cross over the canal.

The Gowanus Canal, completed in the 1860s, was built to allow water access for industrial needs by bulkheading and dredging a tidal creek and wetland that was previously fished for oysters. The canal quickly became one of the nation's busiest industrial waterways and was home to heavy industry such as gas works, coal yards, manufacturers of cement, manufacturers of soap, tanneries, paint and ink factories, machine shops, chemical plants, and oil refineries. It was also used as a repository of untreated industrial wastes, raw sewage, and surface water runoff for many decades. Although much of the industrial activity along the canal has stopped, high contaminant levels remain in the sediments. Despite ongoing pollution problems, some local residents use the Gowanus Canal for recreational purposes such as canoeing and swimming/diving, while others catch and eat fish and crabs.

The City built a “flushing tunnel” in 1911 to replace stagnant canal water with fresh, oxygen-rich water to improve water quality. The tunnel was in operation until the 1960s, when a mechanical failure caused it to shut down and the canal water became stagnant and thus polluted once again. The flushing tunnel was rehabilitated and reactivated in 1999 by the City’s Department of Environmental Protection (NYCDEP) to pump water from Buttermilk Channel to the Gowanus Canal using the 1911 technology. The flushing tunnel

was shut down by NYCDEP on July 19, 2010, for an extended period of facility improvements to modernize the technology and improve operations. In early 2010, an aeration pipe was installed within the canal to circulate superoxygenated water while the flushing tunnel is shut down. The aeration pipe began operation in early July 2010, and repairs were started on the flushing tunnel. The completion date is anticipated to be September 2014.

The canal is part of the New York-New Jersey Estuary, which the U.S. Environmental Protection Agency (USEPA) has designated an Estuary of National Significance under the Clean Water Act Section 320 and USEPA's National Estuary Program.

## SECTION 2

# Conceptual Site Model

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The conceptual site model (CSM) for human receptors presents an overview of site conditions, potential contaminant migration pathways, and potential exposure pathways to potential receptors. Figure 2-1 presents the preliminary CSM developed for the Gowanus Canal. Table 1 in the Attachment summarizes the potential exposure pathways and scenarios that were evaluated as part of the risk assessment.

There have been many sources of contamination to the canal, as discussed in Section 1. Although much of the industrial activity along the canal has stopped, high contaminant levels remain in the sediments.

The watershed drainage area of the Gowanus Canal is 1,758 acres, and the canal waterfront, or riparian area (defined as all blocks wholly or partially within ¼ mile of the canal), is occupied by commercial and industrial properties. The waterfront properties include concrete plants, warehouses, and parking lots. The riparian areas are classified as 18 percent residential, 6 percent park, and 76 percent mixed use. The entire watershed is 53 percent residential, 2 percent park, and 45 percent mixed use (NYCDEP, 2008).

Combined sewers (i.e., sewers that receive both sewage and stormwater flows) serve 92 percent of the Gowanus Canal watershed, while storm sewers serve 2 percent (NYCDEP, 2008). Direct runoff drains 6 percent of the watershed. Three stormwater outfalls also discharge to the canal. During significant rainfall events, the Gowanus Canal receives untreated discharges of combined sewage that exceed the capacity of the Red Hook and Owls Head wastewater pollution control plants (WPCPs); during such events, the canal has overflowed onto neighboring properties and streets. The area surrounding the canal is within a 100-year flood zone, indicating that there is a 1 percent annual chance of flooding in this area (FEMA, 2007).

Currently, the New York State Department of Health (NYSDOH) has fish-consumption advisories for the Upper Bay of the New York Harbor (north of the Verrazano Narrows Bridge), including the Gowanus Canal (NYSDOH, 2010). There are no warning signs concerning the fish-consumption advisories posted along the canal; however, New York City has posted caution signs at locations of combined sewer outfall (CSO) discharges that state during wet weather the CSOs may discharge harmful bacteria to the canal and people should not swim, boat, or fish during these periods. A fishing license is not required to fish in the Gowanus Canal. The main contaminants of concern identified in the NYSDOH health advisories for the waters of the Upper Bay of New York Harbor are polychlorinated biphenyls (PCBs) and dioxin in fish, and cadmium, dioxin, and PCBs in crab and lobster. The NYSDOH advisories include the following advice:

- For women under 50 years and children under 15 years: Do not eat any fish from these waters, eat no more than a few meals per year of crab meat from these waters, and avoid eating the crab tomalley (hepatopancreas) and cooking liquid.

- For all others: Do not eat gizzard shad, white perch, or crab and lobster tomalley (hepatopancreas) and cooking liquid; eat only one meal per month of Atlantic needlefish, bluefish, rainbow smelt, and striped bass; eat no more than four meals per month of all other fish species and blue crab meat.

Potential current and future receptors at the Gowanus Canal may include recreational users, anglers, local residents, and nearby industrial workers. The recreational receptors may contact surface water and sediment through incidental ingestion and dermal absorption and inhale ambient air (volatile and particulate emissions from the surface water and sediment) at canal level while boating, fishing, and crabbing in the canal and potentially, although less common and less likely, while swimming/diving in the canal.

Swimming/diving in the canal, although it does occur (Gowanus Dredgers Canoe Club, 2010; New York Times, 2007; The Gowanus Lounge, 2007), is rare due to the general conditions of the canal, which are largely associated with CSO discharges to the canal. The anglers may also ingest fish or crabs caught in the canal, and share the fish and crabs with family members, including young children and adolescents. Residents and industrial workers may inhale ambient air (associated with volatile and particulate emissions from the canal) at street level. Also, it has been noted that during significant rainfall events the canal may overflow (about two to three times per year), and these residents and industrial workers may contact surface water and sediment (that has overtapped the canal) through incidental ingestion and dermal contact. If any of the sediment that has overtapped the canal is not washed away with rain, it is usually swept up by the local residents or workers and does not accumulate.

Future use of the area surrounding the canal is most likely to remain the same as current use (industrial and residential), with the potential for construction of new housing, which would result in additional residential populations living close to the canal.

## SECTION 3

# Scope of Human Health Risk Assessment

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The primary objective of the baseline HHRA is to assess the potential current and future health risks from the contamination associated with the Gowanus Canal, in the absence of any remedial action. The risk assessment evaluates potential carcinogenic risks and noncarcinogenic hazards for a reasonable maximum exposure (RME) scenario consistent with the National Contingency Plan (NCP) (USEPA, 1994a) and Risk Assessment Guidance for Superfund (RAGS) guidance documents (USEPA, 1989, 1991, 2001a, 2004, 2009). The RME is the highest exposure that is reasonably expected to occur at a site (USEPA, 1989). The risk assessment comprises the following components:

- **Hazard Assessment/Identification of Chemicals of Potential Concern**—Identification of the contaminants found in the canal media and selection of the chemicals of potential concern (COPCs). COPCs represent that subset of the chemicals found at the canal that is expected to contribute the most to the risk estimates for the canal.
- **Exposure Assessment**—Identification of the potential pathways of human exposure and estimation of the magnitude, frequency, and duration of these exposures.
- **Toxicity Assessment**—Assessment of the potential adverse effects of the COPCs and compilation of the noncarcinogenic and carcinogenic toxicity values used for developing numerical risk estimates.
- **Risk Characterization**—Integration of the results of the hazard, exposure, and toxicity assessments to develop numerical estimates of health risks and characterize the potential health risks associated with potential exposure to site-related contamination.
- **Uncertainty Assessment**—Identification and discussion of sources of uncertainty in the risk assessment.

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## SECTION 4

# Hazard Assessment/Identification of COPCs

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The identification of COPCs includes data collection, data evaluation, and data screening to identify those chemicals which would contribute the most to the total risk estimates associated with the site.

The data collection and evaluation involve gathering and reviewing the available site information and compiling a set of data for the purpose of conducting the baseline HHRA. Once the data collection and evaluation are completed, the established data set is further screened to focus the risk assessment efforts on the contaminants that need to be evaluated quantitatively in the risk assessment (data screening).

## 4.1 Data Summary

The purpose of this investigation is to evaluate the potential human health risks associated with contamination in surface sediment and surface water in the Gowanus Canal, in fish and crab caught in the canal, and in air from emissions from the canal media. Surface water and sediment samples collected from the canal, fish and crab tissue samples collected from the canal, and air samples collected at canal level and street level adjacent to the canal were evaluated in the baseline HHRA. Samples for all media have also been collected from reference areas. All of the data are included in Appendix I to the RI report. The samples evaluated in the baseline HHRA and the laboratory analysis performed for these samples are identified in Table 4-1. All data used in the risk assessment were validated in accordance with USEPA Region 2 Data Validation SOPs (USEPA Region 2, 1992, 2001b).

### 4.1.1 Surface Sediment

Surface sediment sampling activities were conducted within the Gowanus Canal and New York Harbor between June 17 and July 1, 2010. Surface sediment samples were collected over the length of the canal from 0 to 6 inches below the top of soft sediment. Some of the surface sediment samples were collected at locations where sediments are exposed at low-tide. Figures 2-3a through 2-3c in the RI report identify the locations where the surface sediment samples were collected. A total of 27 surface sediment samples were collected from the canal. Twelve of the samples were collected from “exposed” locations. All sediment samples were analyzed for Target Compound List (TCL) organics and Target Analyte List (TAL) metals (including mercury and cyanide), and these data were evaluated in the HHRA. Additionally, 19 sediment samples were analyzed for PCB congeners. These 19 locations were selected to provide data for areas with the greatest potential for human exposure (e.g., the canoe launch), to provide data in areas where high PCB concentrations were previously measured in sediment, and to provide spatial coverage throughout the canal. The PCB concentrations and risks were calculated based on total PCBs derived from congener data. Aroclor data were not used in the risk calculations. Ten surface sediment samples were also collected from the Gowanus Bay and Upper New York Bay, outside the mouth of the canal, to provide information for offsite conditions. Figure 2-4 of the RI identifies the reference surface sediment locations.

Two data groupings of surface sediment were evaluated in the HHRA. All of the surface sediment data were included in the data set to evaluate overflow sediment. Only the surface sediment samples collected from “exposed” locations were included in the data set used to evaluate contact with sediment during recreational use of the canal.

#### **4.1.2 Surface Water**

Surface water samples were collected from the length of Gowanus Canal during two sampling events representing dry-weather (June 19, 2010; 27 samples) and wet-weather (July 13, 2010; 26 samples) conditions. Figures 2-5a through 2-5c in the RI identify the locations where the surface water samples were collected. Ten offsite surface water samples were collected from the New York Harbor and Buttermilk Channel. Figure 2-6 identifies the offsite surface water sample locations. The surface water samples were collected from approximately 6 inches below the water surface. Surface water samples from all locations were analyzed for TCL organics and TAL metals (total and dissolved including mercury and cyanide). The total metals data were evaluated to estimate risks associated with metals in the HHRA, as the receptors would come in direct contact with total (rather than dissolved) concentrations in surface water.

#### **4.1.3 Ambient Air**

Air-sampling activities were conducted along the Gowanus Canal and at background locations during two sampling events. Air samples were collected between July 7 and 9, 2010, prior to the start of the canal oxygenation system. A second round of air sampling was conducted July 28 and 29, 2010, following the start-up of the oxygenation system. Air samples were collected at 10 locations along the length of the canal with two samples collected at each location, one at canal level and one at street level. Air samples were also collected at three background locations, with one sample at street level at each location. Figure 2-9 in the RI shows the locations of the air samples. Air samples along the canal were analyzed for VOCs and PAHs. Location 506 at the street level was also analyzed for PCBs during the first sampling event.

#### **4.1.4 Fish Tissue**

Fish and crab samples were collected in the Gowanus Canal and reference locations in Gowanus Bay and Upper New York Bay from June 21 through July 9, 2010. Fish and shellfish were collected in six reaches of the canal and in three reference areas (Figures 2-7 and 2-8 of the RI). Species targeted for sampling and evaluation in the HHRA included blue crab, striped bass, and white perch. Additional species caught and evaluated in the HHRA included American eel. These species were targeted on the basis of previous catch records for the canal, since they are typically consumed by humans, and because they represent different levels within the food web and have potentially different exposure within the ecosystem. These species were selected as the target fish/crab species for the canal in consultation with USEPA.

Both top-level predator (striped bass) and middle-level predator (white perch) fish tissue samples were collected and evaluated in the HHRA. The top-level predators represent species reflecting high-end bioaccumulation due to their position in the food web and are occasionally harvested and consumed by anglers. However, these fish tend to have a relatively large home range, and thus lower site fidelity, and a greater chance of

accumulating contaminants from offsite sources. The midlevel predator represents species most frequently harvested and consumed by anglers and likely retains greater site fidelity. However, the midlevel predator may not reflect maximum bioaccumulation due to its position in the food web. Additionally, because eel were caught during the sampling, and eel are consumed by humans, the eel were evaluated in the HHRA and represent bottom feeders.

Section 2.5 of the RI details the fish sampling, tissue preparation and grouping, and the preparation of fish samples for laboratory analysis. Twelve composite samples of blue crab, six composite samples of eel, five composite samples of striped bass, and two composite samples of white perch were included for quantitative evaluation in the HHRA.

Edible tissue (filet only) samples were analyzed to assess potential human health risks associated with ingestion of striped bass, white perch, and eel. For blue crab, edible portion samples and hepatopancreas samples were analyzed separately and combined to estimate human health risks. The hepatopancreas is a main component of a crab's digestive system. It functions as both the liver and pancreas for the crab, and is involved in producing digestive enzymes and is responsible for filtering impurities from the crab's blood. Some chemicals, such as PCBs and mercury, accumulate in the hepatopancreas. May and Burger (1996) reported that most crabbers in the Newark Bay Complex eat only cleaned crabs, and discard the hepatopancreas, with fewer than 3 percent of those surveyed eating the whole crab. The New Jersey Department of Environmental Protection (NJDEP) (2002) reported that 15 percent of the population surveyed in the Newark Bay Complex ate the hepatopancreas. It is possible that an individual may be exposed to the hepatopancreas during cooking unless actions are taken to remove the hepatopancreas from the crab prior to cooking. Based on the NJDEP report (NJDEP, 2002) and the potential for the hepatopancreas to be released during cooking, an individual may be exposed to both crab edible tissue and hepatopancreas. Therefore, the risk evaluation considered exposures to both the edible tissue and the hepatopancreas as the RME scenario. Risks from ingestion of the edible tissue only are discussed in the Uncertainty Assessment.

## 4.2 Data Evaluation

All of the data included in the risk assessment were evaluated to assess their reliability for use in the quantitative risk assessment. The following criteria were used to assess data usability:

- Estimated values flagged with a J or NJ qualifier were treated as unqualified detected concentrations.
- Data qualified with an R (rejected) were not used in the risk assessment.
- Data qualified with a B (blank contamination) were used in the risk assessment as if the constituents were not detected.
- For duplicate samples, the maximum concentration between the two samples was used as the sample concentration.

- Only the detected congeners were used to calculate the total PCB concentrations for each sample. Undetected congeners and congeners qualified with a B were not included in the calculations (see Section 5.3.1).
- Nondetected values (for all analytes except PCBs) were included in the risk assessment and exposure concentration calculation at the detection limit using ProUCL (USEPA, 2010a).

## 4.3 Selection of COPCs

The identification of COPCs is shown in Tables 2.1 through 2.13 in the Attachment. The methodology used to select the COPCs for quantitative evaluation in the baseline HHRA is outlined below:

- Sediment data were compared to the USEPA Regional Screening Levels (RSLs) for residential exposure to soil (USEPA, 2010b). RSLs that are based on the chemical's noncarcinogenic effects (noted with an ““N” next to the screening toxicity value on the COPC screening table) were divided by 10 to account for potential exposure to multiple constituents. RSLs that are based on carcinogenic effects (noted with a “C”“ next to the screening toxicity value on the COPC screening table) were used without adjustment because the target carcinogenic risk used in the RSL calculations is based on a  $1 \times 10^{-6}$  risk level. Constituents whose maximum detected concentration is below the screening level were not retained as COPCs. As discussed in Section 4.1.1, sediment samples were analyzed for both Aroclors and PCB congeners. Only the PCB congener data were evaluated in the risk assessment so that the risks associated with PCBs would not be double-counted in the HHRA. In accordance with EPA guidance (USEPA, 1996) for PCB congener analysis, the “dioxin-like” PCB toxicity equivalent (TEQ) concentration, the “non-dioxin-like” total PCB concentration, and the total PCB concentration were calculated for each sample using the methods discussed in Section 5.3.1. The dioxin-like PCB TEQ concentration was screened against the 2,3,7,8-TCDD RSL (which was recalculated from the value on the RSL table using the 2,3,7,8-TCDD oral cancer slope factor from USEPA, 1995), the non-dioxin-like PCB concentration was screened against the Aroclor-1260 RSL, and the total PCB concentration was screened against the Aroclor-1254 RSL.
- Surface water data were compared to the USEPA tap water RSLs (USEPA, 2010b). RSLs that are based on the chemical's noncarcinogenic effects were divided by 10 to account for potential exposure to multiple constituents. RSLs that are based on carcinogenic effects were used without adjustment because the target carcinogenic risk used in the RSL calculations is based on a  $1 \times 10^{-6}$  risk level. Constituents whose maximum detected concentration is below the screening value were not retained as COPCs.
- Fish tissue and crab sample data were compared to the USEPA Region 3 fish ingestion RSLs (USEPA Region 3, 2010c). RSLs that are based on noncarcinogenic effects were divided by 10 to account for exposure to multiple constituents. RSLs that are based on carcinogenic effects were used without adjustment. Constituents whose maximum detected concentrations are below the screening level were not retained as COPCs. In accordance with USEPA guidance (USEPA, 1996) for PCB congener analysis, the “dioxin-like” PCB TEQ concentration, the “non-dioxin-like” total PCB concentration,

and the total PCB concentration were calculated for each sample using the methods discussed in Section 5.3.1. The dioxin-like PCB TEQ concentration was screened against the 2,3,7,8-TCDD RSL (which was re-calculated from the value on the RSL table using the 2,3,7,8-TCDD oral cancer slope factor from USEPA, 1995), the non-dioxin-like PCB concentration was screened against the Aroclor-1260 RSL, and the total PCB concentration was screened against the Aroclor-1254 RSL.

- Air data were compared to the USEPA resident air RSLs (USEPA, 2010b). RSLs that are based on noncarcinogenic effects were divided by 10 to account for exposure to multiple constituents. RSLs that are based on carcinogenic effects were used without adjustment. Constituents whose maximum detected concentrations are below the screening level were not retained as COPCs.
- Essential human nutrients (calcium, magnesium, potassium, and sodium) that are toxic at concentrations significantly higher than those found in canal media, and whose concentrations in canal media are only slightly higher than the concentrations at which they occur naturally, were not retained as COPCs.
- Lead concentrations less than 0.015 mg/L in surface water (the Safe Drinking Water Act action level for lead in potable water) and less than 400 mg/kg in sediment (USEPA, 1994b) are considered adequately protective of human health under residential land-use conditions. Lead was retained as a COPC when exceeding these values. Lead was not detected in fish tissue.
- Constituents that were detected in less than 5 percent of the samples were not retained as COPCs (USEPA, 1989).
- Detected constituents classified as USEPA Class A carcinogens (known human carcinogens) were retained as COPCs for evaluation regardless of the comparison of the concentration to the screening level (USEPA, 1989).
- For constituents detected in canal samples that do not have established USEPA RSLs, the RSL values for surrogate constituents were selected based on guidance from the Superfund Technical Support Center and used in the COPC screening process.

## 4.4 Constituents of Potential Concern

Table 4-2 lists the chemicals that were identified as COPCs for site media:

- Surface sediment (exposed and near-shore) – 11 PAHs [acenaphthene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, indeno(1,2,3-cd)pyrene, naphthalene, and pyrene], one additional SVOC [bis(2-ethylhexyl)phthalate], PCBs, and 10 metals (aluminum, arsenic, cadmium, chromium, cobalt, copper, iron, lead, manganese, and vanadium).
- Surface sediment (overflow scenario) – the chemicals indicated above (exposed and near-shore) plus one additional PAH (fluorene).
- Surface water (dry event) – one VOC (benzene), four PAHs [benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene], one additional

SVOC [bis(2-ethylhexyl)phthalate], and four metals (arsenic, chromium, copper, and selenium).

- Surface water (wet event and overflow)—the chemicals indicated above (dry event) plus four additional VOCs (1,4-dichlorobenzene, chloroform, ethylbenzene, and tetrachloroethene), two additional PAHs [dibenz(a,h)anthracene and naphthalene] , two additional metals (cobalt and lead), and one less metal (copper).
- Ambient air (street level)—benzene, chloroform, ethylbenzene and naphthalene.
- Ambient air (canal level)—the chemicals indicated above (street level) plus xylene.
- Fish tissue (striped bass)—two pesticides (p,p'-DDE and p,p'-DDT), PCBs, and three metals (arsenic, mercury, and selenium).
- Fish tissue (white perch)—PCBs and two metals (mercury and selenium)
- Fish tissue (eel)—six pesticides (alpha-chlordane, gamma-chlordane, dieldrin, p,p'-DDD, p,p'-DDE, and p,p'-DDT), PCBs, and six metals (arsenic, chromium, copper, cyanide, mercury, and selenium).
- Blue crab—five PAHs (benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene), PCBs, and three metals (arsenic, copper, and mercury).

## SECTION 5

# Exposure Assessment

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Exposure assessment is the estimation of the likelihood, magnitude, frequency, duration, and routes of potential exposure to a chemical. Exposure refers to the potential contact of an individual (or receptor) with a chemical. Exposure can occur when contaminants migrate from a source to an exposure point, or when a receptor comes into direct contact with contaminated media.

The components of exposure assessment include the following:

- Characterization of exposure setting
- Identification of exposure pathways, and evaluation of whether they are complete
- Quantification of exposure

## 5.1 Characterization of Exposure Setting

Characterization of exposure setting consists of two parts: (1) characterization of the site with respect to its physical characteristics and (2) characterization of the site with respect to human populations at or near the site.

### 5.1.1 Physical Setting

The physical setting is described in the Section 2, the CSM. The Gowanus Canal is a 1.8-mile-long canal located in the New York City borough of Brooklyn, Kings County, New York. The canal borders several residential neighborhoods including Red Hook, Cobble Hill, Carroll Gardens, and Park Slope. The waterfront properties abutting the canal are primarily commercial and industrial.

The Gowanus Canal is a tidally influenced dead-end channel that opens to Gowanus Bay and Upper New York Bay. The canal experiences a semidiurnal tidal cycle (i.e., two high tides and two low tides each tidal day). The entire canal is classified as a saline tributary to Upper New York Bay, and the reach between the head of the canal and 22nd Street is classified as a “minor river, tidal tributary,” according to Title 6 of the New York Code of Rules and Regulations, Chapter X, Part 890. The only freshwater inflows to the canal are wet-weather CSO and stormwater discharges. Because of its narrow width, limited freshwater input, and enclosed upper end, the canal has low current speeds and limited tidal exchange with Gowanus Bay, though these are enhanced by additional circulation from the flushing tunnel when it is operating (NYCDEP, 2008).

### 5.1.2 Potentially Exposed Populations

As discussed above, the area around the Gowanus Canal consists of industrial, commercial, and residential properties. The Gowanus Canal itself may be used for recreational activities such as boating, swimming/diving, fishing, and crabbing (Gowanus Dredgers Canoe Club, 2010; New York Times, 2007; The Gowanus Lounge, 2007). Despite current fish advisories for the Upper New York Bay, which includes the Gowanus Canal, there have been observations of fishing/crabbing along the canal (The Gowanus Lounge, 2007). Although

most properties abutting the canal are commercial and industrial, there are residential neighborhoods within a few blocks of the canal. Therefore, potential current receptors for the surface water, sediment, air, and/or fish and crabs include recreationalists, anglers (and their families), local residents, and industrial workers. Table 1 in the Attachment identifies the potential receptors.

Future use of the canal and use of the surrounding area will likely remain the same as current use. Additionally, there have been discussions concerning construction of residential housing complexes adjacent to the canal. Therefore, the future potentially exposed populations are assumed to be the same as the current potentially exposed populations.

## **5.2 Identification of Exposure Pathways**

An exposure pathway may be described as the physical course that a COPC takes from the point of release to a receptor. To be complete, an exposure pathway must have all of the following components present:

- A source
- A mechanism of a chemical release and transport
- An environmental transport medium
- An exposure point (receptor location)
- An exposure route (inhalation, dermal absorption, ingestion)
- A receptor or exposed population

In the absence of any one of these components, an exposure pathway is considered incomplete and, by definition, there is no risk or hazard. In some cases, a receptor may contact a source directly, eliminating the release and transport pathway.

The potential exposure pathways for Gowanus Canal are identified in the preliminary CSM (Figure 2-1) and Table 1 in the Attachment.

### **5.2.1 Contaminant Sources**

The potential contaminant sources are identified in the preliminary CSM, in Section 2, and include past use of the areas abutting the canal by gas works, coal yards, manufacturers of cement, manufacturers of soap, tanneries, paint and ink factories, machine shops, chemical plants, and oil refineries. The canal was also used as a repository of untreated industrial wastes, raw sewage, and surface water runoff for many decades.

### **5.2.2 Release and Transport Mechanisms**

The primary contaminant release and transport mechanism at the Gowanus Canal site appears to be the discharge from past industrial activities into the canal surface water, accumulation in the sediment, and bioaccumulation into fish and crabs. The transport pathway from emissions from surface water and sediment to ambient air will be evaluated in the risk assessment using the ambient air data collected along the canal. Additionally, the canal has been known to overflow during heavy precipitation events, resulting in transport of surface water and sediment out of the canal onto properties and streets adjacent to the canal. The surface water and sediment remains on the properties and streets adjacent to the canal only for short periods of time during the overflow and flooding events.

### **5.2.3 Exposure Points and Exposure Routes**

Potential exposure points include the surface water and sediment within the canal, surface water and sediment temporarily deposited on properties adjacent to the canal when the canal overflows its banks during heavy precipitation events, ambient air at canal level and street level adjacent to the canal, and fish and crab caught in the canal. Table 1 in the Attachment lists all of the potential exposure pathways that were considered for evaluation in the risk assessment.

### **5.2.4 Summary of Potential Exposure Pathways**

In summary, the potential pathways that were evaluated under the current/future land use scenario include the following:

- Adult, adolescent (12–18 years old), and child (1–6 years old) recreational: incidental ingestion and dermal contact with canal surface water (during both wet- and dry-sampling events); incidental ingestion and dermal contact with exposed and near-shore sediment in the canal; inhalation of ambient air at canal level (both before and during aeration system operation).
- Adult and adolescent (12–18 years old) angler and child (1–6 years old) of angler: ingestion of fish (striped bass, white perch, and eel) and crab caught in the canal.
- Adult and child (1–6 years) residents: inhalation of ambient air at street level (both before and during aeration system operation); incidental ingestion and dermal contact with canal overflow surface water (using surface water collected during wet events); incidental ingestion and dermal contact with sediment deposited adjacent to the canal (using surface sediment data) during canal overflow events.
- Adult industrial worker: inhalation of ambient air at street level (both before and during aeration system operation); incidental ingestion and dermal contact with canal overflow surface water (using surface water collected during wet events); incidental ingestion and dermal contact with sediment deposited adjacent to the canal (using surface sediment data) during canal overflow events.

## **5.3 Quantification of Exposure**

Quantification of exposure involves estimating the exposure point concentration (EPC) and chemical intake.

### **5.3.1 Exposure Concentrations**

The EPC is the estimated concentration at the point of contact. EPCs may be measured directly (i.e., surface water concentration) or calculated using fate and transport models. No fate and transport modeling was conducted for the Gowanus Canal risk assessment since data were collected at potential exposure points.

The EPCs for each COPC were calculated as the 95 percent upper confidence limit (UCL) of the mean concentration. The maximum detected concentration was used in place of the UCL as the EPC when the calculated UCL was greater than the maximum detected value or less than eight samples were available for the data grouping.

ProUCL, Version 4.00.05 (USEPA, 2010a), was used to calculate the UCLs and determine the data distribution. The data distribution identified was used to compute the most appropriate UCL for that specific data set. The recommendations in the ProUCL output file and outlined in the ProUCL model documentation were used to select the appropriate UCL.

In accordance with USEPA guidance (USEPA, 1996), EPCs were calculated separately for each medium (sediment and fish tissue) and data groupings for which PCB congeners were analyzed for (1) “dioxin-like” PCB TEQ concentrations, (2) “non-dioxin-like” total PCB concentrations, and (3) total PCB concentrations. These three concentrations were calculated for each sample using the methods discussed below. The concentrations were calculated using only the detected PCB congeners in each sample. Nondetected congeners and B-flagged congeners were not included in the individual sample sums. The calculations are included in Tables 2.8 Supplement A, 2.11 Supplement A, and 2.12 Supplement A in the Attachment for fish and crab, and for sediment, respectively. Detection limits are not shown on these tables for all of the dioxin congeners. The reporting limits are unreliable because during data validation the reporting limits were not provided correctly if the sample was reanalyzed or diluted. Appendix H presents the data quality evaluation. The uncertainties associated with the EPC calculation approach for PCBs are discussed in Section 8.

TEQ concentrations for “dioxin-like” PCBs were calculated for each sample in accordance with the World Health Organization (WHO) toxicity equivalency factor (TEF) approach (USEPA, 2010d). The purpose of using the WHO TEF approach is to adjust the relative carcinogenic potency of specific dioxin-like PCB congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), the most potent dioxin congener. TEFs are published by WHO for mammals (including humans), fish, and wildlife. PCB TEQs were calculated using the mammal TEFs. Using the measured concentration values for each congener and the TEF for that congener, the TEQ for a mixture of PCB congeners in a specific sample is calculated as follows:

$$TEQ = \sum (TEFi \times Ci)$$

where:

$TEFi$  = Toxicity equivalency factor for congener  $i$

$Ci$  = Concentration of congener  $i$

Risks from the calculated dioxin-like PCB TEQ are therefore based on the toxicity factors for 2,3,7,8-TCDD. The TEFs used to calculate the dioxin-like PCB TEQ concentrations are provided in Table 5-1. Although there are also noncancer toxicity factors for 2,3,7,8-TCDD, there is greater uncertainty associated with evaluating the noncancer risk for the dioxin-like PCB TEQ concentrations using these values. Therefore, the evaluation of noncancer hazards associated with the dioxin-like PCB TEQ concentrations is presented in the uncertainty evaluation (Section 8).

The non-dioxin-like PCB concentrations (the sum of all detected non-dioxin-like PCB congeners) were also calculated and evaluated for carcinogenic risks using carcinogenic toxicity factors for total PCBs. The total PCB concentrations (the sum of all detected PCB congeners, not adjusted using TEFs) were also calculated for each sample and were used to evaluate noncarcinogenic effects.

Most arsenic present in fish and crab tissue is present in the organic form of arsenic (organoarsenical compounds), which is relatively nontoxic to humans and is excreted rapidly and unchanged in urine (U.S. Food and Drug Administration [FDA], 1993; ATSDR, 2007). Some regulatory agencies (for example, FDA) regulate arsenic in fish based on a default but conservative assumption that 10 percent of the total arsenic in fish tissue is potentially in the toxic (carcinogenic) form. Therefore, for this HHRA, it was assumed that 10 percent of the measured total arsenic in fish and crab tissue was in the inorganic form of arsenic. To account for this adjustment, the EPCs for arsenic used in the intake calculations are 10 percent of the total measured arsenic concentrations. The total measured arsenic concentrations are shown in Tables 3.8 through 3.11 in the Attachment, while 10 percent of the total measured values are shown in the Tables 7.8.RME through 7.10.RME and 7.7.CTE through 7.9.CTE in the Attachment.

### **5.3.2 Estimation of Chemical Intakes**

The quantification of exposure is based on an estimate of the chronic daily intake (CDI), the average amount of the chemical contaminant entering the receptor's body per day.

Chemical intake estimates for the ingestion and dermal exposure pathways are generally expressed as follows:

$$CDI = \frac{C \times CR \times EF \times ED}{BW \times AT}$$

Where:

- CDI = chronic daily intake (mg/kg-day)
- C = chemical concentration (mg/L, mg/kg)
- CR = contact rate (L/day, mg/day)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- BW = body weight (kg)
- AT = averaging time (days)

For the dermal pathway, the contact rate usually incorporates the skin surface area in contact with the exposure medium (water or sediment), and an absorption factor. The intake equation for the dermal exposure pathway is shown in the Attachment, Tables 4.1 (RME and CTE) and 4.5 (RME and CTE) for sediment and Tables 4.2 (RME and CTE) and 4.6 (RME and CTE) for surface water.

Chemical exposure estimates for the inhalation pathway are generally expressed as follows:

$$EC = \frac{Ca \times ET \times EF \times ED \times CF}{AT}$$

Where:

- EC = exposure concentration ( $\text{mg}/\text{m}^3$ )
- Ca = chemical concentration in air ( $\text{mg}/\text{m}^3$ )
- ET = exposure time (hours/day)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- CF = conversion factor (day/24 hours)
- AT = averaging time (days)

The intake and exposure equations require exposure parameters that are specific to each exposure pathway. Many of the exposure parameters have default values, which were used for this assessment. These assumptions, based on estimates of body weights, media intake levels, and exposure frequencies and duration, are provided in USEPA guidance. Other assumptions (e.g., for the recreational scenarios) require consideration of location-specific information and were determined using professional judgment. Tables 4.1.RME through 4.6.RME in the Attachment present the exposure parameters that were used for the exposure scenarios that were evaluated in the risk assessment. RME scenario exposure parameters were compiled for all scenarios; central tendency exposure (CTE) parameters were compiled only for scenarios where the RME risk for an environmental medium is greater than 'USEPA's noncarcinogenic hazard or carcinogenic risk target levels (target organ-specific hazard index (HI) >1.0, and excess lifetime cancer risk (ELCR) >1 × 10<sup>-4</sup>). The exposure parameters selected for each receptor are discussed below.

For the fish consumption evaluation, the fish ingestion rate for each of the species of fish evaluated was adjusted to a percentage of the total fish ingestion rate assumed, as reported in literature for the State of New York. The percentage of ingestion of each fish type was obtained from the 1991 New York State Angler Report (Connelly et al., 1992). It was assumed that eel represent bottom feeders with 44 percent of the total fish consumption, striped bass represent the intermediate level with 47 percent consumption, and white perch represent the remaining 9 percent. A 100 percent crab ingestion rate was assumed for blue crab. Burger (2002) found that individuals typically consumed either fish or crabs, and that those who did consume both fish and crabs had lower consumptions of each. Therefore, the total noncarcinogenic hazards and carcinogenic risks were not combined for consumption of both fish and crabs.

For the fish and crab ingestion scenarios, an additional exposure parameter was used in the CDI calculation (USEPA, 2000) for the CTE scenario. During cooking and fish preparation, concentrations of some lipophilic contaminants decrease. Therefore, if appropriate for the COPC, a preparation/cooking loss factor (PCF) was applied to the equation for the CTE assessment.

Appendix C of USEPA's *Guidance for Assessment Chemical Contaminant Data for Use in Fish Advisories, Volume 2: Risk Assessment and Consumption Limits* (USEPA, 2000) summarizes many of the studies that have evaluated loss of PCBs and pesticides from fish during food preparation and cooking. A review of the available literature found the results range considerably, both between various cooking methods and within the same method. Cooking losses, expressed as percent loss based on total PCB mass before and after cooking, as high as 74 percent were reported in one study (Skea et al., 1979). Other studies reported net gains of PCBs (Moya et al., 1998; Armbruster et al., 1987). PCB losses from cooking may be a function of the cooking method (baking, frying, broiling, etc.), cooking duration, temperature during cooking, preparation techniques (i.e., trimmed vs. untrimmed, with or without skin), lipid content of the fish, the fish species, magnitude of the PCB contamination in the raw fish, the extent to which lipids separated during cooking are consumed, reporting method, and/or the experimental study design. Personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings) may result in consumption of PCBs "lost" from the fish upon cooking.

A review of literature found that PCB cooking losses may range from 0 to 74 percent. Despite the wide range of cooking loss estimates, most PCB losses were between 10 and 40 percent. A value of 20 percent (midpoint of 0 – 40 percent) was selected as the CTE estimate for cooking loss. For the RME, a cooking loss factor was not used to include the possibility that pan drippings are consumed along with the fish. Table 5-2 identifies the PCFs used for the CTE evaluation.

### **Recreational Adult, Adolescent, and Child**

The recreational adult, adolescent (ages 12–18), and child (ages 1–6) are individuals who may use the canal for boating, fishing, crabbing, or—although unlikely—swimming/diving and be exposed to the surface water, sediment, and air at canal level. It is assumed that the RME recreational user would engage in recreational activities in the canal for 1 day per week for one-half of the year, from May through October (26 days/year). For estimating the exposure to the surface water and sediment during recreational activities, swimming was assumed. It was assumed that the RME recreational user would be exposed to surface water and sediment for 2.6 hours during each exposure, the national average time spent swimming per swimming event (USEPA, 1989). It was assumed that the RME ingestion rate of sediment during swimming would be 50 percent of the resident ingestion rate of soil (50 mg/day for adults and adolescents and 100 mg/day for children [USEPA, 1991]). The RME ingestion rate of surface water was assumed to equal 0.05 L/day, the surface water ingestion rate for swimming activities (USEPA, 1989). For dermal exposure to sediment, it was assumed that the head, hands, forearms, lower legs, and feet could come into contact with the sediment (i.e., skin surface areas of 6,925 cm<sup>2</sup> [adult], 10,470 cm<sup>2</sup> [adolescent; includes full arms and legs since information was not available for this age group for forearms and lower legs alone], and 2,800 cm<sup>2</sup> [child] [USEPA, 2004]). A sediment-to-skin adherence factor of 0.3 cm<sup>2</sup>, the average adherence factor for reed gatherers (USEPA, 2004), was selected for the adult, and a sediment-to-skin adherence factor of 0.2 cm<sup>2</sup>, the average adherence factor for children in wet soil (USEPA, 2004), was selected for the adolescent and child. For surface water, it was assumed the full body (18,000 cm<sup>2</sup> [adult], 15,758 cm<sup>2</sup> [adolescent], and 6,600 cm<sup>2</sup> [child] [USEPA, 2004]) would be exposed. The default exposure durations of 24 years (adult), 6 years (adolescent ages 12–18), and 6 years (child ages 1–6) and default body weights of 70 kg (adult), 57 kg (mean body weight of girls and boys ages 12–18), and 15 kg (child ages 1–6) were used for exposure to all media.

For the CTE scenarios, the exposure frequency was assumed to be one-half of the RME exposure frequency (or 1 day every other week for one-half of the year). It was assumed that the CTE recreational user would be exposed to the surface water and sediment for one-half of the RME exposure time, or 1.3 hours during each exposure. It was also assumed that the ingestion rates of sediment and surface water would be half the RME values, or 25 mg/day for adults and adolescents and 50 mg/day for children for sediment, and 0.025 L/day for all recreational receptors for surface water. The CTE skin surface areas for exposure to surface water and sediment, and the sediment-to-skin adherence factors were assumed to be the same as the RME values. The total CTE exposure duration for the recreational receptor was assumed to be 9 years (USEPA, 1997), the 50<sup>th</sup> percentile value for living at one residence, therefore, for each of the receptors (adult, adolescent, and child) the exposure duration was assumed to be 3 years. The default body weights of 70 kg (adult), 57 kg (mean body weight

of girls and boys from 12–18), and 15 kg (child ages 1–6) were used to evaluate CTE exposure to all media.

### **Adult Angler**

It was assumed the adult angler would ingest the fish or crabs they catch in the canal. There is not a lot of published literature concerning ingestion rates of self-caught crabs. Burger (2002) reported results of an angler study for fish and crabs in the Newark Bay Complex, and has estimated a yearly consumption rate for self-caught crab by multiplying the number of crab meals eaten per month by the number of crabs eaten at each meal by the number of months per year crabs are caught. Burger assumed the average size of one crab is 70 g. In the Burger (2002) study, it was noted that most of the people interviewed mainly fished or mainly crabbed, and that more than 30 percent of the people who fished and crabbed in the Newark Bay Complex did not eat their catch. People reported crabbing for only 3 months of the year, which is why data from only the 3-month period were used to calculate the annual ingestion rate. This may underestimate the risks and hazards associated with crab ingestion if crabs are actually caught in the canal and eaten for longer than 3 months a year. The yearly consumption rate of self-caught crab estimated from Burger (2002) is 5,760 grams (g), or 16 g/day. A 95% UCL of the yearly consumption value was estimated on the basis of this to be 23 g/day, and an average yearly consumption value was estimated to be 16 g/day. The 95% UCL value was used for the RME scenario for crab ingestion, and the average value was used for the CTE scenario.

Burger (2002) also presented a yearly consumption rate of self-caught fish (based on the Newark Bay Complex study) of 8,210 g, or 22 g/day. This is similar to the value of 26 g/day, the 95th percentile recreational freshwater angler's fish ingestion rate, in the *Exposure Factors Handbook* (USEPA, 1997) and is also similar to the 1991 New York State Angler Survey derived rate of 31.9 g/day (Connelly et al., 1992). The fish ingestion rate of 26 g/day (USEPA, 1997) was used for an RME adult. The mean recreational freshwater angler's fish ingestion rate of 8 g/day (USEPA, 1997) was used for the CTE adult angler.

### **Adolescent Angler**

It was assumed that the adolescent angler, ages 12–18 years, would fish or crab in the canal and ingest the fish and crabs caught in the canal. In addition to eating the fish or crab caught by an adult family member, several studies have found that children begin fishing at approximately 10 years of age (Connelly et al., 1992). There is not a lot of published literature concerning ingestion rates of self-caught crabs by adults, and even less for children. Therefore, the fish ingestion rate for the adolescent was estimated assuming that the intake for an adolescent is about two-thirds of that of an adult. This approach yields ingestion rates generally consistent with the limited information provided in USEPA's *Exposure Factors Handbook* (USEPA, 1997). For adolescents ages 10–19 years, the 95th percentile intake is 26.8 g/day (USEPA, 1997), which is comparable with the 17 g/day used in the HHRA for adolescents ages 12–18 years. The RME ingestion rate of crabs by adolescents was assumed to be 15 g/day (two-thirds the adult rate).. Similarly, the CTE ingestion rate of fish by adolescents was assumed to be 5.3 g/day, and the CTE ingestion rate of crabs by adolescents was assumed to be 10.7 g/day.

### **Child of Angler**

It was assumed that the child (ages 1–6 years) of an angler would ingest the fish and crabs caught by a parent/relative in the canal. There is not a lot of published literature concerning ingestion rates of self-caught crabs by adults, and even less for children. However, based on information presented in USEPA's *Exposure Factors Handbook* (USEPA, 1997) for fish ingestion rates for children, children ingest about one-third the amount of fish that adults ingest. This approach yields ingestion rates generally consistent with the limited information provided in USEPA *Exposure Factors Handbook* (USEPA, 1997). According to Table 10-1 of the *Exposure Factors Handbook* (USEPA, 1997), the 95th percentile intake for children ages 0–9 years is 16.5 g/day, compared to the RME value used in the HHRA of 9 g/day for children ages 1–6 years. The RME ingestion rate of crabs by children was assumed to be 8 g/day. Similarly, the CTE ingestion rate of fish by children was assumed to be 2.7 g/day and the CTE ingestion rate of crabs by children was assumed to be 5.3 g/day.

### **Industrial Adult**

The industrial adult is an individual who works near the canal and may be exposed to ambient air (collected at street-level stations) 250 days/year, 8 hours/day. This industrial adult may also be exposed to canal overflow surface water and sediment. It was conservatively assumed that the canal overflows three times per year and remains in overflow conditions 3 days during each overflow event, for a total of 9 days per year of exposure to the overflowed surface water and surface sediment. It is assumed that the adult industrial worker could be exposed to the overflow water and sediment 8 hours/day during these 9 days. It was assumed that the ingestion rate of the canal overflow sediment would be equal to the adult industrial worker ingestion rate of soil, or 100 mg/day (USEPA, 2002). The ingestion rate of surface water was conservatively assumed to equal 0.05 L/day, the ingestion rate of surface water for swimming activities (USEPA, 1989). For dermal exposure to sediment, it was assumed that the head, hands, and forearms could come into contact with the sediment, for a surface area of 3,300 cm<sup>2</sup> (USEPA, 2004). For dermal exposure to surface water, it was assumed the head, hands, forearms, and lower legs could come into contact with the surface water, for a surface area of 5,700 cm<sup>2</sup> (USEPA, 2004). A sediment-to-skin adherence factor of 0.2 cm<sup>2</sup>, the soil adherence factor for industrial workers (Exhibit 3-5 in USEPA, 2004), was selected. The default exposure duration of 25 years for an adult industrial worker and default body weight of 70 kg were used for exposure to media.

### **Residential Adult and Child**

The residential adult and child are individuals who live near the canal and may be exposed to ambient air (collected at street-level stations) 350 days/year, 24 hours/day (RME scenario). This resident may also be exposed to canal overflow surface water and sediment. It was conservatively assumed that the canal overflows three times per year and remains in overflow conditions 3 days during each overflow event, for a total of 9 days per year of exposure to the overflowed surface water and surface sediment. It was assumed the RME ingestion rate of the canal overflow sediment would be equal to the resident ingestion rates of soil (100 mg/day [adult] and 200 mg/day [child] [USEPA, 2002]). The RME ingestion rate of surface water was assumed to equal 0.05 L/day, the surface water ingestion rate for exposure scenarios involving swimming activities (USEPA, 1989), and the RME exposure time for exposure to the surface water was assumed to equal 2.6 hours during each exposure, the national average time spent swimming per swimming event (USEPA, 1989).

For dermal exposure to sediment and the surface water, it was assumed that the head, hands, forearms, and lower legs could come into contact with the sediment, for a surface area of 5,700 cm<sup>2</sup> (adult) and 2,800 cm<sup>2</sup> (child, including feet) (USEPA, 2004). A sediment-to-skin adherence factor of 0.2 cm<sup>2</sup>, the average adherence factor for children in wet soil (USEPA, 2004), was selected. Default exposure durations of 24 years (adult) and 6 years (child) and default body weights of 70 kg (adult) and 15 kg (child) were used for exposure to all media.

For the CTE scenario, it was assumed the residential receptors would be exposed to ambient air 234 days/year (USEPA, 1993). It was assumed that the CTE resident would be exposed to the surface water for one-half of the RME exposure time, or 1.3 hours during each exposure. It was also assumed that the ingestion rates of sediment and surface water would be one-half the RME value, or 50 mg/day for adults 100 mg/day for children for sediment, and 0.025 L/day for both adults and children for surface water. The CTE skin surface areas for exposure to surface water and sediment and the sediment-to-skin adherence factors were assumed to be the same as the RME values. The CTE exposure duration for the residential receptor was assumed to be 9 years (USEPA, 1997), the 50<sup>th</sup> percentile value for living at one residence; therefore, the CTE exposure duration for the adult was assumed to be 3 years and the CTE exposure duration for the child was assumed to be 6 years, for a total residential exposure duration of 9 years. The default body weights of 70 kg (adult), and 15 kg (child ages 1–6) were used to evaluate CTE exposure to all media.

## SECTION 6

# Toxicity Assessment

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Toxicity assessment defines the relationship between the magnitude of exposure and possible severity of adverse effects, and it weighs the quality of available toxicological evidence. Toxicity assessment generally consists of two steps: hazard identification and dose-response assessment. Hazard identification is the process of determining the potential adverse effects from exposure to the chemical and the type of health effect involved. Dose-response assessment is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the constituent administered or received and the incidence of adverse health effects in the exposed population. Toxicity criteria (e.g., reference doses [RfDs], inhalation reference concentrations [RfCs], cancer slope factors [CSFs], and inhalation unit risk factors [IURs]) are derived from the dose-response relationship.

USEPA recommends that a tiered approach be used to obtain the toxicity values (RfDs, RfCs, CSFs, and IURs) that are used to estimate noncarcinogenic and carcinogenic risks (USEPA, 2003a). The hierarchy of toxicity value sources is the following:

1. Integrated Risk Information System (IRIS) (USEPA, 2010e)
2. Provisional Peer-Reviewed Toxicity Values (PPRTV)
3. Other USEPA and non-USEPA sources, including the National Center for Environmental Assessment (NCEA), Agency for Toxic Substances and Disease Registry (ATSDR), Health Effects Assessment Summary Tables (HEAST), California EPA (Cal EPA), and USEPA's Office of Water

The use in an HHRA of toxicity values from sources other than IRIS increases the uncertainty of the quantitative risk estimates. Some of the COPCs elicit both systemic (noncarcinogenic) toxic effects and cancer (carcinogenic) effects. Because of this, these constituents are evaluated as both noncarcinogens and carcinogens. The health risks for carcinogenic and noncarcinogenic effects were estimated separately based on different toxicity values.

Chromium is a COPC for sediment, surface water, and fish tissue (eel). It was assumed that all of the chromium detected in these media is hexavalent chromium, or Cr(VI), both for determining if chromium was a COPC (comparing the total chromium concentrations to the Cr(VI) RSLs) and for calculating the risks associated with exposure to chromium in these media. Cr(VI) is unstable in the body (in biological tissues) and is ultimately reduced to trivalent chromium, or Cr(III), by a variety of reducing agents, including ascorbate and glutathione (ATSDR, 2008; USEPA, 2010e). Cr(VI) exerts toxicity through direct contact mechanisms rather than bioaccumulation to a critical concentration in tissue. Therefore, all of the chromium detected in fish tissue samples is most likely Cr(III). However, the Cr(VI) toxicity values were used to evaluate the risks associated with chromium in fish tissue and all media. An RfD and RfC for Cr(VI) are available in IRIS; however, IRIS does not include a

CSF or IUR for CR(VI). The CSF for CR(VI) used in the HHRA is the same one included on the RSL Table (USEPA, 2010b) from NJDEP.

## 6.1 Toxicity Information for Noncarcinogenic Effects

Noncarcinogenic health effects include a variety of toxic effects on body systems, ranging from toxicity to the kidneys to central nervous system disorders. The toxicity of a chemical is assessed through a review of toxic effects noted in short-term (acute) animal studies, long-term (chronic) animal studies, and epidemiological investigations.

USEPA (1989) defines the chronic RfD as a dose that is likely to be without appreciable risk of deleterious effects during a lifetime of exposure. Chronic RfDs are specifically developed to be protective for long-term exposure to a chemical or compound (for example, 7 years to a lifetime), and consider uncertainty in the toxicological database and sensitive receptors. Chronic RfDs were used to evaluate noncarcinogenic risks to all receptors evaluated in the risk assessment.

In the development of RfDs, all available studies examining the toxicity of a chemical following exposure are considered on the basis of scientific merit. The lowest dose level at which an observed toxic effect occurs is identified as the lowest observed adverse effect level (LOAEL), and the dose at which no effect is observed is identified as the no observed adverse effect level (NOAEL). Several uncertainty factors (UFs) may be applied to account for uncertainties such as limited data, extrapolation of data from animal studies to human exposures, or the use of subchronic studies to develop chronic criteria. These UFs range from 10 to 10,000, and are based on professional judgment. Consequently, there are varying degrees of uncertainty in the toxicity criteria, which range from 1 to 3,000 for the COPCs identified for this site.

USEPA-derived oral RfDs and inhalation RfCs, and associated UF and modifying factor (MF) values, available for the COPCs are presented in Tables 5.1 and 5.2 in the Attachment. The RfD for Aroclor 1254 was used to estimate the noncarcinogenic effects associated with exposure to total PCBs.

Per USEPA guidance, oral RfDs were adjusted from administered dose (oral) to absorbed dose (dermal) to evaluate dermal toxicity. When appropriate, the RfDs were adjusted using oral absorption factors (USEPA, 2004). This adjustment is shown in Table 5.1 in the Attachment.

## 6.2 Toxicity Information for Carcinogenic Effects

Potential carcinogenic effects are quantified as CSFs or IURs that convert estimated exposures directly to incremental lifetime carcinogenic risks.

CSFs and IURs may be derived from the results of chronic animal bioassays, human epidemiological studies, or both. Animal bioassays are usually conducted at dose levels that are much higher than are likely to be encountered in the environment. This design detects possible adverse effects in the relatively small test populations used in the studies. The actual risks from exposure to a potential carcinogen are not likely to exceed the estimated risks and are probably much lower or even zero. USEPA-derived CSFs and IURs are presented in the Attachment, Tables 6.1 and 6.2. The CSF for 2,3,7,8-TCDD (dioxin) was

used to estimate the carcinogenic risks associated with the dioxin-like PCBs and the CSF for total PCBs was used to estimate the carcinogenic risks associated with the non-dioxin-like PCBs. As was done for oral RfDs, oral CSFs were adjusted from administered dose (oral) to absorbed dose (dermal) to evaluate dermal toxicity. When appropriate, the CSFs were adjusted using oral absorption factors (USEPA, 2004). This adjustment is shown in Table 6.1 in the Attachment. The IRIS toxicity profile for PCBs (USEPA, 2010e) presents a tiered approach for evaluating carcinogenic risks associated with exposure to PCBs. IRIS recommends that the first tier upper-bound CSF of 2.0 (mg/kg-day)<sup>-1</sup> be used to evaluate upper-bound risks estimates for exposures to PCBs through ingestion of fish and sediment, and dermal contact with sediment, and that the central-estimate of 1.0 (mg/kg/day)<sup>-1</sup> be used to evaluate cancer risks for the CTE estimate.

### **6.3 Approach for Potential Mutagenic Effects**

For COPCs that act via a mutagenic mode of action (MMOA), cancer risks were estimated using age-dependent adjustment factors (ADAFs), as is consistent with cancer guidelines and supplemental guidance (USEPA, 2005a, b). Consistent with the Superfund guidance on MMOA (<http://www.epa.gov/oswer/riskassessment/sghandbook/chemicals.htm>), the carcinogenic PAHs are considered to act via a MMOA. Additionally, chromium is also categorized as a chemical with a MMOA (McCarrol, et al., 2010). The calculation of cancer risk using ADAFs is presented in Tables 7s of the Attachment. As chemical-specific data are not available for the carcinogenic PAHs or chromium, default ADAFs, as included in the USEPA Region 3 memorandum *Derivation of RBCs for Carcinogens That Act via a Mutagenic Mode of Action and Incorporate Default ADAFs* (USEPA, 2006), were used for the MMOA evaluation. The default ADAFs used to adjust the CSFs are 10 for 0-2 year olds, 3 for 2-6 year olds, 3 used for 6-12 year olds, and 1 for 16-30 year olds. The CSF was multiplied by the appropriate ADAF to derive the age-specific CSF for a receptor to calculate the total carcinogenic risk. Additionally, the exposure factors for children 0-2 years old and 2-6 years old were assumed to be the same as the parameters for a child 0-6 years old, with the exception of the exposure duration, which was instead 2 years and 4 years, respectively. The exposure factors for the adult residential receptor were used for residents 6-16 years old and 16-30 years old, with the exception of the exposure durations, which were 10 years and 14 years, respectively. For the adolescent receptors, the exposure factors for the adolescents 12-16 years old and 16-18 years old were assumed to be the same as the parameters for an adolescent 12-18 years old, with the exception of the exposure duration, which was 4 years and 2 years, respectively.

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## SECTION 7

# Risk Characterization

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Risk characterization combines the results of the previous elements of the risk assessment to evaluate the potential health risks associated with exposure to the COPCs. The risk characterization is then used as an integral component in risk management decision-making.

## 7.1 Methods for Estimating Risks

Potential human health risks are discussed independently for carcinogenic and noncarcinogenic effects because of the different toxicological endpoints, relevant exposure duration, and methods used to characterize risk. The methodology used to estimate noncarcinogenic hazards and carcinogenic risks are described below.

### 7.1.1 Noncarcinogenic Hazard Estimation

Noncarcinogenic health risks are estimated by comparing the calculated exposures to RfDs (or RfCs). The calculated intake divided by the RfD (or exposure concentration divided by the RfC) is equal to the hazard quotient (HQ):

$$HQ = \text{Intake} / \text{RfD} \text{ or } \text{Exposure Concentration} / \text{RfC}$$

The intake and RfD (or exposure concentration and RfC) represent the same exposure route (i.e., oral intakes are divided by oral RfDs, inhalation exposure concentrations are divided by inhalation RfCs). An HQ that exceeds 1.0 (i.e., intake exceeds the RfD) indicates that there is a potential for adverse health effects associated with exposure to that constituent.

To assess the potential for noncarcinogenic health effects posed by exposure to multiple constituents, an HI approach is used (USEPA, 1986). This approach assumes that noncarcinogenic hazards associated with exposure to more than one constituent are additive (HI = sum of the HQs). Synergistic or antagonistic interactions between constituents are not considered. The HI may exceed 1.0 even if all of the individual HQs are less than 1. HIs may be added across exposure routes to estimate the total noncarcinogenic health effects to a receptor posed by exposure through multiple routes. If the HI is greater than 1, separate HIs are estimated for each target organ to assess whether the HI for a specific target organ is greater than 1. A target-organ-specific HI greater than 1 indicates that there is some potential for adverse noncarcinogenic health effects associated with exposure to the COPCs, possibly warranting remedial action. If the HI for each target organ does not exceed 1, noncarcinogenic hazards are not expected.

### 7.1.2 Carcinogenic Risk Estimation

The potential for carcinogenic effects due to exposure to site-related constituents is evaluated by estimating the ELCR, which is the incremental increase in the probability of developing cancer during one's lifetime in addition to the probability of getting cancer associated with exposure to all non-site-related sources of carcinogens.

Carcinogenic risk is calculated by multiplying the intake by the CSF (or exposure concentration by the IUR).

$$ELCR = \text{Intake} \times \text{CSF} \text{ or } \text{Exposure Concentration} \times \text{IUR}$$

The combined risk from exposure to multiple constituents was evaluated by adding the risks from individual constituents. Risks were also added across the exposure routes if an individual would be exposed through multiple routes.

As required under the NCP (USEPA, 1994a) "[f]or known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between  $10^{-4}$  to  $10^{-6}$  using information on the relationship between dose and response." When a cumulative carcinogenic risk to a receptor under the assumed RME exposure conditions exceeds 1 in 10 thousand (i.e.,  $10^{-4}$  ELCR), the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) generally requires remedial action to reduce risks at the site.

### 7.1.3 Lead

Lead concentrations less than 0.015 mg/L in surface water (the Safe Drinking Water Act action level for lead in potable water) and less than 400 mg/kg in sediment (USEPA, 1994b) are considered adequately protective of human health under residential land-use conditions. Lead concentrations less than 800 mg/kg in sediment are considered adequately protective of human health under industrial land-use conditions. Lead was retained as a COPC when exceeding the residential values. Lead was identified as a COPC for exposed and near-shore surface sediment, surface sediment for the overflow scenario, and surface water during the wet event. Lead does not have available published toxicity factors, and therefore potential risks associated with lead are evaluated differently than the other COPCs. The toxicity of lead is evaluated by USEPA based on blood-lead uptake using a physiologically based pharmacokinetic model called the Integrated Exposure Uptake Biokinetic (IEUBK) model.

The IEUBK model is used to evaluate lead exposure to children. An interim approach to assessing risks associated with adult exposures to lead was developed by USEPA's Technical Review Workgroup for Lead (USEPA, 2003b) and updated in 2005. This methodology is a variation of the IEUBK model. The adult lead methodology (ALM) is used to evaluate risks associated with nonresidential adult exposures to lead in soil (or sediment for this assessment). The model focuses on estimating fetal blood concentrations in women exposed to lead in soil (USEPA, 2003b). It was used in this risk evaluation to be protective of potentially sensitive receptors within the industrial worker population and recreational population that may be exposed to overflow sediment. Because the lead model is a probabilistic model, several of the USEPA default parameters are based on central tendency (i.e., average) values (USEPA, 2003b). Therefore, the arithmetic means for sediment concentration served as the input value for the soil (sediment) concentrations.

The exposure parameters used in the ALM for ingestion and exposure frequency are the same as those that were used to evaluate direct contact with overflow sediment by the industrial worker and direct contact with exposed and near-shore sediment by the recreational adult. The sediment ingestion rate of 100 mg/day and exposure frequency of 9 days/year was assumed for the adult industrial worker. For the recreational adult, a

sediment ingestion rate of 50 mg/day and an exposure frequency of 26 days/year were used.

The ALM uses different sets of geometric standard deviations (GSDs) and baseline blood lead levels based on different studies. The GSD is a measure of the inter-individual variability in blood lead concentrations in a population whose members are exposed to the same nonresidential environmental lead levels. The baseline blood lead concentration is intended to represent the best estimate of a reasonable central value of blood lead concentrations in women of child-bearing age that are not exposed to lead-contaminated nonresidential soil (sediment) or dust at the site (USEPA, 2003b, c). In this analysis, geometric means were used.

ALM spreadsheets provided by USEPA (version date June 21, 2009) were used to calculate blood lead concentrations for the industrial worker. The model results are expressed as the predicted geometric mean blood lead level for adults (that is, women of child-bearing age) and the corresponding 95th percentile fetal blood lead concentrations and the percent of the population potentially experiencing concentrations above 10 micrograms per deciliter ( $\mu\text{g}/\text{dL}$ ), below which adverse manifestations are not expected.

The potential risks associated with residential/recreational exposures to lead are addressed using the IEUBK Lead Model for Windows, Version 1.1, Build 11 (USEPA, 1994c, 2010f). The IEUBK model was designed to provide predictions of the probability of elevated blood lead levels for children. This model addresses three components of environmental risk assessments: the multimedia nature of exposures to lead, lead pharmacokinetics, and significant variability in exposure and risk, through estimation of probability distributions of blood lead levels for children exposed to similar environmental concentrations. Although use of the IEUBK model to evaluate exposure to lead in the sediment and surface water for recreational and residential receptors is extremely conservative, there is currently no better method available. The arithmetic mean of the lead concentration in sediment and surface water was used with the default input parameters to represent site-specific exposures to lead.

## 7.2 Risk Assessment Results

The results of the risk characterization are presented below by receptor group. The risk estimates are calculated in the Attachment, Tables 7.1.RME through 7.10.RME and 7.1.CTE through 7.9.CTE, and summarized in the Attachment, Tables 9.1.RME through 9.10.RME and 9.1.CTE through 9.9.CTE. A summary of the RME results is shown in Table 7-1, and a summary of the CTE results is shown in Table 7-2. CTE risks were calculated when the total RME HI to a receptor group exceeded 1.0 and/or the total RME ELCR exceeded  $1 \times 10^{-4}$ . The risk estimates for each receptor group are summarized below and compared to USEPA's target HI of 1 and target ELCR range of  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$ . Noncarcinogenic hazards less than 1 are below USEPA's noncarcinogenic goal of protection of an HI of 1. Carcinogenic risks between or below  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$  are within USEPA's acceptable carcinogenic risk levels.

Noncarcinogenic hazards and carcinogenic risks are calculated for each of the receptors. However, carcinogenic risks are combined for the adult, adolescent, and child to estimate the carcinogenic risks to the recreational user, angler, and resident.

### **7.2.1 Recreational Adults**

Tables 9.1.RME and 9.1.CTE in the Attachment summarize the HIs (and ELCRs, which are discussed in Section 7.2.4) for recreational adult exposure to Gowanus Canal exposed and near-shore surface sediment, Gowanus Canal surface water, and air at canal level. The higher of the risks and hazards estimated for the two surface water scenarios and the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 0.2, below USEPA's target.
- Total HI (CTE) = 0.08, below USEPA's target.
- Lead was identified as a COPC for sediment and surface water (wet-weather event only). Exposure to lead in sediment was evaluated using the adult lead model. The model results are presented in Table 11.1 (Attachment). The mean sediment lead concentration of 660 mg/kg results in geometric mean blood levels ranging from 1.1 to 1.6 microgram per deciliter ( $\mu\text{g}/\text{dL}$ ) for women of child-bearing age. The corresponding 95th percentile fetal blood lead levels range from 2.6 to 4.9  $\mu\text{g}/\text{dL}$ . The probabilities that the fetal blood lead levels exceed 10  $\mu\text{g}/\text{dL}$  range from 0.005 to 0.5 percent. These values are less than the blood lead goal as described in the 1994 OSWER Directive (USEPA, 1994b) of no more than 5 percent of children (fetuses of exposed women) exceeding 10  $\mu\text{g}/\text{dL}$  blood lead.

### **7.2.2 Recreational Adolescents**

Table 9.2.RME in the Attachment summarizes the HIs (and ELCRs, which are discussed in Section 7.2.4) for recreational adolescent exposure to Gowanus Canal exposed and near-shore surface sediment, Gowanus Canal surface water, and air at canal level. The higher of the risks and hazards estimated for the two surface water scenarios and the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 0.2, below USEPA's target.
- Total HI (CTE) = 0.1, below USEPA's target.
- Lead was identified as a COPC for sediment and surface water (wet-weather event only). Exposure to lead in sediment was evaluated using the adult lead model, as discussed for the recreational adults. The probabilities that the fetal blood lead levels exceed 10  $\mu\text{g}/\text{DL}$  range from 0.005 to 0.5 percent, which is less than the blood lead goal as described in the 1994 OSWER Directive (USEPA, 1994b) of no more than 5 percent of children (fetuses of exposed women) exceeding 10  $\mu\text{g}/\text{dL}$  blood lead.

### **7.2.3 Recreational Children**

Tables 9.3.RME and 9.3.CTE in the Attachment summarize the HIs (and ELCRs, which are discussed in Section 7.2.4) for recreational child exposure to Gowanus Canal exposed and near-shore surface sediment, Gowanus Canal surface water, and air at canal level. The higher of the risks and hazards estimated for the two surface water scenarios and the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 0.5, below USEPA's target.
- Total HI (CTE) = 0.2, below USEPA's target.
- Lead was identified as a COPC for sediment and surface water (wet-weather event only). Site-specific lead exposures were evaluated for recreational children using the IEUBK model. This calculation was based on the site-specific arithmetic mean concentration of lead detected in the sediment (660 mg/kg) and surface water (13 mg/L). The results of the model, along with the probability distribution plot are presented in Table 11.2 in the Attachment. The predicted geometric mean blood lead level for a young child was 7.1 µg/dL, with 23 percent of the population potentially experiencing concentrations exceeding 10 µg/dL, below which adverse manifestations are not expected. These results indicate that if a child was exposed to the sediment and surface water at levels consistent with residential exposure to soil and potable use of surface water, the percent of the exposed population with a blood lead level exceeding 10 µg/dL would be above the 5 percent level that USEPA considers being protective of human health. However, it is unlikely that a child recreational receptor would be exposed to the sediment and surface water at the levels estimated in the IEUBK model, and it is more likely there would be no adverse effects associated with exposure to the lead.

#### **7.2.4 Recreational Receptors – Lifetime Carcinogenic Risks**

Carcinogenic risks were estimated for the combined lifetime (young child/adolescent/adult) recreational receptor, following USEPA risk assessment methodology. The total carcinogenic risk to a recreational receptor was calculated by adding the risks calculated for the young child, adolescent, and adult. The RME carcinogenic risks are summarized in Table 7-4, the CTE carcinogenic risks are summarized in Table 7-5, and the risks are discussed below. The higher of the risks estimated for the two surface water scenarios and the two ambient air scenarios were included in the total estimated risks.

- Total ELCR (RME) =  $1 \times 10^{-3}$ , above USEPA's target risk range. The risk is associated primarily with exposure to surface water ( $7 \times 10^{-4}$ ) and sediment ( $7 \times 10^{-4}$ ), and the risk drivers are carcinogenic PAHs. The carcinogenic PAHs were highest in the surface water samples collected at sampling locations 319 and 325 during the dry-weather sampling event and locations 309 and 319 during the wet-weather sampling event. The carcinogenic PAHs were highest (10 times higher than the next highest detection) in sediment sample 314, at the head of the 6th Street basin (see Figure 2-3a in the RI), and were therefore associated primarily with this location. The concentrations of the PAHs in the canal surface water during both dry- and wet-weather events and in the sediment were statistically significantly greater than in the reference area (see Sections 4.3.2 and 4.4.5 of the RI)
- Exposed and Near Shore Sediment ELCR (RME) =  $7 \times 10^{-4}$ , above USEPA's target risk range
- Surface water, dry weather event ELCR (RME) =  $6 \times 10^{-4}$ , above USEPA's target risk range

- Surface water, wet weather event ELCR (RME) =  $5 \times 10^{-4}$ , above USEPA's target risk range
- Air at canal level, prior to aeration system operation ELCR (RME) =  $4 \times 10^{-7}$ , below USEPA's target risk range
- Air at canal level, after to aeration system operation started ELCR (RME) =  $4 \times 10^{-7}$ , below USEPA's target risk range
- Total ELCR (CTE) =  $3 \times 10^{-4}$ , above USEPA's target risk range
- Exposed and Near Shore Sediment ELCR (CTE) =  $1 \times 10^{-4}$ , above USEPA's target risk range
- Surface water, dry weather event ELCR (CTE) =  $1 \times 10^{-4}$ , above USEPA's target risk range
- Surface water, wet weather event ELCR (CTE) =  $9 \times 10^{-5}$ , within USEPA's target risk range

## **7.2.5 Industrial Workers**

Table 9.4.RME in the Attachment summarizes the HIs and ELCRs for industrial worker exposures to ambient air at street level and surface water and sediment associated with canal overflow. The higher of the risks and hazards estimated for the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 0.8, below USEPA's target HI.
- Total ELCR (RME) =  $8 \times 10^{-5}$ , within USEPA's target risk range.
- Lead was identified as a COPC for sediment and surface water. Exposure to lead in sediment was evaluated using the adult lead model. The model results are presented in Table 11.3 of the Attachment. The mean sediment lead concentration of 533 mg/kg results in geometric mean blood levels ranging from 1.1 to 1.6 ug/dL for women of child-bearing age. The corresponding 95th percentile fetal blood lead levels range from 2.5 to 4.8  $\mu\text{g}/\text{dL}$ . The probabilities that the fetal blood lead levels exceed 10  $\mu\text{g}/\text{DL}$  range from 0.003 to 0.4 percent. These values are less than the blood lead goal as described in the 1994 OSWER Directive (USEPA, 1994b) of no more than 5 percent of children (fetuses of exposed women) exceeding 10  $\mu\text{g}/\text{dL}$  blood lead.

## **7.2.6 Adult Residents**

Tables 9.5.RME and 9.4.CTE, in the Attachment summarize the HIs for adult resident exposures to ambient air at street level and surface water and sediment associated with canal overflow. The higher of the hazards estimated for the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 1 (calculated HI value is 1.1), equals USEPA's target of HI of 1. The HI is associated with inhalation of ambient air. The HIs for all target organs are below USEPA's target, and therefore, the hazard is within USEPA acceptable levels.
- Surface water and sediment HIs (RME) are below 0.1, below USEPA's target.

- Total HI (CTE) = 0.7, below USEPA's target.
- Lead was identified as a COPC for sediment and surface water. Exposure to lead in sediment was evaluated using the adult lead model. The model results are presented in Table 11.3 in the Attachment. The mean sediment lead concentration of 533 mg/kg results in geometric mean blood levels ranging from 1.1 to 1.6 µg/dL for women of child-bearing age. The corresponding 95th percentile fetal blood lead levels range from 2.5 to 4.8 µg/dL. The probabilities that the fetal blood lead levels exceed 10 µg/DL range from 0.003 to 0.4 percent. These values are less than the blood lead goal as described in the 1994 OSWER Directive (USEPA, 1994b) of no more than 5 percent of children (fetuses of exposed women) exceeding 10 µg/dL blood lead.

### **7.2.7 Child Residents**

Tables 9.6.RME and 9.5.CTE in the Attachment summarize the HIs for child resident exposures to ambient air at street level and surface water and sediment associated with canal overflow. The higher of the hazards estimated for the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total HI (RME) = 1 (calculated HI value is 1.3), equals USEPA's target HI of 1. The HI is associated with inhalation of ambient air. The HIs for all target organs are below USEPA's target.
- Surface water and sediment HIs (RME) are below 0.3, below USEPA's target.
- Total HI (CTE) = 0.8, below USEPA's target.
- Lead was identified as a COPC for overflow sediment and surface water. Site-specific lead exposures were evaluated for recreational children using the IEUBK model. This calculation was based on the site-specific arithmetic mean concentration of lead detected in the sediment (533 mg/kg) and surface water (13 mg/L). The results of the model, along with the probability distribution plot are presented in Table 11.2 in the Attachment. The predicted geometric mean blood lead level for a young child was 6.1 µg/dL, with 14.9 percent of the population potentially experiencing concentrations exceeding 10 µg/dL, below which adverse manifestations are not expected. These results indicate that if a child was exposed to the sediment and surface water at levels consistent with residential exposure to soil and potable use of surface water, the percent of the exposed population with a blood lead level exceeding 10 µg/dL would be above the 5 percent level that USEPA considers to be protective of human health. However, it is unlikely that a child recreational receptor would be exposed to the sediment and surface water at the levels estimated in the IEUBK model, and it is more likely there would be no adverse effects associated with exposure to the lead.

### **7.2.8 Child/Adult (Lifetime) Residents**

Carcinogenic risks were estimated for the combined lifetime (child/adult) resident, following USEPA risk assessment methodology. Tables 9.7.RME and 9.6.CTE in the Attachment summarize the ELCRs for lifetime resident exposures to ambient air at street level and surface water and sediment associated with canal overflow. The higher of the risks

estimated for the two ambient air scenarios were included in the total estimated risks and hazards for the receptor group.

- Total ELCR (RME) =  $3 \times 10^{-4}$ , above USEPA's target risk range. The ELCR is associated primarily with exposure to overflow sediment ( $2 \times 10^{-4}$ ), with smaller contributions from exposure to overflow surface water ( $6 \times 10^{-5}$ ) and ambient air after the aeration system started operating ( $4 \times 10^{-5}$ ). The sediment risk drivers are carcinogenic PAHs, with highest concentrations in samples 314, at the head of the 6th Street basin, and 315, at the mouth of the 7th Street turning basin (see Figure 2-3a in the RI). PAH concentrations were variable in the reference area sediment, but concentrations observed in the canal were generally higher than those in the reference area (see discussion in Section 4.3.2 of the RI). Additionally, the statistical comparison of the PAH data from the canal and the reference area surface sediment indicated concentrations in the canal were statistically significantly greater than the reference area.
- Total ELCR (CTE) =  $8 \times 10^{-5}$ , within USEPA's target risk range.

## 7.2.9 Adult Anglers

Tables 9.8.RME and 9.7.CTE in the Attachment summarize the HIs (and ELCRs, which are discussed in Section 7.2.12) for angler adults who ingest fish and crab caught from the canal. Striped bass are representative of upper level predator fish, white perch are representative of middle level predator fish, and eel are representative of bottom feeders.

- Total Fish HI (RME) = 17, above USEPA's target HI. The HIs for ingestion of striped bass and eel exceed USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from mercury. About 70 percent of the total HI is contributed by assumed consumption of American eel. The average concentration of total PCBs in the eel from the canal is about two times higher than the average concentration of total PCBs in the reference samples (see Table 7-3 for average concentrations in canal and reference fish tissue samples). The average concentration of mercury in the eel from the canal samples is slightly lower than the average concentration in the eel from the reference samples.
- Total Crab HI (RME) = 3, above USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from arsenic and mercury. The average concentration of PCBs in blue crab from the canal is almost twice the average concentration of PCBs in blue crab from the reference samples (Table 7-3); however, the average concentrations of arsenic and mercury in blue crab from the reference samples are slightly higher than the average concentrations in the canal samples.
- White perch HI (RME) is below USEPA's target.
- Total Fish HI (CTE) = 2, above USEPA's target HI. The hazard is primarily associated with PCBs in fish (mainly eel).
- Total Crab HI (CTE) = 2, above USEPA's target HI. The hazard is primarily associated with PCBs.

## **7.2.10 Adolescent Anglers**

Tables 9.9.RME and 9.8.CTE in the Attachment summarize the HIs (and ELCRs, which are discussed in Section 7.2.12) for adolescent anglers who ingest fish and crab caught from the canal. Striped bass are representative of upper level predator fish, white perch are representative of middle level predator fish, and eel are representative of bottom feeders.

- Total Fish HI (RME) = 13, above USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from mercury (in striped bass and eel). The HIs for ingestion of striped bass and eel exceed USEPA's target HI. About 74 percent of the total HI is contributed by assumed consumption of American eel. The average concentration of total PCBs in the eel from the canal is about two times higher than the average concentration of total PCBs in the reference samples (see Table 7-3 for average concentrations in canal and reference fish and crab tissue samples). The average concentration of mercury in the eel from the canal samples is slightly lower than the average concentration in the eel from the reference samples.
- Total Crab HI (RME) = 3, above USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from arsenic and mercury. The average concentration of PCBs in blue crab from the canal is almost twice the average concentration of PCBs in blue crab from the reference samples. The average concentrations of arsenic and mercury in the blue crab from the reference samples are slightly higher than the average concentrations in the canal samples.
- White perch HI (RME) is below USEPA's target.
- Total Fish HI (CTE) = 1 (calculated HI value is 1.3), equals USEPA's target HI.

Total Crab HI (CTE) = 0.6, below USEPA's target HI.

## **7.2.11 Children of Anglers**

Tables 9.10.RME and 9.9.CTE in the Attachment summarize the HIs (and ELCRs, which are discussed in Section 7.2.12) for children of anglers who ingest fish and crab caught from the canal. Striped bass are representative of upper level predator fish, white perch are representative of middle level predator fish, and eel are representative of bottom feeders.

- Total Fish HI (RME) = 27, above USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from mercury (in striped bass and eel). About 70 percent of the total HI is contributed by assumed consumption of American eel. The average concentration of total PCBs in the eel from the canal is about two times higher than the average concentration of total PCBs in the reference samples (see Table 7-3 for average concentrations in canal and reference fish and crab tissue samples). The average concentration of mercury in the eel from the canal samples is slightly lower than the average concentration in the eel from the reference samples.
- Total Crab HI (RME) = 5, above USEPA's target HI. The hazard is associated with PCBs, with smaller contributions (below 1) from arsenic and mercury. The average concentration of PCBs in blue crab from the canal is almost twice the average concentration of PCBs in blue crab from the reference samples. The average

concentrations of arsenic and mercury in the blue crab from the reference samples are slightly higher than the average concentrations in the canal samples.

- Total Fish HI (CTE) = 3, above USEPA's target HI, primarily associated with PCBs in eel.
- Total Crab HI (CTE) = 4, above USEPA's target HI, primarily associated with PCBs.

### **7.2.12 Anglers – Lifetime Carcinogenic Risks**

Carcinogenic risks were estimated for the combined lifetime (child/adolescent/adult) angler, following USEPA risk assessment methodology. The total carcinogenic risk to a angler was calculated by adding the risks calculated for the young child, adolescent, and adult. The RME carcinogenic risks are summarized in Table 7-4 and the CTE carcinogenic risks are summarized in Table 7-5, and are presented below. Striped bass are representative of upper level predator fish, white perch are representative of middle level predator fish, and eel are representative of bottom feeders.

- Total Fish ELCR (RME) =  $7 \times 10^{-4}$ , above USEPA's target risk range. The risk from ingestion of eel (bottom feeders) and striped bass (top level predators) exceed USEPA's target risk range. The risk is associated primarily with ingestion of eel (74 percent). The risk drivers are PCBs. The dioxin-like PCBs and nondioxin-like PCBs contributed to the risk at similar risk levels. The average concentration of non-dioxin-like PCBs and dioxin-like PCB TEQs in the eel from the canal is almost two times higher than the average concentrations in the reference samples (see Table 7-3 for average concentrations in canal and reference fish and crab tissue samples).
- Total white perch (middle level predators) ELCR (RME) is within the target risk range .
- Total Crab ELCR (RME) =  $3 \times 10^{-4}$ , above USEPA's target risk range. The risk drivers are PCBs. The average concentration of non-dioxin-like PCBs and dioxin-like PCB TEQs in blue crab from the canal (see Table 7-3 for average concentrations in canal and reference fish and crab tissue samples) is almost twice the average concentration of PCBs in blue crab from the reference samples.
- Total Fish ELCR (CTE) =  $2 \times 10^{-5}$ , within USEPA's target risk range.
- Total Crab ELCR (CTE) =  $6 \times 10^{-5}$ , within USEPA's target risk range.

## SECTION 8

# Uncertainty Assessment

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The risk measures used in HHRA are not fully probabilistic estimates of risk, but are conditional estimates given that a set of assumptions about exposure and toxicity are realized. Thus, it is important to specify the assumptions and uncertainties inherent in the risk assessment to place the risk estimates in proper perspective (USEPA, 1989).

## 8.1 Uncertainty in Data Evaluation and COPC Identification

The sampling conducted along the canal for use in the HHRA focused on areas where contact with the canal is most likely to occur (shallow and exposed sediment and surface water and air samples collected from similar locations) to estimate the most realistic exposure and risk to potential receptors. Additional surface water and surface sediment samples were collected from locations where historic operations and discharges to the canal have most likely occurred. Only the surface sediment samples from the exposed and shallow areas were used to evaluate the recreational exposure risks, as this is the sediment that these receptors are most likely to contact. All of the surface water samples were included in the recreational scenario evaluations since the receptors could be exposed to the water throughout the canal while boating. All of the surface water and surface sediment samples were used to evaluate risks to the residential and industrial worker receptors associated with exposure to canal overflow water and sediment. Therefore, the available data were evaluated in the HHRA in data groupings for each receptor group to estimate the reasonable maximum exposures and risks.

The uncertainty associated with the data analysis is minimal, and all of the data were validated prior to being used in the HHRA. A data quality evaluation was performed on all analytical data evaluated in the HHRA, as discussed in Section 4.1 of the RI.

A few constituents (mainly pesticides) were not detected in any of the fish or crab tissue samples but had detection limits above the human health risk-based screening level. This may result in slightly underestimating the risk associated with ingestion of fish and crab. However, it should be noted that risks above acceptable levels were identified for the fish and/or crab tissue and risks were primarily associated with the PCBs detected in the fish and crab tissue. The pesticides detected in the fish and crab tissue did not contribute significantly to the risk.

A comparison of data collected from the canal to data collected from the reference locations was not used to identify the COPCs for the canal media. This may result in including COPCs related to background conditions in the risk estimates.

All of the canal data were screened against residential screening levels to select the COPCs. The use of residential screening levels is conservative, as not all the scenarios evaluated in the HHRA are residential (i.e., the industrial worker scenario). This may result in including COPCs with small contributions to overall risk estimates.

## **8.2 Uncertainty Associated with Exposure Assessment**

The exposure factors used for quantifying exposure were conservative and reflect upper-bound assumptions. The reliability of the values chosen for the exposure factors also contributes substantially to the uncertainty of the resulting risk estimates. Because most of the exposure factors are upper-bound assumptions, the resulting risks are likely overestimates.

The most conservative recreational scenario (swimming) was used to evaluate potential exposure and risks for recreational adults, adolescents, and children. Based on the current conditions of the canal, it is likely that this is an overestimate of actual exposure to recreational receptors. Although possible, it is highly unlikely that a recreational receptor would swim in the canal for 26 days per year for 2.6 hours per day. Although the RME scenario indicated a slight risk above USEPA target levels, the CTE scenario (using more realistic but conservative assumptions of an exposure frequency of 13 days per year and an exposure time of 1.3 hours per day) indicated a risk within USEPA acceptable risk levels.

For the purposes of this risk assessment, the angler population is defined as those individuals who consume self-caught fish from the Gowanus Canal at least once per year, in the absence of a fishing ban or fish consumption advisories. Sources of uncertainty in the PCB concentrations in fish used in the assessment include the fact that concentrations were averaged over location, and weighted by species. The weighting of species intake in order to derive an average EPC in fish is a source of uncertainty because there are limited site-specific data available to estimate the species ingestion preferences (e.g., weighting factors).

Cancer risks and noncancer health hazards were not specifically quantified for subsistence anglers, or other subpopulations of anglers who may be highly exposed. Although there are no known distinct subpopulations that may be highly exposed, there is some degree of uncertainty as to whether these subpopulations have been adequately addressed in this risk assessment.

As described in Section 5.3.2, reported cooking losses vary considerably among the numerous studies reviewed. However, little information is available to quantify personal preferences among anglers for various preparation and cooking methods and other related habits (such as consumption of pan drippings). The assumption that there is no loss of PCBs during cooking or preparation, used in the RME cancer risk and noncarcinogenic hazard calculations, is conservative and could overestimate cancer risks and noncarcinogenic hazards.

The sediment/skin adherence factor represents the amount of sediment that adheres to skin and is available for dermal exposure. Because this value is likely to vary based on one's activity, the values used for this parameter, which are estimates from single activities, are somewhat uncertain. For dermal contact with canal sediments, published adherence factors for adults gathering reeds and for children playing in wet soils were used as a surrogate for recreational children and adults and children. Although it is somewhat uncertain whether these scenarios are representative of contact with canal sediments, they appear to be a reasonable use of available data.

Commercial PCB mixtures tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain, so there is a potential that

carcinogenic risks and noncarcinogenic hazards have been underestimated. However, since the CSFs are based on animal exposures to a group of PCB mixtures (i.e., Aroclor 1260, 1254, 1242, and 1016) that contain overlapping groups of congeners spanning the range of congeners most often found in environmental mixtures, this source of potential uncertainty is unlikely to have a significant impact.

The fact that any previous exposures (either background, or past consumption of contaminated fish) may still be reflected in an individual's body burden today is an additional source of uncertainty, and may result in an underestimate of noncarcinogenic health hazards.

The risk assessment assumed that people would consume both the combined muscle (edible portion) and hepatopancreas from blue crab. This may result in an overestimation of risk, as many crabbers do not consume the hepatopancreas, and some chemicals, such as PCBs and mercury, accumulate in the hepatopancreas. However, the hepatopancreas is small compared to the edible portion of the fish, and therefore, although concentrations may be higher in the hepatopancreas, it contributes a small amount of the total amount of crab consumed by the receptor. Therefore, it would not significantly change the total amount of contaminants consumed.

Dioxin-like PCB TEQ concentrations, non-dioxin-like PCB concentrations, and total PCB concentrations were calculated for each sample using detected PCB congeners only. In general, if the nondetected dioxin congeners were included in the sample concentration calculations, the non-dioxin-like PCB concentrations and the total PCB concentrations would be similar to those used in the risk assessment and the resulting risks would not differ significantly. The dioxin-like PCB TEQ concentrations would not always be similar, since if the most toxic congener (3,3',4,4',5-Pentachlorobiphenyl [126]) was not detected in a sample, inclusion of this congener at the detection limit times the TEF would contribute significantly to the dioxin-like PCB TEQ, even if it was not detected. Therefore, this may result in an underestimation of actual risk if this congener was present in the sample at a concentration below the reporting limit. However, when this congener was detected in a sample, it was generally flagged as detected below the reporting limit, so it is unlikely that it would be present in the samples at concentrations similar to reporting limits, and it would likely be present at much lower concentrations and not contribute significantly to the dioxin-like PCB TEQ concentration for the sample. Additionally, congeners that were B qualified, indicating they were detected in a laboratory or field blank at a concentration similar to that in the sample, were not included in the concentration calculation. There were a number of samples with B-qualified congeners, which may result in an underestimation of risk if these congeners are present in the samples at a concentration below the blank contamination level. However, the B-qualified concentrations were generally below the reporting limits.

There are inherent limitations and uncertainties associated with estimating health risks on the basis of fish and crab consumption that should be considered when interpreting the results of this HHRA. Factors contributing to these uncertainties include the following:

- Game fish and blue crab ranges are not limited to the canal, but reflect cumulative uptake from all areas they traverse. Blue crab and some of the game fish species kept by anglers have relatively large home ranges, and those caught within the canal are likely, at least in part, to have inhabited areas outside the canal and therefore to have been

potentially exposed to contaminants not related to the canal. However, differences between canal and reference concentrations of PCBs in fish and crab tissue and sediment were identified, with canal media having higher concentrations.

Assumptions regarding fishing/crabbing frequency and fish/crab consumption rates are variable and affect the estimates of exposure and associated risk.

### **8.3 Uncertainty Associated with Toxicity Assessment**

Uncertainty associated with the noncarcinogenic toxicity factors is included in Table 5.1 in the Attachment. The USEPA applies several UFs to extrapolate doses from animal studies to humans. The UFs for the COPCs range from 1 to 3,000. Therefore, there is a high degree of uncertainty in the noncarcinogenic toxicity criteria based on the available scientific data. The noncarcinogenic toxicity factors used in the HHRA are expected to be overestimates of actual toxicity.

CSFs and IURs developed by USEPA represent upper-bound estimates. Carcinogenic risks generated in this assessment should be regarded as an upper-bound estimate on the potential carcinogenic risks. The true ELCR is likely to be less than the predicted value (USEPA, 1989).

Use of provisional or withdrawn toxicity factors increases the uncertainty of the quantitative hazard and risk estimates. Provisional toxicity values (from Cal EPA, PPRTV, ATSDR, and NCEA) were used in the HHRA. The provisional values were used to provide a quantitative estimate rather than a merely qualitative risk discussion; however, USEPA has not fully promulgated these toxicity values.

The TEFs used to adjust the concentrations of the dioxin-like PCB congeners to TEQ of dioxin for the sediment and fish/crab also results in uncertainty in the risk assessment. In particular, although the TEF scheme and TEQ methodology is intended primarily for estimating exposure and risk through ingestion (primarily dietary intakes; USEPA, 2010c), it was also used to estimate exposure and risk through dermal contact. Additionally, not using the dioxin-like PCB congener TEQ to evaluate noncarcinogenic risks may underestimate the noncarcinogenic hazard associated with exposure to the PCBs. However, there is a lot of uncertainty with use of this process, and even more uncertainty with use of it for noncarcinogenic hazards since the TEFs are based on the relationship of the PCB congeners to the carcinogenic risks associated with dioxin. Although, there is a large degree of uncertainty associated with use of this method for noncarcinogenic-hazard evaluation, noncarcinogenic hazards were estimated as part of the uncertainty evaluation.

Noncarcinogenic hazards were calculated for the dioxin-like PCB TEQ concentrations for sediment and fish/crab using the RfD for 2,3,7,8-TCDD from the 1998 dioxin ATSDR toxicity profile. It should be noted that use of this RfD in itself presents a source of uncertainty, as the ATSDR toxicity profiles are a Tier 3 source of toxicity information. Noncarcinogenic hazards were calculated for the non-dioxin-like PCB concentrations using the RfD for Aroclor 1254. The sum of the noncarcinogenic hazards associated with the dioxin-like PCB TEQ concentrations and the non-dioxin-like PCB concentrations for all receptors for sediment and fish/crab are below 1 and in most cases are less than the noncarcinogenic hazards associated with the total PCB concentration, which were estimated using the total PCB concentrations and the RfD for Aroclor 1254. Therefore, evaluation of

the noncarcinogenic hazards for the dioxin-like PCB TEQ concentrations does not change the conclusions of the HHRA.

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## SECTION 9

# Human Health Risk Summary

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The risk assessment was conducted to evaluate the potential human health risks associated with direct contact with sediment and surface water in the Gowanus Canal, with ingestion of fish and crabs from the canal, with direct contact of sediment and surface water that overtops the canal during significant rainfall events, and with inhalation of emissions from the canal into the ambient air near the canal.

Table 5 and Tables 9.1.RME through 9.10.RME in the Attachment summarize the RME ELCRs and HIs. Table 6 and Tables 9.1.CTE through 9.9CTE in the Attachment summarize the CTE ELCRs and HIs. Estimated HIs and ELCRs associated with exposure to canal-related media indicate the potential for unacceptable risks based on exposure to some media by some receptor groups. Risk estimates are summarized below:

- Recreational receptors (adult, adolescent, and child)
  - Exposures are to surface water and surface sediment (from exposed and near-shore locations) in the canal and to ambient air at canal level while swimming in the canal for 26 days per year for 2.6 hours each day (very conservative assumptions given the nature of the canal and current CSO discharges).
  - HIs and ELCRs (RME) for inhalation of canal level air are within USEPA acceptable levels.
  - Total HI (RME) (all media combined) for all recreational receptors is within acceptable risk levels.
  - Total ELCR (RME) for recreational receptor ( $1 \times 10^{-3}$ ) exceeds USEPA's target risk range, primarily because of carcinogenic PAHs in surface water and sediment above background levels (see Sections 4.3.2 and 4.4.5 of the RI).
  - Total HI (CTE) for all recreational receptors are below USEPA's target level.
  - Total ELCR (CTE) for recreational receptor ( $3 \times 10^{-4}$ ) exceeds USEPA's target levels and ranges; the CTE scenario may be more representative of likely exposure (although still conservative, at an assumed swimming frequency of 13 days per year for 1.3 hours per day). The risk is primarily associated with exposure to the young child (67 percent of the risk).
  - Exposure to lead in sediment by adult and adolescent recreational receptors was demonstrated not to be a concern using the adult lead model. The IEUBK model indicated potential adverse effects for children exposed to lead in sediment and surface water; however, the model is based on residential exposure to these media, including using the surface water as a potable water supply. Therefore, it is unlikely, based on a more-realistic recreational exposure, that there would be any adverse effects based on exposure to recreational children.
- Industrial workers and residential receptors (adult and child)

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- Exposures are to ambient air at street level and surface water and sediment associated with canal overflow.
    - Total HI and ELCR (RME) for industrial workers are below or within USEPA's target levels.
    - Total HIs (RME) for residential adults (Total HI equals 1, calculated value is 1.1) and for children (Total HI equals 1, calculated value is 1.3) do not exceed USEPA's target level of 1. Inhalation of ambient air at street level contributes over 75 percent of the hazard. No target organ HIs exceed USEPA's target levels.
    - HIs (RME) for exposure to surface water and sediment are within USEPA acceptable levels.
    - ELCRs (RME) for inhalation of street level air are within USEPA acceptable levels.
    - ELCR (RME) for lifetime (child/adult) resident ( $3 \times 10^{-4}$ ) exceeds USEPA's acceptable level primarily associated with carcinogenic PAHs in sediment (with a smaller contribution from surface water below  $1 \times 10^{-4}$ ) that were above background levels (see Sections 4.3.2 and 4.4.5 of the RI).
    - Exposure to lead in sediment by industrial workers was demonstrated not to be a concern using the adult lead model. The IEUBK model indicated potential adverse effects for children exposed to lead in sediment and surface water; however, the model is based on residential exposure to these media, including using the surface water as a potable water supply. Therefore, it is unlikely, on the basis of a more realistic exposure, that there would be any adverse effects based on recreational children's exposure to lead.
  - Anglers (adult, adolescent, and child)
    - Exposure is from ingestion of fish (striped bass [representative of top level predators], white perch [representative of middle level predators], and eel [representative of bottom feeders]) and crab caught in the Gowanus Canal. Assuming that fishing/crabbing and ingestion of fish/crab from the Gowanus Canal occur at typical recreational angler fish consumption rates is very conservative given the nature of the canal and current CSO discharges to the canal.
    - Total Fish HIs (RME) (all fish types) for adults (17), adolescents (13), and children (27) exceed acceptable risk levels, primarily because of PCBs. The ingestion of striped bass and eel contributed higher HIs than ingestion of white perch (which for the child only did contribute to HI above 1). Additionally, mercury contributed to the HIs, at HIs above 0.1 but below 1.
    - Total Crab HIs (RME) for adults (37), adolescents (3), and children (5) exceed acceptable risk levels, primarily because of PCBs. Additionally, mercury and arsenic contributed to the HIs, at HIs above 0.1 but below 1.0.
    - Average PCB concentrations in canal samples are about twice the average PCB concentrations in reference samples; however, concentrations of PCBs in reference samples would also result in HIs and ELCRs above acceptable risk levels.

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- Total Fish ELCR (RME) for angler ( $7 \times 10^{-4}$ ) exceeds USEPA's target risk range, primarily because of PCBs.
  - Total Crab ELCR (RME) for angler ( $3 \times 10^{-4}$ ) exceeds USEPA's target risk range, primarily because of PCBs.
  - Total Fish HIs (CTE) for adults (2.0), adolescents (1.7), and children (3.2) exceed USEPA's target level; these are associated with PCBs in fish and crab.
  - Total Crab HIs (CTE) for adults (2) and children (3) exceed USEPA's target level; these are associated with PCBs. Total HI (CTE) for adolescents (Total HI equals 1, calculated as 1.3) does not exceed USEPA's target level.
  - Total Fish ELCR (CTE) for angler receptor is within USEPA's target range; the CTE scenario may be more representative of consumption of fish for the Gowanus Canal (although still conservative).
  - Total Crab ELCR (CTE) for angler receptor is within USEPA's target range; the CTE scenario may be more representative of consumption of crab for the Gowanus Canal (although still conservative).
  - A primary contributor to fish consumption risk is PCBs in tissues of fish caught from the canal. This conclusion is supported by NYDOH's decision to issue fish consumption advisories for the Upper Bay of the New York Harbor (north of the Verrazano Narrows Bridge), including the Gowanus Canal (NYSDOH, 2010) in part on the basis of PCB concentrations present in fish. Reference area average PCB fish concentrations are about one half the average concentrations identified in canal fish. However, these would also result in risks above acceptable risk levels.

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## SECTION 10

# References

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- Ambruster, G., K.G. Gerow, W.H. Gutenmann, C.B. Littman, and D.J. Lisk. 1987. The effects of several methods of fish preparation on residues of polychlorinated biphenyls and sensory characteristics in striped bass. *Journal of Food Safety* 8:235-243.
- ATSDR (Agency for Toxic Substances and Disease Registry). 2007. Toxicological Profile for Arsenic. U.S. Department of Health and Human Services. August.
- ATSDR (Agency for Toxic Substances and Disease Registry). 2008. Toxicological Profile for Chromium. U.S. Department of Health and Human Services. September.
- Burger, J. 2002. Consumption Patterns and Why People Fish. *Environmental Research Section A* 90, 125-135.
- Connelly, Nancy A., Barbara A. Knuth, and Carole A. Bisogni. 1992. *Effects of the Health Advisory and Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries*. Report for New York Sea Grant Institute Project No. R/FHD-2=PD. September.
- FEMA (Federal Emergency Management Agency). 2007. Flood Insurance Rate Map 360490211F. September 5.
- Gowanus Dredgers Canoe Club. 2010. Welcome to the Gowanus Dredgers Canoe Club. Available at <http://www.waterfrontmuseum.org/dredgers/home.html>.
- Gowanus Lounge. November 6, 2007. Yummy Blue Plate Special: Gowanus Canal Blue Fish. Available at <http://gowanuslounge.blogspot.com/2007/11/yummy-blue-plate-special-gowanus-canal.html>.
- May, H., and J. Burger. 1996. Fishing in a Polluted Estuary: Fishing Behavior, Fish Consumption, and Potential Risk. *Risk Analysis*. Vol. 16, no. 4. pp. 459-471.
- McCarroll N., N. Keshava, J. Chen, G. Akerman, A. Kligerman, and E. Rinde. 2010. An evaluation of the mode of action framework for mutagenic carcinogens case study II: Chromium (VI), *Environmental and Molecular Mutagenesis* Volume 51, Issue 2, pages 89-111, March.
- Moya, J., K.G. Garrahan, T.M. Poston, G.S. Durell. 1998. Effects of cooking on levels of PCBs in the fillets of winter flounder. *Bull. Environ. Contamin. Toxicol.* 60:845-851.
- New York Times. 2007. Divers Who Jump in to Take the Mystery out of City Waterways. Available at [http://www.nytimes.com/2007/11/27/nyregion/27divers.html?\\_r=1](http://www.nytimes.com/2007/11/27/nyregion/27divers.html?_r=1). November 27.
- NJDEP (New Jersey Department of Environmental Protection). 2002. Estimate of Cancer Risk to Consumers of Crabs Caught in the Area of the Diamond Alkali Site and Other Areas of the Newark Bay Complex from 2,3,7,8-TCDD and 2,3,7,8-TCDD Equivalents. Prepared by

the Division of Science, Research and Technology (April 25). Available at <http://www.state.nj.us/dep/dsr/craboutreach/crabsra.pdf>.

NYCDEP (New York City Department of Environmental Protection). 2008. *Gowanus Canal Waterbody/Watershed Facility Plan Report*. New York City Department of Environmental Protection, Bureau of Engineering Design and Construction. City-wide Long Term CSO Control Planning Project. August.

NYSDOH (New York State Department of Health). 2010. Chemicals in Sportfish and Game, 2010-2011 Health Advisories. Available at <http://www.health.state.ny.us/environmental/outdoors/fish/docs/fish.pdf>.

Skea, J.C., H.A. Simonin, E.J. Harris, S. Jackling, and J.J. Spagnoli. 1979. Reducing levels of mirex, arochlor 1254, and DDE by trimming and cooking Lake Ontario brown trout (*Salmo trutta L.*) and smallmouth bass (*Micropterus dolomieu lacepede*). *J Great Lakes Res.* 5(2):153-159.

USEPA. 1986. *Guidelines for Health Risk Assessment of Chemical Mixtures*. Federal Register, Vol. 51 34041. September.

USEPA. 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual, Part A, Interim Final. Office of Solid Waste and Emergency Response. EPA/540/1-89/002.

USEPA. 1991. Risk Assessment Guidance for Superfund. Vol. 1: Human Health Evaluation Manual-Supplemental Guidance, Standard Default Exposure Factors. Interim Final. OSWER Directive 9285.6-03.

USEPA Region 2. 1992. *Evaluation of Metals Data for the Contract Laboratory Program (SOP HW-2, Revision 11)*. January.

USEPA. 1993. Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure. Washington, DC.

USEPA. 1994a. National Oil and Hazardous Substances Contingency Plan. September 15.

USEPA. 1994b. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive 9355.4-12. July 14.

USEPA. 1994c. Guidance Manual for the IEUBK Model for Lead in Children. PB93-963510, OSWER 9285.7-15-1. February.

USEPA. 1995. Health Assessment Document for Polychlorinated Dibenzo-p-Dioxins. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, OH. EPA 600/8-84-014F.

USEPA. 1996. PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures. EPA/600/P-96/001F. September.

USEPA. 1997. *Exposure Factors Handbook*. EPA/ 600/P-95/Fa, -Fb, and -Fc.

USEPA. 2000. *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume 2: Risk Assessment and Fish Consumption Limits - Third Edition*. Appendix C. Dose Modifications Due to Food Preparation and Cooking. EPA 823-B-00-008. November.

- USEPA. 2001a. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments. Office of Solid Waste and Emergency Response. OSWER 9285.7-47. December.
- USEPA Region 2. 2001b. *Data Validation SOP for Statement of Work OLM04.2* (SOP HW-6, Revision 12). March.
- USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24. December.
- USEPA. 2003a. Human Health Toxicity Values in Superfund Risk Assessments. OSWER Directive 9285.7-53. December.
- USEPA. 2003b. Recommendations of the Technical Review Workgroup for Lead for an Approach to Assessing Risks Associated with Adult Exposure to Lead in Soil. Office of Solid Waste and Emergency Response. OSWER 9285.7-54. January.
- USEPA. 2004. *Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual* (Part E, Supplemental Guidance for Dermal Risk Assessment) Interim. OSWER 9285.7-02EP. July.
- USEPA. 2005a. Guidelines for Carcinogenic Risk Assessment. EPA/630/P-03/001F. March.
- USEPA. 2005b. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F. March.
- USEPA. 2006. Derivation of RBCs for Carcinogens that Act Via a Mutagenic Mode of Action and Incorporate Default ADAFs. Region 3. October.
- USEPA. 2009. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment). Final. EPA-540-R-070-002. OSWER 9285.7-82. Office of Superfund Remediation and Technology Innovation. January.
- USEPA. 2010a. ProUCL, Version 4.00.05. Prepared by Lockheed Martin Environmental Services. May.
- USEPA. 2010b. EPA Regional Screening Level Summary Table. May
- USEPA Region 3. 2010c. Regional Screening Level Fish Ingestion Table November 2010. Available at <http://www.epa.gov/reg3hwmd/risk/human/index.htm>. December.
- USEPA. 2010d. Recommended Toxicity Equivalence Factors (TEFs) for Human Health Risk Assessments of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Dioxin-Like Compounds. Risk Assessment Forum, Washington, DC. EPA/100/R-10/005.
- USEPA. 2010e. Integrated Risk Information System database.
- USEPA. 2010f. IEUBKWin32 Lead Model for Windows, Version 1.1, Build 11. <http://www.epa.gov/superfund/lead/products.htm#guid>
- U.S. Food and Drug Administration (U.S. FDA). 1993. Guidance Documents for Trace Elements in Seafood. Center for Food Safety and Applied Nutrition. January.