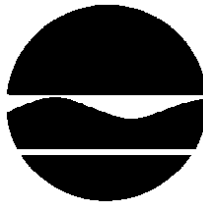


**New York State
Department of Environmental Conservation
Division of Environmental Remediation
625 Broadway
Albany, New York 12233-7016**

**ONONDAGA LAKE
HUMAN HEALTH RISK ASSESSMENT
Volume 1 of 2
(Text, Tables, Figures, and Appendices A through D)**



**Onondaga Lake Project
Site No. 7-34-030-002
Contract Number C004365, Task Order 1**

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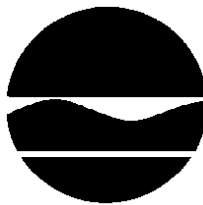
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East Syracuse, NY

December 2002

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Department of Environmental Conservation
Division of Environmental Remediation
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**ONONDAGA LAKE
HUMAN HEALTH RISK ASSESSMENT
Volume 2 of 2
(Appendix E)**



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Onondaga Lake HHRA – Contents

(Volume 1 of 2)

	<u>Page</u>
List of Figures	x
List of Tables	xi
Acronyms and Abbreviations	xiii
Glossary	xvi
Executive Summary	ES-1
1. Introduction	1-1
1.1 Guidance Documents	1-3
1.2 Organization	1-4
2. Background	2-1
2.1 Site and Vicinity Description	2-1
2.2 Climate	2-1
2.3 Groundwater and Surface Water	2-2
2.4 Population and Demographics	2-3
2.5 Site History	2-3
2.6 Potential Use and Exposures	2-5
3. Identification of Contaminants of Potential Concern	3-1
3.1 Fish Tissue	3-6
3.2 Onondaga Lake Nearshore Surface Sediment	3-8
3.2.1 Northern Basin Nearshore Sediments	3-9
3.2.2 Southern Basin Nearshore Sediments	3-10
3.3 Wetland Sediments	3-11
3.3.1 Northern Basin Wetland SYW-6	3-11
3.3.2 Northern Basin Wetland SYW-10	3-12
3.3.3 Southern Basin Wetland SYW-12	3-12
3.3.4 Southern Basin Wetland SYW-19	3-12

3.4	Dredge Spoil Soils	3-12
3.4.1	Dredge Spoils – Surface Soils	3-13
3.4.2	Dredge Spoils – Surface and Subsurface Soils	3-13
3.5	Onondaga Lake Surface Water	3-13
4.	Exposure Assessment	4-1
4.1	Exposure Setting and Receptor Populations	4-2
4.1.1	Current Land and Site Use	4-2
4.1.1.1	Drinking Water	4-3
4.1.1.2	Fishing	4-3
4.1.1.3	Beaches and Swimming	4-3
4.1.1.4	Potential Receptors	4-4
4.1.2	Future Land and Site Use	4-4
4.2	Potential Exposure Pathways	4-5
4.2.1	Consumption of Fish Tissue	4-6
4.2.2	Lake and Wetland Surface Sediments and Dredge Spoil Soils ..	4-8
4.2.3	Surface Water	4-8
4.2.4	Consumption of Game	4-9
4.2.5	Inhalation of Volatile Organic Compounds	4-9
4.3	Quantitation of Exposure	4-9
4.3.1	Fish Consumption	4-10
4.3.1.1	Fish Consumption Rates	4-11
4.3.1.2	Fractional Intake of Fish	4-12
4.3.1.3	Cooking Loss from Fish	4-12
4.3.1.4	Exposure Frequency and Duration for Fish Consumption	4-13
4.3.2	Exposure Frequency and Duration and Receptor Characteristics for All Media Except Fish	4-13
4.3.2.1	Recreational Scenarios	4-14

4.3.2.2	Construction Worker Scenario	4-15
4.3.3	Incidental Ingestion of Sediments and Soils	4-15
4.4	Dermal Contact with Surface Sediments and Soils	4-16
4.4.1	Skin Surface Area Available for Contact	4-16
4.4.2	Soil-to-Skin Adherence Factors	4-17
4.5	Incidental Ingestion of Surface Water	4-17
4.6	Dermal Contact with Surface Water	4-18
4.7	Exposure Point Concentrations	4-18
4.7.1	Sample Data Used for Calculation of Exposure Point Concentrations in Fish	4-19
4.7.2	Exposure Point Concentration Calculations in Fish – Special Considerations for Mercury and Arsenic	4-19
4.7.2.1	Mercury	4-19
4.7.2.2	Arsenic	4-20
4.7.3	Determination of Data Distribution Type	4-20
4.7.4	Calculation of Exposure Point Concentrations	4-21
4.7.5	Calculation of Length-Weighted Average for Sediment Samples	4-22
4.8	Summary of Exposure Assessment	4-23
5.	Toxicity Assessment	5-1
5.1	Derivation of Toxicity Values	5-1
5.1.1	USEPA-Derived Oral Toxicity Values	5-1
5.1.2	Toxicity Assessment for Dermal Exposure	5-2
5.2	Chemical-Specific Summaries and Toxicity Values for Onondaga Lake Contaminants of Potential Concern	5-3
5.2.1	Metals and Organometallic Compounds	5-4
5.2.1.1	Aluminum	5-4
5.2.1.2	Antimony	5-5

5.2.1.3 Arsenic	5-5
5.2.1.4 Barium	5-6
5.2.1.5 Cadmium	5-6
5.2.1.6 Chromium	5-7
5.2.1.7 Copper	5-8
5.2.1.8 Cyanide	5-8
5.2.1.9 Iron	5-9
5.2.1.10 Lead	5-9
5.2.1.11 Manganese	5-10
5.2.1.12 Mercury – General	5-10
5.2.1.13 Methylmercury	5-11
5.2.1.14 Mercury – Inorganic	5-12
5.2.1.15 Nickel	5-12
5.2.1.16 Selenium	5-13
5.2.1.17 Thallium	5-13
5.2.1.18 Vanadium	5-14
5.2.1.19 Zinc	5-14
5.2.2 Volatile Organic Compounds	5-14
5.2.2.1 Benzene	5-15
5.2.2.2 Bromodichloromethane	5-16
5.2.2.3 Chlorobenzene	5-16
5.2.2.4 Chloroform	5-16
5.2.2.5 Methylene Chloride (Dichloromethane)	5-17
5.2.2.6 Xylenes	5-17
5.2.3 Semivolatile Organic Compounds	5-17
5.2.3.1 Bis(2-ethylhexyl)phthalate or Di(2-ethylhexyl)phthalate	5-18
5.2.3.2 Dibenzofuran	5-18
5.2.3.3 1,2-Dichlorobenzene	5-19

5.2.3.4	1,3-Dichlorobenzene	5-19
5.2.3.5	1,4-Dichlorobenzene	5-20
5.2.3.6	Hexachlorobenzene	5-20
5.2.3.7	1,2,4-Trichlorobenzene	5-21
5.2.4	Polycyclic Aromatic Hydrocarbons	5-21
5.2.4.1	Carcinogenic Polycyclic Aromatic Hydrocarbons	5-22
5.2.4.2	Other Polycyclic Aromatic Hydrocarbon	
	Contaminants of Potential Concern	5-23
5.2.5	Pesticides	5-24
5.2.5.1	Aldrin	5-24
5.2.5.2	delta-Benzene Hexachloride (δ -BHC)	
	(delta-Hexachlorocyclohexane; delta-HCH)	5-24
5.2.5.3	Chlordane	5-25
5.2.5.4	DDT and Related Compounds – General	5-26
5.2.5.5	2,4'-DDE (o,p'-DDE)	5-26
5.2.5.6	4,4'-DDD (p,p'-DDD)	5-26
5.2.5.7	4,4'-DDE (p,p'-DDE)	5-27
5.2.5.8	4,4'-DDT (p,p'-DDT)	5-27
5.2.5.9	Dieldrin	5-27
5.2.5.10	Heptachlor Epoxide	5-28
5.2.6	Polychlorinated Biphenyls	5-28
5.2.6.1	Cancer Risk	5-29
5.2.6.2	Non-Cancer Toxicity	5-30
5.2.7	PCDD/PCDFs	5-30
6.	Risk Characterization	6-1
6.1	Cancer Risks	6-2
6.1.1	Methods	6-2
6.1.2	Quantitation of Cancer Risks by Pathway	6-2

6.1.3	Quantitation of Cancer Risks by Receptor	6-4
6.2	Non-Cancer Hazards	6-5
6.2.1	Methods	6-5
6.2.2	Quantitation of Non-Cancer Hazards by Pathway	6-6
6.2.3	Quantitation of Non-Cancer Hazards by Receptor	6-7
6.3	Summary of Risk Characterization	6-7
7.	Uncertainty Assessment	7-1
7.1	Uncertainties Associated with Selection of Substances	7-1
7.1.1	Selection of Analytical Parameters	7-1
7.1.1.1	Fish Fillets	7-2
7.1.1.2	Lake Sediments – Northern Basin	7-3
7.1.1.3	Lake Sediments – Southern Basin	7-3
7.1.1.4	Wetland Sediments (SYW-6, 10, 12, and 19)	7-3
7.1.1.5	Dredge Spoils Area Soils	7-4
7.1.1.6	Surface Water	7-5
7.1.2	Contaminant Screening	7-6
7.1.2.1	Uncertainty Associated with Screening Out Contaminants	7-6
7.1.2.2	Uncertainty Associated with Naturally Occurring Chemicals in Soil/Sediment	7-7
7.2	Uncertainties Associated with Exposure Point Concentrations	7-8
7.2.1	Uncertainties Related to Calculation Procedure for Exposure Point Concentrations	7-8
7.2.2	Uncertainties Related to Assigned Data Distribution Types and Resultant Upper Confidence Limit-Based Exposure Point Concentrations	7-9
7.2.3	Effect of Cooking Loss on PCB and PCDD/PCDF Concentrations in Fillets	7-10

7.3	Uncertainties Related to Fish Consumption Rates	7-10
7.3.1	Fish Consumption Rates for Adults	7-11
7.3.2	Fish Consumption Rates for Children	7-11
7.3.3	Subsistence Fishers	7-13
7.3.4	Fractional Intake	7-13
7.4	Uncertainties Associated with Exposure Assessment Assumptions ..	7-14
7.4.1	Dermal Absorption Pathway Assumptions	7-14
7.4.1.1	Soil-Skin Adherence Factor	7-15
7.4.1.2	Uncertainties Related to Dermal Absorption Factors ..	7-15
7.4.1.3	Route-to-Route Extrapolation	7-16
7.4.1.4	Dermal Absorption of Contaminants from Water	7-16
7.4.1.5	Skin Surface Area Available for Dermal Contact	7-17
7.4.1.6	Contaminants of Potential Concern Evaluated in the Dermal Pathway	7-17
7.4.2	Uncertainties Associated with Ingestion of Soil/Sediment and Surface Water	7-17
7.4.2.1	Soil/Sediment Ingestion Rates	7-17
7.4.2.2	Fraction Ingested from Contaminated Site	7-18
7.4.2.3	Surface Water Ingestion Rates	7-18
7.4.2.4	Duration of Swimming Events	7-19
7.4.3	Body Weight	7-19
7.4.4	Exposure Duration and Frequency	7-19
7.5	Uncertainties Related to Available Toxicity Data	7-22
7.5.1	Accuracy of Quantitative Cancer and Non-Cancer Toxicity Values	7-23
7.5.1.1	Polychlorinated Biphenyls	7-23
7.5.1.2	Methylmercury Non-Cancer Reference Dose	7-24
7.5.1.3	Dioxins and Furans	7-26

7.5.2	Lack of Quantitative Toxicity Values for Detected Chemicals	7-28
7.5.2.1	Use of Surrogate Toxicity Data for Polycyclic Aromatic Hydrocarbons	7-28
7.5.2.2	Surrogate Toxicity Data for Other Contaminants of Potential Concern	7-29
7.5.2.3	Contaminants of Potential Concern Without Quantitative Toxicity Data	7-30
7.5.3	Lack of Analytical Data on the Specific Form of Contaminants of Potential Concern	7-31
7.5.3.1	Mercury and Methylmercury	7-31
7.5.3.2	Arsenic	7-35
7.5.3.3	Chromium	7-36
7.5.3.4	Polychlorinated Biphenyls	7-36
8.	Conclusions	8-1
8.1	Exposure Pathways	8-1
8.2	Contaminant Screening and Identification of Contaminants of Potential Concern	8-2
8.3	Toxicity Assessment	8-2
8.4	Risk Characterization	8-3
8.4.1	Fish Ingestion	8-3
8.4.2	Sediment/Soil Pathways	8-3
8.4.3	Surface Water Pathway	8-4
8.4.4	Receptor-Specific Risks and Hazards Across Pathways	8-4
8.5	Uncertainty Assessment	8-4
8.6	Summary of Risks and Hazards Exceeding Target Levels	8-6
9.	References	9-1

Appendices (contents for appendices follow appendix cover page):

Appendix A. Summary of Site Data Used in the HHRA

Appendix B. RAGS Part D Tables

Appendix C. USEPA Regions 3 and 9 Screening Values

Appendix D. Comparison of ProUCL and Default Data Distributions for Calculation of Exposure Point Concentrations and Risks

Appendix E. Toxicity Profiles for Contaminants of Potential Concern *(Volume 2 of 2)*

List of Figures *(figures follow text of each chapter)*

- | | |
|------------|---|
| Figure 1-1 | Locations of Honeywell Former Facilities and Referenced Disposal Areas near Onondaga Lake |
| Figure 1-2 | Onondaga Lake and Vicinity |
| Figure 1-3 | Major Components of the Baseline Human Health Risk Assessment for Onondaga Lake |
| Figure 4-1 | Exposure Areas |
| Figure 4-2 | Dredge Spoils Area |
| Figure 4-3 | Eastern Shore Near Marina |
| Figure 4-4 | Southwest Area |

List of Tables *(tables follow text and figures of each chapter)*

Table ES-1	Summary of Contaminant Screening
Table ES-2	Selection of Exposure Pathways – Onondaga Lake Human Health Risk Assessment
Table ES-3	Summary of Cancer Risks and Non-Cancer Hazards
Table ES-4	Summary of COPCs Contributing to Cancer Risks
Table ES-5	Summary of COPCs Contributing to Non-Cancer Hazards
Table ES-6	Summary of Cancer Risks and Non-Cancer Hazards Exceeding Target Levels
Table 1-1	Summary of Data Sources Used in the Onondaga Lake Human Health Risk Assessment
Table 3-1	Summary of Contaminant Screening
Table 3-2	Summary of Lake Fish Samples (Fillets) Used in the HHRA
Table 4-1	Summary of Factors Used to Assess Dermal Exposures via Soil/Sediment and Surface Water
Table 5-1	WHO TEFs for Human Health Risk Assessment
Table 6-1	Summary of Cancer Risks and Non-Cancer Hazards
Table 6-2	Reasonable Maximum Exposure – Receptor-Specific Risk and Hazard Estimates
Table 6-3	Central Tendency Exposure – Receptor-Specific Risk and Hazard Estimates
Table 6-4	Summary of COPCs Contributing to Cancer Risks
Table 6-5	Summary of COPCs Contributing to Non-Cancer Hazards
Table 6-6	Summary of Target Organs for Non-Cancer Hazards
Table 7-1A	Comparison of Wetland SYW-6 EPC Calculations Based on Length-Weighted Average and Discrete Samples
Table 7-1B	Comparison of Risk and Hazard Calculations for Wetland SYW-6 Using Alternate Approaches
Table 7-2	Comparison of 2002 Wetland SYW-6 Sample W6-3 to Nearby Sample Data
Table 7-3	Age-Adjusted Fish and Shellfish Consumption, Based on USEPA Onondaga Lake (Region 2) Default Assumptions and Data from Rupp (1980), Javitz (1980), and Pao (1982)

Table 7-4	Comparison of Risk and Hazard Estimates Using Alternate Ingestion Rate Assumptions
Table 7-5	Comparison of Total Mercury and Methylmercury Concentrations in Onondaga Lake Deeper Sediments
Table 7-6A	Arsenic Speciation Data, Willamette River Composite Fish Samples
Table 7-6B	Arsenic Speciation Data, Columbia River Bi-State Program Fish Samples
Table 8-1	Summary of Cancer Risks and Non-Cancer Hazards Exceeding Target Levels
Table 8-2	Receptor-Specific Risk and Hazard Estimates Exceeding Target Levels

Acronyms and Abbreviations

ABS _{GI}	gastrointestinal absorbance
ACJ	Amended Consent Judgment
AF	adherence factor
ATSDR	Agency for Toxic Substances and Disease Registry
BEHP	bis(2-ethylhexyl)phthalate
BMD	benchmark dose
BMDL	benchmark dose limit
BTEX	benzene, toluene, ethylbenzene, and xylenes
CDC	Centers for Disease Control
CDI	chronic daily intake
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CLP	Contract Laboratory Program
COPC	contaminant of potential concern
CSF	carcinogenic slope factor
CSO	combined sewer overflow
CT	central tendency
DAF	dermal absorption factor
DDT	dichlorodiphenyl trichloroethane
DEHP	di(2-ethylhexyl)phthalate
DHHS	Department of Health and Human Services
EPC	exposure point concentration
FI	fraction ingested
FS	feasibility study
GI	gastrointestinal
GLSFATF	Great Lakes Sport Fish Advisory Task Force
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard index
HPV	health-protective values
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
LCP	Linden Chemicals and Plastics
LDC	Lakefront Development Corporation
LOAEL	lowest observed adverse effect level
LOEL	lowest observed effect level
LWA	length-weighted average
Metro	Metropolitan Syracuse Sewage Treatment Plant

mgd	million gallons per day
MOE	margin-of-exposure
MRL	minimal risk level
MVUE	minimum variance unbiased estimator
MWB	Metropolitan Water Board
NCEA	National Center for Environmental Assessment
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NOAA	National Oceanic and Atmospheric Administration
NOAEL	no observed adverse effect level
NPL	National Priorities List
NRC	National Research Council
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
NYSDDL	New York State Department of Law
OCPD	Onondaga County Parks Department
OCWA	Onondaga County Water Authority
ODH	Oregon Department of Health
ODHS	Oregon Department of Human Services
OEHHA	Office of Environmental Health Hazard Assessment [California]
OLMC	Onondaga Lake Management Conference
OLP	Onondaga Lake Partnership
OSWER	Office of Solid Waste and Emergency Response
OU	Operable Unit
PAH	polycyclic aromatic hydrocarbon
PBPK	physiologically based pharmacokinetic
PCB	polychlorinated biphenyl
PCDD/PCDF	polychlorinated dibenzo- <i>p</i> -dioxin and polychlorinated dibenzofuran
PRG	preliminary remediation goal
PSA	preliminary site assessment
PWG	Pathology Working Group
RAGS	Risk Assessment Guidance for Superfund
RBC	risk-based concentration
RDI	recommended daily intake
RfC	reference concentration
RfD	reference dose
RI	remedial investigation
RME	reasonable maximum exposure
ROD	Record of Decision
SA	surface area
SDW	Syracuse Department of Water
SQL	sample quantitation limit
SSAF	soil-to-skin adherence factor

SVOC	semivolatile organic compound
SYW	Syracuse West [USGS quadrant sheet]
TAL	Target Analyte List
TCB	trichlorobenzene
TCDD	tetrachlorodibenzo- <i>p</i> -dioxin
TCL	Target Compound List
TEC	toxic equivalent concentration
TEF	toxicity equivalence factor
TEQ	toxicity equivalence quotient
TIC	tentatively identified compound
UCL	upper confidence limit
UF	uncertainty factor
UFI	Upstate Freshwater Institute
USDA	United States Department of Agriculture
USDOI	United States Department of Interior
USEPA	United States Environmental Protection Agency
USFDA	United States Food and Drug Administration
USGS	United States Geological Survey
VOC	volatile organic compound
WDOH	Washington Department of Health
WHO	World Health Organization

Glossary

Cancer Slope Factor (CSF). A plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The CSF is used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular concentration of a specific potential carcinogen (cancer-causing chemical).

Deterministic Analysis (Deterministic Risk Assessment). Calculation and expression of health risks as a single numerical value or “single point” estimate of risk. In a deterministic risk assessment, uncertainty and variability are discussed separately in a qualitative or semi-quantitative manner.

Exposure Medium. The contaminated environmental medium to which an individual may be exposed, including the transfer of contaminants from one medium to another (e.g., volatilization of chemicals from water into air).

Exposure Pathway. The course (path) a contaminant takes from the source to the exposed individual. An exposure pathway analysis links the sources, locations, and types of environmental contamination with population locations and activity patterns (“receptors”) to determine the significant pathways of human exposure. As shown on RAGS Table 1 in Appendix B of this HHRA, an exposure pathway is defined as each unique combination of scenario time frame, medium, exposure medium, exposure point, receptor population, receptor age, and exposure route.

Exposure Point. An exact location of potential contact between a person and a contaminant within an exposure medium.

Exposure Point Concentration (EPC). The value (concentration) that represents an estimate of the contaminant concentration to which an individual may be exposed. For the Onondaga Lake HHRA, the exposure point concentration is either a statistically calculated estimate of the arithmetic mean (generally the 95 percent upper confidence limit [UCL] on the mean) or the maximum detected concentration.

Exposure Route. The manner in which a contaminant comes in contact with a person (receptor); i.e., ingestion, dermal contact, or inhalation.

Hazard Index (HI). The sum of more than one hazard quotient for multiple substances and/or multiple exposure pathways.

Hazard Quotient (HQ). The ratio of a single substance exposure level over a specified period of time to the reference dose (RfD) for the same substance over the same time.

Intake. A measure of exposure expressed as the mass of a contaminant in contact with the exchange medium per unit body weight per time; expressed as mg contaminant per kg body weight per day (mg/kg-day). For dermal pathways, it is the absorbed dose of the contaminant.

Medium. The environmental substance or matrix (fish, water, soil/sediment) that is a potential source of contaminants in the exposure medium. For the Onondaga Lake HHRA (but not necessarily for all risk assessments), the “medium” and the “exposure medium” are the same.

Reasonable Maximum Exposure (RME). The maximum exposure that is reasonably expected to occur at a site, calculated individually for each pathway. The RME is not a “worst case” estimate but combines mean or 50th percentile values for some parameters in the intake equation with higher-end estimates (90th or 95th percentile values) for a few of the more sensitive parameters in the intake equation.

Receptor Age. The description of the exposed individual. For this HHRA, the receptor ages assessed were young child (under six years old), older child (six to less than 18 years old) and adult (including construction worker; all individuals 18 or older).

Receptor Population. The exposed individual relative to the exposure pathway considered. Examples of receptor populations include recreational users, trespassers, construction workers, residents, and the like.

Reference Dose (RfD). An estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of harmful non-cancer health effects during a lifetime. Chronic RfDs (as used in this HHRA) are specifically developed to be protective for long-term exposure to a contaminant.

Scenario Time Frame. The time period (current or future, or both) considered for the exposure pathway.

Upper Confidence Limit (on the Mean) (UCL). A statistical estimate of the likelihood that the “true” mean or average does not exceed the estimate of the mean (average) concentration. For example, the 95 percent UCL on the mean, which is used extensively in this HHRA, is an estimate of the mean, calculated from site data, for which there is a 95 percent likelihood that the calculated value is equal to or greater than the true mean. Expressed another way, there is only a 5 percent chance that the true mean of the sampled medium exceeds the 95 percent UCL.

EXECUTIVE SUMMARY

This document presents the quantitative baseline human health risk assessment (HHRA) for the remedial investigation (RI) and feasibility study (FS) for Onondaga Lake, located in Onondaga County, New York. The objective of the HHRA is to evaluate the potential for adverse human health effects associated with current or potential future exposures to chemicals present in Onondaga Lake surface water, fish, and certain nearshore sediments, wetlands sediments, and dredge spoils area soils, in the absence of any action to control or mitigate those chemicals (i.e., under the no action alternative).

As defined in the Consent Decree, the site includes Onondaga Lake, its outlet, and tributaries that may have been directly affected by Honeywell operations. The tributaries directly affected by Honeywell include Ninemile Creek and its tributaries, Geddes Brook and the West Flume; Tributary 5A; the East Flume; and Harbor Brook (Chapter 1, Figure 1-1). As discussed below, these tributaries are not included in this HHRA since they are being covered as part of other investigations. The New York State Department of Environmental Conservation (NYSDEC) has also required that Wetlands SYW-6 and SYW-12 be included in the site (Chapter 4, Figure 4-1).

In addition to the areas of the site listed above, this HHRA includes an evaluation of limited data that were collected in Wetlands SYW-10 and SYW-19 and an upland area associated with the dredge spoils area located north of the mouth of Ninemile Creek (Chapter 4, Figure 4-1). Human health risks associated with Wetlands SYW-10 and SYW-19 and the dredge spoils area will be further evaluated as part of separate sites and, therefore, the risk analysis associated with these areas in this HHRA is considered preliminary, pending the finalization of the HHRA's associated with these other sites. Specifically, Wetland SYW-10 will be further evaluated as part of the RI/FS for the Geddes Brook/Ninemile Creek site; Wetland SYW-19 will be further evaluated as part of the RI/FS for the Wastebed B/Harbor Brook site; and the dredge spoils area will be further evaluated as a separate site with its own investigation.

The areas and media covered by this HHRA include Onondaga Lake fish, surface water, and sediments and shoreline areas directly abutting the lake – specifically, four New York State-regulated wetland areas (Wetlands SYW-6, 10, 12, and 19), and the dredge spoils area situated on the west side of the lake, north of Ninemile Creek between Wetlands SYW-6 and 10 (collectively, the “Onondaga Lake Study Area”). Other Honeywell sites in the vicinity of Onondaga Lake, including the Wastebed B/Harbor Brook site (including Harbor Brook and the East Flume), Willis Avenue Chlorobenzene site (including Tributary 5A), Willis Avenue Ballfield site, LCP Bridge Street site (including the West Flume), and Geddes Brook/Ninemile Creek site, are the subject of ongoing investigations and have been or will be addressed in separate reports. This HHRA addresses risk associated with contamination within the Onondaga Lake Study Area, without regard to the source of the contamination.

The HHRA, and this executive summary, follow the USEPA Risk Assessment Guidance format and sequence. The HHRA consists of the following chapters and appendices:

- Chapter 1, Introduction – Discusses the general framework and format of the document.
- Chapter 2, Background – Provides background information on the site, such as site history, features, and climate.
- Chapter 3, Identification of Contaminants of Potential Concern – Discusses the available data for all site media (e.g., fish tissue, sediment, water) for each exposure area (e.g., northern basin); discusses the results of the contaminant screening process, and identifies the contaminants that are considered contaminants of potential concern (COPCs) in each site medium after the screening.
- Chapter 4, Exposure Assessment – Presents the exposure setting and exposed populations (receptors); in other words, what types of people may be exposed to contaminants in various site media (e.g., adult construction workers exposed to subsurface contaminants in soil by dermal contact and incidental ingestion). Next, the exposure is quantified (estimates of how much of a contaminated medium to which each receptor may be exposed). Finally, the calculations of the exposure point concentrations (EPCs) of each COPC in each contaminated medium are discussed.
- Chapter 5, Toxicity Assessment – Discusses the chemical-specific cancer risk or non-cancer hazard toxicity data used to calculate the potential adverse health effects from exposure to site contaminants.
- Chapter 6, Risk Characterization – Presents the results of the quantitative risk assessment, including estimates of both cancer risks and non-cancer hazards for each medium and each receptor population.
- Chapter 7, Uncertainty Assessment – Discusses aspects of the HHRA that are likely to overestimate or underestimate site risks.
- Chapter 8, Conclusions.
- Chapter 9, References.
- Appendix A, Summary of Site Data Used in the HHRA – Includes discussion and tabulation of data collected by Honeywell and NYSDEC that are used in this HHRA.
- Appendix B, RAGS Part D Tables.

- Appendix C, USEPA Region 3 and Region 9 Screening Values.
- Appendix D, Comparison of ProUCL and Default Data Distributions for Calculation of Exposure Point Concentrations and Risks.
- Appendix E, Toxicological Profiles for Contaminants of Potential Concern.

1. Introduction

This HHRA was conducted in accordance with the Onondaga Lake RI/FS Work Plan (PTI, 1991) approved by the NYSDEC, as amended, and with applicable guidance documents (see Chapter 1, Section 1.1 for details) from the US Environmental Protection Agency (USEPA). As science and policy evolve over time, some of the guidance documents used in this HHRA were superseded or supplemented during the time this HHRA was being prepared. To the extent practical, the most current USEPA guidance documents and data have been utilized. For example, all of the USEPA screening values were updated in 2002 prior to performing the screening for this HHRA (Appendix C), and the toxicity files on USEPA's Integrated Risk Information System (IRIS) were all accessed in 2002 to verify that current peer-reviewed toxicity data were being used in the HHRA (Chapter 5 and Appendix E). The only known exception to this approach involves the format of the RAGS Part D tables presented in Appendix B. In June 2002, USEPA indicated that the December 2001 revision of RAGS Part D was to be used for all new risk assessments (superseding the January 1998 version of RAGS Part D). However, RAGS Part D is merely a standardized reporting format (utilized by USEPA to generate consistency among risk assessments at different sites and in different regions), and does not affect how risks are calculated. This risk assessment was initiated prior to issuance of the new guidance and, therefore, the 1998 version was used for this HHRA with USEPA's concurrence; however, not utilizing the December 2001 revision has no impact on the findings of this document.

Risk assessments conducted for regulatory purposes, such as this HHRA, are designed to be protective of human health and consistent with requirements for risk assessment provided by USEPA. Two different types of exposure scenarios are presented in this HHRA – the reasonable maximum exposure (RME) scenario, and the central tendency (CT; sometimes referred to as the “typical”) scenario. For the RME scenario, two or three of the most sensitive input parameters (typically the intake rate, such as the amount of fish consumed) are set to the 90th or 95th percentile values, while the rest of the inputs to the risk calculation are set to the average or median (50th percentile) value. As such, the RME is not a “worst case” scenario. Although the cumulative impact of the 95th percentile exposure and toxicity assumptions used in the RME scenario may overestimate risks for many site users (receptors), there could be some receptors for whom exposure and risks are underestimated even in the RME scenario.

For the CT scenario, all variables in the risk calculations are set to the average or median values. (The same toxicity values and EPCs are almost always used for both the RME and CT scenarios.) Factors that may overestimate or underestimate risks are discussed in Chapter 7, Uncertainty Assessment, in this HHRA.

2. Background

2.1 Site and Vicinity Description

Onondaga Lake is located in Onondaga County in central New York State, adjacent to the city of Syracuse. The lake covers an area of approximately 4.6 square miles (sq mi) (12 sq kilometers [km]), or 3,000 acres, and has a maximum length of 4.7 mi (7.5 km) and width of 1.2 mi (1.9 km), and has about 11.7 mi (18.8 km) of shoreline (based on PTI, 1991). As shown in Chapter 1, Figure 1-2, Onondaga Lake is divided into a northern basin and a southern basin; this division roughly corresponds to the surface water classifications of the lake and also to the exposure scenarios established for this HHRA (discussed further in Chapter 4).

The southern end of Onondaga Lake borders the city of Syracuse and is a heavily developed urban area. The town of Salina and the village of Liverpool border the north and northeast edges of the lake, respectively, while the town of Geddes and the village of Solway border the west and southwest edges, respectively. Onondaga Lake is encircled by major roadways: Interstate 90 (I-90) runs along the northwest tip of the lake, Interstate 690 (I-690) runs along the west and southwest edges of the lake, Interstate 81 (I-81) is near the southeast corner of the lake, and New York Route 370 runs along the north and northeast edges (see Chapter 4, Figure 4-1 of this HHRA). More than 75 percent of the shoreline of Onondaga Lake, most of which is classified as parkland, is owned by Onondaga County.

2.2 Climate

The Onondaga Lake region's climate is strongly influenced by its geographic proximity to Lake Ontario (Effler, 1996), which moderates air temperatures. Normal temperatures in the Syracuse area (measured at Hancock International Airport, about 3 mi [4.8 km] east-northeast of Onondaga Lake) range from 23.2°F (-4.9°C) in January to 71.1°F (21.7°C) in July (National Oceanic and Atmospheric Administration [NOAA], 2002). Based on data from the period from 1971 to 2000, the average first occurrence of 32°F (0°C) in the fall is November 15, and the average last occurrence of 32°F (0°C) in the spring is April 8 (NOAA, 2002). Precipitation falls an average of 171 days per year, and during the summer months (i.e., June through August), precipitation falls, on average, 10 to 11 days per month (NOAA, 1993).

2.3 Groundwater and Surface Water

Groundwater within the Onondaga Lake drainage basin generally flows into the tributaries and then flows from the tributaries to the lake, following the topography of the area. Onondaga Lake's water surface is at a lower static head than nearby wells; therefore, the lake does not recharge the aquifer under non-flood conditions (NYSDEC, 1989). Paths of groundwater flow and exchanges with surface water depend on local geologic conditions within each tributary basin. Local groundwater mounding (with groundwater elevations up to approximately 65 ft [20 m] higher than the surrounding water table) occurs under some of the Solway Wastebeds (Geraghty & Miller, 1982; Blasland & Bouck, 1989). Groundwater discharges to the lake contribute a significant but relatively minor amount of water to the total water budget. However,

as noted in the RI report (TAMS, 2002b), groundwater is a significant source of several COPCs to the lake, including mercury; benzene, toluene, ethylbenzene, and xylenes (BTEX); chlorinated benzenes; and polycyclic aromatic hydrocarbons (PAHs). The groundwater is classified as Class GA (defined as a source of drinking water [6 NYCRR Part 701.15]) by New York State regulations; however, some areas of groundwater in the vicinity of the lake may not currently be appropriate for use as sources of potable water due to natural occurrences of salinity (e.g., brine fields in the area) and contributions from anthropogenic salinity sources, including Honeywell. Contamination of the lake prevents the lake water from being used as a drinking water source.

The surface waters of the lake currently violate NYSDEC standards for clarity (turbidity) and bacterial contamination (coliform), but an Amended Consent Judgment (ACJ) is in place with Onondaga County under which the lake is to meet NYSDEC surface water criteria by 2012, as further discussed in Chapter 2, Section 2.5. As discussed in Chapter 2, Section 2.6, most of the northern two-thirds of the lake is classified as Class B water, and the southern third of the lake is Class C.

2.4 Population and Demographics

The population of Onondaga County grew to 469,000 in 1970; since that time, according to the most recent census data, it has decreased to about 458,000 (US Census Bureau, 2000). Most of the population currently is, and historically has been, concentrated in the Syracuse metropolitan area. This area includes Solway, Westvale, and Fairmount on the western side of the lake and Galeville, Liverpool, Mattydale, and North Syracuse on the eastern side of the lake (see Chapter 1, Figure 1-1 of this HHRA). Other population centers in the Onondaga Lake drainage basin are the villages of Camillus, Marcellus, Otisco, LaFayette, and East Syracuse, and the Onondaga Nation Territory.

More details on the demographics of the city of Syracuse and Onondaga County are provided in Chapter 4, Exposure Assessment.

2.5 Site History

Historically, Onondaga Lake has received loadings of industrial wastes and wastewater and sewage effluent. Sources of chemicals to Onondaga Lake include the following:

- Multiple industrial sources, including Honeywell.
- Sources related to the population in the area (e.g., sewage and landfills).
- Natural sources (e.g., inorganic constituents occurring naturally in soil or groundwater).

Honeywell's predecessor companies began manufacturing operations in Solway, New York, in the late 1800s. Operations occurred at three principal plants (Chapter 1, Figure 1-1): the Main Plant, which

manufactured soda ash and related products from 1884 until 1986 and benzene, toluene, and xylenes from 1917 to 1970; the Willis Avenue plant, which manufactured chlorinated benzenes and chlor-alkali products from 1918 until its closure in 1977; and the Bridge Street plant (sold to Linden Chemicals and Plastics [LCP] in 1979), which manufactured chlor-alkali products from 1953 until 1988. Two areas of the former Main Plant (i.e., the Petroleum Storage Area and the Chlorobenzene Hot Spot Area) are being investigated as part of the Willis Avenue site RI/FS.

The Bridge Street and Willis Avenue plants are sources of COPCs, including mercury, BTEX, chlorinated benzenes, PAHs, and polychlorinated biphenyls (PCBs). The Bridge Street plant discharged into the West Flume, which flows to Geddes Brook, which is a tributary to Ninemile Creek, which in turn is a tributary to the lake. A remedy has been selected by NYSDEC for the Bridge Street plant and a remedial design work plan, prepared by Honeywell, is under review.

Honeywell also disposed of Solvay wastes in numerous wastebeds covering more than 3.1 sq mi (8.1 sq km, or 2,000 acres), and disposed of organic wastes in the Semet Residue Ponds in Wastebed A; organic wastes have also been disposed of in Wastebed B near Harbor Brook. An RI/FS has been completed at the Semet Residue Ponds site and an RI/FS is underway at the Wastebed B/Harbor Brook and Willis Avenue Ballfield sites. In addition, Honeywell disposed of large quantities of combined Solvay wastes and mercury and organic wastes into the lake (e.g., via the East Flume; see RI Chapter 4, Section 4.5.1 [TAMS, 2002b]). Further discussion of these sources is provided in the RI report. A discussion of potential contaminant sources from non-Honeywell industrial sites in the area is also provided in the RI.

In the early 1800s, Onondaga Lake was receiving untreated industrial and domestic wastes. Around the turn of the twentieth century, a combined sewer system, a single system that transmits a combination of domestic and industrial flows as well as stormwater originating from various sources, was installed that discharged into tributaries and ultimately the lake.

The first primary sewage treatment facility in the Syracuse area was constructed in 1925 at the southern end of Onondaga Lake. An additional major treatment plant was built in 1940 on Ley Creek. During the 1950s, Onondaga County established a sewer district that encompassed the City of Syracuse and some surrounding suburban areas. A new primary treatment plant, the Onondaga County Metropolitan Syracuse Wastewater Treatment Plant (Metro), was constructed in 1960 with a 50 million gallons per day (mgd) design capacity (Onondaga Lake Management Conference [OLMC], 1993).

The Metro sewage treatment plant, which serves the city of Syracuse and several surrounding towns, is currently permitted (NY-0027081) to discharge an average of 80 mgd through its main outfall to Onondaga Lake. The plant provides tertiary treatment for flows up to 120 mgd. For combined stormwater and industrial/domestic sewage flow up to 220 mgd, the incremental flow above 120 mgd receives primary treatment and seasonal chlorination prior to discharge into the lake through a second outfall.

The sewers contain hydraulic relief structures otherwise known as combined sewer overflows (CSOs), which have historically allowed diluted sewage (due to the mixing of stormwater and sewage) to discharge

to several tributaries of Onondaga Lake during high flow events. In 1985, Phase I of a program to abate CSOs was implemented. The second phase of the CSO abatement program began in 1990. Additional abatement activities associated with the CSOs are underway as discussed below.

In January 1998, an ACJ (88-CV-0066) was executed by NYSDEC, the State Attorney General, Atlantic States Legal Foundation, and Onondaga County. The ACJ evolves from a 1989 Judgment on Consent (88-CV-0066) settling litigation between the State of New York and the county relating to state and federal water pollution control regulations.

The ACJ, which is designed to improve the water quality of Onondaga Lake, specifically includes a listing of over 30 projects to be undertaken by Onondaga County over a 15-year period. Although completion of the entire project is not required until 2012, many of these county projects are scheduled for completion by 2009 (Onondaga County Department of Water Environment Protection [OCDWEP], 2002) .

The projects may be grouped into three categories, including:

- Improvement and upgrading of the county's main sewage treatment plant (Metro).
- Eliminating and/or decreasing the effects of the CSOs on the lake and its tributaries.
- Performance of a lake and tributary monitoring program designed to evaluate the effects of the improvement projects on the water quality of the lake and its tributaries.

2.6 Previous and Ongoing Investigations of Related Sites

Other studies, both historical and ongoing, of sites near Onondaga Lake include:

- An RI has been completed for Honeywell's Semet Residue Ponds (O'Brien & Gere, 1991) site. This RI has been approved by NYSDEC. The RI report contained a risk assessment evaluating potential exposures from the residue ponds, groundwater, and ambient air (the studies of Tributary 5A sediment and surface water have now been incorporated into the Willis Avenue investigation). A Record of Decision (ROD) for the site has been finalized and was issued on March 28, 2002.
- An RI was prepared by NYSDEC for Honeywell's LCP Bridge Street site (Operable Unit [OU]-1) (NYSDEC/TAMS, 1998a). This RI also included a risk assessment of the facility and nearby tributaries (i.e., the West Flume). In addition, an RI is underway for OU-2 of the LCP Bridge Street site.

- The Willis Avenue RI report, including the HHRA for that site (O'Brien & Gere, 2002), is under review by NYSDEC.
- An RI and HHRA for Geddes Brook/Ninemile Creek were submitted by Honeywell in November 2001 and are being revised by NYSDEC.
- Honeywell has also commenced a preliminary site assessment (PSA) at the Mathews Avenue Landfill site and RI/FSSs, including HHRAs, at the Wastedbed B/Harbor Brook and Willis Avenue Ballfield sites near the southwestern corner of the lake (see Chapter 1, Figure 1-1). The East Flume (formerly investigated during the Willis Avenue RI) and the lower reach of Harbor Brook are now part of the Wastedbed B/Harbor Brook site.

3. Contaminants of Potential Concern

The HHRA uses a screening process to select COPCs that is structured to minimize the likelihood of eliminating contaminants from further analysis that could be of concern. All available contaminant concentration data were reviewed for lake fish (fillets only; limited to species likely to be consumed by humans), and for water and sediments in the northern and southern basins of the lake, for sediments in four adjacent wetlands, and for dredge spoils area soils. Lake sediments at water depths of more than about 6.5 ft (2 m) were not included, as it is unlikely that humans would have much, if any, direct contact with such sediments.

Site concentration data were compared with risk-based concentrations developed by USEPA Regions 3 and 9. For the screening, the highest concentration of a contaminant in a specific medium (e.g., southern basin sediments) was compared to the more conservative of the Region 3 or Region 9 screening criteria. The published screening criteria for carcinogens are set at a cancer risk level of 10^{-6} ; these criteria were used as published. However, USEPA Region 2 (along with many other risk assessors) utilizes a hazard index (HI) of 0.1 for screening non-cancer hazards; as the Region 3 and Region 9 screening criteria are based on a HI of 1.0, the published values were divided by 10 prior to use in screening non-carcinogenic effects for this risk assessment.

In addition to mercury (including methylmercury), which was identified in the RI/FS Work Plan (PTI, 1991) as one of the principal COPCs to be evaluated in this HHRA, a total of 60 other contaminants were identified as COPCs (as chemicals or chemical mixtures) in one or more site media and were retained for further analysis in the HHRA and are listed on Table ES-1.

4. Exposure Assessment

Onondaga Lake is surrounded by lands used for industrial, commercial, and recreational purposes. No residential property directly abuts the lake. Recreational visitors to Onondaga Lake are the receptors with the greatest potential for exposure to COPCs. Thus, this HHRA focuses mainly on recreational visitors to

the site, although it also evaluates potential exposures to construction workers who may contact contaminated media during work in these areas. Under current conditions, potential exposures for recreational visitors to the site are limited by the lack of public swimming areas. The New York State Department of Health (NYSDOH) has also issued specific, restrictive fish consumption advisories for Onondaga Lake. This HHRA, however, assesses risk in the absence of institutional controls under both current and future use scenarios. As a result, this baseline HHRA evaluates current and potential future uses under the assumption that there are no restrictions, advisories, or limitations in place. Human health risks associated with Wetlands SYW-10 and SYW-19 and the dredge spoils area will be further evaluated as part of separate sites and, therefore, the risk analysis associated with these areas in this HHRA is considered preliminary, pending the finalization of the HHRAs associated with these other sites. Exposure pathways quantitatively evaluated are shown on Table ES-2 and include the following:

- Consumption of fish from Onondaga Lake.
- Incidental ingestion of and dermal contact with COPCs in nearshore surface sediments in the northern and southern basins of the lake and surface sediments in the four wetlands that are of concern in this HHRA adjacent to the lake.
- Incidental ingestion of and dermal contact with COPCs in surface and subsurface soil in the dredge spoils area located along the shoreline of the lake north of Ninemile Creek.
- Incidental ingestion of and dermal contact with COPCs in Onondaga Lake surface water.

An initial PSA conducted for Onondaga Lake by NYSDEC (NYSDEC, 1989a, as cited in PTI, 1991) concluded that there was little potential for releases of contaminants to air. The data for volatile organic compounds (VOCs) in surface water and near-surface soils and sediments were reviewed as part of this HHRA, and the initial conclusion by NYSDEC is considered to still be appropriate for recreational users and nearby residents. In addition, there are currently no structures on the site nor are any likely to be built, due to regulatory restrictions (e.g., zoning and wetlands) and the nature of the area (e.g., much of the lake shoreline area is owned by or under the jurisdiction of the Onondaga County Parks Department [OCPD], and the wetlands areas are generally unsuitable for construction, even absent regulatory restrictions). Therefore, the inhalation pathway was considered to be incomplete for all media and was not assessed further in this report.

The RME and CT scenarios were evaluated for each of the complete pathways summarized above and listed in Table ES-2. Consumption of fish from the lake was determined to be the pathway with the highest potential for exposure to COPCs.

As site-specific information was not available for all the input parameters for exposure assessment or risk calculation, assumptions based on professional judgment or USEPA-recommended generic default values

were used in the exposure assessment. For example, the RME fish consumption rate of 25 grams per day (g/day) applied in the RME risk calculations is the default 95th percentile recommendation in the USEPA Exposure Factors Handbook (1997a). This fish consumption rate is equivalent to approximately 40 eight-ounce meals from Onondaga Lake per year. The uncertainties associated with the use of this fish consumption rate and other exposure assumptions are discussed in Chapter 7, Uncertainty Assessment.

5. Toxicity Assessment

Risk estimates for all COPCs were based on use of toxicity values, including carcinogenic slope factors (CSFs) to assess potential carcinogenic effects and reference doses (RfDs) to assess potential non-cancer effects, that were derived by USEPA and published on its peer-reviewed IRIS database and the USEPA Health Effects Assessment Summary Tables (HEAST), and were supplemented by additional guidance from USEPA National Center for Environmental Assessment (NCEA), USEPA Region 2, NYSDOH, and NYSDEC. The three COPCs (or COPC groups) responsible for a majority of estimated site risks are PCBs, polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/PCDFs), and methylmercury.

- PCBs – RME carcinogenic risk estimates for PCBs were based on the CSF of 2 (mg/kg-day)⁻¹, which is the highest of a range of upper-bound CSFs derived from studies in rats. This value is recommended by USEPA for evaluating food chain exposures, sediment or soil ingestion, and dermal contact exposures (if a dermal absorption factor is used) for all Aroclors (except for certain PCB mixtures with very low chlorine content; however, such mixtures were not detected in samples included in this HHRA). CT carcinogenic risk estimates for PCBs were based on the CSF of 1 (mg/kg-day)⁻¹, which is the central estimate CSF cited in IRIS. Non-cancer effects for PCBs were evaluated for the two groups of Aroclors – less chlorinated PCBs (using the toxicity data for Aroclor 1016) and highly chlorinated PCBs (using the toxicity data for Aroclor 1254) – for which USEPA has published RfDs.
- PCDD/PCDFs – Carcinogenic risk estimates for PCDD/PCDFs were based on a toxicity equivalent (TEQ) approach. USEPA does not currently have any quantitative toxicity factors (e.g., oral RfD) for the non-cancer health effects of PCDD/PCDFs; therefore, no quantitative assessment of non-cancer health hazards associated with PCDD/PCDFs is provided in this HHRA. However, a qualitative assessment is provided in Chapter 7, Uncertainty Assessment, along with alternate cancer risks estimates based on the current peer-review draft of USEPA's dioxin reassessment document.
- Methylmercury – USEPA's RfD for methylmercury of 0.0001 mg/kg-day has been applied in estimates of non-cancer hazards for the fish consumption pathway and for Onondaga Lake surface water and sediments in which methylmercury was

detected and for the fraction of total mercury in the wetlands sediments that is assumed to be methylmercury. The USEPA RfD of 0.0003 mg/kg-day has been applied for evaluation of non-cancer hazards of mercury (as inorganic mercury) in other media. Methylmercury/mercury has not been assessed quantitatively for cancer risks in this HHRA as no oral CSFs have been established by USEPA.

6. Risk Characterization

USEPA toxicity values (i.e., CSFs or RfDs) were combined with exposure estimates to derive estimates of potential health risks related to exposure to COPCs in Onondaga Lake media. Cancer risk estimates were compared to a target risk range of 1×10^{-4} to 1×10^{-6} . A 1×10^{-6} excess cancer risk represents an additional one-in-one-million probability that an individual may develop cancer over a 70-year lifetime as a result of the exposure conditions evaluated. Non-cancer effects are expressed as the ratio of the estimated exposure, or intake rate over a specified exposure period, to the RfD derived for a similar exposure period. This ratio is termed a hazard quotient (HQ). HQs for multiple COPCs or pathways are summed to generate an HI for a specific exposure route or receptor. Exposures resulting in an HI less than or equal to 1.0 are unlikely to result in non-cancer health effects. Estimated cancer risks and non-cancer hazards for both RME and CT scenarios for the 31 pathways evaluated in this HHRA are summarized in Table ES-3.

6.1 Cancer Risks

The RME cancer risks for fish ingestion ranged from 2.4×10^{-4} for young children to 7.8×10^{-4} for adults, all of which exceed the upper end of the target risk range (1×10^{-4}). RME cancer risks for older child exposure to Wetland SYW-6 sediments also exceed 1×10^{-4} . With these exceptions, the cancer risk estimates for the other exposure pathways and scenarios, both RME and CT (including the CT scenario for fish ingestion), were less than 1×10^{-4} , although cancer risk estimates exceeded 10^{-6} for many pathways, as summarized on Tables ES-3 and ES-6. The CT cancer risk for the fish consumption pathway scenario for all recreational anglers (adults and children) is about 4.5×10^{-5} , and RME risks to at least one receptor for each of the sediment and dredge spoils exposure areas exceeded 10^{-6} . Cancer risks associated with the fish ingestion pathways were due primarily to exposure to PCBs, PCDD/PCDFs, and, to a lesser extent, arsenic, as shown on Table ES-4.

RME cancer risk estimates associated with several other exposure pathways related to lake sediments and wetland sediments in recreational scenarios were greater than 1×10^{-6} . The highest of these was about 2.6×10^{-4} for older child exposure to Wetland SYW-6 sediments, followed by risks greater than 10^{-5} for one or more recreational receptor's exposure (ingestion and dermal combined) to southern basin sediments and Wetlands SYW-6, 10, 12, and 19 (see Table ES-6). In CT scenarios, the highest excess cancer risk (other than fish consumption) was about 1.4×10^{-5} for the older child recreational exposure to Wetland SYW-6 sediments. All RME and CT risks associated with Onondaga Lake surface water pathways were below the 1×10^{-6} risk level.

6.2 Non-Cancer Hazards

The RME HI for the recreational angler fish consumption pathway was approximately 18 for adults, 28 for young children, and 20 for older children. CT HIs ranged from approximately 4.5 for adults to 7.0 for young children. The elevated HIs for the fish consumption pathways were primarily related to PCBs (low and high molecular weight, assessed as Aroclors 1016 and 1254), methylmercury, and, to a lesser extent, arsenic. The COPCs contributing the largest amount of non-cancer hazard for each pathway are summarized on Table ES-5, and include PCBs and methylmercury. All other HIs for pathways other than fish ingestion were less than 1.0, although the cumulative RME HI for an older child who frequently accessed all the contaminated areas (all four wetlands, northern and southern basin sediments, and the dredge spoils area) closely approaches 1.0. However, as discussed in Chapter 7, Uncertainty Assessment, it is not considered likely that an individual would be exposed to all the contaminated media sites at the RME frequency.

The risks to children for the fish consumption pathway (presented above) are based on the assumption that older children consume two-thirds as much fish as adults, and young children (under age six) consume one-third as much fish as adults. As there are only limited data on which this assumption of children's fish ingestion rates could be based, it is possible that the ingestion rates for children may be higher or lower than those used in this HHRA; therefore, risks to children may be higher or lower than those presented in this HHRA and shown on Table ES-3.

Based on the exposure assumptions and toxicity values used in the risk evaluations, these results indicate the potential for adverse non-cancer health effects as a result of long-term exposures via ingestion of lake fish. This conclusion is consistent with the fact that PCB and methylmercury concentrations for some lake fish exceed US Food and Drug Administration (US FDA) action limits.

7. Uncertainty Assessment

As described above, the USEPA risk assessment methodology used in this HHRA is designed to be protective of human health. Thus, site risks may be less than the risks estimated using standard risk assessment methods for most, though not necessarily all, receptors. Several key factors in the risk assessment methods used are likely to result in some overestimates or underestimates of potential risks for most visitors to Onondaga Lake. These include, but are not limited to, the following:

- Application of an assumed RME fish consumption rate of 25 g/day, which is USEPA's default 95th percentile consumption rate and was derived from three key studies. Individual studies have suggested RME fish consumption rates both higher (e.g., up to 170 g/day for subsistence fishers; 32 g/day for Hudson River anglers) and lower (e.g., less than 25 g/day) than the RME fish consumption rate used in this HHRA. In addition, the consumption rate utilized is derived from studies on adults; only limited data were available for estimating fish consumption by children (see Chapter 4, Section 4.2.1 and Chapter 7, Section 7.3.2).

- The assumptions that all freshwater fish consumed come from Onondaga Lake (i.e., application of a fractional intake of one) and that no PCBs or PCDD/PCDFs are lost during cooking. These assumptions may overestimate risk to some receptors. However, as Onondaga Lake is a highly desirable fishing location, and it is not known to what extent persons who consume Onondaga Lake fish adhere to the NYSDOH recommendations to remove the skin and fat and not consume the drippings, it is likely that these assumptions are realistic for at least some of the potentially exposed recreational angler population.
- There is some uncertainty in the USEPA CSF of $2 \text{ (mg/kg-day)}^{-1}$ for PCBs; however, as discussed in Chapter 7, it is not clear whether the uncertainty may lead to an underestimate or overestimate of cancer risks associated with PCBs.
- The application of toxicity values for PCDD/PCDFs that are currently being reassessed by USEPA may underestimate cancer risks from these compounds, if the conclusions of the preliminary reassessment are unchanged after peer-review and finalization of the reassessment.
- The lack of published non-cancer toxicity values for PCDD/PCDFs may underestimate non-cancer hazards from dioxins. The reassessment currently being conducted by USEPA suggests that there are likely non-cancer hazards from these compounds, in addition to cancer risks. Therefore, the absence of non-cancer toxicity values for PCDD/PCDFs precluded their inclusion in the quantitative HHRA and may result in an underestimate of non-cancer hazards in media in which these COPCs are present.
- The lack of peer-reviewed cancer and non-cancer toxicity values for some of the PAH compounds detected in Onondaga Lake sediments, wetlands, and dredge spoils may result in a slight underestimation of risks or hazards.

Derivation of appropriate and protective toxicity values for mercury/methylmercury, PCDD/PCDFs, and PCBs in human populations is the subject of extensive study and debate. The toxicity values derived by USEPA and used in this HHRA represent a protective interpretation of the available toxicological data, and incorporate uncertainty and modifying factors to account for the need to extrapolate from animal studies to humans, among other issues. Chapter 7, Uncertainty Assessment, provides a discussion of the basis for and the reliability of the toxicity values used in this risk assessment. In general, confidence in the methylmercury toxicity data is considered high, and the IRIS value has recently been confirmed by a study conducted by the National Research Council (NRC).

The CSF used for PCDD/PCDFs was published in USEPA's HEAST a number of years ago, and is currently under reassessment. The preliminary dioxin reassessment, which has not yet been peer-reviewed or finalized, suggests that the CSF used in this HHRA may underestimate cancer risks from PCDD/PCDFs.

The potential magnitude of this is discussed quantitatively in Chapter 7. In addition, the preliminary dioxin reassessment also suggests that there are non-cancer toxicity effects from PCDD/PCDFs; the published (final) toxicity data available for this HHRA do not include a means of assessing non-cancer toxicity of dioxins and as such may underestimate non-cancer hazards to receptors in media in which dioxins are present (also discussed in Chapter 7).

Although the CSFs and RfDs used for quantitative assessment of PCBs were taken from USEPA's peer-reviewed IRIS database, there is more uncertainty about the PCB toxicity data than for the methylmercury data. (For example, USEPA characterizes the confidence in the oral RfDs for Aroclors 1016 and 1254 as medium, while the confidence in the oral RfD for methylmercury is high.) A number of factors contribute to the relative uncertainty of the PCB toxicological data, including the fact that Aroclors are a mixture of many (typically 30 or more) individual chlorinated biphenyl compounds ("congeners"); the commercial mixtures studied in the laboratory are altered when released to the environment by physical, biological, and metabolic processes; there are a wide range of observed effects and concentrations at which effects were observed in laboratory studies; as well as the issues associated with most chemicals in extrapolating toxicological data from animal studies.

Although there are incidences of human exposure to PCBs, data from human exposure are only useful on a qualitative basis due to lack of information about the specific composition of the mixture to which persons were exposed, exposure concentrations, and route of exposure, as well as a lack of long-term monitoring data in a number of these cases. Recent studies also suggest that some PCB congeners have dioxin-like effects and may contribute to PCDD/PCDF-related health effects; however, the lack of PCB congener-specific data precluded any assessment of this possibility.

8. Conclusions

The objective of this HHRA was to evaluate the potential for adverse human health effects associated with current or potential future exposures to chemicals present in Onondaga Lake surface water, sediments, fish, certain portions of the adjacent wetlands, and the dredge spoils area in the absence of any action to control or mitigate those chemicals. Under this "no remedial action" scenario, the HHRA principally focused on future lake conditions that further assumed unrestricted recreational use of the lake and the absence of a specific, restrictive fish consumption advisory. A total of 60 COPCs or groups of COPCs (including mercury and methylmercury) were identified for further analysis in the HHRA. Consistent with USEPA guidance, RME and CT scenarios for these COPCs were evaluated for several pathways, including a recreational fish consumption pathway, as summarized below:

- Cancer risks and non-cancer hazards calculated for the consumption of Onondaga Lake fish exceeded the upper end of the target risk levels (Table ES-6), as follows:

- The calculated RME cancer risks (ranging from 2.4×10^{-4} to 7.8×10^{-4}) exceeded the high end of the target risk range (10^{-4}), and exceeded the low end of the target cancer risk (10^{-6}) by more than two orders-of-magnitude. The CT fish ingestion cancer risk (about 4.5×10^{-5} for all recreational receptors) was below the upper end of the target range.
- The RME non-cancer HIs (ranging from about 18 to 28) exceeded the target HI (1.0) by a factor of almost 20 or more. The calculated CT non-cancer HIs (4.5 to 7 for adults and children) also exceeded the target.
- RME cancer risks for 21 of the 28 pathways other than fish ingestion equaled or exceeded the low end of the target risk range of 1×10^{-6} , with the highest of these being about 2.6×10^{-4} for older child exposure to Wetland SYW-6 sediments.
- For the CT cancer risk calculations, the low end of the 10^{-6} target range was equaled or exceeded in 8 of the 28 pathways other than fish ingestion, with a maximum CT risk of about 1.4×10^{-5} for older child exposure to Wetland SYW-6 sediments.
- None of the calculated non-cancer hazards (for both RME and CT scenarios) associated with pathways other than fish ingestion exceeded the target threshold of 1.0. The highest RME hazard other than fish ingestion was about 0.54 for young child exposure to southern basin sediments. The calculated non-cancer CT hazards for all pathways other than fish ingestion were all less than 0.1.

Cumulative risks and hazards were calculated for receptors who may be exposed to COPCs in multiple site media—for example, eating contaminated fish and being exposed to contaminated sediments. The receptors evaluated were adult recreators, young child recreators, older child recreators, and construction workers. For all cumulative risk and hazard calculations including fish ingestion, the cumulative risk or hazard was essentially the same as that associated with the fish ingestion pathway alone. Therefore, to assess the cumulative risks associated with pathways other than fish ingestion (i.e., exposure to lake sediment, wetlands sediment, dredge spoils soil, and lake surface water), the cumulative risk for each receptor was also calculated excluding the fish ingestion pathway, as summarized below:

- Cumulative RME cancer risks for adults (excluding fish ingestion) were calculated as 1×10^{-4} .
- Cumulative RME cancer risks (excluding fish ingestion) were calculated as about 3.5×10^{-5} for young children. In addition, the receptor-specific RME HI was calculated as about 0.8 for the young child.

- Cumulative RME cancer risks (excluding fish ingestion) were calculated as about 3.8×10^{-4} for older children. In addition, the calculated value of the cumulative RME HI, excluding fish ingestion, was 0.98 for the older child recreator.
- Cumulative RME cancer risks (excluding fish ingestion) were calculated as 2×10^{-5} for construction workers. In addition, the receptor-specific RME HI was calculated as about 0.8 for construction workers.

It should be noted that these cumulative estimates are probably unrealistically high, especially for the adult and older child recreational receptors, as the cumulative risk calculation assumes RME exposure frequencies summing to 218 days per year to Onondaga Lake sediments, wetlands, and dredge spoils. Cumulative RME HIs calculated in the same manner (excluding fish ingestion) generally did not exceed 1.0, although some approached 1.0, as indicated above.

Chapter 7, Uncertainty Assessment, of this HHRA provides a discussion of the reliability of the input parameters to the quantitative risk calculations, and provides a qualitative and, in some cases, semi-quantitative assessment of the effect of alternative values in risk calculations. As indicated there, actual cancer risks and non-cancer hazards may vary from those presented in the quantitative risk characterization tables.

1. INTRODUCTION

The objective of the Onondaga Lake baseline human health risk assessment (HHRA) is to quantify human health risks associated with contaminants of potential concern (COPCs) in Onondaga Lake and related areas (wetlands and the dredge spoils area) in the absence of any remedial action (i.e., under the no-action alternative). This HHRA focuses on Onondaga Lake (water, sediments, and fish) and select shoreline areas directly abutting the lake – specifically, four New York State-regulated wetland areas (Wetlands SYW-6, SYW-10, SYW-12, and SYW-19) and the dredge spoils area on the west side of the lake, north of Ninemile Creek between Wetlands SYW-6 and SYW-10.

A draft HHRA report for Onondaga Lake was submitted by Honeywell in May 1998. NYSDEC disapproved this draft document and provided comments to Honeywell in October 1998. After completing additional sampling in 1999 and 2000, Honeywell submitted a revised draft HHRA report in March 2001. This revised report was disapproved by NYSDEC in July 2001. The reasons for disapproval are outlined in the accompanying determination. This HHRA is the NYSDEC/TAMS Consultants, Inc. (TAMS) rewrite of Honeywell's revised draft HHRA report.

Information in this HHRA regarding site history, including historical sources of contamination, is summarized from the accompanying Onondaga Lake Remedial Investigation (RI) report (TAMS, 2002b). NYSDEC/TAMS obtained the information in the RI report (and this HHRA report) from, among other sources, reports and materials prepared by Honeywell and its consultants. While the accuracy of the information provided by Honeywell and its consultants is accepted for purposes of the several reports, it must be noted that pursuant to paragraph 68 of the Consent Decree, discovery in the underlying litigation has been stayed. Consequently, the information furnished by Honeywell and its consultants, as well as information provided by third-party sources, has not been verified through the formal discovery process. The State reserves the right, consistent with and without limitation to its rights under paragraphs 33 and 34 of the Consent Decree and under state and federal law, to correct or amend any information in the RI and risk assessment reports if, without limitation: (a) discovery is conducted, and (b) that discovery reveals information supporting such correction or amendment.

As defined in the Consent Decree, the site includes Onondaga Lake, its outlet, and tributaries that may have been affected by Honeywell operations. The tributaries directly affected by Honeywell are Ninemile Creek and its tributaries, Geddes Brook and the West Flume; Tributary 5A; the East Flume; and Harbor Brook (Figures 1-1 and 1-2). As discussed below, these tributaries are not included in this HHRA since they are being covered as part of other investigations. NYSDEC has also required that Wetlands SYW-6 and SYW-12 be included in the site. Risks associated with Wetlands SYW-10 and SYW-19 and the dredge spoils area will be further evaluated as part of separate sites and therefore the risk analysis associated with these areas in this HHRA is considered preliminary, pending the finalization of the HHRA's associated with these other sites. Specifically, Wetland SYW-10 will be further evaluated as part of the RI/Feasibility Study (FS) for the Geddes Brook/Ninemile Creek site; Wetland SYW-19 will be further evaluated as part of the RI/FS for the Wastebed B/Harbor Brook site; and the dredge spoils area will be further evaluated as a

separate site with its own investigation. This quantitative Onondaga Lake HHRA utilizes the sample data collected to date in these areas.

As this HHRA is focused on Onondaga Lake and its shoreline, the tributaries directly affected by Honeywell are not included in this HHRA since they are being covered as part of other investigations, as noted below:

- Limited sampling was conducted in the West Flume as part of the RI for Onondaga Lake. The West Flume has been investigated further as part of a separate RI for the Honeywell LCP Bridge Street site. Potential human health risks related to exposures to COPCs in the West Flume were evaluated in the RI and HHRA for the LCP Bridge Street facility (NYSDEC/TAMS, 1998a).
- Limited sampling was conducted in Geddes Brook and Ninemile Creek as part of the RI for Onondaga Lake. These tributaries have been investigated further as part of a separate RI for Geddes Brook and Ninemile Creek. The Geddes Brook/Ninemile Creek work plan was submitted by Honeywell in April 1998, and rewritten by NYSDEC. The first phase of sampling was completed in October 1998. The draft HHRA was submitted to NYSDEC in July 2000 (Exponent, 2000a). Based on NYSDEC comments, additional data were collected in 2001 for sediments and floodplain soils in Geddes Brook and Ninemile Creek. The revised draft RI report and risk assessments were submitted to NYSDEC in November 2001, rejected on February 15, 2002, and are currently being rewritten by NYSDEC. Additional floodplain soils sampling was performed by Honeywell in Ninemile Creek in 2002. NYSDEC is scheduled to complete the rewrites by July 31, 2003.
- Risks associated with Harbor Brook and the East Flume are being addressed under a separate investigation for the Wastebed B/Harbor Brook site.
- Risks associated with Tributary 5A are being addressed under a separate investigation for the Willis Avenue site.

This HHRA was conducted in accordance with the Onondaga Lake RI Work Plan (PTI, 1991), approved by NYSDEC, as amended, and the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) and other applicable guidance documents from the US Environmental Protection Agency (USEPA) (see Section 1.1). This HHRA addresses the key aspects of the human health risk assessment task as specified in the Onondaga Lake RI/FS Work Plan (PTI, 1991). The specifics of implementation are not identical to those outlined in the work plan due to subsequent changes in regulations and guidance documents, the acquisition of additional data, and advancements in analytical and risk assessment science.

This HHRA was conducted independently of the public health assessment for Onondaga Lake that was prepared by the New York State Department of Health (NYSDOH) and the Agency for Toxic Substances and Disease Registry (ATSDR) (NYSDOH and ATSDR, 1995). All available sources of data were reviewed; however, data collected by Honeywell during Phase 1 (1992) and Phase 2A (1999 and 2000) of the RI and fish data collected by NYSDEC from 1992 to 2000 were the primary data used in the HHRA. Table 1-1 summarizes the data used in this HHRA.

This HHRA was conducted under USEPA's Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) of 1980 guidance and protocols and, as such, only quantifies excess (incremental) risk associated with this site. It does not quantify risks associated with other Honeywell Onondaga Lake-related sites nor does it include evaluation of risks associated with non-Honeywell contaminant sources at other upland locations. Therefore, the total risk to individuals resulting from all these sources is likely higher than the risks associated with the Onondaga Lake site, as quantified in this report. Because this HHRA represents baseline conditions, it does not reflect reduced risk associated with any future remedial actions.

1.1 Guidance Documents

The following sources of guidance were used for the HHRA:

- Risk Assessment Guidance for Superfund (RAGS): Volume 1. Human Health Evaluation Manual (Parts A, B, and D) (USEPA, 1989, 1991a,b, 1998).
- Supplemental Guidance to RAGS: Calculating the Concentration Term (USEPA, 1992a).
- Risk Assessment Guidance for Superfund (RAGS): Volume 1. Human Health Evaluation Manual (Part E), Supplemental Guidance for Dermal Risk Assessment (USEPA, 2001a).
- Supplemental Guidance to RAGS: Standard Default Exposure Factors (USEPA, 1991b) and Exposure Factors Handbook (USEPA, 1997a).
- USEPA Region 3 risk-based concentration (RBC) table, April 2002 update (USEPA, 2002a).
- USEPA Region 9 preliminary remediation goals (PRGs) table, October 2002 Update (USEPA, 2002b).
- Guidance for Data Usability in Risk Assessment (USEPA, 1990).

- USEPA Office of Solid Waste and Emergency Response directives (USEPA, 1991c, 1992b) on characterizing risks and uncertainties in quantitative risk assessments.

Other guidance documents utilized for specific aspects of this HHRA are identified where referenced in the text and included in Chapter 9, References.

1.2 Organization

Chapter 2 of this HHRA contains a brief summary of site background information that includes site history, climate, land use, and demographics. Additional site background information is provided in the accompanying RI report (TAMS, 2002b). The subsequent chapters of this HHRA describe the results of the four steps recommended in USEPA guidance for risk assessment, as shown in Figure 1-3: identification of COPCs (Chapter 3), exposure assessment (Chapter 4), toxicity assessment (Chapter 5), and risk characterization (Chapter 6). An uncertainty assessment, which discusses HHRA assumptions and other factors that may over- or underestimate potential site risks, is provided as Chapter 7. The risk assessment results are summarized in Chapter 8, and references for the entire document are provided in Chapter 9. All of the data used in the analyses and detailed discussions of technical issues in the HHRA are provided in the following appendices to the HHRA:

- Appendix A, Summary of Site Data Used in the HHRA, presents the analytical results for recent and historical samples from the Onondaga Lake system used in this HHRA.
- Appendix B, RAGS Part D Tables, presents all the tables (Tables 1 through 10) required by RAGS Part D (USEPA, 1998).
- Appendix C, USEPA Region 3 and Region 9 Screening Values, provides tables of USEPA-derived RBCs used in the selection of COPCs, as well as information from the regions explaining the derivation and use of these criteria.
- Appendix D, Comparison of ProUCL and Default Data Distributions for Calculation of Exposure Point Concentrations and Risks, presents a comparison of exposure point concentrations (EPCs) and risks calculated using the default data distribution assumptions (USEPA, 1989) and the recent USEPA statistical software ProUCL 2.1 (USEPA, 2002c).
- Appendix E, Toxicity Profiles for Contaminants of Potential Concern, includes the available USEPA Integrated Risk Information System (IRIS) files for the COPCs assessed in this HHRA, as well as additional files from the ATSDR and the USEPA National Center for Environmental Assessment (NCEA).

2. BACKGROUND

Information about the Onondaga Lake site and its vicinity, climate, groundwater and surface water, population and demographics, history, and the site's potential use and exposures is provided below.

2.1 Site and Vicinity Description

Onondaga Lake is located adjacent to the city of Syracuse in Onondaga County in central New York State. The lake covers an area of approximately 4.6 square miles (sq mi) (12 sq kilometers [km]), or 3,000 acres, and has a maximum length of 4.7 mi (7.5 km) and width of 1.2 mi (1.9 km), and has about 11.7 mi (18.8 km) of shoreline (based on PTI, 1991). The southern end of Onondaga Lake borders the city of Syracuse and is a heavily developed urban area. The town of Salina and the village of Liverpool border the north and northeast edges of the lake, while the town of Geddes and the village of Solvay border the west and southwest edges, respectively. Onondaga Lake is encircled by major roadways: Interstate 90 (I-90) runs along the northwest tip of the lake, Interstate 690 (I-690) runs along the west and southwest edges of the lake, Interstate 81 (I-81) is near the southeast corner of the lake, and New York Route 370 runs along the north and northeast edges (see Chapter 4, Figure 4-1 of this HHRA). More than 75 percent of the shoreline of Onondaga Lake, most of which is classified as parkland, is owned by Onondaga County. As shown in Chapter 1, Figure 1-2 and Chapter 4, Figure 4-1, Onondaga Lake is divided into a northern basin and a southern basin; this division roughly corresponds to the surface water classifications of the lake and also to the exposure scenarios established for this HHRA (discussed further in Chapter 4).

Onondaga Lake is surrounded by industrial, commercial, and recreational areas. Most of the northern half of the shoreline is parkland. Commercial and industrial areas near Onondaga Lake are concentrated around the southern end, in the Syracuse metropolitan area. Residential areas are located along the northeast and west lakeshores, although no residential property directly abuts the lake. A dredge spoils area is located on the western side of the lake just north of the mouth of Ninemile Creek. While I-690 and a railroad line separate the residential area of the village of Lakeland from the dredge spoils area and the lake, a pedestrian bridge over I-690 and the railroad line provides access to the dredge spoils area and the lake in this area (see Chapter 4, Figures 4-1 and 4-2).

There are four state-regulated wetland areas adjacent to the lake on the southern, western, and northwestern edges of the lake. Wetlands SYW-6 and SYW-12 are part of the Onondaga Lake site and are included in this HHRA. Wetlands SYW-10 and SYW-19 and the dredge spoils area, while not considered part of the site, are included in this HHRA based on data collected during the Phase 2A Onondaga Lake investigation in 2000; however, these areas are being further evaluated as part of other investigations (see Chapter 1) and, therefore, risk estimates associated with these areas may change.

2.2 Climate

The climate in the Onondaga Lake area is strongly influenced by its geographic proximity to Lake Ontario (Effler, 1996), which moderates air temperatures. The average monthly temperatures in the

Syracuse region (measured at Hancock International Airport, about 3 mi (4.8 km) east-northeast of Onondaga Lake) range from 23.2°F (-4.6°C) in January to 71.1°F (21.7°C) in July (National Oceanic and Atmospheric Administration [NOAA], 2002). Based on data from the period from 1971 to 2000, the average first occurrence of freezing temperatures (daily low of 32°F [0°C]) in the fall is November 15, and the average last occurrence of freezing temperatures in the spring is April 8 (NOAA, 2002). Precipitation falls an average of 171 days per year, and during the summer months (i.e., June through August), precipitation falls, on average, 10 to 11 days per month (NOAA, 1993).

2.3 Groundwater and Surface Water

Onondaga Lake receives surface water runoff from a drainage basin estimated to cover 248 sq mi (642 sq km) (Effler, 1996). Surface water flows into the lake from Ninemile Creek, Onondaga Creek, Ley Creek, Harbor Brook, Tributary 5A, the East Flume, Bloody Brook, and Sawmill Creek (Chapter 1, Figure 1-1). Water is also added to the lake by the Metropolitan Syracuse Sewage Treatment Plant (Metro), which is located in the southeast corner of the lake, and through intermittent bidirectional flow from the Seneca River at the outlet of the lake (Effler and Driscoll, 1986, as cited in Onondaga Lake Management Conference [OLMC], 1989).

A small amount of water enters the lake through the East Flume, an excavated drainage ditch that receives stormwater from the village of Solvay and process water from General Chemical Corp. and Salt City Energy Venture, L.P. The East Flume historically served as a major point of discharge for Honeywell wastes and wastewater, and now conveys groundwater and runoff from Honeywell properties. As indicated in Chapter 1, the East Flume is being evaluated as part of a separate RI/FS for the Wastebed B/Harbor Brook site.

Ninemile Creek and Onondaga Creek account for most of the inflow to the lake, together comprising approximately 65 percent of the total inflow for the period from 1976 to 1989. During the same period, Metro and Ley Creek accounted for nearly 25 and 10 percent of the total inflow, respectively. Contributions from all other tributaries were less significant. In 1992, when most of the RI field investigations were conducted, Ninemile Creek and Onondaga Creek accounted for 30 and 31 percent of total inflow, respectively, while Metro and Ley Creek accounted for 18 and 7.8 percent, respectively (NYSDEC/TAMS, 1998b).

Groundwater within the Onondaga Lake drainage basin generally flows into the tributaries and then from the tributaries to the lake, following the topography of the area. Onondaga Lake's water surface is at a lower static head than nearby wells; therefore, the lake does not recharge the aquifer under non-flood conditions (NYSDEC, 1989). Paths of groundwater flow and exchanges with surface water depend on local geologic conditions within each tributary basin. Local groundwater mounding (with groundwater elevations up to approximately 65 ft [20 m] higher than the surrounding water table) occurs under some of the Solvay Wastebeds (Geraghty & Miller, 1982; Blasland & Bouck, 1989). Groundwater discharges to the lake contribute a significant but relatively minor amount of water to the total water budget. However, as noted in the RI report (TAMS, 2002b), groundwater is a significant source of several COPCs to the lake, including mercury; benzene, toluene, ethylbenzene, and xylenes (BTEX); chlorinated benzenes; and polycyclic aromatic hydrocarbons (PAHs). The groundwater is classified as Class GA (defined as a source of drinking water [6 NYCRR Part 701.15]) by New York State regulations; however, some areas of groundwater

in the vicinity of the lake may not currently be appropriate for use as sources of potable water due to natural occurrences of salinity (e.g., brine fields in the area) and contributions from anthropogenic salinity sources, including Honeywell. Contamination of the lake prevents the lake water from being used as a drinking water source.

2.4 Population and Demographics

The population of Onondaga County grew to 469,000 in 1970; since that time, according to the most recent census data, it has decreased to about 458,000 (US Census Bureau, 2000). Most of the population currently is, and historically has been, concentrated in the Syracuse metropolitan area (see Chapter 1, Figure 1-1). This area includes Solvay, Westvale, Lakeland, and Fairmount on the western side of the lake and Galeville, Liverpool, Mattydale, and North Syracuse on the eastern side of the lake (Murphy, 1978). Other population centers in the Onondaga Lake drainage basin are the villages of Camillus, Marcellus, Otisco, LaFayette, and East Syracuse. In addition, the Onondaga Nation Territory is located about 6 mi (9.6 km) south of the lake in the towns of Onondaga and LaFayette. The population of the Onondaga Reservation includes about 800 persons who identified themselves on the most recent census as “American Indian or Alaska Native” (US Census Bureau, 2000).

2.5 Site History

Historically, Onondaga Lake has received loadings of industrial wastes and wastewater and sewage effluent. Sources of chemicals to Onondaga Lake include the following:

- Multiple industrial sources, including Honeywell and its predecessors.
- Sources related to the population in the area (e.g., sewage and landfills).
- Natural sources (e.g., inorganic constituents occurring naturally in soil or groundwater).

Honeywell’s predecessor companies began manufacturing operations in Solvay, New York, in the late 1800s. Operations occurred at three principal plants (Chapter 1, Figure 1-1): the Main Plant, which manufactured soda ash and related products from 1884 until 1986 and benzene, toluene, and xylenes from 1917 to 1970; the Willis Avenue plant, which manufactured chlorinated benzenes and chlor-alkali products from 1918 until its closure in 1977; and the Bridge Street plant (sold to Linden Chemicals and Plastics [LCP] in 1979), which manufactured chlor-alkali products from 1953 until 1988. Two areas of the former Main Plant (i.e., the Petroleum Storage Area and the Chlorobenzene Hot Spot Area) are being investigated as part of the Willis Avenue site RI/FS.

The Bridge Street and Willis Avenue plants are sources of COPCs including mercury, BTEX, chlorinated benzenes, PAHs, and polychlorinated biphenyls (PCBs). The Bridge Street plant discharged into the West Flume, which flows to Geddes Brook, which is a tributary to Ninemile Creek, which in turn is a tributary to the lake.

The Main Plant and the Willis Avenue plant initially discharged waste directly into the lake and, once the waste buildup was too great, Honeywell began dredging a channel through the waste to allow discharge. This channel became known as the East Flume. Such dredging likely occurred numerous times over a period of decades, with the result that the discharge point progressively moved east. Further information on Honeywell facilities and waste disposal is presented in the Onondaga Lake RI/FS Site History Report (PTI, 1992) and in the RI report (TAMS, 2002b).

Honeywell also disposed of Solvay wastes in numerous wastebeds covering more than 3.1 sq mi (8.1 sq km, or 2,000 acres), and disposed of organic wastes in the Semet Residue Ponds in Wastebed A; organic wastes have also been disposed of in Wastebed B near Harbor Brook. An RI/FS has been completed at the Semet Residue Ponds site and an RI/FS is underway at the Wastebed B/Harbor Brook and Willis Avenue Ballfield sites. In addition, Honeywell disposed of large quantities of combined Solvay wastes and mercury and organic wastes into the lake (e.g., via the East Flume; see RI Chapter 4, Section 4.5.1 [TAMS, 2002b]). Further discussion of these sources is provided in the RI report. A discussion of potential sources of COPCs from non-Honeywell industrial sites in the area is also provided in the RI.

In the early 1800s, Onondaga Lake was receiving untreated industrial and domestic wastes. Around the turn of the twentieth century, a combined sewer system, a single system that transmits a combination of domestic and industrial flows as well as stormwater originating from various sources, was installed that discharged into tributaries and ultimately the lake.

The first primary sewage treatment facility in the Syracuse area was constructed in 1925 at the southern end of Onondaga Lake. An additional major treatment plant was built in 1940 on Ley Creek. During the 1950s, Onondaga County established a sewer district that encompassed the City of Syracuse and some surrounding suburban areas. A new primary treatment plant, the Onondaga County Metropolitan Syracuse Wastewater Treatment Plant (Metro), was constructed in 1960 with a 50 million gallons per day (mgd) design capacity (OLMC, 1993).

The Metro sewage treatment plant, which serves the city of Syracuse and several surrounding towns, is currently permitted (NY-0027081) to discharge an average of 80 mgd through its main outfall to Onondaga Lake. The plant provides tertiary treatment for flows up to 120 mgd. For combined stormwater and industrial/domestic sewage flow up to 220 mgd, the incremental flow above 120 mgd receives primary treatment and seasonal chlorination prior to discharge into the lake through a second outfall.

The sewers contain hydraulic relief structures otherwise known as combined sewer overflows (CSOs), which have historically allowed diluted sewage (due to the mixing of stormwater and sewage) to discharge to several tributaries of Onondaga Lake during high flow events. In 1985, Phase I of a program to abate CSOs was implemented. The second phase of the CSO abatement program began in 1990. Additional abatement activities associated with the CSOs are underway as discussed below.

In January 1998, an Amended Consent Judgment (ACJ) (88-CV-0066) was executed by NYSDEC, the State Attorney General, Atlantic States Legal Foundation, and Onondaga County. The ACJ

evolves from a 1989 Judgment on Consent (88-CV-0066) settling litigation between the State of New York and the county relating to state and federal water pollution control regulations.

The ACJ, which is designed to improve the water quality of Onondaga Lake, specifically includes a listing of over 30 projects to be undertaken by Onondaga County over a 15-year period. Although completion of the entire project is not required until 2012, many of these county projects are scheduled for completion by 2009 (Onondaga County Department of Water Environment Protection [OCDWEP], 2002).

The projects may be grouped into three categories, including:

- Improvement and upgrading of the county's main sewage treatment plant (Metro).
- Eliminating and/or decreasing the effects of the CSOs on the lake and its tributaries.
- Performance of a lake and tributary monitoring program designed to evaluate the effects of the improvement projects on the water quality of the lake and its tributaries.

2.6 Potential Use and Exposures

Onondaga Lake water is classified as follows:

- Approximately the northern two-thirds of the lake are classified as Class B water. The best usages of Class B waters are "primary and secondary contact recreation and fishing. These waters shall be suitable for fish propagation and survival" (6 NYCRR Part 701.7).
- Approximately the southern third of the lake and the area at the mouth of Ninemile Creek (see Appendix A, Figure A-1) are classified as Class C water. The best usage of Class C waters is "fishing. These waters shall be suitable for fish propagation and survival. The water quality shall be suitable for primary and secondary contact recreation, although other factors may limit the use for these purposes" (6 NYCRR Part 701.8).

Recreational fishing and recreational fish harvest are allowed in Onondaga Lake, subject to NYSDEC regulations on minimum fish lengths, fishing seasons, possession limits, etc. The NYSDOH issued an advisory which recommends that the public should eat no walleye (*Stizostedion vitreum*), and to limit consumption of all other species to no more than once per month. The advisory also carries the stipulation that infants, children under the age of 15, and women of childbearing age should eat no fish from the lake (NYSDOH, 2002a). Onondaga Lake and its tributaries do not serve as drinking water sources (Syracuse Department of Water, 2000).

No permitted swimming beaches or sanctioned swimming areas exist at Onondaga Lake due to, in part, elevated bacterial counts and the turbidity of the lake water (Effler, 1996). Boating is allowed in all parts of the lake. In 1990, more than one million people used Onondaga Lake County Park (located along the northern half of the lake) for recreational activities such as boating (Moore, 1991, pers. comm.). The shoreline of the lake is used for water-related recreation and cycling/jogging trails.

The State of New York, Onondaga County, and the city of Syracuse have jointly sponsored the preparation of a land-use master plan to guide future development of the Onondaga Lake area (Reimann-Buechner Partnership, 1991). The primary objective of land-use planning efforts is to enhance the quality of the lake and lakeshore for recreational and commercial uses. Anticipated recreational uses of the lake include fishing without consumption restrictions and swimming.

The proximity of the lake to numerous centers of human activity, as well as the presence of designated recreational areas on its shores, indicate that several receptor populations could potentially have contact with the lake and the adjoining wetlands and dredge spoils area. These receptor groups could include recreational visitors, nearby residents, and workers in the industrial and commercial facilities near the lake. Chapter 4, Exposure Assessment, provides information on exposure scenarios, including estimates of the magnitude, frequency, duration, and routes of exposure for both recreational users of the lake and construction workers.

3. IDENTIFICATION OF CONTAMINANTS OF POTENTIAL CONCERN

The HHRA uses a screening process to select and provide a full evaluation of contaminants of potential concern (COPCs). The available contaminant concentration data were reviewed to identify COPCs in the following site media:

- Fish (fish fillets).
- Lake sediments (divided into two subsets, the northern and southern basins).
- Lake surface water (0 to 3 meter [m] depth).
- Selected wetland sediments (divided into four subsets, one for each of the four adjacent wetland areas).
- Dredge spoils soil (spoils area situated north of Ninemile Creek).

The data used in the HHRA are presented in Appendix A. Sample location maps are also included in Appendix A (Figures A-2 through A-6). Appendix B provides a summary of site data and data analyses. Tables 2.1 through 2.10 in Appendix B have been prepared in the format of RAGS Part D, Table 2, and present the occurrence, distribution, and selection of COPCs in Onondaga Lake and provide the following information, as specified by USEPA (1998):

- Chemicals detected and not detected in each medium.
- Frequency of detection of chemicals in each medium.
- Range of detected concentrations for each chemical in each medium.
- Range of detection limits for the chemicals in each medium.
- Background screening values for metals in sediment from a local, noncontiguous water body (Otisco Lake) were used as a basis for comparison to site concentrations; however, no contaminants were screened out (i.e., determined not to be COPCs) based on this comparison.
- Screening toxicity values (i.e., risk-based criteria [RBCs]), when available, for fish consumption, for exposure to residential soil, and for residential use of drinking water.

Appendix B also presents exposure point concentrations (EPCs) calculated for each COPC in each exposure medium, along with additional supporting documentation consistent with USEPA (1998).

Table 3-1 provides a summary of all COPCs in all site media, based on Tables 2.1 through 2.10 in Appendix B. The following sections describe how site data were used to identify COPCs.

Site data were reviewed to determine a list of contaminants that may be of concern for human health. The methods used to select COPCs were consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) and intended to include all contaminants detected at levels of potential health concern. For metals, sediment concentrations in Onondaga Lake were compared with background concentrations in sediment from the reference lake (Otisco Lake). For the comparison, the maximum Onondaga Lake concentration was compared to two times the average of the Otisco Lake concentrations. The Otisco Lake sediment data were used as a basis for comparison with inorganics data (only) in the Onondaga Lake sediment and wetland sediment matrices. However, based on recent USEPA Region 2 guidance (M. Sivak, pers. comm., 2002), background concentrations were not used to screen out any contaminants that exceeded applicable risk-based screening criteria (i.e., USEPA Region 3 RBCs or USEPA Region 9 Preliminary Remediation Goals [PRGs]). Therefore, compounds that exceed risk screening levels but are within the background screening range have been carried through as COPCs, but are flagged on the COPC screening tables (Appendix B, Table 2). Such compounds are discussed further (i.e., the extent to which they pose risk, and the extent to which such risk may be similar to background risk) in Chapter 6, Risk Characterization and Chapter 7, Uncertainty Assessment.

Concentrations of chemicals in all media were compared with human health-based screening criteria derived by USEPA Regions 3 and 9. These screening criteria, along with information on their development and use, are included in Appendix C and were used in this HHRA for:

- Ingestion of fish.
- Ingestion of soil/sediment.
- Ingestion of water.

USEPA Region 3 values for consumption of fish were used in identifying COPCs in fish tissues. The more conservative of either USEPA Region 3 RBCs or USEPA Region 9 PRGs for residential soil and for tapwater were used in screening soil, sediment, and water. The USEPA Region 9 PRGs for soil account for three potential exposure routes: ingestion, inhalation of particles or vapors, and dermal contact. The PRGs for tap water account for ingestion of water and inhalation of volatile organics from water (USEPA, 2002b).

In addition, because the USEPA Region 3 and Region 9 values do not address dermal contact with chemicals detected in water, detected chemicals were also compared with the list of chemicals identified in USEPA guidance (i.e., flagged "Y" in Exhibits B-3 and B-4 in USEPA, 2001a) where dermal exposure has a substantial contribution to exposure. These chemicals were also considered for inclusion as COPCs, even if the detected concentrations in water were below the screening level concentration for ingestion.

Tables 2.1 through 2.10 in Appendix B show the minimum and maximum detected concentrations of chemicals in each environmental medium, detection frequencies, minimum and maximum detection limits, background screening values (two times the mean background concentration for

metals in sediments and soils only), and the applicable health-based screening criteria noted above. The RBCs correspond to either a 1×10^{-6} excess cancer risk (for carcinogens) or a hazard quotient (HQ) of 0.1 (for non-carcinogens), whichever is more stringent (USEPA, 2002a,b). (The USEPA Region 3 and Region 9 screening criteria for non-carcinogens were developed for an HQ of 1.0, so the regional criteria for such chemicals were divided by 10 for use in the Onondaga Lake HHRA for use as conservative screening criteria. The residential soil criterion for lead, 400 mg/kg, was developed as a conservative residential screening criterion by USEPA and was not subject to any further modification.) In addition, Appendix C includes copies of the original sources of USEPA RBCs used in screening COPCs.

Chemical concentrations in fish were compared with RBCs derived using a fish consumption rate of 54 g/day (USEPA, 2002b). This consumption rate is higher than would be expected for most anglers at the lake and provides a conservative means for selecting COPCs in fish from the lake. It is possible that this screening level may underestimate risks if there are users (e.g., subsistence fishers) consuming lake fish at a higher level. However, the actual level of consumption used for the quantitative assessment, in accordance with USEPA's Exposure Factors Handbook (1997a), is based on a lower consumption rate. The use of the higher rate in the screening process is conservative so that chemicals which may be of concern are included in the quantitative assessment; it is not a determination that these contaminants are, in fact, a significant contributor to risk.

Site data for lake and wetland sediments and from the dredge spoils area were compared with USEPA screening criteria for residential soil (USEPA, 2002a,b) as a conservative means to evaluate direct contact with these media. Use of the residential soil screening criteria for COPCs in these media is conservative because these values are based on daily contact with soil in a residential scenario, whereas exposures to lake sediments, wetland sediments, or dredge spoil soils would be restricted to occasional contact during recreational activities, or short durations during construction activities. Such exposures would be expected to occur less frequently than exposures that a child might receive at a residence. Consequently, the total exposure to these sediments or soils would be expected to be less than exposure to soil in a residential scenario.

Contaminant concentrations in Onondaga Lake surface water were compared with screening criteria derived by USEPA, based on assumed levels of exposure resulting from use of water as the source of residential drinking water. This method is conservative because the current classification of Onondaga Lake by NYSDEC (generally, Class B in the north and Class C in the south) does not include drinking water supply as one of the designated best usages. As described above, detected chemicals in water were also compared with the USEPA list of chemicals (USEPA, 2001a) identified as having potential for substantial exposure through dermal contact in water.

Consistent with USEPA guidance (1989), data were also evaluated in light of the following considerations:

- Although USEPA indicates that contaminants can be excluded based on frequency of detection, no detected contaminants were excluded solely on this basis. Contaminants that were not detected in any sample in a particular medium were eliminated from consideration in that medium.

- A chemical can be eliminated from consideration if it is an essential nutrient, present at low concentrations, and toxic only at high doses. Consistent with USEPA guidance (1989), several essential nutrients were not included as COPCs (i.e., calcium, magnesium, potassium, and sodium).
- Risk assessment guidance (USEPA, 1989) states that if common laboratory contaminants (e.g., acetone, methylene chloride, toluene, phthalate esters) are found at less than ten times the maximum concentration detected in any blank, or if other chemicals are found at less than five times the maximum concentration detected in any blank, these chemicals can be eliminated. The data used for the screening and subsequent quantitative uses in the HHRA were validated and contaminants attributable to blank contamination were rejected or negated during validation; thus, the database used for the HHRA was pre-screened to remove the blank contaminants. As a result, no contaminants were excluded during the HHRA process on this basis.
- Only contaminants for which chemical-specific data are available were considered. In other words, analytes that were only analyzed as a class, such as total petroleum hydrocarbons, are not evaluated as potential COPCs in the risk assessment (although individual petroleum hydrocarbon constituents, such as benzene and naphthalene, are included in the assessment). It should be noted that this exclusion does not apply to some contaminants for which isomers or related individual compounds were summed for convenience and consistency in evaluating data (e.g., chlordane isomers were summed for total chlordane, various polychlorinated biphenyl [PCB] Aroclors were summed to derive total PCBs [for use in calculating cancer risk], or highly chlorinated PCBs or less chlorinated PCBs [for calculating non-cancer hazards], and the various polychlorinated dibenzo-*p*-dioxins and furans [PCDD/PCDFs] were summed to derive a total toxic equivalence quotient [TEQ]). These issues are discussed in greater detail in Chapter 5, Toxicity Assessment.
- Unknowns, non-target compounds, and tentatively identified compounds (TICs), such as 1-phenyl-1-(4-methylphenyl)-ethane and 1-phenyl-1-(2,4-dimethylphenyl)-ethane (PTE and PXE, respectively), are not included in the evaluation, as the identification and concentration of these compounds is not certain. The potential impact of exclusion of unknowns, non-target compounds, and TICs on the HHRA is discussed in Chapter 7, Section 7.1.1.
- Due to high concentrations of some target compounds, some samples require dilution and, subsequently, the sample quantitation limits (SQLs; analogous to reporting limits) may be unusually high (higher by a factor of 10 or more compared to the “usual” quantitation limit for that analyte). Data with non-detected values at high SQLs were evaluated and, in accordance with USEPA guidance (Section 5.3.2 in USEPA, 1989), eliminated from further

consideration where their use would result in non-detected values driving the calculated EPC.

After consideration of the issues described above, organic contaminants were identified as COPCs in a medium in which the maximum concentration exceeded the lowest (i.e., most health-protective) screening value, which was usually a USEPA screening criterion. Metals were identified as COPCs if the maximum concentration exceeded the screening value. Metals data were compared to twice the mean of the background concentration (where available); if the site concentration was less than or equal to twice the mean of the background concentration, the metal was flagged "BKG" but was not screened out of the quantitative risk assessment.

Table 3-1 presents the COPCs identified for the various media and includes most, though not all, of the COPCs identified in the Public Health Assessment for Onondaga Lake (NYSDOH and ATSDR, 1995), but is more extensive than that list. The list of COPCs derived through the identification process described here is distinct from the list of substances described in the Consent Decree and updates the COPC list presented in the Onondaga Lake RI/FS Work Plan (PTI, 1991). A summary of the data and results of the screening for COPCs for each medium is also provided in the sections below.

In the medium-specific discussion that follows, quantitative discussions of the number of COPCs determined are based on those that will be carried through in the HHRA as discrete contaminants. In a few cases, due to the manner in which samples were analyzed or data presented, there are COPCs listed in Table 3-1 that are not carried through individually, but are included in a group or class of compounds for subsequent quantitative assessment. These include:

- **PCBs.** Eight specific Aroclors were analyzed for in at least one medium; in addition, the NYSDEC fish fillet data include Aroclor 1254/1260. Cancer risk quantitation is based on total PCBs; for non-cancer hazards, individual Aroclors were categorized as either less chlorinated PCBs (for evaluation using the reference dose [RfD] for Aroclor 1016), or highly chlorinated PCBs (for evaluation based on the RfD for Aroclor 1254). The discussion below includes all the individual Aroclors (1016 through 1268), but does not include Aroclor 1254/1260 or total PCBs. Aroclor 1254/1260 is not included as it is an analytical construct to represent higher molecular weight Aroclors, and total PCBs are not included as they are, by definition, a COPC if any individual Aroclor exceeded screening criteria.
- **Mercury.** Methylmercury and total mercury are considered as separate and discrete analytes in the discussion below. However, some fish fillets were analyzed for several forms of mercury (e.g., total mercury, ionic mercury, and methylmercury). The various forms of mercury analyzed and detected in fish tissue were combined into a single "mercury (as methylmercury)" value (as shown in the Appendix B tables), as the data indicate that virtually all the mercury in fish tissue is in the form of methylmercury (see discussion in Section 3.1, below).

- **PCDD/PCDFs.** The PCDD/PCDF constituents are interpreted as 2,3,7,8-TCDD equivalents (the “Total PCDD/PCDF TEQ”) and considered a single contaminant. The TEQ value is derived from the PCDD/PCDF analytical data using the World Health Organization (WHO; 1998) toxicity equivalence factors (TEFs), as described in Chapter 5.

3.1 Fish Tissue

Fish tissue data are presented in Appendix A (Tables A-1A through A-1E), and the screening is summarized in Appendix B, Table 2.1. Data from all fish species commonly consumed (i.e., carp [*Cyprinus carpio*], channel catfish [*Ictalurus punctatus*], white perch [*Morone americana*], bluegill [*Lepomis macrochirus*], largemouth bass [*Micropterus salmoides*], smallmouth bass [*Micropterus dolomieu*], northern pike [*Esox lucius*], and walleye [*Stizostedion vitreum*]) were considered in the risk assessment. All available fish data meeting the following criteria were utilized:

- The fish sample must be in fillet form.
- Fish must be legal size (within 0.5 inch) for those species with legal size requirements (i.e., walleye – legal size 15 inches, with the minimum for inclusion in the HHRA at 14.5 inches; and smallmouth and largemouth bass – legal size 12 inches, with the minimum for inclusion in the HHRA at 11.5 inches).
- For species with no minimum legal size (e.g., bluegill), 6 inches was used as the minimum size, as it is not expected that fish smaller than 6 inches would be filleted for consumption.
- Data from samples collected prior to 1992 were not included in the quantitative risk assessment. These data were excluded for several reasons, including their age (data from more than ten years ago may not be representative of current conditions); inconsistencies in the species and analytical parameters of the older data; and lack of documentation in at least some cases of the field and laboratory quality assurance protocols utilized for generation of the data. The sampling associated with the 1992 RI was a large effort and conducted under a NYSDEC-approved work plan (PTI, 1991), and subsequent data used have been generated either directly by NYSDEC or its contractors or under work plans approved by NYSDEC.
- Honeywell’s PCB data (PTI samples) from 1992 were not utilized; further review of these data by NYSDEC indicated potential problems. Specifically, a low bias to the PCBs in fillet data was suspected, due to concerns about a high percentage of non-detects coupled with poor surrogate recovery. A more detailed discussion of this issue is provided in Appendix A (Section A.4.1, discussion of Table A-1C) of this report.

The data sets used in this HHRA for fish tissue are summarized in Table 3-2. The 26 COPCs identified in lake fish tissue include the following:

- Ten inorganics (antimony, arsenic, chromium, cyanide, manganese, methylmercury, total mercury, selenium, vanadium, and zinc).
- Two semivolatile organic compounds (SVOCs) (bis[2-ethylhexyl]phthalate and hexachlorobenzene).
- Nine pesticides (aldrin, delta-BHC, four DDT-related compounds [2,4'-DDE; 4,4'-DDD; 4,4'-DDE; and 4,4'-DDT], dieldrin, sum of chlordanes, and heptachlor epoxide).
- Four PCBs (Aroclors 1016, 1242, 1248, and 1260).
- PCDD/PCDFs.

Lead, which was also detected in fish tissue, will be evaluated qualitatively because it lacks a toxicity value in fish tissue. The screening value for tetraethyl lead was not considered to be appropriate for screening lead in fish tissue; lead is discussed further in Chapter 5, Toxicity Assessment and Chapter 7, Uncertainty Assessment.

It should be noted that the evaluation for volatile organic compounds (VOCs) and SVOCs (including polycyclic aromatic hydrocarbons [PAHs]) in fish fillets was based on a very limited data set; specifically, four composite adult fillets collected and analyzed for Target Compound List (TCL) and Target Analyte List (TAL) constituents in 1992. The samples in the 1992 composites were reported as being collected in the nearshore (littoral) area in the southern part of Onondaga Lake (PTI, 1993a).

The evaluation for TAL inorganics was also based on a limited data set (11 samples), consisting of the four 1992 composite fillets along with seven individual (not composite) fillets collected in 2000. Eleven of the 29 adult fish collected by Honeywell in 2000 as part of the Onondaga Lake Phase 2A investigation were filleted. Seven of these fish were collected in the lake and four were collected in lower Ninemile Creek. All data from the seven lake fish, including PCDD/PCDF data, were included in this HHRA. Data from the four fish collected in lower Ninemile Creek are used in the Geddes Brook/Ninemile Creek HHRA.

Mercury in Fish Fillets

The mercury data were generated by various sources, primarily Exponent/PTI for Honeywell and NYSDEC. The data were entered into the database in the manner in which they were reported; e.g., as methylmercury, ionic mercury, and mercury.

The methylmercury data were generated from fillet samples submitted by Honeywell/PTI specifically for methylmercury analysis. Several (i.e., 16) samples submitted for methylmercury analysis by Honeywell/PTI were also analyzed for ionic mercury, which has been interpreted in this HHRA as

inorganic mercury. Ionic mercury concentrations were all low, averaging about four percent of the methylmercury concentration determined for the same fillet. Although the ionic (inorganic) form of mercury is less toxic than methylmercury, and averages about four percent of the methylmercury in the samples for which data for it exist, the ionic mercury has been included in the calculation of the EPCs for this HHRA. In other words, the ionic mercury data (for the 16 samples for which the data are available) are summed with the methylmercury values to generate the total mercury concentration used for the fish ingestion pathway in this HHRA.

Fish filets submitted for analysis by NYSDEC were analyzed for mercury, which is interpreted in this HHRA as total mercury (i.e., the analytical result includes mercury in any form – methyl or inorganic). As noted in the literature and supported by the low concentrations of ionic mercury in the Honeywell/PTI fish sample data, the mercury result reported by NYSDEC is considered to be functionally equivalent to methylmercury. Therefore, the total mercury concentration used in the HHRA (Appendix A, Table A-1A) includes both the Honeywell/PTI methylmercury data and the NYSDEC mercury data.

3.2 Onondaga Lake Nearshore Surface Sediments

The HHRA evaluated only nearshore sediment data (within the 0 to 30 cm profile), defined as sediment data located between the shoreline and a water depth of up to 2 m. The nearshore sediments (0 to 30 cm) are the only sediments that could reasonably be expected to be contacted by individuals wading or swimming. As a result, 61 samples from the 1992 data set are evaluated in the HHRA (Appendix A, Figure A-3). In addition, 123 nearshore sediment samples collected by Honeywell/Exponent in 2000 from the 0 to 30 cm profile were included in the HHRA (Appendix A, Figure A-4).

It should be noted that the depth interval of samples used in this HHRA is inconsistent with that currently considered by USEPA for direct contact (i.e., 0 to 6 inches or 0 to 15 cm). The 0 to 2 cm and 0 to 30 cm intervals were used in this HHRA, as there were no 0 to 15 cm data from the 1992 investigation. In order to assess the effect of data from intervals other than 0 to 15 cm, the Phase 2A investigation included a set of samples from multiple intervals (0 to 2 cm, 2 to 15 cm, and 15 to 30 cm) at the same location. Review of these data suggests that there is no clear relationship between contaminant concentration and depth in the 0 to 30 cm interval in the nearshore environment; therefore, all data from less than 30 cm (including the 15 to 30 cm data from the Phase 2A investigation) have been used in the HHRA. All individual (discrete depth intervals) samples were assessed, without averaging over the length of the core, for the purposes of the COPC screening (Appendix B, Table 2). However, for the contaminants identified as COPCs in this chapter, a different approach (a length-weighted average for each sediment sample location) was then utilized in the development of EPCs in Appendix B, Table 3 (see Chapter 4, Exposure Assessment).

Surface sediment data for the northern and southern basins of Onondaga Lake are considered separately here because these parts of the lake have been classified for different uses by New York State (NYCRR Chapter X, Subchapter B, Title 14, Part 895; see also Appendix A, Figure A-1 of this HHRA). This differentiation is also consistent with the actual uses and development patterns along the lake, with the southern basin being more industrialized and the northern basin being less

developed, with more parkland along the edge of the lake. Definitions of the use categories are as follows:

- The waters of the northern basin (approximately the northern two-thirds) of the lake are classified as Class B. The best usages of Class B waters are “primary and secondary contact recreation and fishing. These waters shall be suitable for fish propagation and survival” (6 NYCRR Part 701.7).
- The waters of the southern basin (approximately the southern third) of the lake and an area around the mouth of Ninemile Creek are classified as Class C. The best usage of Class C waters is “fishing. These waters shall be suitable for fish propagation and survival. The water quality shall be suitable for primary and secondary contact recreation, although other factors may limit the use for these purposes” (6 NYCRR Part 701.8).

The classification system is used to identify the best uses of surface waters, but does not necessarily mean that the lake currently meets the water quality standards for its best use (NYSDOH and ATSDR, 1995). The water classification regulations for Onondaga lake state that, “[w]hen the waters of that portion of Onondaga Lake which are assigned to class B herein shall have been so improved as to comply with the standards of quality specified for class B, the Water Pollution Control Board at that time will give consideration to reclassification of additional areas of Onondaga Lake to class B” (6 NYCRR Part 895.3). Therefore, for the purposes of this risk assessment, Onondaga Lake is evaluated in its entirety as a Class B water body.

The nearshore sediment sample results were separated according to the sample locations in the northern versus southern basins of the lake, as the different potential uses of the shorelines are expected to result in different exposure potentials, as discussed in Chapter 4. The northern and southern basins of the lake are discussed separately below, with the sample locations in each area shown in Appendix A, Figures A-3 and A-4. The lake sediment data used in this analysis are presented in Appendix A, Tables A-2 and A-3 and summarized in Appendix B, Tables 2.2 and 2.3.

3.2.1 Northern Basin Nearshore Sediments

The 20 contaminants identified as COPCs in the northern basin nearshore sediments, shown in Appendix B, Table 2.2, include:

- Ten inorganics (antimony, arsenic, barium, cadmium, chromium, iron, manganese, mercury, methylmercury [an organometallic compound listed and tabulated with the inorganic compounds in this HHRA], and thallium).
- One VOC (benzene).
- One SVOC (hexachlorobenzene).

- Five PAHs (benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene, and naphthalene).
- Two PCBs (Aroclors 1254 and 1268).
- PCDD/PCDFs.

Of the metals identified as COPCs, four (arsenic, iron, manganese, and thallium) were present at concentrations that exceeded the risk-based screening criteria but were within the background range (i.e., less than two times the average of the Otisco Lake sediment samples). The extent to which these metals represent site-related risk will be discussed further in Chapter 6, Risk Characterization and Chapter 7, Uncertainty Assessment.

While methylmercury concentrations in northern basin sediments did not exceed screening criteria, it is carried through the risk assessment due to its historical significance in the Onondaga Lake environment.

3.2.2 Southern Basin Nearshore Sediments

The 44 contaminants identified as COPCs in the southern basin nearshore sediments, shown in Appendix B, Table 2.3, include:

- 16 inorganics (aluminum, antimony, arsenic, barium, cadmium, chromium, copper, cyanide, iron, lead, manganese, mercury, methylmercury, nickel, thallium, and vanadium).
- Four VOCs (benzene, chlorobenzene, methylene chloride, and xylenes).
- Four SVOCs (dibenzofuran, 1,3- and 1,4-dichlorobenzene, and hexachlorobenzene).
- 13 PAH compounds (2-methylnaphthalene, acenaphthylene, benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, fluoranthene, indeno[1,2,3-cd]pyrene, naphthalene, and phenanthrene).
- One pesticide (dieldrin).
- Five PCBs (Aroclors 1221, 1242, 1248, 1254, and 1260).
- PCDD/PCDFs.

Of the metals identified as COPCs in the southern basin sediments, one (manganese) was present at a concentration exceeding the risk-based screening criterion, but was at a concentration within the background range (i.e., less than two times the average of the Otisco Lake sediment samples). This

issue (i.e., the extent to which manganese represents site-related risk) will be discussed further Chapter 6, Risk Characterization and Chapter 7, Uncertainty Assessment.

While methylmercury concentrations in southern basin sediments did not exceed screening criteria, it is carried through the risk assessment due to its historical significance in the Onondaga Lake environment.

3.3 Wetland Sediments

There are four New York State-regulated wetlands adjacent to Onondaga Lake that are evaluated in this HHRA: Wetlands SYW-6, SYW-10, SYW-12, and SYW-19. Wetland sediments from 0 to 15 cm and 15 to 30 cm were collected from these four areas by Honeywell/Exponent in 2000 (Appendix A, Figure A-4). Five additional locations in Wetland SYW-6 were sampled by NYSDEC/TAMS in 2002 (also shown on Figure A-4). Two of the wetlands are near the northern basin (Wetlands SYW-6 and SYW-10), and two are near the southern basin (Wetlands SYW-12 and SYW-19). Sediment data for the four wetlands are discussed separately below. The data used in this analysis are presented in Appendix A and summarized in Tables A-4 and A-5.

It should be noted that the sediment samples from the four wetlands were analyzed for “total mercury;” none of these wetland samples were analyzed for methylmercury. Based on data from 29 samples collected at the LCP Bridge Street site and an extensive literature review, it was assumed that 1 percent of the total mercury in the wetlands sediments is methylmercury, and the remaining 99 percent of the total mercury is inorganic mercury, as discussed in greater detail in Chapter 6, Section 6.3.1.1 of the BERA (TAMS, 2002a). The uncertainty associated with this assumption is discussed in Chapter 7, Section 7.5.3.1 of this HHRA.

The two southern basin wetlands, SYW-12 (near Ley Creek) and SYW-19 (near Harbor Brook), were evaluated separately due to the locations and characteristics of the two areas, which suggested that contaminant concentrations and associated risks are likely to differ between the two. The two northern basin wetlands, both of which are on the west side of the lake between Ninemile Creek and the lake outlet, were also evaluated separately, although these were expected to more closely resemble each other due to the general similarity of the area in which they are located.

3.3.1 Northern Basin Wetland SYW-6

The 21 contaminants identified as COPCs in northern basin Wetland SYW-6, listed in Appendix B, Table 2.4, consisted of nine metals (aluminum, arsenic, cadmium, chromium, cyanide, iron, manganese, mercury [inorganic and methylmercury], and thallium), 11 PAHs (acenaphthylene, benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, 2-methylnaphthalene, naphthalene, phenanthrene), and PCDD/PCDFs.

3.3.2 Northern Basin Wetland SYW-10

The 15 contaminants identified as COPCs in northern basin Wetland SYW-10, listed in Appendix B, Table 2.5, include eight metals (aluminum, antimony, arsenic, chromium, iron, manganese, mercury [inorganic and methylmercury], and thallium), five PAH compounds (benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene), PCBs (Aroclor 1260), and PCDD/PCDFs.

3.3.3 Southern Basin Wetland SYW-12

The 19 contaminants identified as COPCs in southern basin Wetland SYW-12 (Ley Creek area), listed in Appendix B, Table 2.6, include ten inorganics (aluminum, arsenic, cadmium, chromium, copper, cyanide, iron, manganese, mercury [inorganic and methylmercury], and thallium); six PAH compounds (benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, and phenanthrene); and three PCBs (Aroclors 1242, 1254, and 1260). Wetland SYW-12 sediment samples were not analyzed for PCDD/PCDFs.

3.3.4 Southern Basin Wetland SYW-19

The 25 contaminants identified as COPCs in southern basin Wetland SYW-19 (Harbor Brook/East Flume area), listed in Appendix B, Table 2.7, include eight metals (antimony, arsenic, barium, cadmium, chromium, iron, manganese, and mercury [inorganic and methylmercury]); three SVOCs (1,3-dichlorobenzene, 1,4-dichlorobenzene, and hexachlorobenzene); eight PAH compounds (benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, and phenanthrene); two pesticides (aldrin and dieldrin); three PCBs (Aroclors 1242, 1254, and 1260); and PCDD/PCDFs.

3.4 Dredge Spoil Soils

The dredge spoils area is located along the western shoreline of Onondaga Lake, north of the mouth of Ninemile Creek, between wetland areas SYW-10 and SYW-6. The spoils, dredged from the Ninemile Creek delta in the late 1960s, rest atop native soils and have been covered with soil of unknown origin. The dredge spoil soils were evaluated for a recreational scenario and a construction worker scenario. The surface soils or cover material from the ground surface to about 3 ft deep were screened for the recreational scenario; all surface and subsurface soils, including the cover material, dredge material, and native material, were combined and screened for the construction worker scenario. The depths for this scenario range from 6.7 to 11.7 ft, based on the available data. These two sets of data are discussed separately below. The data used in these analyses are presented in Appendix A (Tables A-6 and A-7) and summarized in Appendix B (Tables 2.8 and 2.9). The locations of these samples are shown on Figure A-4.

The dredge spoil samples were analyzed for full TAL inorganics and TCL organics, except VOCs and pesticides. A subset of the dredge spoil samples (both surface and deeper) were analyzed for PCDD/PCDFs.

3.4.1 Dredge Spoils – Surface Soils

The eight contaminants identified as COPCs in the dredge spoil surface soils (Appendix B, Table 2.8) consist of six metals (aluminum, arsenic, chromium, iron, manganese, and mercury); one PAH compound (benzo[a]pyrene); and one SVOC (hexachlorobenzene). Dredge spoil samples were analyzed only for total mercury (no methylmercury analyses have been conducted on the dredge spoils to date). All the mercury in the dredge spoil samples is assumed to be inorganic mercury; the uncertainty associated with this assumption and an assessment of potential hazards using assumptions of higher rates of methylation are discussed in Chapter 7, Section 7.5.3.1.

It should be noted that the depth intervals included in the surface soils group for the dredge spoils varied, and in many cases included samples from deeper intervals (intervals extending up to 107 cm below the surface) than would ideally be evaluated for exposure to contaminants in surface soils. However, the nature of the available data necessitated the use of data from deeper samples to evaluate exposure to surface soils.

3.4.2 Dredge Spoils – Surface and Subsurface Soils

The samples used in screening COPCs for the dredge spoil soils from all depths include all surface and subsurface intervals, so all eight of the COPCs identified for the surface soils (discussed in Section 3.4.1) are also COPCs for soils from all depths. In addition to the surface soil COPCs, contaminants identified as COPCs due to their presence in deeper samples include:

- Three inorganics (cadmium, cyanide, and thallium).
- Eight PAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene, naphthalene, and phenanthrene).
- Two PCBs (Aroclors 1254 and 1268).
- PCDD/PCDFs.

The screening for the dredge spoils surface and subsurface soils is presented in Appendix B, Table 2.9.

3.5 Onondaga Lake Surface Water

Onondaga Lake surface water samples have been analyzed for 11 TAL metals, plus five forms of mercury (methyl-, dimethyl-, ionic, elemental, and total mercury), as shown in Appendix A, Table A-8A. Organic analytes for which the surface water samples were analyzed (Table A-8B) consisted of VOCs and a limited suite of SVOCs, including chlorinated benzenes (dichlorobenzene, trichlorobenzene, and hexachlorobenzene). Surface water samples from 0 to 3 m water depths were not analyzed for other SVOCs (including PAHs), pesticides, PCBs, or PCDD/PCDFs.

Contaminant concentrations in surface water were compared with USEPA Region 3 RBCs and Region 9 PRGs for residential drinking (tap) water consumption, as shown in Appendix B, Table 2.10. Lead was screened against the 15 µg/L "action level" identified in the federal drinking water regulations (40 CFR Part 141). Cadmium and manganese exceeded the risk-based screening concentrations for drinking water and were, therefore, identified as COPCs. Methylmercury and total mercury did not exceed risk-based screening criteria but are included as COPCs for lake surface water, due to their historical significance in the Onondaga Lake environment. Organic compounds detected in lake surface water and identified as COPCs consist of four VOCs (benzene, bromodichloromethane, chlorobenzene, and chloroform) and two SVOCs (1,3- and 1,4-dichlorobenzene).

In addition to the ten COPCs identified based on the criteria discussed above, detected contaminants were also evaluated for the dermal pathway. Based on Exhibits B-3 and B-4 of recent USEPA guidance (2001a), five additional chemicals (chromium, toluene, xylene, 1,2-dichlorobenzene, and 1,2,4-trichlorobenzene) were evaluated as potential COPCs in surface water. However, toluene and xylene were not retained as COPCs due to a combination of infrequent detection (each of these chemicals was detected in only one out of 48 samples) and low concentrations (the one detection of each was less than 0.5 µg/L, and at least two orders-of-magnitude below the ingestion screening criterion). The other three chemicals (chromium, 1,2-dichlorobenzene, and 1,2,4-trichlorobenzene) have been added to the COPC list, for a total of 13 COPCs (as summarized in Table 3-1) in Onondaga Lake surface water.

4. EXPOSURE ASSESSMENT

Exposure assessment is the process of identifying human populations that could contact site-related contaminants, and estimating the magnitude, frequency, duration, and route(s) of such exposures under current and potential future land use scenarios. In this HHRA, potential site risks are evaluated for current and, as appropriate, future recreational exposure scenarios. In addition, potential exposure to lake and wetland sediments, dredge spoil soils, and surface water were also considered for a construction worker scenario.

Other receptor populations typically considered in a risk assessment (i.e., residents and long-term workers) were not considered here because the site (i.e., the lake and Wetlands SYW-6 and SYW-12) and the additional areas evaluated in this HHRA (i.e., Wetlands SYW-10 and SYW-19 and the dredge spoils area) are undeveloped and, given the nature of the areas, are unlikely to be developed in the future. Potential occupational exposures associated with the Honeywell properties were evaluated in the Semet Residue Ponds (O'Brien & Gere, 1991), LCP Bridge Street (NYSDEC/TAMS, 1998a), and Willis Avenue (O'Brien & Gere, 2002 [under NYSDEC review]) remedial investigations (RIs) and associated HHRA's. In addition, RIs and risk assessments are currently being performed by NYSDEC at the Geddes Brook/Ninemile Creek site and by Honeywell at the Wastebed B/Harbor Brook and Willis Avenue Ballfield sites.

This chapter describes how the exposure scenarios for the Onondaga Lake HHRA were selected as a means of estimating current and future exposures and potential risks, as follows:

- In Section 4.1, the exposure setting is characterized and potentially exposed populations are identified (e.g., recreational visitors).
- In Section 4.2, potential exposure pathways are identified.
- In Section 4.3, quantitation of exposure to various contaminated media, including fish consumption rates, exposure frequency and duration, and incidental ingestion of sediments and soils are addressed.
- In Section 4.4, dermal contact with surface sediments and soils is addressed for the potential receptor populations and scenarios.
- In Section 4.5, incidental ingestion of surface water for the potential receptor populations and scenarios is addressed.
- In Section 4.6, dermal contact with surface water for the potential receptor populations and scenarios is addressed.

- In Section 4.7, the methods for calculating exposure point concentrations (EPCs) are presented.
- In Section 4.8, a brief summary of the chapter is presented.

4.1 Exposure Setting and Receptor Populations

The first step in evaluating exposures at a site is to characterize the site with respect to its physical characteristics, current and potential future land uses, and human populations on or near the site. A summary of this information is provided in Chapter 2 of this HHRA and Chapters 1 and 3 of the RI report (TAMS, 2002b). Additional information on current (Section 4.1.1) and potential future (Section 4.1.2) land and site use is provided below. This information is used to identify possible exposure pathways for potentially exposed populations and to determine appropriate exposure intake variables to quantify exposure.

4.1.1 Current Land and Site Use

Onondaga Lake lies within the jurisdictional boundaries of the city of Syracuse, the towns of Salina and Geddes, and the villages of Solvay and Liverpool in Onondaga County, New York. Onondaga Lake is located in an urban area and is surrounded by industrial, commercial, and recreational areas. Most of the northern half of the shoreline is parkland owned by Onondaga County. The primary land use around the lake is classified as parks and recreation (Reimann-Buechner Partnership, 1991). The areas along the northeast and west lakeshores are zoned as “residential open land” or “residential A,” although no residential property directly abuts the lake, as these areas are actually parkland. The residential properties closest to the lake, in Liverpool and Lakeland, are less than 0.1 mile (mi) (150 meters [m]) from the lakeshore but are separated from the lake by parkland and/or highways. There are no residential properties on the wetlands and dredge spoils area evaluated in this HHRA. Commercial and industrial areas near Onondaga Lake are concentrated around the southern end of the lake, in the Syracuse metropolitan area.

With the exception of the parkland, marina, and pedestrian and bicycle paths on the northern end of the lake (see Figure 4-1), all of which are administered by the Onondaga County Parks Department (OCPD), the majority of the lakefront of Onondaga Lake remains undeveloped (Effler, 1996). New development has occurred near the southern shore of Onondaga Lake, along Onondaga Creek and the Barge Canal, as part of the Syracuse Inner Harbor Project, as approximately 42 acres of land, owned by the New York State Canal Corporation, are being developed for recreational and commercial uses by the Lakefront Development Corporation (LDC) (LDC, 2001). The only private property on the lake is owned by commercial interests (e.g., Conrail, Honeywell, Crucible Materials Corporation) (Reimann-Buechner Partnership, 1991). Farming areas and open space are located predominantly in the southern half of the drainage basin. Chapter 3, Figure 3-11 of the RI report presents the covertypes within 0.5 mi (0.8 km) of Onondaga Lake (TAMS, 2002b).

4.1.1.1 Drinking Water

Onondaga Lake is currently neither used nor permitted as a drinking water source. As noted in Chapters 2 and 3, Onondaga Lake is a Class B and Class C water body; the best usages for these classifications include fishing and recreation but not use as a potable water source. The Syracuse Department of Water (SDW) provides drinking water for the entire city of Syracuse and portions of the towns of DeWitt, Onondaga, Camillus, Skaneateles, and Salina and the villages of Jordan and Elbridge. The SDW obtains most of its water from Skaneateles Lake, about 20 mi (32 km) southwest of Syracuse, but supplements its supply during times of drought or other conditions with water from Lake Ontario or Otisco Lake. Water from Lake Ontario is treated and provided by the Metropolitan Water Board (MWB). In addition, in the year 2000, about 5 percent of the SDW's water was obtained from Otisco Lake through the Onondaga County Water Authority (OCWA) (SDW, 2001).

The OCWA provides water for the remainder of Onondaga County not serviced by SDW, in addition to emergency connections to some of the entities served by the SDW (e.g., the city of Syracuse). The OCWA obtains the majority of its water from Lake Ontario (purchased from the MWB) and Otisco Lake, although it obtains some water for a few communities from Skaneateles Lake through the SDW (SDW, 2001).

4.1.1.2 Fishing

The current fish population in Onondaga Lake is vastly improved over conditions in the 1950s, when over 90 percent of the fish in the lake were common carp (*Cyprinus carpio*) (Onondaga Lake Partnership [OLP], 2002). Recent surveys have identified over 50 species of fish in the lake, including sport fish such as bass, walleye (*Stizostedion vitreum*), and northern pike (*Esox lucius*), as well as panfish such as perch, bluegill (*Lepomis macrochirus*), and pumpkinseed (*Lepomis gibbosus*) (OLP, 2002). Onondaga Lake has been recently called one of the “finest July bass lakes” in New York State, and is a “favorite weekend destination for many central New York bass fishermen,” according to *Game and Fish Magazine* (<http://gameandfish.about.com>, accessed May 2002), although the magazine does note that “... anglers who fish it would be well advised to release their catch.” There have been bass fishing tournaments on the lake in 2001 and 2002.

Although there are New York State Department of Health (NYSDOH) fish advisories in place for Onondaga Lake (discussed in greater detail in Section 4.2.1, below), fishing is a significant recreational activity and is addressed in this HHRA.

4.1.1.3 Beaches and Swimming

There are no permitted swimming beaches or sanctioned swimming areas, as Onondaga Lake is generally not fit for swimming due to elevated levels of coliform bacteria and turbidity (Effler, 1996); the last public beach on Onondaga Lake was closed in 1940. However, there is evidence of primary contact recreation, such as water skiing, and secondary contact recreation, such as boating and canoeing. Water skiing, boating, and canoeing are allowed in all parts of the lake. Access to nearshore sediment along the

southwestern portion of the lake is restricted by Interstate 690 (I-690), which runs along the southwestern shore of the lake between Honeywell's hazardous waste sites (e.g., Semet Residue Ponds and Willis Avenue) and the lakeshore. As noted in the RI (TAMS, 2002b), the sediments in this area of the lake contain the highest concentrations of contaminants of potential concern (COPCs).

The tributaries directly impacted by Honeywell are Ninemile Creek and its tributaries, Geddes Brook and the West Flume; Tributary 5A; the East Flume; and Harbor Brook. Potential human health risks related to exposures to COPCs in the West Flume were evaluated in the RI and HHRA for the LCP Bridge Street site (NYSDEC/TAMS, 1998a). Geddes Brook and Ninemile Creek are the subject of an ongoing investigation and are being further evaluated in a separate HHRA being rewritten by NYSDEC. Potential human health risks associated with the remaining tributaries (i.e., the East Flume, Harbor Brook, and Tributary 5A) are being addressed by Honeywell (with NYSDEC oversight) in other documents in preparation for other Onondaga Lake subsites. Tributary 5A is being addressed in the HHRA for the Willis Avenue site and both the East Flume and Harbor Brook are being addressed in the HHRA for the Wastebed B/Harbor Brook site.

4.1.1.4 Potential Receptors

Under current conditions, the most likely potential receptors for Onondaga Lake are recreational users (including nearby residents). Recreational visitors or residents could include both children and adults. In addition, it is conceivable that a construction worker might contact dredge spoil soils, lake and wetland sediments, or surface water during work in these areas.

4.1.2 Future Land and Site Use

In recognition of the scope of present and future development opportunities adjoining Onondaga Lake, various local and state agencies jointly sponsored the preparation of a land use master plan that will serve as a guide to lakefront development (Reimann-Buechner Partnership, 1991). Development for recreational and commercial purposes along Onondaga Creek and the Barge Canal in the Syracuse Inner Harbor is ongoing, including the September 2001 opening of the most recent phase of improvements to the Inner Harbor (LDC, 2001). It is anticipated that the existing bicycle/jogging trail will be expanded to encircle the entire lake, although formal planning (and construction) of the section between the State Fairgrounds and Syracuse awaits completion of environmental studies of properties in the area, including the Honeywell sites (J. Eallonardo, 2002, pers. comm.).

The planned recreational uses include swimming and fishing in the lake without health advisories. Planned future amenities include a pleasure boat marina, restaurants, hotels, a freshwater education and research center, retail establishments, and water-oriented recreation and culture parks (Effler, 1996). Anticipated development includes remediation and commercial development of industrial areas at the southern end of the lake, including the Carousel Mall on the southeast shore of the lake, and relocation of "Oil City," an area near the Barge Canal terminal used for storage of petroleum products.

The objectives of the land-use planning efforts for Onondaga Lake are to enhance the quality of the lake and its shore for primarily recreational or commercial uses. Hence, potential future receptors would be recreational visitors to the lake or possibly workers at commercial establishments by the lakefront who might visit the lake occasionally. However, the potential for nearby workers to be exposed to COPCs in the lake is expected to be less than the exposure potential for people who might visit the lake for recreation. Thus, recreational users are considered to be the most likely receptors and have the greatest potential for exposure. Although employees of recreational facilities (e.g., workers at a marina) might be present at the lakefront more frequently, it was not considered likely that an employee's exposure to contaminated media would be greater than those of recreational users.

Under potential future land use conditions, the HHRA assumes that fishing and swimming would occur and that the fish consumption advisory would be lifted. The HHRA also assumes that the lake will still be surrounded by recreational and commercial facilities and that the greatest potential for exposure will result from recreational uses of the lake.

On January 20, 1998, Onondaga County, the State of New York, and other parties entered an Amended Consent Judgment (ACJ) requiring Onondaga County to upgrade the existing sewage treatment plant (Metro) and to reduce the impact of combined sewer overflows (CSOs). This ACJ is designed to achieve full compliance with federal Clean Water Act requirements by December 1, 2012, although, under the current schedule, over three-fourths of the improvements will be completed by 2008 (OLP, 2002).

However, the possibility of the lake serving as a drinking water source in the future is considered highly unlikely. The lake is not classified as a potable water source, due in part to natural and anthropogenic sources of salinity and the combined effects of municipal and industrial pollution. In addition, Skaneateles Lake currently serves as the primary source of drinking water for the city of Syracuse. Although future use of the lake as a drinking water source is highly unlikely, the risk assessment did apply screening values based on consumption of drinking water to identify COPCs. Groundwater is not withdrawn from the lake, wetlands, or dredge spoils area, nor is it used in those areas, so groundwater ingestion was not evaluated as an exposure medium.

4.2 Potential Exposure Pathways

This section identifies potential exposure pathways for COPCs found in environmental media. An exposure pathway is the course a contaminant takes from a source to an exposed receptor. A complete exposure pathway consists of the following four elements:

- A source for the contaminant (i.e., contaminated medium).
- A mechanism of release, retention, or transport of a contaminant in a given medium (e.g., air, water, soil).
- A point of human contact with the medium (i.e., exposure point).

- A route of exposure at the point of contact (e.g., incidental ingestion, dermal contact).

If any one of these elements is missing, the pathway is considered incomplete and does not present a means of exposure. Only those exposure pathways judged to be potentially complete are quantified in this HHRA. RAGS Table 1 in Appendix B shows the conceptual model used to identify the exposure pathways evaluated in this HHRA.

Contaminants of potential concern have been detected in fish tissue, lake and wetland sediments, dredge spoils soil, and surface water in or near Onondaga Lake. As described above, the most likely means for exposure to these COPCs is through recreational use of the lake, wetlands, and dredge spoils area. Under current conditions, exposure to COPCs in these media may be affected by advisories regarding the consumption of fish from the lake, the lack of public swimming beaches on the lake, and limited access to the lakefront in some areas. However, this HHRA quantified the following exposure pathways in the absence of any institutional controls or other restrictions, and therefore uses the same exposure assumptions for current and potential future recreational scenarios:

- Consumption of fish from Onondaga Lake.
- Incidental ingestion of and dermal contact with surface sediments from Onondaga Lake and wetland areas (i.e., the top 30 cm of sediments under water less than 2 m deep in the lake, and from the top 30 cm in wetland sediments) during recreational uses and occupational contact (construction worker).
- Incidental ingestion of and dermal contact with surface soils of dredge spoils (i.e., 0 to 3 ft [about 100 cm]) by visitors or construction workers, or incidental ingestion of and dermal contact with deeper dredge spoil soils (i.e., 0 to 11.7 ft [about 360 cm]) by construction workers.
- Incidental ingestion of and dermal contact with surface water from Onondaga Lake during recreational uses (e.g., wading, boating, or swimming).

The following subsections describe the potentially complete exposure pathways associated with each medium evaluated in the HHRA.

4.2.1 Consumption of Fish Tissue

There is currently a specific, restrictive fish consumption advisory for Onondaga Lake (NYSDOH, 2002a), but no ban on fishing. As discussed previously in this chapter, fishing is a sanctioned and significant recreational activity at Onondaga Lake. The health advisory is based in part on findings of mercury and PCBs in fish tissues at concentrations exceeding US Food and Drug Administration (FDA) tolerance limits.

For example, the average concentration of mercury in the 728 fish tissue samples used in this HHRA, 1.05 mg/kg, exceeds the FDA tolerance limit of 1.0 mg/kg.

The current advisory for Onondaga Lake includes the following recommendations (NYSDOH, 2002a):

- **Walleye** – All persons should eat none (due to mercury contamination).
- **Carp and channel catfish** (*Ictalurus punctatus*) – All persons should eat no more than one meal (8 oz) per month (due to mercury, dioxin, and PCB contamination).
- **All other species** – All persons should eat no more than one meal (8 oz) per month (due to mercury contamination).
- Infants, children under 15 years of age, and women of childbearing age should not eat any fish whatsoever from Onondaga Lake.

However, because this HHRA focuses on future land use conditions and assumes that all fish caught from the lake will be consumed, future recreational visitors were assumed to be the receptor with the most exposure to chemicals in fish.

Although there are no studies by USEPA which specifically evaluate consumption by children of recreationally caught fish, extrapolation or estimates from other data suggest that consumption of fish by children (especially younger children) may be higher – perhaps up to four times higher – than that by adults, on a body-weight normalized basis. For this HHRA, the fish ingestion rates for children were based on those recommended by USEPA Region 2. Rates were also recently used for the Hudson River PCBs site HHRA (TAMS/USEPA, 2000). For the older and young child, fish ingestion rates were estimated to be two-thirds that of an adult for an older child (age six to less than 18) and one-third that of an adult for the younger child (up to six years old). However, given the limited data on fish consumption rates for children, there is a fair amount of uncertainty in this estimated consumption rate. Therefore, the potential for children to consume fish at a greater or lesser rate, and the risks and hazards associated with other consumption rates, are discussed in greater detail in Section 7.3.2 of Chapter 7, Uncertainty Assessment.

The potential existence of a subsistence fishing population was evaluated but there was considered to be insufficient evidence to warrant its inclusion in the HHRA as a complete pathway. Although the Onondaga Nation has a reservation south of Syracuse, its northernmost boundary is about 6 mi (9.6 km) south of the southernmost edge of the lake. The Onondaga Reservation is actually slightly closer to Otisco Lake, the reference lake. Due to the distance from Onondaga Lake, and a lack of any anecdotal accounts of subsistence fishing occurring at the lake, this pathway was not considered complete. However, as there is not firm evidence that subsistence fishing does not occur, the potential risks and hazards associated with consumption rates appropriate for subsistence fishers are presented in Chapter 7, Uncertainty Assessment.

4.2.2 Lake and Wetland Surface Sediments and Dredge Spoil Soils

Exposure to COPCs in surface sediments within Onondaga Lake, wetland sediments, or dredge spoil soils could occur via incidental ingestion of, or dermal contact with, these media. Both of these exposure routes were quantitatively evaluated for each of the sediment and soils data sets for the recreational and construction worker scenarios.

As indicated above, nearshore lake surface sediment is defined here as sediments under water less than 2 m deep. In evaluation of lake and wetland sediments, data from sample profiles from the 0 to 30 cm depth were used; where samples from multiple depths were available, they were length-weighted and a single concentration (a length-weighted average) was developed for each sediment sampling location. (The procedure and a sample calculation are shown in Section 4.7.) For dredge spoil soils, a surface soil evaluation was conducted using the samples of the material placed on the dredge spoil soils; these sample depths (up to about 3 ft) often exceed those generally used to quantify exposure to surface soil. For the construction worker scenario, data from the surface down to a depth of 11.7 ft within dredge spoil soils (including both the samples evaluated for dredge spoil surface soil exposure, as well as the samples from greater depths, including the contaminated spoil material from the Ninemile Creek delta) were used to calculate the EPCs. The soil depth interval evaluated for the construction worker scenario is a function of the depths of the samples (i.e., the available data), which is in turn based on the depth (thickness) of the dredge spoils at specific locations.

In evaluating recreational uses, adults, young children (up to age six), and older children (age 6 to less than 18) were considered, as these groups might come into contact with sediments in the lake. The potential for anyone to contact sediments at areas other than the park areas along the northern half of the lake is currently limited. However, because recreational uses of the lake may increase in the future, potential risks for adults and children (both young and older) who may contact lake sediments were evaluated for both the northern and southern portions of the lake in the HHRA. As access to the wetlands and the dredge spoils area is currently limited and, for the purposes of this study, development of these areas is assumed to be unlikely, only older children and adults (including construction workers) were assumed to be potentially exposed to the wetland sediments or dredge spoils area soils. As there is some potential for workers to access site media during construction activities, construction worker scenarios were evaluated for incidental ingestion of and dermal contact with lake and wetland sediments and for dredge spoil soils.

4.2.3 Surface Water

Potential exposures that could result from incidental ingestion of and dermal contact with chemicals in surface water were evaluated in the HHRA through recreational and construction worker scenarios. In consideration of planned recreational uses of Onondaga Lake described in Section 4.1.2, Future Land and Site Use (and the planned achievement of Clean Water Act public beach criteria for coliform and water clarity), adults and children were evaluated in a swimming scenario for Onondaga Lake. Application of exposure estimates for swimming also provides a protective means to evaluate exposure and risks associated with any other water activities, such as boating, water skiing, and sailboarding.

4.2.4 Consumption of Game

The NYSDOH has issued statewide advisories regarding the consumption of snapping turtles and wild waterfowl (NYSDOH, 2002a). The advisory for snapping turtles recommends that women of childbearing age and children under 15 eat no turtles or turtle soup, and that others who consume snapping turtles should remove the fat, liver, and eggs. The NYSDOH advisory for waterfowl is that mergansers (a subfamily of diving duck) not be eaten by any person, and that other waterfowl species should be skinned and have the fat removed, and no more than two meals per month of wild waterfowl should be eaten.

NYSDOH advisories are based on statewide PCB contamination in snapping turtles and statewide PCB, mirex, chlordane, and DDT contamination in waterfowl (NYSDOH, 2002a). All of these COPCs have been detected in one or more media (e.g., sediments, fish) at Onondaga Lake. However, there are no data on the extent of hunting or consumption of snapping turtles or waterfowl at Onondaga Lake, and there are no site-specific analytical data for turtles or waterfowl to quantify exposure point concentrations. Although the hunting of waterfowl on Onondaga Lake is permitted under New York State law, the hunting season is significantly shorter than the fishing season. In addition, some (but by no means all) of the waterfowl are migratory, and attribution of contamination to Onondaga Lake is difficult. Therefore, the consumption of game is not included in the quantitative HHRA, although there is a potential for increased risk to consumers of snapping turtles and waterfowl, including nearby Native American populations.

4.2.5 Inhalation of Volatile Organic Compounds

An initial preliminary site assessment (PSA) conducted for Onondaga Lake by NYSDEC (NYSDEC, 1989, as cited in PTI, 1991) concluded that there was little potential for releases of contaminants to air. The data for volatile organic compounds (VOCs) in surface water and near-surface soils were reviewed as part of this HHRA, and the initial conclusion by NYSDEC is considered to still be appropriate for recreational users and nearby residents. In addition, there are currently no structures on the site nor are any likely to be built, due to regulatory restrictions (e.g., zoning and wetlands). Therefore, the inhalation pathway was considered to be incomplete for all media and was not assessed further in this report.

4.3 Quantitation of Exposure

In this section, COPC intakes for chronic exposures are estimated for the exposure pathways identified in the previous section. COPC intakes are based on estimates of exposure concentrations at the exposure point (i.e., EPCs) and on the estimated magnitude of exposure to COPC-containing media. Exposure estimates for ingestion, termed chronic daily intakes (CDIs), are defined as the mass of a contaminant taken into the body, per unit of body weight, per unit of time. For dermal contact, exposures are expressed as absorbed dose rather than administered dose.

The averaging time used to determine a CDI depends on the type of toxic effect being assessed. For carcinogenic effects, CDIs are calculated by averaging the total cumulative dose over a lifetime. The

estimate of the average lifespan is assumed to be 70 years, based on USEPA (1991b) guidance.¹ For assessing non-cancer effects, CDIs are calculated by averaging intakes only over the period of exposure. The distinction between these two approaches is based on USEPA's conclusion that the toxicological mechanisms of action are different for carcinogenic and non-carcinogenic processes.

Intakes of COPCs were estimated using algorithms and assumptions consistent with USEPA guidance (e.g., USEPA, 1989) for the potential exposure pathways: consumption of fish, incidental ingestion of and dermal contact with surface sediments in the lake and wetlands and with surface and subsurface dredge spoil soils, and incidental ingestion of and dermal contact with surface water. Both central tendency (CT, also known as typical) and reasonable maximum exposure (RME) estimates were calculated.

In RAGS Part 3, Exhibit 3-2, USEPA defines CT exposure as "a risk descriptor representing the average or typical individual in the population, usually considered to be the arithmetic mean or median of the risk distribution" (USEPA, 2001b). For this HHRA, the CT was calculated using average, or 50th percentile (median), values for exposure factors, as shown on RAGS Tables 4.1 through 4.31 of Appendix B. (The 50th percentile value is the preferred value for the CT scenario; however, for some parameters this value is not reported and the average value is used instead.)

The RME is defined as "the highest exposure that is reasonably expected to occur at a site" (Section 6.1 of USEPA, 1989). The RME provides an estimate of a conservative case (greater than the CT, or typical) that is still within the range of possible exposures; it is not a "worst-case scenario." Normally, only two or three of the variables in the exposure equation are set to high-end (90th or 95th percentile) values; typically, these are contact rate and exposure frequency or duration, for the RME case, while CT (50th percentile, or average) values are used for other variables (e.g., body weight, skin surface area).

A summary of the exposure assumptions used in the exposure assessment and the exposure algorithms and assumptions used to calculate CDIs for all potentially complete exposure pathways (as identified in RAGS Table 1) are provided in RAGS Tables 4.1 through 4.31 (Appendix B). Table 4-1, accompanying this chapter, provides chemical-specific dermal absorption factors.

The following subsections present methods used to quantify exposure via each of the pathways and the methods used to calculate EPCs for CT and RME scenarios for these pathways.

4.3.1 Fish Consumption

Under current land use conditions, fishing is a significant recreational activity on Onondaga Lake. Although specific, restrictive health advisories exist (recommending people limit their consumption of lake fish, or for some subgroups, not consume any) that may limit the extent of consumption of fish from the lake, the extent

¹ USEPA's *Exposure Factors Handbook* (USEPA, 1997a) recommends use of 75 years for the average value for life expectancy; however, the original 70-year value is used in this HHRA for consistency among risk assessments, and the difference (error) between the two values is low.

to which these advisories are adhered to is unknown. The HHRA also focuses on future land use conditions and assumes that fish caught from the lake will be consumed. Hence, current and future recreational visitors were assumed to be the receptors with exposure to COPCs in fish. The algorithm and assumptions used to estimate risks associated with consumption of fishes from Onondaga Lake are presented in Appendix B, RAGS Tables 4.1 through 4.3.

4.3.1.1 Fish Consumption Rates

No fish consumption data specific to Onondaga Lake were available. In accordance with USEPA guidance (1997a), the HHRA uses a fish consumption rate of 8 g/day for the CT, or typical, exposure scenario and 25 g/day for the RME scenario. These values were derived by USEPA (1997a) from the mean and the 95th percentile fish consumption rates identified in surveys of anglers on Lake Michigan (West et al., 1989, 1993), on Lake Ontario (Connelly et al., 1996), and in Maine (Ebert et al., 1993), and are USEPA's recommended default rates for recreational freshwater anglers. Uncertainties associated with the use of these fish consumption rates and an evaluation of alternative rates are presented in Chapter 7, Uncertainty Assessment.

As noted in Section 4.2.1, there are limited data on the consumption of sport-caught fish by children. However, estimates based on limited literature data and other assumptions suggest that consumption rates for young children (under age six) may be as much as four times that of adults on a body weight-normalized basis (see Chapter 7, Table 7-3). For this HHRA, the fish ingestion rates for children were based on those recommended by USEPA Region 2. These rates were also recently used for the Hudson River PCBs Superfund site HHRA (TAMS/USEPA, 2000). For the older and young child, fish ingestion rates were estimated to be two-thirds that of an adult for an older child (age six to less than 18) and one-third that of an adult for a young child (less than six years old). Given the limitations in existing data on rates of consumption by children of recreationally caught fish, exposure estimates applied to children in deriving risk estimates are somewhat uncertain. A more detailed assessment of the various approaches to estimating the fish ingestion rate for children, and the effect of different estimates on the risk to children, is presented in Chapter 7, Uncertainty Assessment.

The potential for Onondaga Lake to serve as a subsistence source of food was also considered. Studies evaluating subsistence-level fish consumption have primarily identified such use among concentrated Native American populations; a consumption rate of 170 g/day is suggested for this subpopulation (USEPA, 1997a). According to the most recent census data (US Census Bureau, 2000), there are about 4,000 persons in Onondaga County, or about 0.9 percent of the population of the county, who identify themselves as Native Americans (the official census category is "American Indian and Alaskan Native;" however, the Alaskan Native component is assumed to be negligible in Onondaga County). The percentage of Native Americans is somewhat higher (about 1.1 percent) in the city of Syracuse.

The Onondaga Reservation, which includes about 800 Native Americans, is located about 6 mi (9.6 km) south of Onondaga Lake. It is important to note that these figures are for individuals identifying themselves as entirely of one race or ethnic group; for Native Americans, the numbers almost double when also

considering persons of multiple race or ethnicity who include Native American as one of the components. Since the demographics of the Onondaga Lake area suggest that the presence of a subsistence fishing subpopulation is at least possible, it will be evaluated quantitatively in Chapter 7, Uncertainty Assessment.

4.3.1.2 Fractional Intake of Fish

A second major component of fish consumption rates is the fractional intake, or the percentage of the fish consumed that may originate from the affected area. Because of the absence of consumption data specific to Onondaga Lake, both the RME and CT estimates calculated in this HHRA incorporate the conservative assumption that all fish consumed are caught at Onondaga Lake (i.e., there is no adjustment for fractional intake – referred to as “fraction ingested” [from contaminated source], or FI, in risk assessment terminology).

The USEPA default fish consumption rates used in the HHRA represent consumption from all freshwater resources fished during the season. Similar to anglers evaluated in the studies from other lakes (see Section 4.3.1.1) used to derive USEPA’s 25 g/day intake rate, however, several other desirable fishing locations that are not subject to health advisories are available to anglers who live near Onondaga Lake. Although some anglers may fish in more than one water body (and thus have an FI of less than 1.0), it is equally conceivable that some anglers, especially those who live near the lake (e.g., within walking distance), may fish in Onondaga Lake almost exclusively (and have an FI of 1.0). Chapter 7, Uncertainty Assessment, includes a discussion of risk estimates derived through application of an FI of less than 1.0.

4.3.1.3 Cooking Loss from Fish

The human health risks from exposure to chemicals in fish depend entirely on the amount of chemical actually ingested, rather than the amount present in the aquatic species. Contrary to the default exposure assessment assumptions used in many risk assessments that evaluate the potential hazards associated with fish consumption, most anglers are unlikely to be exposed to the concentrations measured in the raw fillet. There is evidence that preparation and cooking of edible fish tissue can result in loss of lipophilic contaminants such as PCBs and PCDD/PCDFs, although the extent of such loss varies according to the specific study and methods of cooking (grilling, pan-frying, etc.) and preparation (skinning, filleting, etc.).

Cooking methods that result in significant fat loss from fish tissue or allow for the transfer of lipophilic contaminants to cooking oil resulted in the greatest PCB losses. Due to the nature of mercury in fish, no loss of mercury during cooking is expected.

Wilson et al. (1998) recently conducted a review of the literature on loss of contaminants from fish during cooking; literature on cooking loss was also reviewed by USEPA for the Hudson River PCBs site HHRA (TAMS/USEPA, 2000). These reviews, of 12 to 14 studies published between 1972 and 1998, found that mean reductions of PCBs ranged from no loss (a few gains were reported, but these are likely to be within the range of experimental error and were considered to be zero loss) to a 74 percent reduction. Despite the wide range of data reviewed, it is not possible to determine the key factors that control the extent of

cooking loss, especially in view of the wide range of cooking loss values reported in the literature. PCB losses from cooking may depend on the cooking method, length and temperature of cooking, preparation method (extent of skin removal and trimming, etc.), species of fish, lipid content of the individual fish cooked, the initial PCB concentration, whether or not cooking liquids are discarded or consumed, and the experimental design and analytical reporting method.

The “Uniform Sport Fish Consumption Advisory for the Great Lakes Region,” issued by the Great Lakes Sport Fish Advisory Task Force (GLSFATF, 1993), includes an assumed 50 percent reduction factor for PCBs in fish fillets. This value may not be conservative, as it assumed that advisories on trimming and cooking fish were adhered to by the public. Based on USEPA’s review of the literature, the Hudson River HHRA included only an assumed 20 percent cooking loss (CT only; zero loss was assumed for the RME). Reviewing the results of the cooking loss literature surveys, as well as previous USEPA precedent and comments from NYSDEC and NYSDOL (1998a) on the draft Onondaga Lake HHRA, led to the use of a cooking loss of 33 percent for PCBs for the CT for this Onondaga Lake HHRA. For the RME scenario, no cooking loss adjustments have been applied.

There are a few cooking loss studies that included dioxins and furans (PCDD/PCDFs). Zabik and Zabik (1996) reviewed the degree of cooking loss for a variety of organochlorine compounds in fish tissue, including tetrachlorodibenzo-*p*-dioxin (TCDD). These limited data do confirm what has been indicated in most literature – that there is some loss of the studied compounds during cooking, but the data are not considered sufficiently robust to establish a separate estimate of cooking loss for PCDD/PCDFs. The cooking loss for PCDD/PCDFs has, therefore, been assumed to be the same as the cooking loss for PCBs (i.e., 33 percent for CT, 0 percent for RME).

4.3.1.4 Exposure Frequency and Duration for Fish Consumption

Exposure frequency generally reflects an estimate of the number of times or days per year that an exposure occurs. However, most fish consumption rates, including those described above, reflect a daily average over an entire year. Therefore, an exposure frequency of 365 days/year was applied to this exposure pathway. Exposure duration is an estimate of the number of years that exposure can occur. In this risk assessment, default values for mean and upper-bound exposure durations of nine (50th percentile) and 30 years (90th percentile), respectively (USEPA, 1989, 1991b), were applied for the CT and RME estimates.

4.3.2 Exposure Frequency and Duration and Receptor Characteristics for All Media Except Fish

As described above, the most likely human populations to visit and recreate in the lake are adults and children. Younger children (i.e., under six years old) would not be expected to visit the wetlands or dredge spoil areas, given the limited access; thus, adults and older children (i.e., six to less than 18 years old) were evaluated for these areas. The construction worker scenarios are evaluated for adult workers who may contact any of the site media. In this assessment, visitors or workers are assumed to contact either soils, sediments, or water on a given visit.

Figure 4-1 presents the areas evaluated in the HHRA, including the wetlands and dredge spoils area, and physical features that affect site accessibility (e.g., highways, railroad lines, paths, pedestrian bridges). Aerials of three shoreline areas shown on Figure 4-1 are included in Figures 4-2 for the dredge spoils area, 4-3 for the eastern shore/marina area, and 4-4 for the southwest corner of the lake near Harbor Brook.

4.3.2.1 Recreational Scenarios

The HHRA recreational scenarios assume that contact with lake and wetland sediments, as well as the dredge spoils area soils, will occur only during the months of May through September, when the average daily maximum temperature reaches at least 70° F (21° C).

The HHRA assumes that individuals of all ages may contact the nearshore surface sediments of Onondaga Lake, and risk estimates are provided for adults, older children, and young children contacting lake sediments. The HHRA assumes a CT exposure frequency of 32 days/year and a RME frequency of 44 days/year for contact with surface sediment in the northern part of the lake, both based on professional judgment. The average of 32 days would approximate two visits per week during the three summer months (i.e., June, July, and August) and one visit per week during one spring and one fall month (i.e., May and September). The upper-bound value of 44 days approximates three visits per week during the three summer months and one visit per week during the two months in spring and fall. These values (32 days CT and 44 days RME) are applicable to the northern basin lake sediments, the two northern wetlands (Wetlands SYW-6 and SYW-10), and the dredge spoils area (which is located adjacent to Wetland SYW-10 and has similar accessibility characteristics; see Figure 4-2). However, as these exposure estimates are assumptions based on professional judgment, the potential for higher frequency of exposure to these areas is assessed in Chapter 7, Uncertainty Assessment, as an avid recreator scenario.

In the southern part of the lake there are no public parks and, like the northern part of the lake, there are no beaches. Access to the southern part of the lake is restricted by freeways, industrial sites, the Metro plant, and heavy undergrowth (see Figure 4-4). For these reasons, visits to the southern part of the lake are expected to be less frequent than those to the northern part of the lake. The HHRA assumes, for the CT scenario, an average of one visit per month to the southern end of the lake for the three summer months and one spring and one fall month, for a total of five visits per year. For the RME scenario, the HHRA assumes one visit per week for the three summer months and one visit per month for two additional months in spring and fall, for a total of 14 visits per year. These values (five days CT and 14 days RME) are applicable to the southern basin lake sediments and the two southern wetlands, Wetlands SYW-12 and SYW-19.

Based on USEPA (1991b), for the RME case, the HHRA assumes that adults might visit the area over a period of 30 years, while the CT exposure scenario assumes that both adults and older children may visit the lake and associated areas for a shorter period of nine years. Both RME and CT estimates of exposure duration for young children and older children are limited by the age range evaluated (i.e., six years for young children and 12 years for older children [ages 6 to under 18]). Children are assumed to visit the lake as frequently as adults, but have a somewhat higher dose due to their lower body weight (i.e., 15 kg

average for children under 6, and 43 kg average for ages 6 to under 18, in comparison to 70 kg average for adults).

4.3.2.2 Construction Worker Scenario

For the construction worker scenario, workers were assumed to contact soils or sediments for 25 days/year in the RME or 10 days/year in the CT scenario over a two-year construction period, based on the assumption that construction in the area considered (lake, wetlands, or dredge spoils) is likely to consist of smaller projects, such as sewer line or extraction trench installation, rather than a large, lengthier project such as construction of warehouses, factories, or residential units. The worker's body weight is assumed to be 70 kg. Large projects were considered unlikely due to the characteristics of the exposure areas (e.g., wetlands) and foreseeable potential development of the lakeshore area, which focuses on paths, trails, and relatively small park facilities. However, it is not impossible that a larger project such as a marina could be undertaken in areas covered by this HHRA; therefore, potential risks associated with a project of longer duration are discussed in Chapter 7, Uncertainty Assessment.

4.3.3 Incidental Ingestion of Sediments and Soils

RAGS Tables 4.4 through 4.27 (Appendix B) present the exposure factors and algorithms for ingestion of nearshore lake surface sediments, and for wetlands surface sediments and dredge spoil surface and subsurface soils. Potential recreational receptor populations include adults, young children (up to six years old), and older children (6 to less than 18 years old) for lake surface sediments, and adults and older children for surface sediments in wetlands and surface soils in the dredge spoils areas. The construction worker scenario is also evaluated for contact with lake surface sediments, wetland surface sediments, and surface and subsurface soils in the dredge spoils area. The exposure evaluated for these media represents the incidental ingestion of sediments or soils as a result of direct contact with sediments or soils on the hands, followed by hand-to-mouth activity (either inadvertent or associated with eating or smoking).

No data exist in the literature that are specifically applicable to sediment ingestion rates. USEPA default values for ingestion of soils were assumed to represent ingestion rates of sediments from Onondaga Lake and the associated areas considered here (i.e., 100 percent of total daily soil ingestion is attributed to ingestion of sediments evaluated in this HHRA).

The Exposure Factors Handbook (USEPA, 1997a) identifies mean and upper-bound soil ingestion rates for young children of 100 and 400 mg/day, respectively. However, USEPA indicates that the 400 mg/day intake rate is based on short-term exposure and may not be appropriate for evaluating chronic exposure. Therefore, the 200 mg/day intake rate for young children identified in previous USEPA guidance (USEPA, 1991b) and discussed in USEPA (1997a) is used in the RME scenario, and 100 mg/day is used in the CT scenario for young children.

The Exposure Factors Handbook (USEPA, 1997a) does not provide a recommendation for an upper-bound value for soil ingestion by adults and older children. However, previous USEPA guidance

(USEPA, 1991b) identified 100 mg/day as an upper-bound intake rate. Therefore, this value was used as the intake rate for older children and adults in the RME scenario. Consistent with USEPA guidance, the mean value for adults of 50 mg/day was used in the CT recreational scenario for adults and older children (USEPA, 1997a).

A value of 330 mg/day is used for the sediment/soil ingestion rate for construction workers for both RME and CT exposures, as presented in recent soil screening guidance (USEPA, 2001c). This value represents USEPA's recent reassessment of the 480 mg/day ingestion rate (based on Hawley, 1985, as cited in USEPA, 1997a), which had been used previously (e.g., for the LCP Bridge Street site HHRA [NYSDEC/TAMS, 1998a]) for the construction worker ingestion rate.

4.4 Dermal Contact with Surface Sediments and Soils

Dermal exposure and risk calculations are based on the most recent USEPA guidance; i.e., Risk Assessment Guidance for Superfund, Volume I, Part E – Supplemental Guidance for Dermal Risk Assessment (USEPA, 2001a). The exposure assumptions and algorithms for dermal contact with sediments and soils are presented in RAGS Tables 4.4 through 4.27 (Appendix B). Dermal exposure was expressed as an absorbed dose by incorporating a contaminant-specific dermal absorption factor into the exposure equation. Dermal absorption factors reflect the desorption of the contaminant from soil and the absorption of the contaminant across the skin and into the bloodstream (USEPA, 1997a). The dermal absorption factors used in the HHRA are presented in Table 4-1.

Dermal exposures result in an estimate of absorbed dose, not the amount of contaminant that comes in contact with the skin (i.e., intake). Because oral toxicity values (i.e., carcinogenic slope factors and reference doses [CSFs and RfDs]) are usually expressed as intakes, they must be adjusted with oral absorption factors to obtain reference toxicity values expressed as an absorbed dose. In accordance with RAGS Part E, this adjustment is performed only when the oral absorption efficiency is less than 50 percent. No adjustment to the absorbed dose is made for chemicals for which the absorption efficiency is 50 to 100 percent. To calculate an adjusted toxicity value, a CSF is divided by the oral absorption factor, and an RfD is multiplied by the oral absorption factor (USEPA, 1989). Table 4-1 of this HHRA provides the oral absorption factors used for relevant COPCs in this HHRA; adjusted toxicity values are shown in the risk characterization tables presented in Appendix B.

4.4.1 Skin Surface Area Available for Contact

The skin surface area available for contact (referred to as surface area, or SA) reflects the amount of skin exposed to a contaminant in the exposure scenario. For outdoor sediment/soil exposure, USEPA dermal guidance recommends using 5,700 cm² for adults, 5,400 cm² for older children, and 2,800 cm² for young children for residential scenarios (USEPA, 2001a); these skin surface areas will be used for evaluation of recreational exposure in this HHRA. In accordance with USEPA recommendations, the same skin surface area is used for both the RME and CT scenarios.

As the construction of factories or commercial establishments is not anticipated in the areas covered by this HHRA, the USEPA default SA for commercial/industrial workers is not used, and so the adult SA value of 5,700 cm² (the same as the recreational scenario) was used for construction worker dermal exposure to sediments/soils.

4.4.2 Soil-to-Skin Adherence Factors

The soil-to-skin adherence factor (AF) refers to the amount of soil that remains deposited on the skin after contact. Adherence factors vary by soil type (e.g., moisture content, particle size), by the body part coming in contact with the soil, and by the activity being conducted while in contact with the soil. Although USEPA (1997a) reports that AFs for sediments are likely to be less than for soils because contact with water may wash the sediment off the skin, effects of particle size and washing are not specifically addressed in the following soil-to-skin AFs.

Based on data presented in USEPA's dermal guidance (USEPA, 1999) that was in effect at the time of the review of the draft Onondaga Lake HHRA and based on discussions among NYSDEC, USEPA Region 2 staff, and Honeywell, consensus AFs for adults of 0.15 mg/cm² for CT and 0.3 mg/cm² for RME were developed. While these values are slightly different than the current values presented in RAGS Part E as defaults (Exhibit C-3), the AFs used in this HHRA are consistent with the underlying studies cited in RAGS Part E (Exhibit C-2).

Considering data for children playing in wet soil (USEPA, 1999), consensus AFs of 0.2 mg/cm² for CT and 2.7 mg/cm² for RME were developed for young and older children for the recreational scenario, based on discussions among NYSDEC, USEPA Region 2 staff, and Honeywell. USEPA does not provide any specific guidance (default) AF values for the recreational scenario.

For the construction worker scenario, the AFs for utility workers of 0.9 mg/cm², which was the 95th percentile AF, was used for the RME scenario, and 0.2 mg/cm², which was the geometric mean AF (USEPA, 2001a; Exhibit 3-3), was used for the CT scenario. As both of these factors were based on data from utility workers, they are considered to be a reasonable representation of data for the type of construction that could occur in the lake, wetlands, or dredge spoils area.

4.5 Incidental Ingestion of Surface Water

RAGS Tables 4.28 through 4.31 present the exposure assumptions and algorithm for incidental ingestion of surface water while swimming or wading in Onondaga Lake (i.e., recreational exposure), or incidental ingestion of surface water while working in the lake (i.e., construction exposure). RAGS (Section 6.6.1 of USEPA, 1989) recommends a value of 50 mL/hour as the amount of water ingested while swimming, which was used to assess ingestion of lake water in the HHRA. Although visitors to the lake may not swim on every visit to the lake, exposure to surface water may also occur during other recreational activities such as water skiing. In addition, since recreational users who are boating in the lake can access surface waters in both the northern and southern portions of the lake, the exposure frequency for surface waters will be

the same in both the northern and southern portions of the lake. Therefore, the exposure frequency for swimming in the lake was assumed to be the same as exposure to northern basin sediments; i.e., 32 days (CT scenario) and 44 days (RME scenario).

The exposure time (event duration) for swimming is assumed to be 2.6 hours for the RME (based on the value presented in Exhibit 6-13 of RAGS Part A [USEPA, 1989]) and one hour for the CT (based on the Exposure Factors Handbook, Table 15-176 [USEPA, 1997a]).

For the construction worker scenario, the worker is assumed to ingest one-quarter the incidental ingestion amount of surface water (i.e., 12 mL/hr) as adults in the recreational scenario (i.e., 50 mL/hr). The exposure time is assumed to be one hour for the RME and one-half hour for the CT, based on professional judgment. Other exposure frequencies and durations for the construction worker are as described above for the recreational scenario.

4.6 Dermal Contact with Surface Water

RAGS Tables 4.28 through 4.31 (Appendix B) present the exposure assumptions and algorithm for calculating the absorbed dose from dermal contact with surface water in the lake. The risk assessment assumes that young children, older children, and adults may swim in Onondaga Lake, fully immersing their bodies. In accordance with RAGS Part E, the 50th percentile skin surface area is used for both the RME and CT exposure assessment. The total body surface areas used for the HHRA are 18,000 cm² for adults, 13,000 cm² for older children (age 6 to under 18), and 6,600 cm² for young children (up to six years old). As discussed in Section 4.4.1, the SA for the construction worker used in this HHRA is 5,700 cm².

The permeability constant reflects the rate of movement of the contaminant across the skin. Permeability constants for all the COPCs in surface water were taken from Exhibit B-2 of RAGS Part E (USEPA, 2001a) and are shown in Table 4-1 herein. For inorganic COPCs without experimentally measured permeability constants, the USEPA-recommended default value of 0.001 cm/hour was used. All other exposure assumptions are the same as discussed above for ingestion of surface water.

4.7 Exposure Point Concentrations

The EPC, or the concentration term in the exposure equation, is meant to reflect a representative concentration at the exposure point or points over the exposure period (USEPA, 1989). In evaluating the RME scenario, USEPA guidance specifies the use of the 95 percent upper confidence limit (UCL) on the mean concentration. (USEPA considers the 95 percent UCL to be the best estimate of the average concentration. As the number of data points increases, the 95 percent UCL approaches the arithmetic mean.) In most situations, assuming long-term contact with the maximum concentration in any exposure medium is not reasonable. USEPA's RAGS states that, although the average concentration (estimated by the 95 percent UCL) does not reflect the maximum concentration that could be contacted at any one time, it is regarded as a reasonable estimate of the concentration likely to be contacted over time (USEPA, 1989). It does not, however, represent a worst case or maximum possible exposure.

4.7.1 Sample Data Used for Calculation of Exposure Point Concentrations in Fish

Exposure to COPCs in fish tissue (fish tissue EPCs) was evaluated using data on measured concentrations in fillet samples from all fish of legal size from Onondaga Lake based on data collected from 1992 to 2000. As previously noted, fish species evaluated in the risk assessment include bluegill, carp, channel catfish, largemouth bass (*Micropterus salmoides*), northern pike, smallmouth bass (*Micropterus dolomieu*), walleye, and white perch (*Morone americana*). Although most species evaluated in the assessment have no minimum size limit identified by New York State, only fillets from fish approximately 6 inches long or larger were included in the risk assessment. Because concentrations of contaminants which tend to bioaccumulate (e.g., PCBs) tend to increase with fish size (i.e., in older fish), this provides a conservative means to evaluate risks for anglers who may consume smaller fish. The species evaluated, the legal size limits, and the minimum size included in the risk assessment are as follows:

Species	New York State Minimum Limit (inches)	Minimum Size in Risk Assessment (inches [cm])
Bluegill	No size limit	5.9 (15.1)
Carp	No size limit	18.0 (45.6)
Channel catfish	No size limit	14.2 (36.1)
Largemouth bass	12	11.6 (29.4)
Northern pike	No size limit	35.5 (90.2)
Smallmouth bass	12	11.5 (29.3)
Walleye	15	16.3 (41.3)
White perch	No size limit	6.3 (16.0)

A summary of the fish fillet data used in this HHRA was presented previously (see Chapter 3, Table 3-2). Detailed tables presenting all Honeywell and NYSDEC fish data used in the risk assessment are provided in Appendix A.

4.7.2 Exposure Point Concentration Calculations in Fish – Special Considerations for Mercury and Arsenic

The toxicity of two COPCs in fish tissue, mercury and arsenic, is affected by the specific contaminant form present in fish tissue.

4.7.2.1 Mercury

As noted previously, the predominant form of mercury in fish tissue is organic (methylmercury), and all mercury data in fish are considered to be methylmercury for risk assessment purposes. A more complete discussion of the form and toxicity of mercury in fish tissue is presented in Chapter 5, Toxicity Assessment

and Chapter 7, Uncertainty Assessment. Mercury in other media, such as sediment and water, is assumed to be predominantly in the inorganic form for this risk assessment (this is further discussed in Chapter 5, Section 5.2.1.12 and Chapter 7, Section 7.5.3.1).

4.7.2.2 Arsenic

The toxicity of arsenic varies significantly depending on whether it is in the organic or inorganic form. Based on the review of data on arsenic speciation in fish samples from two freshwater rivers in the northwest (Willamette and Columbia Rivers) and discussions with environmental officials in this area (from USEPA Region 10, Oregon Health Division and the Washington Department of Health), it has been assumed for this HHRA that 10 percent of the total arsenic found in Onondaga Lake fish samples is in the more toxic inorganic form. The remainder of the arsenic in fish is assumed to be in the form of relatively non-toxic organic compounds such as arsenobetain. Further discussion of this issue, including presentation of the data used to support the assumption used for this HHRA, is provided in Chapter 5, Toxicity Assessment and Section 7.5.3.2 of Chapter 7, Uncertainty Assessment.

4.7.3 Determination of Data Distribution Type

In order to utilize the appropriate equations for calculating the 95 percent UCL (USEPA, 1992a), the type of distribution of the data (normal or lognormal distribution) must be determined. Therefore, as part of data analyses conducted during the HHRA, data distributions in each affected environmental medium were evaluated and distributions of each data set in each medium were determined. In accordance with guidance from NYSDEC and USEPA Region 2 (NYSDEC and NYSDOL, 2000) and USEPA RAGS guidance (USEPA, 1992a), where data sets had fewer than ten samples, UCLs were not calculated and the maximum concentration was used as the EPC for both the RME and CT scenarios. Where data sets were larger than or equal to ten samples, the data distributions were statistically tested for normality or lognormality using the Shapiro-Wilks (for data sets of less than 50) or the D'Agostino (for data sets of 50 or more) tests. (Specific statistical tests were obtained from Gilbert, 1987, consistent with the guidance in USEPA, 1989 [RAGS Part A] and USEPA, 1992a.) Data sets were identified as best fitting either a normal or lognormal distribution (the higher W-statistic for data sets with $n < 50$, and the Y-statistic for data sets closer to zero for $n > 50$); all data sets were assigned to one of these two distribution types.

Although all data were assigned to either a normal or lognormal distribution in order to utilize the UCL calculation equations (USEPA, 1992a), not all data sets fit neatly into one of these two categories. For these non-parametric data sets, USEPA has developed a software program called ProUCL (version 2.1 was available as this HHRA was written) that first screens (tests) the data set for the data distribution type, and then calculates the UCL using a variety of approaches (USEPA, 2002c). The UCL calculations for normal and lognormal distributions are the same as those in the RAGS guidance, but several non-parametric calculations are performed that can be utilized for the 95 percent UCL where data sets are neither normally nor lognormally distributed.

The issue of the assignment of data distribution type and the effect of alternate procedures for calculation of the 95 percent UCL or other appropriate statistical estimates of the upper bound of the mean concentration are discussed in greater detail in Section 7.2.2 of Chapter 7, Uncertainty Assessment and in Appendix D.

4.7.4 Calculation of Exposure Point Concentrations

RAGS (USEPA 1989) recommends use of the 95 percent UCL of the arithmetic mean concentration to estimate the EPCs for all RME and CT scenarios because USEPA considered it to be the best estimate of the mean. More recently, USEPA has developed additional statistical tools (ProUCL) which include other estimates of the mean, which may include the 97.5 or 99 percent UCL (or some other estimate), depending on the specific characteristics of the data set. As noted above, for this HHRA, the EPC estimates were based on the 95 percent UCL; the potential effect of using (or not using) alternate statistical methods such as those included in the ProUCL software is discussed in Chapter 7, Section 7.2.2 and Appendix D.

For data sets assigned to a lognormal distribution and in accordance with supplemental guidance to RAGS (USEPA, 1992a), the UCL on the mean concentration was calculated using Land's H-statistic as follows:

$$UCL = \exp\left(\bar{y} + \frac{S_y^2}{2} + \frac{S_y \times H}{\sqrt{n-1}}\right)$$

where:

n	=	number of observations.
H	=	H-statistic for a given confidence level, n, and S _y (interpolated from statistics lookup tables in Gilbert, 1987).
exp	=	exponential function.
\bar{y}	=	average of the log-transformed data ($y = \ln[x]$).
S _y	=	standard deviation of the log-transformed data.

For normally distributed data sets, the UCL on the mean concentration is calculated using the appropriate equation from the RAGS guidance (USEPA, 1992a):

$$UCL = \bar{X} + t * \frac{s}{\sqrt{n}}$$

where:

\bar{X}	=	arithmetic mean of the (untransformed) sample data set for the compound of concern.
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s	=	sample standard deviation of the (untransformed) sample data set for the compound of concern.
t	=	the Student's t-statistic for the 95 percent confidence interval for a one-tailed distribution (taken from Gilbert, 1987). The t-statistic is a function of the number of samples collected.
n	=	number of samples in the data set.

The use of the 95 percent UCL of the mean concentration is defined as a value that, when calculated repeatedly for randomly drawn subsets of site data, equals or exceeds the true mean 95 percent of the time. Although the 95 percent UCL of the mean provides a conservative estimate of the true mean, it should not be confused with the 95th percentile of site concentration data (USEPA, 1992a). The 95 percent UCL becomes a less conservative estimate with larger data sets, for which the 95 percent UCL on the mean and the arithmetic average tend to converge. For example, for mercury in fish where there are a large number of data points, the 95 percent UCL is 1.08 mg/kg, and the arithmetic average is 1.05 mg/kg (see Appendix B, RAGS Table 3.1).

The results of the calculation of the EPCs used in the HHRA are presented in Appendix B (RAGS Tables 3.1 through 3.10) for each of the ten exposure points (i.e., fish fillets; northern basin lake sediments; southern basin lake sediments; Wetlands SYW-6, SYW-10, SYW-12, and SYW-19; surface soils from the dredge spoils area; subsurface soils from the dredge spoils area; and lake surface water).

In summary, consistent with guidance from NYSDEC and USEPA Region 2 (NYSDEC and NYSDOL, 2000), the EPC applied for both the RME and CT estimates was the lower of either the maximum value or the UCL value calculated for data sets with more than ten samples. For samples in which a COPC was not detected, a numerical value of one-half the quantitation limit was used in calculations of the average and the 95 percent UCL. In some samples, however, the quantitation limit was unusually high, with the result that one-half the quantitation limit exceeded many, if not all, of the detected values for a particular COPC. In such cases, consistent with USEPA guidance (1989), these high quantitation limits were not used; that is, the data point was excluded for that analyte and the number of samples ("n") was reduced accordingly. Similarly, consistent with USEPA guidance, where the 95 percent UCL on the mean exceeded the maximum value, the maximum value was used in the RME and CT calculations.

As noted in Section 4.7.3 above and discussed in greater detail in Appendix D, some (three of 18) chemical-specific EPC calculations generated from the ProUCL software differed by more than 50 percent from the EPC values used in this HHRA, which were calculated as described in this section. However, as the difference in the EPCs for these chemicals did not significantly change the resultant risk and hazard estimates, the ProUCL software was not used to recalculate all the EPCs.

4.7.5 Calculation of Length-Weighted Averages for Sediment Samples

As noted in Chapter 3, Section 3.2 and in Section 4.2.2, a length-weighted average (LWA) was generated as an intermediate step prior to calculating the EPCs for sediment samples from which multiple (two or

three) depth intervals were analyzed at a given location. This was done to generate a single concentration assumed to be representative of the contaminant concentration for the entire sample (core length) used in the HHRA, which was the upper 30 cm (maximum; not all samples used extended to this depth) at each location. The LWA is different than a straight arithmetic average of the data for a given location in that the LWA apportions (weighs) the concentration more heavily to the larger sample intervals, and less so to the shorter sample intervals.

For example, in a sediment sample location (core) with three sample intervals consisting of 0 to 2, 2 to 15, and 15 to 30 cm, the 0 to 2 cm concentration accounts for 2/30 (0.067) of the LWA; the 2 to 15 cm concentration accounts for 13/30 (0.433) of the LWA; and the 15 to 30 cm concentration accounts for 15/30 (0.500) of the LWA. This calculation assumes a homogenous distribution of contamination throughout each sample interval, and is consistent with how contamination is evaluated in other media. An example of this calculation, using the mercury data from southern basin sediment Station S325 (see Appendix A, Table A-3A), is shown below.

Sample Interval	Fraction of Total	Mercury Concentration	Weighted Concentration
0-2 cm	2/30 (0.067)	0.072 mg/kg	0.0048 mg/kg [0.072 × 0.067]
2-15 cm	13/30 (0.433)	0.17 mg/kg	0.0737 mg/kg [0.17 × 0.433]
15-30 cm	15/30 (0.500)	0.24 mg/kg	0.12 mg/kg [0.24 × 0.500]
Total (0-30 cm)	30/30 (1.0)	—	0.198 mg/kg [0.0048 + 0.0737 + 0.12]

Therefore, the LWA for Station S325 is 0.198 mg/kg.

For many sample locations there are only two data points. For many of these, the lengths of the sample intervals are identical – e.g., the two samples are both 15 cm (0 to 15 and 15 to 30 cm intervals); for these samples, the LWA is identical to the arithmetic average of the two concentrations. At a few stations, the two intervals are not identical; for example, at southern basin Station S402, the two intervals were 0 to 2 and 2 to 10 cm. As the total depth of that core was 10 cm, the 0 to 2 cm interval sample weighting factor is 2/10 (0.2), and the 2 to 10 cm sample interval weighting factor is 8/10 (0.8).

4.8 Summary of Exposure Assessment

The values used in the exposure assessment are provided in RAGS Tables 4.1 through 4.31 (Appendix B). The results of the exposure assessment for each potential future exposure pathway (estimates of EPCs and CDIs) are provided in the risk characterization in Appendix B, RAGS Tables 7 and 8. The CDIs calculated in this exposure assessment were combined with COPC-specific toxicity values (described in Chapter 5 and Appendix B, RAGS Tables 5 and 6) to characterize potential risks.

Several sources of uncertainty affect the estimates of exposure and, consequently, the final estimates of potential risk. All exposure assessments have uncertainties associated with the sampling and analytical data

(e.g., how well the data represent actual site conditions) and the exposure assumptions (e.g., how well the assumptions reflect actual exposure conditions). Because of these uncertainties, COPC intakes calculated for the RME scenario used reasonable conservative intake variables so that potential exposure is not underestimated. A more detailed discussion of uncertainties associated with this exposure assessment is presented in Chapter 7, Uncertainty Assessment.

5. TOXICITY ASSESSMENT

The purpose of a toxicity assessment is to evaluate the potential for contaminants of potential concern (COPCs) to cause adverse health effects in exposed persons and to define the relationship between the extent of exposure to a chemical and the likelihood and severity of any adverse health effects. The standard procedure for a toxicity assessment is to identify toxicity values for carcinogenic and non-carcinogenic effects and to summarize other relevant toxicity information. This chapter provides background on methods used to evaluate toxicity that could result following oral or dermal exposure to COPCs. Appendix E provides detailed toxicity profiles on key COPCs in this risk assessment, and Chapter 7, Uncertainty Assessment, includes a discussion of uncertainties related to the toxicity values utilized (or not utilized) for some of the site COPCs, including:

- Arsenic.
- Chromium.
- Manganese.
- Mercury.
- Methylmercury.
- 4,4'-DDD.
- Polychlorinated biphenyls (PCBs).
- Polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs).
- Polycyclic aromatic hydrocarbons (PAHs).

5.1 Derivation of Toxicity Values

The derivation of the USEPA toxicity values for the oral and dermal routes are discussed below. As the inhalation pathway is not complete at the Onondaga Lake site (see Chapter 4, Section 4.2.5 and Appendix B, RAGS Table 1), toxicity values for inhalation are not discussed.

5.1.1 USEPA-Derived Oral Toxicity Values

USEPA-derived toxicity values used in risk assessments are termed carcinogenic slope factors (CSFs) and reference doses (RfDs). Carcinogenic slope factors are used to estimate the incremental lifetime risk of developing cancer, corresponding to the chronic daily intakes (CDIs) calculated in the exposure assessment. The potential for non-carcinogenic health effects is typically evaluated by comparing estimated daily intakes with RfDs, which represent daily intakes at which no adverse effects are expected to occur over a lifetime of exposure, including exposure to sensitive subpopulations. Both CSFs and RfDs are specific to the route of exposure (e.g., by ingestion, or oral exposure). Currently, there are no CSFs or RfDs specifically for dermal exposure; therefore, oral absorption factors were used to adjust oral CSFs and RfDs to assess dermal exposure, as described in Section 5.1.2.

As indicated in RAGS (USEPA, 1989a), the primary source for toxicity values is USEPA's Integrated Risk Information System (IRIS). The IRIS database contains USEPA-verified toxicity values, in addition to

up-to-date health risk and USEPA regulatory information for many contaminants commonly detected at hazardous waste sites. USEPA extensively reviews and verifies CSFs and RfDs derived for risk assessment and, once verified and posted in IRIS, these CSFs and RfDs represent agency consensus.

USEPA's Health Effects Assessment Summary Tables (HEAST) (USEPA, 1997b), also provide USEPA-derived toxicity values that may or may not be verified at the time of publication. However, very few of the quantitative toxicological data for this HHRA were obtained from HEAST (see the COPC-specific discussion below).

Additional criteria were obtained through USEPA's National Center for Environmental Assessment (NCEA), either directly or as cited in either the USEPA Region 3 Risk-Based Criteria (RBC) tables or USEPA Region 9 Preliminary Remediation Goals (PRG) tables. Confirmation of these values, as well as additional guidance for toxicity data not available elsewhere, was obtained from NCEA through USEPA Region 2 risk assessment staff. The specific NCEA source of each quantitative toxicity value used is noted in the COPC-specific discussion below, typically as NCEA, 2002a and 2002b. The NCEA documents referenced in these two citations are also available in Appendix E of this HHRA.

The IRIS toxicity profiles, which summarize toxicity information and USEPA's derivation of oral toxicity values for COPCs, are presented for all COPCs addressed in Appendix E. Uncertainties associated with toxicity assessment and with the quantitative toxicity values used in this HHRA are discussed in Chapter 7, Uncertainty Assessment.

Chemical-specific summaries, including identification and discussion of the toxicity values used for Onondaga Lake COPCs, are presented in Section 5.2.

5.1.2 Toxicity Assessment for Dermal Exposure

With the publication of RAGS Part E (USEPA, 2001a), USEPA updated its dermal risk assessment guidance; the guidance in RAGS Part E supersedes the algorithms and parameter values in previous guidance (including both RAGS Part A [USEPA, 1989] and previous dermal guidance documents, such as the Dermal Exposure Assessment: Principles and Applications [USEPA, 1992c]).

RAGS Part E includes dermal absorption fractions from soil (RAGS Exhibit 3-4 [USEPA, 2001a], presented in HHRA Chapter 4, Table 4-1), which are used for calculation of the CDI by the dermal pathway. The current guidance includes a default value for semivolatile organic compounds (SVOCs), but no longer recommends a default dermal absorption fraction for inorganics (metals).

In the absence of dermal toxicity factors, USEPA has developed a procedure for making route-to-route (i.e., oral-to-dermal) extrapolation for systemic effects. This procedure accounts for the fact that oral RfDs and CSFs are generally expressed as the amount of substance administered (per body weight per time unit), while exposure estimates for the dermal route are expressed as an absorbed dose. The procedure uses dose-response relationships from oral administration studies and adjusts for absorption efficiency to

represent the toxicity factor in terms of absorbed dose. Where oral absorption is essentially complete (near 100 percent), the absorbed dose is essentially the same as the administered dose. However, where the gastrointestinal absorption of a particular chemical is low (e.g., 1 percent), the absorbed dose is much smaller than the administered dose, and toxicity factors based on the absorbed dose need to be adjusted to account for the difference between absorbed and administered doses.

In effect, the magnitude of the toxicity factor adjustment is inversely proportional to the absorption fraction in the critical study. As recommended by RAGS Part E (Section 4.2 of USEPA, 2001a), there are two basic criteria for adjusting the oral toxicity factor:

- The toxicity value is based on administered dose (e.g., ingestion [diet] or gavage).
- There are scientifically defensible data showing that the gastrointestinal absorption of the chemical is significantly less than 100 percent (50 percent is used by USEPA as the threshold for this factor).

If both these criteria are not met, then the USEPA guidance suggests that the assumption of complete (100 percent) absorption may be made (i.e., no adjustment to the oral toxicity factor). USEPA also specifically suggests that default assumptions of 100 percent absorption be made for inorganics and organics for which specific values are not available (see RAGS E, Exhibit 4-1 [USEPA, 2001a], summarized in this HHRA in Chapter 4, Table 4-1). Note that the importance of the dermal pathway, relative to ingestion, increases as the gastrointestinal absorbance (ABS_{GI}) value decreases; therefore, the default assumption of 100 percent absorption for chemicals without chemical-specific data may result in an underestimate of risk via the dermal pathway for these chemicals.

A CSF is divided by the oral absorption factor, and an RfD is multiplied by the oral absorption factor to calculate the adjusted toxicity value. The oral absorption values assumed in this HHRA are presented in Chapter 4, Table 4-1; adjusted toxicity values are presented in Appendix B, RAGS Tables 5.1 and 6.1.

5.2 Chemical-Specific Summaries and Toxicity Values for Onondaga Lake Contaminants of Potential Concern

Summary information for all the identified COPCs, as presented in Chapter 3, Table 3-1, for this HHRA is provided below. The summaries are organized in the following parameter groupings:

- Metals and organometallic compounds (Section 5.2.1).
- Volatile organic compounds (VOCs) (Section 5.2.2).
- SVOCs (Section 5.2.3).
- PAHs (Section 5.2.4).
- Pesticides (Section 5.2.5).
- PCBs (Section 5.2.6).
- PCDD/PCDFs (Section 5.2.7).

5.2.1 Metals and Organometallic Compounds

The discussion below provides brief summaries for the metals and organometallic compounds identified as COPCs for this HHRA. The full IRIS summaries and related Agency for Toxic Substances and Disease Registry (ATSDR) and NCEA documents are provided in Appendix E. These metals and organometallic compounds include:

- Aluminum.
- Antimony.
- Arsenic.
- Barium.
- Cadmium.
- Chromium.
- Copper.
- Cyanide.
- Iron.
- Lead.
- Manganese.
- Mercury – General.
- Methylmercury.
- Mercury – Inorganic.
- Nickel.
- Selenium.
- Thallium.
- Vanadium.
- Zinc.

5.2.1.1 Aluminum

Aluminum is an abundant element in the earth's crust, and is a constituent of many commercial drug and cosmetic products (e.g., antacids, buffered aspirin, and anti-perspirants in the form aluminum chlorhydrate). Although low-level exposure to aluminum is not known to be harmful, and aluminum is present at low concentrations in water and many foods, it is not a necessary human nutrient (ATSDR, 1999a,b).

Aluminum was identified as a COPC in soils/sediments. It is not expected to be a significant contaminant in Onondaga Lake surface water. Although there are no data from the 0 to 3 m depth interval, aluminum concentrations in samples from greater depths (6 and 12 m below the lake surface) were below screening criteria. There are no quantitative risk or toxicity factors for aluminum published in IRIS or HEAST. The oral RfD utilized for the Onondaga Lake HHRA, 1 mg/kg-day, is a provisional value from NCEA, as shown on the USEPA Region 3 RBC and Region 9 PRG tables (USEPA, 2002a,b).

5.2.1.2 Antimony

Antimony has been identified as a COPC in both fish tissue and soils/sediments. Although antimony is not expected to be a significant contaminant in surface water, no samples from the 0 to 3 m interval were analyzed. Antimony was detected in two out of four samples from deeper intervals (6 m) at concentrations (about 25 µg/L) exceeding the drinking water screening criterion (Region 3 tap water RBC of 15 µg/L). Antimony is normally present at only very low concentrations in the environment (typically less than 1 mg/kg in soil). It is also present at very low concentrations in foods (about 1 µg/kg or less), with a typical dietary intake of about 5 µg/day (ATSDR, 1992). Antimony is widely used in the production of alloys (including, among many others, pewter and storage battery plates); antimony compounds have a wide variety of uses, including as a flame retardant in textile and plastic products (Sittig, 1991; ATSDR, 1995a).

Generally, the primary exposures to antimony are via skin contact and inhalation of dust and fumes. Oral ingestion may occur, causing headache, nausea, sleeplessness, and dizziness, with liver and kidney degeneration as later manifestations. With some exceptions (antimony trisulfide and antimony pentachloride), antimony compounds are less toxic than antimony (Sittig, 1991). However, the exact form in which antimony occurs at the Onondaga Lake site is unknown.

The oral RfD for antimony published in IRIS is 4×10^{-4} mg/kg-day; due to the limitations of the data on which the RfD is based, confidence in this RfD is considered low (IRIS, 2000a).

5.2.1.3 Arsenic

Arsenic was identified as a COPC in both fish and soils/sediments. Although arsenic is not expected to be a significant contaminant in surface water, no samples from the 0 to 3 m interval were analyzed. Arsenic was detected in one out of four samples from deeper intervals (12 m) at a concentration (1.1 µg/L) just over the detection limit (1 µg/L) and exceeding the Region 3 tap water screening criterion of 0.045 µg/L. Arsenic is a naturally occurring element in the earth's crust and in soil; although natural soil concentrations are usually low, they vary locally. Arsenic compounds have a variety of uses, including agricultural (insecticides, herbicides, larvicides, and pesticides), as a wood preservative, in leather tanning, some specialty paints and enamels, and to prevent sludge formation in lubricating oils (ATSDR, 2001a; Sittig, 1991).

As IRIS publishes a CSF and an oral RfD (IRIS, 2000b), arsenic was evaluated both as a toxic and a carcinogenic chemical. The quantitative data in IRIS are for "Arsenic, inorganic;" however, the specific form of arsenic, especially in fish, is unknown. Organic compounds of arsenic are generally less toxic than inorganic compounds (ATSDR, 2001b).

The oral RfD utilized is 3×10^{-4} mg/kg-day; confidence in this value is described as "medium." The oral CSF is $1.5 \text{ (mg/kg-day)}^{-1}$. Arsenic is classified as Group A – known human carcinogen (IRIS, 2000b).

As noted in Chapter 4, Section 4.7.2.2, the toxicity of arsenic varies significantly depending on whether it is in the organic or inorganic form. Some studies suggest that the majority of arsenic (over 90 percent) typically found in fish may be in the less toxic (organic) form. The Oregon Department of Human Services (ODHS), for example, notes that “[m]ost of the arsenic in fish is in the relatively non-toxic form called arsenobetain (fish arsenic)” (ODHS, 2002). Recent data for fish tissue samples that were analyzed for both total arsenic and inorganic arsenic were received from USEPA and reviewed for this HHRA. These data, which are discussed in greater detail in Chapter 7, Section 7.5.3.2, support an estimate of 10 percent as the assumed fraction of arsenic in fish as inorganic arsenic. The arithmetic average of the 42 samples reviewed was 6.4 percent inorganic arsenic, with a 95 percent UCL on the arithmetic mean of 10.7 percent inorganic arsenic. Thus, it has been assumed for this HHRA that 10 percent of the total arsenic found in Onondaga Lake fish samples is in the more toxic inorganic form. The remainder of the arsenic in fish is assumed to be in the form of relatively non-toxic organic compounds such as arsenobetain. The RfD of 3×10^{-4} mg/kg-day is applied to the 10 percent of the arsenic assumed to be inorganic. The organic fraction of the arsenic in fish tissue is assumed to be non-toxic for the purposes of risk quantitation.

5.2.1.4 Barium

Barium was identified as a COPC in northern and southern basin sediments and in Wetland SYW-19 sediments. It is not expected to be a significant contaminant in Onondaga Lake surface water. Although there are no data from the 0 to 3 m depth interval, barium concentrations in samples from greater depths (6 and 12 m below the lake surface) were below screening criteria. Barium is a naturally occurring element in soils, normally in the form of a barium compound, rather than as pure or metallic barium. Barium sulfate is used medically for x-ray testing; other barium compounds are used for the manufacture of some white pigments; chlorine; sodium hydroxide; rubber vulcanizing; papermaking; and in the brick and tile, pyrotechnic, and electronic industries. Barium compounds are present in a wide variety of commercial and household products, including paints, paper, soap, rubber, and linoleum (Sittig, 1991; ATSDR, 1995b).

Toxicological data for barium are present in IRIS (“Barium and Compounds”). The oral RfD is 7×10^{-2} mg/kg-day; confidence in this value is reported as medium (IRIS, 1999).

Barium has also been reviewed for carcinogenicity. Under the 1986 guidelines for carcinogenic risk assessment, barium is classified as Group D – not classifiable as to human carcinogenicity. Under the 1996 guidelines, barium is considered “not likely to be carcinogenic to humans following oral exposure” (IRIS, 1999).

5.2.1.5 Cadmium

Cadmium is a naturally occurring element in soil, typically in the forms of cadmium oxide, cadmium chloride, or cadmium sulfate. Cadmium is corrosion-resistant and is used as a protective coating for metals (e.g., galvanization). It is also used in storage batteries; as a stabilizer in PVC; electronics; and in aircraft and automobile manufacturing. Cadmium compounds are also used as fungicides, insecticides, and nematocides (Sittig, 1991; ATSDR, 1999c).

Although inhalation is typically the primary exposure route for cadmium, long-term ingestion of low concentrations of cadmium leads to accumulation of cadmium in the kidneys and may cause kidney disease (ATSDR, 1999c). Other effects may be lung damage and bone weakness. Adverse effects from skin (dermal) contact with cadmium have not been established.

Cadmium has been identified as a COPC in soil/sediment and in surface water. There are two different oral RfDs for cadmium, depending on the medium of ingestion (IRIS, 1998a). The RfD for “food,” 1×10^{-3} mg/kg-day, is used in this HHRA for soil/sediment ingestion; the RfD for “water,” 5×10^{-4} mg/kg-day, is used for the surface water ingestion pathway.

Cadmium is classified by USEPA as Group B1 – probable human carcinogen, based on animal data and limited human data. USEPA has concluded that the available data are sufficient only for quantitative carcinogenicity assessment by inhalation (IRIS, 1998a); no oral CSF has been established and, therefore, cadmium will not be assessed quantitatively as a carcinogen for this HHRA.

5.2.1.6 Chromium

Chromium has been identified as a COPC in fish tissue, soil/sediment, and Onondaga Lake surface water. Chromium is a naturally occurring element in soils and rocks. It is typically found in the environment as chromium(III); metallic chromium (chromium[0]) and chromium(VI) are generally produced by industrial processes. Chromium(0) is typically used in making steel alloys. Chromium(III) and (VI) are used for dyes and pigments, leather tanning, wood preserving, and chrome plating (ATSDR, 2001b; Sittig, 1991).

Chromium is considered an essential nutrient, with a recommended daily intake (RDI) of 120 µg/day (0.120 mg/day) (US Food and Drug Administration [US FDA]; 21 CFR 101.9[c][8][iii]), and is found in grains, egg yolks, meats, and nuts. However, ingestion of large quantities of chromium(III) or (VI) may cause health problems.

USEPA has established quantitative toxicity factors for chromium(III) and (VI). The oral RfD for chromium(III) is 1.5 mg/kg-day; confidence in this value is considered low (IRIS, 1998b). The oral RfD for chromium(VI) has been set at 3×10^{-3} mg/kg-day; confidence in this RfD is also low (IRIS, 1998c).

Chromium(VI) has been established as a known human carcinogen (Group A) by inhalation. However, the carcinogenicity of chromium(VI) by ingestion (oral route) has not been determined, and is classified as Group D – not classifiable as to human carcinogenicity (IRIS, 1998c). There is no oral CSF for chromium(VI) in IRIS, HEAST, or other agency source; therefore, ingestion of chromium will not be assessed quantitatively in the Onondaga Lake HHRA for carcinogenicity.

The carcinogenic potential for chromium(III) has been reviewed by USEPA. Chromium(III) has been assigned to Group D – not classified, due to inadequate data (IRIS, 1998b). The classification of chromium(VI) as a known human carcinogen raises concern for the carcinogenic potential of chromium(III)

(IRIS, 1998b); nonetheless, there are no quantitative data for the assessment of chromium(III) for carcinogenicity by the oral route.

5.2.1.7 Copper

Copper has been established as a COPC in soil/sediment; however, it was not identified as a COPC in fish tissue or in surface water. Copper occurs naturally in soils, and is present in plants and animals. It is used to make metal (e.g., pipes, pennies, wire); it is also used in water treatment; to treat some plant diseases; and to preserve wood, leather, and fabrics (ATSDR, 1999d).

Copper is considered an essential nutrient (ATSDR, 1999d), with an RDI of 2.0 mg/day (US FDA; 21 CFR 101.9[c][8][iii]).

An RfD of 3.7×10^{-2} mg/kg-day is reported in the USEPA Region 9 PRG tables (USEPA, 2002b), with a citation of HEAST as the source of the value; essentially the same value (4×10^{-2}) is listed in the Region 3 RBC tables (USEPA, 2002a), with the same attribution to HEAST. However, the HEAST data (USEPA, 1997b) consulted for this HHRA did not contain an oral RfD for copper. NCEA has confirmed an oral RfD of 4×10^{-2} for copper (NCEA, 2002a; see also Appendix E of this HHRA).

The carcinogenic potential for copper has been reviewed by USEPA. Copper has been assigned to Group D – not classified, due to inadequate data; copper is being reassessed under the IRIS program (IRIS, 1998d).

5.2.1.8 Cyanide

Cyanide is a COPC in fish, sediment, and dredge spoils. It is not expected to be a significant contaminant in Onondaga Lake surface water. Although there are no data from the 0 to 3 m depth interval, cyanide was detected in one of four samples from greater depths (6 and 12 m below the lake surface) at a concentration (171 µg/L) below screening criteria for most cyanide salts (e.g., potassium cyanide). Toxicity data are available in IRIS for free cyanide (IRIS, 1997a) and a number of individual cyanide compounds (e.g., sodium cyanide and calcium cyanide [IRIS, 1997b,c]). The cancer potential of these compounds has not been determined (i.e., they have been assigned to Group D), although oral RfDs are available. The exact form in which cyanide exists in the Onondaga Lake environment is unknown. The toxicity data for the three forms of cyanide that were considered most likely to be present (i.e., free cyanide, calcium cyanide, and sodium cyanide) were reviewed; the oral RfDs ranged from 4×10^{-2} mg/kg-day for calcium cyanide and sodium cyanide (IRIS, 1997b,c) to 2×10^{-2} for free cyanide (IRIS, 1997a). The RfD for free cyanide has been selected for use in the quantitative HHRA.

The carcinogenic potential for cyanide has been reviewed by USEPA. Cyanide has been assigned to Group D – not classified, due to inadequate data (IRIS, 1997a), and is not assessed for cancer toxicity in this HHRA.

5.2.1.9 Iron

Iron was identified as a COPC in all the sediment and dredge spoil exposure areas, but not in fish or surface water. Iron occurs naturally, typically as iron(II) (ferrous iron) and iron(III) (ferric iron), in the form of iron oxides. Iron is the fourth most abundant element, comprising about four percent of the earth's crust (Standen, 1967). Iron is considered an essential nutrient, with an RDI of 18 mg/day (US FDA; 21 CFR 101.9[c][8][iii]). However, short-term ingestion of large amounts of iron may cause drowsiness, sluggishness, vomiting, and diarrhea; ingestion of a half-ounce (about 14 g) may cause death in an adult (Sittig, 1991). Excessive long-term ingestion of iron may result in accumulation in the body, especially in the liver, spleen, and lymphatic system.

There is no IRIS toxicological summary for iron, and toxicity data for iron are not present in HEAST. The RfD for iron used for this HHRA, 0.3 mg/kg-day, was developed by NCEA, as reported in the both the Region 3 RBC and Region 9 PRG tables (USEPA, 2002a,b). As there are no data on the cancer potential for iron, it is not assessed for cancer toxicity in this HHRA.

5.2.1.10 Lead

Lead has been identified as a COPC in southern basin sediments, although not in fish or surface water. Historically, lead was used in many products, including paint, gasoline, ceramics, caulking, and solder; however, most of these uses have been dramatically reduced due to bans and restrictions on the use of lead in consumer products (ATSDR, 1999e). Lead is still present in the environment as a result of these uses, and is still used in batteries, ammunition, x-ray shielding, and solder and pipes, although at reduced levels.

Lead can affect most organs and systems of the body, but the greatest concern with regard to lead is its effect on the development of young children. The Centers for Disease Control (CDC) considers a blood level of 10 µg/dL to be elevated. However, no RfD has been established for lead. Based on an uptake model for lead, USEPA has established a residential soil screening level for lead of 400 mg/kg (OSWER directive 9355-4.12, USEPA, 1994). While the absence of an RfD precludes a quantitative assessment, lead was screened qualitatively in this HHRA by comparison with the residential soil screening level.

USEPA (IRIS, 1997d) considers lead to be a Group B2 carcinogen, based on sufficient animal data, but inadequate human data. However, no CSF has been established.

Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to this contaminant. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, USEPA's Carcinogen Assessment Group recommends that a numerical estimate not be used (IRIS, 1997d).

5.2.1.11 Manganese

Manganese has been identified as a COPC in fish tissue, soil/sediment, and Onondaga Lake surface water. Manganese is a nutrient, as discussed below, and is present at low levels in food, air, and water. Found naturally in the environment, typically as compounds with sulfur, oxygen, chlorine, or other elements, manganese is used in the manufacture of some pesticides and gasoline additives (ATSDR, 2001c).

Manganese is considered an essential nutrient, with an RDI of 2 mg/day (US FDA; 21 CFR 101.9[c][8][iii]). As with most essential nutrients, too much or too little intake of manganese may cause health problems. USEPA has established an oral RfD of 0.14 mg/kg-day for manganese. The IRIS RfD includes manganese from all sources, including diet. The explanatory text in IRIS recommends using a modifying factor of three when calculating risks associated with non-food sources (IRIS, 1997e). IRIS also recommends subtracting dietary exposure (default assumption of 5 mg/day [USEPA Region 3 default {RBC introductory text; FAQ #2, USEPA Region 3; provided in Appendix C of this HHRA}], consistent with the high end of the safe range [2 to 5 mg/day] for persons over ten years old, from the National Research Council [NRC] as cited in ATSDR, 2001c). Thus, the IRIS RfD of 0.14 mg/kg-day has been lowered by a factor of 2×3 , or 6, to reflect manganese from both food and non-food, or most environmental, sources. Therefore, the RfD for manganese from non-dietary sources utilized in this HHRA is 0.023 mg/kg-day.

USEPA considers the confidence level of the oral RfD to be medium (IRIS, 1997e). The USEPA Region 3 RBC tables list the oral RfD for “Manganese – Food” at 0.14 mg/kg-day, but list an oral RfD of 2×10^{-2} mg/kg-day for “Manganese – Non-Food” (USEPA, 2002a). The Region 9 PRG tables have only a single entry for manganese, “Manganese and Compounds,” with an RfD of 2.4×10^{-2} mg/kg-day (USEPA, 2002b). This is essentially the same value as used for non-dietary ingestion of manganese in this HHRA (2.3×10^{-2} mg/kg-day); however, the unmodified IRIS RfD (0.14 mg/kg-day) is used for fish ingestion and incidental ingestion of surface water.

Manganese has been classified in Group D – not classifiable as to human carcinogenicity. USEPA states that “existing studies are inadequate to assess the carcinogenicity of manganese” (IRIS, 1997e).

5.2.1.12 Mercury – General

Mercury is a principal COPC in environmental media in the Onondaga Lake National Priorities List (NPL) site. Historically, mercury has been used in the production of chlorine gas and caustic soda (several of these facilities were present in the Onondaga Lake vicinity, as noted in the remedial investigation [RI] report), and also in thermometers, dental fillings, and batteries (ATSDR, 1994, 1999). Analytical data for mercury are typically for total mercury; that is, the specific form (organic or inorganic, or specific valence state) is unknown. However, it is understood that mercury is present in biota in organic form as methylmercury.

Mercury in other matrices (i.e., soil/sediment and surface water) is evaluated in the form reported (i.e., total mercury, assumed to be inorganic, or methylmercury, assumed to be organic) for matrices for which data

for both forms are available (northern basin sediments, southern basin sediments, and Onondaga Lake surface water). For the four wetland exposure areas (Wetlands SYW-6, 10, 12, and 19) and the dredge spoils area, no analyses for methylmercury were conducted.

For the Onondaga Lake BERA (TAMS, 2002a), it has been assumed, based on a literature review, mercury/methylmercury data from the nearby LCP Bridge Street site, and Onondaga Lake sediment data (see Appendix B, RAGS Tables 3.2 and 3.3), including sediment data from areas that are not included in this HHRA, that 1 percent of the total mercury in wetland sediments is methylmercury, a value considered protective of the ecosystem. The BERA assumed that the mercury in soils – e.g., dredge spoils – is entirely in the inorganic form. The basis for the estimates of the fraction of methylmercury is discussed in greater detail in Chapter 6, Section 6.3.1.1 of the Onondaga Lake BERA (TAMS, 2002a) and summarized in Chapter 7, Section 7.5.3.1 of this HHRA. For the HHRA, differences in the assumed fraction of mercury which is present as methylmercury in wetland sediments has no significant impact on hazard calculations (see discussion in Chapter 7, Section 7.5.3.1 of this HHRA).

5.2.1.13 Methylmercury

Methylmercury has been identified as a COPC in fish, lake and wetland sediments, and surface water. It is a toxic chemical with which a number of adverse health effects have been associated in both human and animal studies. The largest amount of data exist on neurotoxicity, particularly in developing organisms. USEPA considers the central nervous system the most sensitive target organ on which there are data suitable for development of an RfD (Section I.A of IRIS, 2001a). As noted in Chapter 3, Section 3.1, the available data show that the mercury in Onondaga Lake fish samples is 90 percent or more in the form of methylmercury; therefore, the RfD for methylmercury is used in this HHRA for calculation of health risks associated with fish consumption. There are also a limited amount of methylmercury data for the northern and southern basin lake sediments and for surface water; to the extent that data are available, methylmercury is assessed quantitatively for non-cancer hazards in these matrices also.

Estimates of potential risks associated with methylmercury are based on USEPA's current RfD of 1×10^{-4} mg/kg-day (IRIS, 2001a). The current RfD, verified for use in 1995 and reassessed in 2001 by USEPA, is based on protection against adverse effects that may occur following prenatal exposure during gestation. USEPA initially derived the current RfD value from data for Iraqi infants accidentally exposed to alkyl mercury in grain during gestation in 1971 (Marsh et al., 1987, as cited in IRIS, 2001a). In this population, delayed walking was reported in infants whose mothers had elevated hair methylmercury concentrations. USEPA subsequently applied analyses of more recent studies as reported by the NRC (NRC, 2000). NRC (2000) considered three epidemiological longitudinal developmental studies suitable for quantitative risk assessment: the Seychelles Islands; the Faeroe Islands; and New Zealand studies. The Seychelles study has yielded no evidence of impairment related to methylmercury exposure thus far, while the other two studies have found adverse effects for some neuropsychological endpoints. The Faeroe Islands study is the larger of the latter two studies, and was therefore recommended by NRC for use in derivation of an RfD.

USEPA agreed with the NRC's conclusions, and has proposed the same numeric RfD (0.1 µg/kg-day) based on neuropsychological findings from the Faeroe Islands data. USEPA used a benchmark dose (BMD) approach to quantify a dose-effect relationship between methylmercury in cord blood and a neurological endpoint. A BMD limit of 58 µg/L cord blood was estimated based on findings from the Boston Naming Test, a neuropsychological evaluation. A methylmercury intake level associated with a blood level of 58 µg/L was calculated to be 1.0 µg/kg-day. A total uncertainty factor of 10 was then applied, with the resulting RfD (i.e., 0.0001 mg/kg-day), as derived from the Faeroe Islands data, unchanged from the RfD derived from the Iraqi data.

Methylmercury has been classified as Group C – possible human carcinogen, based on inadequate data in humans and limited evidence of carcinogenicity in animal studies (IRIS, 2001a). No oral CSF has been established by USEPA, and, therefore, methylmercury is not assessed quantitatively for cancer risks in this HHRA.

5.2.1.14 Mercury – Inorganic

Inorganic mercury has been identified as a COPC in all matrices except fish. As discussed above, all mercury in fish is assumed to be methylmercury. Published toxicity data are available in IRIS for elemental mercury (Hg[0]) (IRIS, 1997f) and mercuric chloride (HgCl₂) (IRIS, 1997g). As elemental mercury has not been observed in water or sediments, and the toxicity data for elemental mercury does not include an oral RfD, the IRIS (1997g) toxicity data for mercuric chloride (i.e., oral RfD of 3×10^{-4} mg/kg-day) has been used for this quantitative HHRA.

USEPA has identified mercuric chloride as a possible human carcinogen (Group C), based on an absence of data in humans and limited evidence of carcinogenicity in animal studies (IRIS, 1997g). No oral CSF for mercuric chloride has been established by USEPA; therefore, it is not assessed quantitatively for cancer risks in this HHRA.

5.2.1.15 Nickel

Nickel was identified as a COPC in the southern basin sediments; it is not a COPC in other Onondaga Lake soil/sediments, fish tissue, or surface water. Nickel is used in alloys with many other metals, including stainless steel; is used as a catalyst; and is used in the manufacture of coins, batteries, enamels, and glass (Sittig, 1991; ATSDR, 1997a).

Although there is no RDI for nickel, it is thought that a small amount of nickel may be necessary for human health (ATSDR, 1997a), and the average dietary intake of nickel is approximately 300 µg/day (Sittig, 1991).

USEPA has established an oral RfD of 2×10^{-2} mg/kg-day for soluble salts of nickel (IRIS, 1998e). Confidence in this value is considered medium. USEPA has not evaluated the soluble salts of nickel for human carcinogenicity, and is reassessing the toxicity data for nickel (IRIS, 1998e).

5.2.1.16 Selenium

Selenium has been identified as a COPC in fish tissue only; it was not identified as a COPC in soil/sediment or surface water. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for selenium, it was not detected in samples from greater depths in the lake (6 and 12 m).

The major commercial use of selenium is in the manufacture of rectifiers. It is also used as a pigment; a vulcanizing agent for rubber; in the electronics and photographic industries; and in anti-dandruff shampoos (Sittig, 1991; ATSDR, 1997b).

Selenium is considered an essential nutrient, with an RDI of 70 µg/day (0.07 mg/day) (US FDA; 21 CFR 101.9[c][8][iii]). However, ingestion of excess selenium may cause brittle hair and deformed nails, and a loss of feeling in the extremities (ATSDR, 1997b). USEPA has established an RfD of 5×10^{-3} mg/kg-day for "Selenium and Compounds;" confidence in this value is considered high (IRIS, 1997h).

The carcinogenic potential for selenium has been reviewed by USEPA. Selenium has been assigned to Group D – not classifiable, due to inadequate data (IRIS, 1997h), and is not assessed for cancer toxicity in this HHRA. However, it is noted that one selenium compound – selenium sulfide – is classified as Group B2 (probable human carcinogen) (IRIS, 1997i).

5.2.1.17 Thallium

Thallium has been identified as a COPC in soil/sediment; it is not a COPC in fish tissue and is not expected to be a significant contaminant in Onondaga Lake surface water. Although there are no data from the 0 to 3 m depth interval, thallium was not detected in samples from greater depths (6 and 12 m below the lake surface). Thallium and its compounds are used in fungicides, rodenticides, and insecticides; in fireworks; in alloys with mercury in electrical switches; in photoelectric cells; optical instruments; electronics and semiconductors; and dyes and pigments, among other uses (Sittig, 1991; ATSDR, 1995c).

There are no generic toxicity data for thallium (e.g., for thallium or thallium and compounds) in IRIS, although there are toxicity data for a number of specific thallium compounds (e.g., thallium chloride, thallium sulfate). The oral RfD for thallium used in this HHRA is 6.6×10^{-5} mg/kg-day, the source for which is cited as IRIS on the USEPA Region 9 PRG tables (USEPA, 2002b). However, as of June 2002, no entry under the heading "Thallium" or "Thallium and Compounds" was found on the IRIS web site. A similar oral RfD value, 7×10^{-5} mg/kg-day, is listed on the Region 3 RBC tables (USEPA, 2002a), although Region 3 cites the source of the value as "other" (i.e., not IRIS, HEAST, or NCEA). These values are also similar to those for several of the thallium compounds for which there are IRIS RfDs; specifically, thallium carbonate (IRIS, 1997j), thallium sulfate (IRIS, 1997k), and thallium chloride (IRIS, 1997l), all of which have a published oral RfD of 8×10^{-5} mg/kg-day.

The IRIS files for all the thallium compounds reviewed list a classification of Group D – not classifiable as to human carcinogenicity, due to lack of data (IRIS, 1997j,k,l).

5.2.1.18 Vanadium

Vanadium has been identified as a COPC in fish tissue and soil/sediment, but is not expected to be a significant contaminant in Onondaga Lake surface water. Although there are no data from the 0 to 3 m depth interval, vanadium was not detected in samples from greater depths (6 and 12 m below the lake surface). Vanadium is a naturally occurring element that is typically found as a compound with oxygen, sodium, sulfur, or chlorine (ATSDR, 1995d). The principal use of vanadium is in metals used in the automotive and aircraft industries; lesser amounts are used in the manufacture of plastics, rubber, and ceramics (Sittig, 1991; ATSDR, 1995d).

Short-term health effects have been noted from the inhalation of vanadium, although the effects of ingestion are not known (ATSDR, 1995d). The only vanadium compound for which there is an IRIS file is vanadium pentoxide (IRIS, 1997m); however, this compound was not considered to be representative of the forms in which vanadium may be present at the Onondaga Lake site. The oral RfD for vanadium used in this HHRA is 7×10^{-3} mg/kg-day, as published in HEAST (USEPA, 1997). This oral RfD is close to the IRIS value for vanadium pentoxide (9×10^{-3} mg/kg-day [IRIS, 1997m]). NCEA indicated that there was no additional guidance available for vanadium (NCEA, 2002c); therefore, the HEAST value is the best available toxicity data.

Vanadium has not been classified with regard to its human cancer-causing potential by USEPA, the International Agency for Research on Cancer (IARC), or the Department of Health and Human Services (DHHS) (ATSDR, 1995d).

5.2.1.19 Zinc

Zinc has been identified as a COPC in fish tissue only; zinc concentrations did not exceed screening criteria in soil/sediment or surface water.

Zinc is considered an essential nutrient, with an RDI of 15 mg/day (US FDA; 21 CFR 101.9[c][8][iii]). Ingestion of levels substantially higher than this amount (in the 100 to 250 mg/day range) may cause anemia, pancreas damage, and decreased HDL ("good") cholesterol (ATSDR, 1995e). USEPA has established an oral RfD for zinc of 0.3 mg/kg-day; zinc is being reassessed in the IRIS program (IRIS, 1998f). Confidence in the oral RfD is considered medium.

The carcinogenic potential of zinc is assessed as Group D – not classifiable, due to inadequate evidence in humans and animals (IRIS, 1998f).

5.2.2 Volatile Organic Compounds

The discussion below provides brief summaries for the VOCs identified as COPCs for this HHRA. While VOCs were identified as COPCs in soil/sediment and in Onondaga Lake surface water, they were not

identified as COPCs in fish tissue. The full IRIS summaries and related ATSDR documents are provided in Appendix E. These VOCs include:

- Benzene.
- Bromodichloromethane.
- Chlorobenzene.
- Chloroform.
- Methylene chloride (dichloromethane).
- Xylenes.

Although dichlorobenzenes and naphthalene are sometimes analyzed and reported as VOCs, for this HHRA those compounds are discussed as SVOCs (with naphthalene in the PAH subset of SVOCs).

5.2.2.1 Benzene

Benzene is a volatile constituent of crude oil and refined gasoline and motor fuels. It is also used extensively in industry (it is one of the 20 most-produced chemicals in the US); as a raw material or chemical intermediate for the production of other chemicals, such as styrene and phenols; and in the manufacture of plastics, resins, detergents, pharmaceuticals, pesticides, and dyes (ATSDR, 1997c; Sittig, 1991).

Short-term effects of ingesting large amounts of benzene include vomiting, stomach irritation, convulsion, increased heart rate, and ultimately death. The principal long-term toxic effects of ingestion or inhalation of benzene are blood-related, causing several forms of leukemia and harmful effects on the bone marrow, resulting in anemia (IRIS, 2001b; ATSDR, 1997c).

There are no non-cancer toxicity data for benzene published in IRIS. However, both the Region 3 RBC and Region 9 PRG tables, citing NCEA as the source, list an oral RfD of 3×10^{-3} mg/kg-day for benzene; this value is used in the quantitative HHRA. The NCEA issue paper (NCEA, 2002a) confirms this value. NCEA staff indicated that although this paper is no longer current, which is defined as being more than three years old, the provisional RfD has been reviewed by NCEA staff and approved for use on the Onondaga Lake project (NCEA, 2002a; see also Appendix E of this HHRA).

Benzene is classified by USEPA as Group A – known human carcinogen for all routes of exposure. The IRIS oral CSF (USEPA, 2001b) is presented as a range from 1.5 to 5.5×10^{-2} (mg/kg-day)⁻¹. The oral slope factor has been derived by USEPA from the inhalation risk factor. The range of oral slope factors is related to uncertainty regarding the absorption of benzene in the body. As some animal studies have shown that absorption to be as high as 100 percent, the high end, or more conservative, of the range of CSFs (i.e., 5.5×10^{-2} [mg/kg-day]⁻¹) was used for the Onondaga Lake HHRA.

5.2.2.2 Bromodichloromethane

Bromodichloromethane is not produced to any significant extent as a commercial product in the US. It is one of a class of chemicals referred to as trihalomethanes, a class which also includes chloroform, and is formed as a byproduct in water disinfected by chlorination (ATSDR, 1999f).

There are no adequate health studies on the effects of bromodichloromethane on humans; however, based on animal studies, the liver, kidneys, and central nervous system are the likely target organs. On the basis of these data, USEPA has established an oral RfD of 2×10^{-2} mg/kg-day for bromodichloromethane (IRIS, 1997n).

Bromodichloromethane is classified by USEPA as Group B2 – probable human carcinogen, based on animal data. The oral CSF is 6.2×10^{-2} (mg/kg-day)⁻¹ (IRIS, 1997n).

5.2.2.3 Chlorobenzene

Chlorobenzene was once a large-production bulk chemical in the US (including at the Honeywell Willis Avenue Chlorobenzene site), although production has dropped significantly since 1960. It was used as an intermediate in the production of other chemicals, including phenol and DDT, and current uses include the manufacture of anilines, dyes, and pesticides, and as a de-greaser for auto parts (Sittig, 1991; ATSDR, 1999g).

There are no adequate health studies on the effects of chlorobenzene on humans. Animal studies suggest that the liver, kidneys, and central nervous system are the organs most likely to be affected by chlorobenzene (ATSDR, 1999g). The oral RfD for chlorobenzene is 2×10^{-2} mg/kg-day (IRIS, 1997o); USEPA indicates that the confidence in this value is medium.

The cancer-causing potential of chlorobenzene has been reviewed by USEPA, but the data were not sufficient; therefore, chlorobenzene was assigned to Group D – not classified as to human carcinogenicity (IRIS, 1997o).

5.2.2.4 Chloroform

Chloroform was once used as an anaesthetic, but that use has been long discontinued due to its toxicity. It is currently used as a solvent (especially for the lacquer industry); in the preparation of pharmaceuticals; and in the manufacture of other products. It is one of a class of chemicals referred to as trihalomethanes, a class which also includes bromodichloromethane, discussed above, and is formed as a byproduct in water disinfected by chlorination (Sittig, 1991; ATSDR, 1997e).

There are no adequate health studies on the effects of chloroform on humans; however, based on animal studies, the liver and kidneys are the likely target organs (ATSDR, 1997e). The oral RfD for chloroform has been established by USEPA at 1×10^{-2} mg/kg-day (IRIS, 2002a).

Chloroform is classified by USEPA as Group B2 – probable human carcinogen, based on sufficient evidence of carcinogenicity in animals (IRIS, 2002a). However, as no oral CSF has been established for chloroform (IRIS, 2002a) and NCEA has no cancer values for chloroform (NCEA, 2002d), it is not assessed quantitatively for cancer risks in this HHRA.

5.2.2.5 Methylene Chloride (Dichloromethane)

Methylene chloride is used as a paint stripper; de-greaser; extraction solvent; in the manufacture of photographic film; and in some pesticides and aerosol products (Sittig, 1991; ATSDR, 2001d).

The effects of long-term ingestion of methylene chloride by humans are not known, but it may cause harmful effects to the liver, blood, and central nervous system (Sittig, 1991). The oral RfD for dichloromethane has been established by USEPA at 6×10^{-2} mg/kg-day (IRIS, 1997p).

Methylene chloride (dichloromethane) is classified by USEPA as Group B2 – probable human carcinogen, based on animal data. The oral CSF for methylene chloride is 7.5×10^{-3} (mg/kg-day)⁻¹ (IRIS, 1997p).

5.2.2.6 Xylenes

Xylenes are three individual isomers (ortho, meta, and para, which are abbreviated as o-, m-, and p-xylene, respectively). While the chemical properties of these three isomers are not identical, they are considered together as group (i.e., as total xylenes or sum of xylenes). Xylene is one of the major bulk chemicals produced in the US (i.e., in the top 30 in terms of production volume). Like benzene, it is found in crude petroleum and refined motor fuels. It is also used as a paint thinner; a constituent in paints and varnishes; a chemical feedstock; a solvent and cleaning agent; and a number of other industrial and manufacturing uses (Sittig, 1991; ATSDR, 1996a).

Long-term exposure to xylene has been associated with liver and kidney damage (Sittig, 1991) and brain and central nervous system effects (ATSDR, 1996a). The oral RfD for xylene is 2.0 mg/kg-day (IRIS, 1998g); USEPA indicates that the confidence in this value is medium.

The cancer-causing potential of xylenes has been reviewed by USEPA but the data were not sufficient; xylene was therefore assigned to Group D – not classifiable as to human carcinogenicity. Xylene is being reassessed under the IRIS program (IRIS, 1998g).

5.2.3 Semivolatile Organic Compounds

The discussion below provides brief summaries for the SVOCs identified as COPCs for this HHRA, with the exception of PAHs, for which information is presented in Section 5.2.4. The full IRIS summaries and related ATSDR documents are presented in Appendix E. These SVOCs include:

- Bis(2-ethylhexyl)phthalate or di(2-ethylhexyl)phthalate.
- Dibenzofuran.
- 1,2-Dichlorobenzene.
- 1,3-Dichlorobenzene.
- 1,4-Dichlorobenzene.
- Hexachlorobenzene.
- 1,2,4-Trichlorobenzene.

5.2.3.1 Bis(2-ethylhexyl)phthalate or Di(2-ethylhexyl)phthalate

Bis(2-ethylhexyl)phthalate (BEHP) and di(2-ethylhexyl)phthalate (DEHP) are different names for the same chemical, with the former being more common in chemical analysis (e.g., data from the Contract Laboratory Program [CLP] program), and the latter in toxicological data (e.g., ATSDR and IRIS), as discussed below. DEHP was identified as a COPC in fish tissue; it was not identified as a COPC in any sediments or dredge spoils. BEHP is not expected to be a COPC in surface water. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for BEHP, it was detected only at low concentrations (2 to 10 µg/L) in four samples collected in 1992 from greater depths in the lake (6 and 12 m).

DEHP is a synthetic chemical used principally as a plasticizer (an additive to plastics to make them more flexible), and may constitute as much as 40 percent of some PVC products (ATSDR, 1993a). It is also used to a lesser extent in inks, pesticides, cosmetics, and vacuum pump oil (Sittig, 1991).

There is little information regarding the harmful effects of DEHP on humans. Based on animal studies (which showed increased liver weight), an oral RfD of 2×10^{-2} mg/kg-day has been established by USEPA (IRIS, 1998h). Confidence in this value is considered by USEPA to be medium.

DEHP is classified by USEPA as Group B2 – probable human carcinogen, based on animal data. The oral CSF established for DEHP is 1.4×10^{-2} (mg/kg-day)⁻¹ (IRIS, 1998h).

5.2.3.2 Dibenzofuran

Dibenzofuran was identified as a COPC only in southern basin sediments; it was not identified as a COPC in other sediments, fish tissue, or dredge spoils and is not expected to be a COPC in surface water. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for dibenzofuran, it was not detected in four samples collected in 1992 from greater depths in the lake (6 and 12 m). Little information is available for dibenzofuran, also known as diphenylene oxide. It is used as an insecticide and in organic synthesis (Sittig, 1991; Hawley, 1981).

No oral RfD has been established for dibenzofuran (IRIS, 2000c); data are considered inadequate for quantitative risk assessment (HEAST, Table 1 [USEPA, 1997]). This chemical is being reassessed under the IRIS program (IRIS, 2000c). The oral RfD used for the HHRA is 4×10^{-3} mg/kg-day; this value was

developed by NCEA, as cited in the USEPA Region 3 RBC and Region 9 PRG tables (USEPA, 2002, 2000b). The NCEA issue paper for dibenzofuran received from USEPA Region 2 confirms this value (NCEA, 2002a; see also Appendix E of this HHRA).

The cancer-causing potential of dibenzofuran has been reviewed by USEPA, but the data were not sufficient; dibenzofuran was assigned to Group D – not classified as to human carcinogenicity (IRIS, 2000c).

5.2.3.3 1,2-Dichlorobenzene

1,2-Dichlorobenzene, also known as ortho- or o-dichlorobenzene, is one of the three dichlorobenzene isomers (the others being 1,3- [meta-] and 1,4- [para-] dichlorobenzene). All three isomers have been identified as COPCs for the Onondaga Lake HHRA. However, due to differences in toxicity and carcinogenicity, each individual dichlorobenzene isomer is discussed separately, rather than being summed and evaluated as total dichlorobenzenes.

1,2-Dichlorobenzene is used as a process solvent for the manufacture of toluene di-isocyanate and as an intermediate in the manufacture of other chemicals, including dyes, herbicides, and de-greasers (Sitting, 1991).

The oral RfD for 1,2-dichlorobenzene is 9×10^{-2} mg/kg-day (IRIS, 2000d); USEPA indicates that the confidence in this value is low. This chemical is currently being reassessed (IRIS, 2000d). NCEA has developed a provisional RfD for 1,2-dichlorobenzene of 3×10^{-1} mg/kg-day; however, as recommended by USEPA Region 2, the IRIS value will be used for the quantitative HHRA. The potential effect on the results of the hazard calculation of using the IRIS value as opposed to the provisional NCEA value is discussed in Chapter 7, Uncertainty Assessment.

The cancer-causing potential of 1,2-dichlorobenzene has been reviewed by USEPA but the data were inadequate; 1,2-dichlorobenzene was assigned to Group D – not classified as to human carcinogenicity. There are no human data for carcinogenicity, and limited animal data are both positive and negative with regard to trends for carcinogenic responses in rodents (IRIS, 2000d).

5.2.3.4 1,3-Dichlorobenzene

Little information on the use of 1,3-dichlorobenzene was found (Sitting, 1991); it may have some commercial use as an insecticide (Hawley, 1981), and may be present as a contaminant in commercial 1,2- and 1,4-dichlorobenzenes.

No oral RfD has been established for 1,3-dichlorobenzene (IRIS, 2000e); the oral RfD is considered not verifiable (HEAST, Table 1 [USEPA, 1997b]). This chemical is being reassessed under the IRIS program (IRIS, 2000e). NCEA has provided a provisional oral RfD of 9×10^{-4} for 1,3-dichlorobenzene for the

Onondaga Lake HHRA (NCEA, 2002a; see also Appendix E of this HHRA), although confidence in this value is considered low. The oral RfD of 9×10^{-4} is used in this HHRA.

The cancer-causing potential of 1,3-dichlorobenzene has been reviewed by USEPA but the data were inadequate (i.e., there were no human data and no animal data, and limited genetic data); 1,3-dichlorobenzene was assigned to Group D – not classifiable as to human carcinogenicity (IRIS, 2000e).

5.2.3.5 1,4-Dichlorobenzene

1,4-Dichlorobenzene is used primarily as an insecticide (i.e., for mothballs) and as an air deodorizer (ATSDR, 1999i; Sittig, 1991).

Long-term ingestion of 1,4-dichlorobenzene may affect the central nervous system and cause liver and possibly blood problems (ATSDR, 1999i; Sittig, 1991). No oral RfD has been established for 1,4-dichlorobenzene (IRIS, 2000f; HEAST, 1997); it is currently being reassessed in the IRIS program (IRIS, 2000f). The oral RfD used for this HHRA is 3×10^{-2} mg/kg-day; this value was developed by NCEA, as cited in the both the USEPA Region 3 RBC and Region 9 PRG tables (USEPA, 2002a,b). Although the study on which the value is based is no longer considered current, NCEA recommends use of the 3×10^{-2} mg/kg-day oral RfD for the Onondaga Lake site (NCEA, 2002a; see also Appendix E of this HHRA).

The IRIS summary for 1,4-dichlorobenzene notes that this chemical has not undergone a full evaluation under the IRIS program for evidence of human carcinogenic potential (IRIS, 2000f); in other words, it is not classified. However, an oral slope factor of 2.4×10^{-2} (mg/kg-day)⁻¹ is present in HEAST (USEPA, 1997), and this value is used in this HHRA for evaluating the cancer risk of 1,4-dichlorobenzene.

5.2.3.6 Hexachlorobenzene

Hexachlorobenzene has been identified as a COPC in fish tissue and in soil/sediment. Hexachlorobenzene was widely used as a pesticide and fungicide for onions and wheat and other grains until 1965. It was also used in the manufacture of fireworks, ammunition, electrodes, dye, and synthetic rubber, and as a wood preservative (Sitting, 1991; ATSDR, 1997d). There are currently no commercial uses of hexachlorobenzene (ATSDR, 1997d).

Accidental ingestion of hexachlorobenzene-contaminated grain (which occurred in Turkey) resulted in liver damage and shortened life spans, and some children fed contaminated breast milk developed a condition called “pink sore” that was usually fatal. Hexachlorobenzene affected the skin, skeletal system, liver, stomach, and nervous system (Sittig, 1991; ATSDR, 1997d).

USEPA has established an oral RfD of 8×10^{-4} mg/kg-day for hexachlorobenzene, based on liver effects in animal studies (IRIS, 1997q); confidence in this value is considered medium.

Hexachlorobenzene is classified by USEPA as Group B2 – probable human carcinogen, based on animal data (liver, thyroid, and kidney tumors). An oral CSF of $1.6 \text{ (mg/kg-day)}^{-1}$ has been established for hexachlorobenzene (IRIS, 1997q).

5.2.3.7 1,2,4-Trichlorobenzene

Onondaga Lake surface water is the only medium in which 1,2,4-trichlorobenzene (1,2,4-TCB) has been identified as a COPC. It is used as a dielectric fluid- and heat-transfer medium; a dye carrier; a herbicide intermediate; a de-greaser; a lubricant; and a termiticide (Sittig, 1991). 1,2,4-TCB is the only trichlorobenzene isomer with any significant reported commercial uses (Sittig, 1991). There is no ATSDR profile for 1,2,4-TCB.

USEPA has established an oral RfD of $1 \times 10^{-2} \text{ mg/kg-day}$ for 1,2,4-TCB; confidence in this value is considered medium (IRIS, 1997r). It has been assigned to Group D – not classifiable as to human carcinogenicity, due to inadequate animal data and no human data.

5.2.4 Polycyclic Aromatic Hydrocarbons

A total of 13 different PAH compounds have been identified as COPCs in Onondaga Lake soils and sediments (see Chapter 3, Table 3-1). PAH compounds are not expected to be COPCs in surface water. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for PAHs, no PAHs were detected in four samples collected in 1992 from greater depths in the lake (6 and 12 m). The PAH compounds are among a group of over 100 different chemicals; however, conventional chemical analyses typically identify and quantitate only about 17 of the PAH compounds. PAHs (except naphthalene) do not generally have any commercial uses as individual chemical compounds; however, PAHs are naturally occurring components of crude oil and petroleum products, such as fuel oil.

PAHs are also formed as byproducts of combustion processes, including vehicular exhaust, burning coal, and forest fires; the burning of tobacco; and the charbroiling of meat. Due to atmospheric deposition, PAHs can be deposited in the environment at great distances from the original source. Historically, PAHs – which are a constituent of coal tar – were used in some commercial products such as shampoos. Currently, the major commercial occurrences of PAHs are in roofing tar, creosote, and similar products, in addition to the petroleum products mentioned previously, with some lesser uses in dyes, plastics, and pesticides (ATSDR, 1996b; Sittig, 1991).

Chemical-specific toxicity data (CSFs or RfDs) have not been developed for most of the individual PAH compounds. Of the 13 PAHs identified as COPCs for Onondaga Lake, seven are considered carcinogenic (classified as Group B2 – probable human carcinogen) by USEPA:

- Benz(a)anthracene (IRIS, 1997s).
- Benzo(a)pyrene (IRIS, 1998h).
- Benzo(b)fluoranthene (IRIS, 1997t).

- Benzo(k)fluoranthene (IRIS, 1997u).
- Chrysene (IRIS, 1997v).
- Dibenz(a,h)anthracene (IRIS, 1997w).
- Indeno(1,2,3-cd)pyrene (IRIS, 1997x).

The six other PAHs considered here have been evaluated for toxicity (non-cancer):

- Acenaphthylene.
- Benzo(g,h,i)perylene.
- Fluoranthene.
- 2-Methylnaphthalene.
- Naphthalene.
- Phenanthrene.

These six were either not evaluated for carcinogenicity by USEPA (e.g., 2-methylnaphthalene); not classified due to insufficient evidence (assigned to Group D); or, in the case of naphthalene, considered a possible human carcinogen (Group C), but with insufficient data to confirm its carcinogenicity (IRIS, 2002b).

5.2.4.1 Carcinogenic Polycyclic Aromatic Hydrocarbons

Of the seven carcinogenic (Group B2) PAHs identified as Onondaga Lake COPCs, only one – benzo(a)pyrene – has a published CSF in IRIS (IRIS, 1998i). However, USEPA has published provisional guidance that provides order-of-magnitude estimates of the carcinogenic potency of the other carcinogenic PAHs relative to benzo(a)pyrene (USEPA, 1993a). The carcinogenic PAHs, their relative potency, and the CSFs used in this HHRA are shown below. The oral CSFs listed below are identical to those listed in the most recent USEPA Region 3 RBC tables (USEPA, 2002a).

Carcinogenic PAH	Published CSF	Relative Potency ^a	CSF used for HHRA
Benz(a)anthracene	NA	0.1	0.73 (mg/kg-d) ⁻¹
Benzo(a)pyrene	7.3 (mg/kg-d) ^{-1 b}	1.0	7.3 (mg/kg-d) ⁻¹
Benzo(b)fluoranthene	NA	0.1	0.73 (mg/kg-d) ⁻¹
Benzo(k)fluoranthene	NA	0.01	0.073 (mg/kg-d) ⁻¹
Chrysene	NA	0.001	0.0073 (mg/kg-d) ⁻¹
Dibenz(a,h)anthracene	NA	1.0	7.3 (mg/kg-d) ⁻¹
Indeno(1,2,3-cd)pyrene	NA	0.1	0.73 (mg/kg-d) ⁻¹

Notes: ^a Relative potency factor from Table 8 in *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (USEPA, 1993a).

^b IRIS, 1998i.

5.2.4.2 Other Polycyclic Aromatic Hydrocarbon Contaminants of Potential Concern

Of the six other PAH compounds identified as COPCs for Onondaga Lake, RfDs are available in IRIS for only two – naphthalene and fluoranthene. However, at the request of TAMS and USEPA Region 2, NCEA provided additional guidance for some of these PAHs. A brief summary of the available toxicological data is presented below for these six compounds:

- Acenaphthylene has no quantitative oral RfD (IRIS, 1997y) for non-cancer toxicity. It has been assigned to Group D – not classifiable as to human carcinogenicity, due to absence of human data and inadequate animal data. Acenaphthylene is not listed in either the USEPA Region 3 RBC or Region 9 PRG tables. However, NCEA indicated that the oral RfD for pyrene (3×10^{-2} mg/kg-day; IRIS, 1997qq) is appropriate to use for the non-cancer risk assessment of acenaphthylene (NCEA, 2002d).
- Benzo(g,h,i)perylene has no quantitative oral RfD (IRIS, 1997z) for non-cancer toxicity. It has been assigned to Group D – not classifiable as to human carcinogenicity, due to absence of human data and inadequate animal data. Benzo(g,h,i)perylene is not listed in either the USEPA Region 3 RBC or Region 9 PRG tables. However, NCEA indicated that the oral RfD for pyrene (3×10^{-2} mg/kg-day) is appropriate to use for the non-cancer risk assessment of benzo(g,h,i)perylene (NCEA, 2002d).
- Fluoranthene has an oral RfD of 4×10^{-2} mg/kg-day (IRIS, 1997aa), based on animal studies (subchronic toxicity in mice); confidence in this value is considered low. It has been assigned to Group D – not classifiable as to human carcinogenicity, due to absence of human data and inadequate animal data.
- 2-Methylnaphthalene does not have an IRIS substance file and is not listed in HEAST. However, the Region 3 RBC tables list an oral RfD of 2×10^{-2} mg/kg-day, cited as NCEA – provisional, for 2-methylnaphthalene (USEPA, 2002). There is no entry for 2-methylnaphthalene in the Region 9 PRG tables. The NCEA issue paper for 2-methylnaphthalene confirms this value (NCEA, 2002b; see also Appendix E of this HHRA).
- Naphthalene is used in mothballs, insecticides (carbaryl), dyes, resins, and leather tanning agents (ATSDR, 1996c), in addition to being found as noted above in the introductory discussion of PAHs. USEPA has established an oral RfD of 2×10^{-2} mg/kg-day for naphthalene, based on a subchronic toxicity study of rats (IRIS, 2002b). Naphthalene is classified in Group C – possible human carcinogen, based on inadequate data in humans and limited evidence of oral carcinogenicity in animal studies (IRIS, 2002b); no quantitative oral CSF has been established for

naphthalene, although naphthalene is currently being reassessed under the IRIS program.

- Phenanthrene has no quantitative oral RfD (IRIS, 1997bb) for non-cancer toxicity. It has been assigned to Group D – not classifiable as to human carcinogenicity, due to the absence of human data and inadequate animal data. Phenanthrene is not listed in either the Region 3 RBC or Region 9 PRG tables. However, NCEA indicated that the oral RfD for pyrene (3×10^{-2} mg/kg-day) is appropriate to use for the non-cancer risk assessment of phenanthrene (NCEA, 2002d).

5.2.5 Pesticides

The pesticides discussed below are COPCs for Onondaga Lake via the fish ingestion pathway. Only two of the pesticides – the related compounds aldrin and dieldrin – were identified as COPCs in soil or sediment. Pesticide compounds are not expected to be COPCs in surface water. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for pesticides, no pesticides were detected in four samples collected in 1992 from greater depths in the lake (6 and 12 m).

5.2.5.1 Aldrin

Aldrin has been identified as a COPC in fish tissue and in the sediments of Wetland SYW-19 in the southern basin. Aldrin is an insecticide with a structure similar to that of dieldrin, and quickly breaks down to dieldrin in the body and in the environment. From 1950 to 1970, aldrin and dieldrin were popular pesticides for crops like corn and cotton. Because of concerns about damage to the environment and the potential harm to human health, in 1974 USEPA banned all uses of aldrin and dieldrin, except to control termites. In 1987, USEPA banned all uses (ATSDR, 1993b).

USEPA has established an oral RfD of 3×10^{-5} mg/kg-day for aldrin; confidence in this value is considered medium (IRIS, 1997cc). Aldrin is classified as B2 – probable human carcinogen, based on increases in tumors (principally in the liver) in animal studies (IRIS, 1997cc). The oral slope factor for aldrin is 17 (mg/kg-day)⁻¹.

5.2.5.2 Delta-Benzene Hexachloride (δ -BHC) (delta-Hexachlorocyclohexane; delta-HCH)

Delta-Hexachlorocyclohexane (delta-HCH) is one of eight stereo-isomers of HCH, four of which are typically reported in pesticide analyses, with the other three being the alpha, beta, and gamma isomers. The gamma isomer, also known as lindane, is the most potent insecticide (most toxic) of the isomers, but all have some degree of toxicity. Technical HCH is a mixture of the various HCH isomers (Sittig, 1991). Lindane has not been produced in the US since 1977, although it is still imported into and formulated in the US. Former uses included insecticide on fruit and vegetable crops including greenhouse vegetables and forest crops including Christmas trees. Lindane is still used in ointments for the treatment of lice and scabies (ATSDR, 1999j).

USEPA has not established an oral RfD for delta-HCH in either the IRIS program (IRIS, 1997dd) or in HEAST. There is an IRIS oral RfD for gamma-HCH (lindane) of 3.4×10^{-4} mg/kg-day (IRIS, 2002c). However, NCEA indicated that the oral RfD for lindane (3.4×10^{-4} mg/kg-day) is appropriate to use for the non-cancer risk assessment of delta-HCH (NCEA, 2002d).

Delta-HCH is considered not classifiable (Group D) with regard to human carcinogenicity. Therefore, no quantitative cancer assessment of delta-HCH is included in this HHRA.

5.2.5.3 Chlordane

Chlordane is a commercial manufactured chemical that was used as a pesticide in the US from 1948 to 1988, sold under trade names such as Octachlor and Velsicol 1068. Until 1983, chlordane was used as a pesticide on crops like corn and citrus and on home lawns and gardens. Because of concern about damage to the environment and harm to human health, USEPA banned all uses of chlordane in 1983, except to control termites. In 1988, USEPA banned all uses (ATSDR, 1995f).

Technical chlordane is not a single chemical, but a mixture of pure chlordane mixed with many related chemicals (e.g., nonachlor). The total chlordane concentration used for this HHRA is a sum of several of these chemicals, as discussed in Chapter 3 and Appendix A.

Chlordane affects the nervous system, the digestive system, and the liver in both people and animals. Headaches, irritability, confusion, weakness, vision problems, vomiting, stomach cramps, diarrhea, and jaundice have occurred in people who breathed air containing high concentrations of chlordane or swallowed small amounts of chlordane. Ingestion of large amounts of chlordane can cause convulsions and death in humans.

The US FDA limits the amount of chlordane and its breakdown products in most fruits and vegetables to less than 300 µg/kg and in animal fat and fish to less than 100 µg/kg (ATSDR, 1995f).

USEPA has established an oral RfD of 5×10^{-4} mg/kg-day for chlordane, based on animal studies showing toxic effects on the liver. Confidence in this value is considered medium (IRIS, 1998j).

Chlordane is classified as Group B2 – probable human carcinogen (USEPA, 1986); it would be considered a “likely carcinogen by all routes of exposure” under the 1996 Proposed Guidelines (IRIS, 1998j). The International Agency for Research on Cancer has determined that chlordane is not classifiable as to its carcinogenicity to humans. Studies of workers who made or used chlordane do not show that exposure to chlordane is related to cancer, but that information is not definitive. Mice fed low levels of chlordane in food developed liver cancer (ATSDR, 1995j). USEPA has established an oral CSF of 3.5×10^{-1} (mg/kg-day)⁻¹ for chlordane (IRIS, 1998i), based on liver cancer in animal (i.e., mouse) studies.

5.2.5.4 DDT and Related Compounds – General

DDT and related compounds, for the purpose of this HHRA, consist of a group of four chemicals: 2,4'-DDE; 4,4'-DDD; 4,4'-DDE; and 4,4'-DDT. All four were identified as COPCs in Onondaga Lake fish tissue, but were not identified as COPCs in other site media.

DDT (1,1,1-trichloro-2,2-bis[p-chlorophenyl]ethane; alternately, p,p'-dichlorodiphenyl-trichloroethane) was a manufactured chemical widely used to control insects on agricultural crops and insects that carry diseases such as malaria and typhus. Two similar chemicals that sometimes contaminate DDT products are DDD (1,1-dichloro-2,2-bis[p-chlorophenyl]ethane) and DDE (1,1-dichloro-2,2-bis[chlorophenyl]ethylene). DDD was also used to kill pests, and its use has also been banned. One form of it has been used medically to treat cancer of the adrenal gland. DDE has no commercial use (ATSDR, 1995g).

Because of damage to wildlife and the potential harm to human health, the use of DDT has been banned in the US, except for public health emergencies. DDT is still used in some other countries (ATSDR, 1995g).

5.2.5.5 2,4'-DDE (o,p'-DDE)

2,4'-DDE was identified as a COPC in fish tissue based on its detection at concentrations above the screening criterion, which is the USEPA Region 3 fish ingestion RBC for DDE (isomer unspecified) (USEPA, 2001b). However, absent specific toxicity data for 2,4'-DDE, no quantitative assessment of cancer or non-cancer risks associated with this chemical is included in this HHRA. No IRIS, HEAST, or ATSDR files were found for 2,4'-DDE, and neither the Region 3 RBC nor Region 9 PRG tables include an entry specific to this isomer (USEPA, 2002, 2000b). NCEA has made no recommendation for quantitatively assessing 2,4'-DDE.

5.2.5.6 4,4'-DDD (p,p'-DDD)

4,4'-DDD has been identified as a COPC in fish tissue at the Onondaga Lake site. It is typically detected in association with the related compounds 4,4'-DDT and 4,4'-DDE.

USEPA has not established an oral RfD for 4,4'-DDD (IRIS, 1997ee). However, in accordance with the recommendation from USEPA Region 2, the oral RfD for 4,4'-DDT – 5×10^{-4} mg/kg-day (IRIS, 1997ff) – was previously used for the quantitative assessment of 4,4'-DDD (NYSDEC, October 2, 1998). However, based on the NCEA recommendations received for this HHRA, a provisional RfD of 3×10^{-3} mg/kg-day will be used for this HHRA (NCEA, 2002a; see also Appendix E of this HHRA).

4,4'-DDD is considered as Group B2 – probable human carcinogen by USEPA, based on liver tumors and thyroid tumors in animal studies (IRIS, 1997ee). The oral CSF for 4,4'-DDD is 2.4×10^{-1} mg/kg-day.

5.2.5.7 4,4'-DDE (p,p'-DDE)

4,4'-DDE has been identified as a COPC in fish tissue at the Onondaga Lake site. It is typically detected in association with the related compounds 4,4'-DDT and 4,4'-DDD.

USEPA has not established an oral RfD for 4,4'-DDE (IRIS, 1997gg; HEAST in USEPA, 1997). However, based on the NCEA recommendations received for this HHRA, a provisional RfD of 7×10^{-4} mg/kg-day will be used for this HHRA (NCEA, 2002a; see also Appendix E of this HHRA).

4,4'-DDE is considered as Group B2 – probable human carcinogen by USEPA, based on liver tumors and thyroid tumors in animal studies (IRIS, 1997gg). The oral CSF for 4,4'-DDE is 3.4×10^{-1} (mg/kg-day)⁻¹.

5.2.5.8 4,4'-DDT (p,p'-DDT)

4,4'-DDT has been identified as a COPC in fish tissue at the Onondaga Lake site. It is typically detected in association with the related compounds 4,4'-DDT and 4,4'-DDE.

USEPA has established an oral RfD for 4,4'-DDT of 5×10^{-4} mg/kg-day, based on animal studies showing effects on the liver (IRIS, 1997ff). Confidence in this value is considered medium.

4,4'-DDT is considered as Group B2 – probable human carcinogen by USEPA, based on liver tumors in ten animal (i.e., seven mouse and three rat) studies (IRIS, 1997ff). The oral CSF for 4,4'-DDT is 3.4×10^{-1} (mg/kg-day)⁻¹.

5.2.5.9 Dieldrin

Dieldrin has been identified as a COPC in fish tissue and in the sediments of Wetland SYW-19 in the southern basin. Dieldrin is an insecticide with similar structure to that of aldrin, and aldrin quickly breaks down to dieldrin in the body and in the environment. From 1950 to 1970, aldrin and dieldrin were popular pesticides for crops like corn and cotton. Because of concerns about damage to the environment and the potential for harm to human health, USEPA banned all uses of aldrin and dieldrin in 1974, except to control termites. In 1987, USEPA banned all uses (ATSDR, 1993b).

USEPA has established an oral RfD of 5×10^{-5} mg/kg-day for dieldrin; confidence in this value is considered medium (IRIS, 1997hh).

Dieldrin is considered to be Group B2 – probable human carcinogen by USEPA, based on liver tumors in animal (i.e., seven mouse) studies (IRIS, 1997hh). The oral CSF for dieldrin is 16 (mg/kg-day)⁻¹.

5.2.5.10 Heptachlor Epoxide

Heptachlor epoxide, a breakdown product of heptachlor, has been identified as a COPC in fish tissue. The epoxide is more likely to be found in the environment than heptachlor. Heptachlor is a powder that smells like camphor (mothballs). Heptachlor was marketed under trade names including Heptagran, Basaklor, Drinox, Soleptax, Termide, and Velsicol 104. Heptachlor was used extensively in the past for killing insects in homes, buildings, and on food crops, especially corn. Use slowed in the 1970s and stopped in 1988 (ATSDR, 1993c).

USEPA has established an oral RfD of 1.3×10^{-5} mg/kg-day for heptachlor epoxide (IRIS, 1997ii), based on animal studies showing effects on the liver. Confidence in this value is considered low, due to the low quality of the studies from which the RfD was derived.

USEPA classifies heptachlor epoxide as Group B2 – probable human carcinogen, based on animal studies showing liver carcinomas. The oral CSF for heptachlor epoxide is $9.1 \text{ (mg/kg-day)}^{-1}$ (IRIS, 1997ii).

5.2.6 Polychlorinated Biphenyls

Polychlorinated biphenyls as a group, as well as several of the individual Aroclors (i.e., commercial-mixture PCB compounds with different properties, differing among each other principally in the overall level of chlorination of the biphenyl molecules), have been identified as COPCs in fish tissue and soils and sediments at the Onondaga Lake site. Although there were no surface water samples from the 0 to 3 m depth interval analyzed for PCBs, PCBs were not detected in samples from greater depths in the lake (6 and 12 m). The individual Aroclors that were identified as COPCs at Onondaga Lake, as shown in Chapter 3, Table 3-1, include Aroclors 1016, 1221, 1242, 1248, 1254, 1260, and 1268. (Some NYSDEC fish data are reported as Aroclor 1254/1260, as shown in Table 3-1 and Appendix A; this is an analytical/reporting construct and does not represent a unique commercial Aroclor mixture.)

PCBs are mixtures of up to 209 different compounds (referred to as “congeners”) that include a biphenyl and from one to ten chlorine atoms; “Aroclors” were commercial products with differing amounts of the individual congeners marketed in the US (similar mixtures were sold elsewhere under different trade names). PCBs have been used as a dielectric fluid in electrical equipment such as transformers and capacitors due to their heat resistance and insulating properties. PCBs were also used in the ballasts of fluorescent lights and in hydraulic oils. The manufacture of PCBs was terminated in the US in 1977 due to evidence of harmful health effects (ATSDR, 2001e).

Although there are both non-cancer and cancer toxicity data for PCBs, there are not Aroclor-specific data for all the Aroclors detected at the Onondaga Lake site. There are IRIS files for “Polychlorinated Biphenyls (PCBs)” (IRIS, 1998j); Aroclor 1016 (IRIS, 1997jj); Aroclor 1248 (IRIS, 1997kk); and Aroclor 1254 (IRIS, 1997ll). The available toxicity data, and the manner in which it was applied to the Onondaga Lake HHRA, are discussed below.

5.2.6.1 Cancer Risk

The current USEPA CSFs for PCBs are for PCBs as a class; i.e., they are not Aroclor-specific or congener-specific. PCBs are assigned B2 weight-of-evidence classification, as a probable human carcinogen. The Group B2 classification indicates that there is sufficient evidence of PCB carcinogenicity from animal studies, but the human carcinogenicity data are considered inadequate (IRIS, 1998j).

The IRIS files for the individual Aroclors (1016, 1248, and 1254) all state that they have "...not undergone a complete evaluation under USEPA's IRIS program for evidence of human carcinogenic potential" (IRIS, 1997jj, kk, ll); therefore, the cancer toxicity data for "PCBs" are applied to each of the individual Aroclors that have been identified as COPCs at the Onondaga Lake site.

USEPA uses a three-tiered approach for PCB cancer potency in humans (IRIS, 1998k), as follows:

- **High risk and persistence:** Applicable to food chain exposure; sediment or soil ingestion; dermal exposure, where an absorption factor is applied; and early life exposure.
- **Low risk and persistence:** Applicable to ingestion of water-soluble congeners; inhalation of evaporated congeners; and dermal exposure, if no absorption factor is applied.
- **Lowest risk and persistence:** Applicable only when it is known that the PCB mixture contains less than 0.5 percent congeners with more than four chlorines (pentachloro or more highly chlorinated PCB congeners).

For this HHRA, the exposure pathways (soil/sediment ingestion and dermal contact, and fish ingestion) all meet the criteria for the high risk and persistence tier, so those CSFs were applied for calculating the carcinogenic risks for PCBs. PCBs were not identified as a COPC in Onondaga Lake surface water since they were not detected in lake water.

Literature and analytical data were reviewed to determine if any of the Aroclors that are Onondaga Lake site COPCs met the criterion for lowest risk and persistence, based on the absence of more highly chlorinated PCB congeners. Literature data are in agreement that Aroclors 1242 and higher (i.e., 1248, 1254, 1260, and 1268) are composed of 10 percent or more congeners with more than four chlorines; however, the literature data are less consistent with regard to Aroclors 1016 and 1221 (reported values range from zero to about one percent for these two congeners) (Table 1-4 in TAMS/USEPA, 2000).

More recent (i.e., 1994) congener-specific analysis of Aroclor standards data generated for the Hudson River PCBs Site RI/FS were reviewed; these data show that both Aroclor 1016 and 1221 contain slightly more than 1 percent pentachlorobiphenyl and hexachlorobiphenyl (Table 1-5 in TAMS/USEPA, 2000).

Therefore, it was determined that none of the Onondaga Lake Aroclor mixtures met the criteria for “low risk and persistence.”

For each tier, USEPA has developed both an upper-bound slope factor and a central estimate slope factor. For this Onondaga Lake HHRA, the upper-bound CSF ($2 \text{ [mg/kg-day]}^{-1}$) is used for the reasonable maximum exposure (RME) and the central estimate CSF ($1 \text{ [mg/kg-day]}^{-1}$) is used in the central tendency (CT) risk calculation.

5.2.6.2 Non-Cancer Toxicity

There are IRIS files for “Polychlorinated Biphenyls (PCBs);” Aroclor 1016; Aroclor 1248; and Aroclor 1254. The IRIS file for “PCBs” references the individual IRIS files for Aroclors 1016, 1248, 1254, and 1260 (although the file for Aroclor 1260 was not located).

The IRIS file for Aroclor 1016 lists an oral RfD of $7 \times 10^{-5} \text{ mg/kg-day}$, with a confidence level of medium (IRIS, 1997jj). The file for Aroclor 1248 notes that the health effects data were reviewed and “...determined to be inadequate for the derivation of an oral RfD” (IRIS, 1997kk). The file for Aroclor 1254 presents an oral RfD of $2 \times 10^{-5} \text{ mg/kg-day}$, with a confidence level of medium (IRIS, 1997ll).

Based on the fact that the individual Aroclor mixtures differ only in the relative quantities (percentages) of different PCB congeners, it is reasonable to assume that similar Aroclors will have similar toxicological properties, even in the absence of verifiable Aroclor-specific data. For non-cancer toxicity, the Aroclors have been divided into two groups, as follows:

- The lower molecular weight Aroclor group includes Aroclors 1016, 1221, and 1242, and is characterized for the quantitative HHRA by the oral RfD for Aroclor 1016 ($7 \times 10^{-5} \text{ mg/kg-day}$) (IRIS, 1997jj). Aroclor 1232 would also be assigned to this group, except that it was not detected in samples used for this HHRA.
- The higher molecular weight Aroclor group includes Aroclors 1248, 1254, 1260 (including the Aroclor 1254/1260 data), and 1268. The higher molecular weight Aroclor group is characterized in this HHRA by the oral RfD for Aroclor 1254 ($2 \times 10^{-5} \text{ mg/kg-day}$) (IRIS, 1997ll). Aroclor 1248 has been assigned to the higher molecular weight Aroclor group for the HHRA, based on its being predominantly (over 50 percent) composed of tetrachloro- and higher chlorinated congeners. This assignment of Aroclor 1248 is also more conservative; i.e., more protective of human health.

5.2.7 PCDD/PCDFs

PCDD/PCDFs have been identified as COPCs in fish tissue, lake and wetland sediments, and dredge spoils. Surface water samples have not been analyzed for PCDD/PCDFs. PCDD/PCDFs are a group of 210 structurally related chlorinated chemicals that are ubiquitous in the environment. There are a total of

135 possible polychlorinated dibenzofurans (PCDFs), and 75 different polychlorinated dibenzo-*p*-dioxins (PCDDs). Sources of PCDD/PCDFs include incineration of municipal and certain industrial wastes, chlorination processes used in pulp and paper manufacturing and water treatment systems, and the production and use of certain chlorinated pesticides (e.g., 2,4,5-T).

Although PCDD/PCDFs are typically present in the environment as a mixture of many individual components, 2,3,7,8-TCDD (tetrachlorodibenzo-*p*-dioxin) has been extensively studied and is thought to be the most toxic congener within this chemical class. USEPA has not developed quantitative toxicity factors for any other specific PCDD/PCDF congeners because of the limited toxicological information available for these compounds (although there is an IRIS file for “Hexachlorodibenzo-*p*-dioxin, Mixture” [IRIS, 1997mm]).¹ Instead, USEPA adopted a toxicity equivalence factor (TEF) approach for quantitatively estimating the toxicity of other congeners that is based on their likely toxicity relative to that of 2,3,7,8-TCDD (USEPA, 2000a). The TEFs were updated by Van den Berg et al. (1998), and these revised TEFs have been utilized in this HHRA (Table 5-1 herein). In the TEF approach, 2,3,7,8-TCDD is assigned a weighting factor, or TEF, of 1. All other PCDD/PCDFs are assigned weighting factors based on their toxicity relative to 2,3,7,8-TCDD. Only PCDDs and PCDFs with four or more chlorine atoms, and only those with chlorine atoms in the 2,3,7,8 positions, are assigned TEFs; no toxicity factor is assigned to the other PCDD or PCDF congeners.

To apply the TEF approach, the concentrations of individual congeners in a PCDD/PCDF mixture are multiplied by their respective TEFs to yield the equivalent concentration of 2,3,7,8-TCDD. For example, a concentration of 0.2 ng/kg of 1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin, which has a TEF of 0.1, is considered equivalent to a concentration of 0.02 ng/kg of 2,3,7,8-TCDD. The equivalent concentrations of 2,3,7,8-TCDD for all of the PCDD/PCDF congeners in a given mixture are then summed to yield a total 2,3,7,8-TCDD toxic equivalent (TEQ) that is applied in risk calculations. The 2,3,7,8-TCDD TEFs used in this risk assessment to calculate the 2,3,7,8-TCDD TEQ are listed in Table 5-1 herein. The uncertainties related to the use of the TEF approach are discussed in Chapter 7, Uncertainty Assessment.

USEPA classifies 2,3,7,8-TCDD as a probable human carcinogen (i.e., a Group B2 carcinogen), based on inadequate data in human populations but sufficient evidence in laboratory animals. The numerous epidemiological studies undertaken to assess the potential carcinogenicity of PCDD/PCDFs in humans have generally suffered from a lack of accurate exposure data and potentially confounding exposures to other contaminants. There is no USEPA-verified CSF for 2,3,7,8-TCDD in IRIS; however, an oral CSF of 1.5×10^5 (mg/kg-day)⁻¹ is published in HEAST (USEPA, 1997b), based on the occurrence of tumors in a study of female rats (Kociba et al., 1978) and subsequent USEPA reviews. USEPA recently released the public review draft of its exposure and human health reassessment of 2,3,7,8-TCDD and related compounds (USEPA, 2000); however, the conclusions from the draft reassessment are not used in this

¹USEPA has developed a CSF for use in estimating risks associated with ingestion and inhalation exposures to mixtures of hexachlorodibenzo-*p*-dioxins (USEPA, 1997a). Risk assessments for PCDD/PCDFs, however, typically use the TEF approach to estimate the carcinogenic risks associated with these compounds.

quantitative HHRA. Risk estimates derived from the data (proposed CSF) in the draft dioxin reassessment document are presented in Chapter 7, Section 7.5.1.3.

USEPA does not currently have any quantitative toxicity factors (e.g., oral RfD) for the non-cancer health effects of PCDD/PCDFs. The most common and clearly demonstrated adverse systemic health effect that has been observed in humans exposed to PCDD/PCDFs through ingestion or dermal contact is a skin lesion known as chloracne. In addition, there is evidence suggesting that PCDD/PCDFs may cause liver damage, loss of appetite, weight loss, and digestive disorders in humans; however, these effects may have occurred as a result of concomitant exposures to chemicals where PCDD/PCDFs were present as trace contaminants. In some animal species, 2,3,7,8-TCDD caused liver damage and wasting following exposure to lethal or near-lethal doses. Animal studies have also provided evidence that PCDD/PCDFs may cause reproductive, developmental, and immune system toxicity (ATSDR, 1989). The USEPA draft dioxin reassessment (USEPA, 2000a) supports the existence of non-cancer health effects from dioxin. Although an RfD for dioxin is not proposed, several approaches to evaluating non-cancer toxicity of PCDD/PCDFs are discussed in the draft reassessment and summarized in Chapter 7, Section 7.5.1.3 of this HHRA.

6. RISK CHARACTERIZATION

In risk characterization, quantitative exposure estimates and toxicity factors are combined to calculate numerical estimates of potential health risk. In this chapter, potential cancer risks and non-cancer health hazards are estimated assuming long-term exposure to chemicals detected in site media. Risks are calculated for each completed exposure pathway, as discussed in Chapter 4, Exposure Assessment, and summarized in RAGS Table 1 (Appendix B).

As described in Chapter 4, potential risks are estimated for both current and future recreational scenarios. Exposure parameters such as exposure frequency for both current and future recreational use utilize those assumed for future use, as future recreational use of the lake is expected to be more extensive than current use. Thus, potential risk estimates for the future recreational scenario provide a conservative means for evaluating current potential risks posed by site media. Potential future risks are also estimated for construction workers who may contact lake and wetland sediments, dredge spoils area soils, and surface water.

The risk characterization methods described in RAGS Part A (USEPA, 1989a) and Part E (USEPA, 2001a) are used to calculate reasonable maximum exposure (RME) and central tendency (CT) (also known as typical) excess lifetime cancer risks for carcinogens and hazard indices (HIs) for contaminants with non-cancer health effects. These methods and the results of the risk characterization are described below, and are summarized in Table 6-1. In addition, the tables in Appendix B have been prepared consistent with Tables 7 and 8 of RAGS Part D (USEPA, 1998). These tables show detailed results of the risk calculations for each exposure pathway, including exposure point concentrations (EPCs) and intakes (as chronic daily intakes, or CDIs) calculated for the RME and CT scenarios, toxicity values used in risk estimates, and risk estimates for each COPC in each exposure pathway.

It should be noted that USEPA updated the RAGS guidance document as this HHRA was being prepared (K. Martin, pers. comm., 2002); USEPA indicated that the new guidance document (USEPA, 2001d) should be used for new risk assessments. As this HHRA was at a state of substantial completion at the time, the new (i.e., December 2001) RAGS Part D reporting format is not used in this report (with the concurrence of USEPA Region 2 risk assessment staff). The tables in Appendix B of this HHRA are those in the 1998 RAGS Part D guidance.

As the RAGS-specified tables do not provide a mechanism for summing risk across receptors (e.g., the cumulative risk of a receptor to COPCs in more than one medium – for instance, fish tissue, soil/sediment, and surface water), additional tables have been developed for this purpose and are included in this chapter.

Uncertainties associated with the quantitation of cancer risks and non-cancer human health hazards are discussed in Chapter 7 of this HHRA.

6.1 Cancer Risks

The procedures and results of the calculation of risks associated with carcinogenic COPCs are presented in this section. Only COPCs for which there are USEPA oral cancer slope factors (CSFs) are evaluated quantitatively in this HHRA. The CSFs used for each COPC and their sources are presented and discussed in Chapter 5, Toxicity Assessment, and in RAGS Table 6.1 in Appendix B. The potential effect on the quantitative HHRA for known or suspected carcinogens for which oral CSFs are not available is discussed in Chapter 7, Uncertainty Assessment.

It should be noted that the cancer risks discussed in this document represent excess or incremental cancer risks. In other words, the risks presented in this document are the increased cancer risks due to site-related COPCs.

6.1.1 Methods

Quantifying total excess cancer risk requires calculating risks associated with exposure to individual carcinogens and aggregating risks associated with simultaneous exposure to multiple carcinogenic chemicals. A cancer risk estimate for a single carcinogen is calculated by multiplying the carcinogenic chronic daily intake (CDI) of the contaminant by its CSF. A 1×10^{-6} cancer risk represents a one-in-one-million additional probability that an individual may develop cancer over a 70-year lifetime as a result of the exposure conditions evaluated. Because cancer risks are assumed to be additive, risks associated with simultaneous exposure to more than one carcinogen in a given medium are aggregated to determine a total cancer risk for each exposure pathway. Total cancer risks for each pathway are then summed for reasonable combinations of exposure pathways to determine the total cancer risk for the population of concern.

The findings presented here are compared to levels cited in the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), which states that "... acceptable exposure levels are generally concentration levels that represent an excess upper bound cancer to an individual of between 10^{-4} to 10^{-6} ... The 10^{-6} risk level shall be used as the point of departure for determining remediation goals ..." (40 CFR §300.430[e][2][A][2]). These target risk levels are also often applied by other agencies responsible for protecting human health (NYSDOH and ATSDR, 1995).

6.1.2 Quantitation of Cancer Risks by Pathway

Carcinogenic risk estimates were calculated for children and adults in the RME and CT scenarios as the probability of additional cancers associated with the selected exposure pathways (see Appendix B, RAGS Table 1). Separate risk estimates have been calculated for fish ingestion by both older and young children, in addition to adults, utilizing the ingestion rates discussed in Chapter 4, Section 4.2.1.

Based on the exposure assumptions and toxicity values described above, Tables 6-2 and 6-3 provide a summary of risk estimates for all complete exposure pathways in the RME and CT scenarios. Risks

associated with fish consumption by recreational users (adults, as well as young and older children) exceeded the upper end of the 10^{-6} to 10^{-4} target risk range for the RME scenario, ranging from 2.4×10^{-4} (for young children) to 7.8×10^{-4} for adults. These RME risk estimates were principally related to exposure to polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs), each of which individually contributed risks greater than 1×10^{-4} for the adult receptor (Appendix B, RAGS Table 8.1).

The principal chemicals contributing to risk for each pathway, defined as contributing individual (chemical-specific) risks of greater than 10^{-6} , or contributing at least 10 percent of the pathway-specific risk, are shown on Table 6-4 for the RME and CT scenarios. Mercury compounds (inorganic mercury and methylmercury) are identified by USEPA as Class C (possible human) carcinogens; however, there are no cancer toxicity data to quantitatively evaluate this endpoint.

The CT cancer risk for fish consumption was calculated slightly below 1×10^{-4} , at about 4.5×10^{-5} for all recreational receptors (adults, young children, and older children), with the same chemicals (i.e., PCBs and PCDD/PCDFs) contributing the bulk of the risk.

As the risks associated with fish ingestion exceeded risks from other pathways for all receptors by a large margin (typically by an order-of-magnitude or more), Tables 6-2 and 6-3 also show the total receptor risk without the fish ingestion pathway included. Cancer risk estimates for other exposure pathways (excluding fish ingestion) for recreational users and construction workers were less than 10^{-4} . However, RME risk estimates (Table 6-2) for exposure to soils and sediments (lake sediments, wetlands, and dredge spoils) exceeded 10^{-6} for all receptors except construction worker exposure to northern basin sediments and Wetland SYW-10. Central tendency risk estimates (Table 6-3) were much lower, typically by about one order-of-magnitude. Central tendency risk estimates equaled or exceeded 10^{-6} for at least one receptor for southern basin sediments, northern wetlands (Wetlands SYW-6 and SYW-10), and one of the southern wetlands (Wetland SYW-19), ranging from 1×10^{-6} (older child exposure to Wetland SYW-10 sediments) to 1.4×10^{-5} (older child exposure to Wetland SYW-6 sediments) (Table 6-3).

Cancer risks associated with the lake sediments and wetland sediments were primarily from exposure to polycyclic aromatic hydrocarbons (PAHs) (particularly benzo[a]pyrene), as well as hexachlorobenzene, arsenic, and PCDD/PCDFs. Risks associated with exposure to the dredge spoils area are primarily related to arsenic, with lesser contributions from benzo(a)pyrene and hexachlorobenzene. PCBs did not generally contribute greatly to the risks associated with exposure to sediments, wetlands, or dredge spoils. Risks associated with dermal contact typically accounted for 50 to 90 percent of the cancer risk for exposure to sediments and soils.

Cancer risks associated with exposure to surface water were substantially below 10^{-6} for all receptors for both RME and CT scenarios (the highest value was 6.1×10^{-8} for the adult recreational RME scenario).

Although the elevated cancer risk estimates for consumption of fish from the lake were due primarily to exposure to PCBs and PCDD/PCDFs, a number of additional chemicals had RME risk estimates in the

fish consumption pathway that were greater than the lower end of the acceptable range identified by USEPA (i.e., 1×10^{-6}) for carcinogens. These included arsenic, bis(2-ethylhexyl)phthalate, hexachlorobenzene, 4,4'-DDE, aldrin, dieldrin, and heptachlor epoxide. Each of these risk estimates was less than 2×10^{-5} , with the highest risk estimates (for adult recreational receptors) being 1.8×10^{-5} for arsenic and 9.3×10^{-6} for dieldrin.

6.1.3 Quantitation of Cancer Risks by Receptor

The highest site risks and hazards are from the fish ingestion pathway; the fish ingestion pathway represents between 47 and 88 percent of the RME cancer risk for recreational receptors (see Table 6-2). Total risk estimates for each recreational receptor for all pathways combined, as shown on Tables 6-2 (RME) and 6-3 (CT), were essentially the same as the risk estimate associated with fish ingestion (e.g., the adult RME risk estimate for fish ingestion is 7.8×10^{-4} ; and the total adult RME risk across all pathways is 8.8×10^{-4}). The receptor-specific cumulative risk summary tables include both the total cancer risks (including fish ingestion), and also show the cancer risks associated with just the sediment, soil, and surface water pathways (i.e., excluding fish ingestion).

Although the great majority of the risks were related to fish consumption, the total RME risk estimates were greater than the lower end of the target risk range of 1×10^{-6} for all the recreational and construction worker receptors and pathways (either ingestion or dermal contact) for lake sediments, wetland sediments, and dredge spoils. The highest RME risk estimates, excluding fish consumption, were greater than 1×10^{-4} for the older child (3.8×10^{-4}) and adult (1×10^{-4}), based on exposure to the seven soil/sediment media (i.e., the northern and southern basin sediments, each of the four wetlands, and the near-surface dredge spoils) and the lake water. The RME risk from pathways other than fish ingestion for other receptors ranged from 2×10^{-5} for the construction worker to about 3.5×10^{-5} for the young child recreational receptor.

Although both the ingestion and dermal pathways were significant for all receptors, the dermal pathway contributed the majority of the risk for both the recreational and construction worker scenarios. For the older child recreational receptor, the RME dermal risk (about 3.6×10^{-4}) exceeded the ingestion (other than fish) risk (about 2.3×10^{-5}) by an order-of-magnitude. The ingestion risk was a larger fraction of the total RME risk for the other receptors (adults, young children, and construction workers), as shown on Table 6-2.

Central tendency cancer risks, summarized on Table 6-3, were also driven by fish ingestion, the risk from which was calculated as about 4.5×10^{-5} for all recreational receptors (adults, young children, and older children). Total CT risks associated with other pathways (excluding fish ingestion) were within the 10^{-4} to 10^{-6} range, going from about 2.6×10^{-6} for the young child to about 1.8×10^{-5} for the older child recreator. Both the dermal and ingestion pathways contributed to the overall (non-fish-ingestion) risk, with each pathway contributing cancer risks exceeding 1×10^{-6} for most recreational and construction worker receptors.

Detailed tables illustrating the risks for each pathway, receptor, and COPC are provided in Appendix B, RAGS Tables 9 and 10.

6.2 Non-Cancer Hazards

6.2.1 Methods

Unlike carcinogenic effects, potential adverse health effects that are non-carcinogenic are not expressed as a probability. Instead, these effects are expressed as the ratio of the estimated exposure (intake) over a specified time period to the reference dose (RfD) derived for a similar exposure period (e.g., CDI to chronic RfD). This ratio is termed a hazard quotient (HQ). If the CDI exceeds the RfD (i.e., an HQ of greater than 1.0), there may be concern for non-cancer adverse health effects. Exposures resulting in an HQ of less than 1.0 are not likely to result in adverse health effects.

In initial risk calculations, HQs for individual COPCs are summed for each exposure pathway to derive a hazard index (HI). Hazard indices for each exposure pathway are then summed to determine the total HI for each population of concern. The non-cancer hazards are presented in Tables 6-1 (summary), 6-2 (RME), 6-3 (CT), and 6-5 (COPC summary).

In the event a total HI exceeds 1.0, the HI is segregated by primary target organs because adding HQs of compounds that do not affect the same target organ could overestimate the potential for adverse effects. Consistent with the RAGS Part D guidance (USEPA, 1998), HQs are summed across exposure pathways for specific chemicals that share the same critical effect or primary target organ, as reported in USEPA's Integrated Risk Information System (IRIS), Health Effects Assessment Summary Tables (HEAST), or other sources (see Appendix B, RAGS Table 5.1, and additional discussion in Chapter 5 of this report), to determine a total HI for that target organ or critical effect, as shown in Appendix B, RAGS Table 9.

Table 6-6 shows the classification of COPCs by primary target organs (or systems) for calculating the target organ HI. Some COPCs have more than one target organ and, consequently, the HQs associated with these chemicals are summed under more than one target organ HI. Because the HQs for these chemicals are "double-counted," the sum of the target organ HIs may exceed the total HI.

Although the target organ HIs provide more information about the potential for adverse effects to result from the exposure conditions evaluated in each scenario, it is important to note that all chemicals may have health effects on organs or systems other than the primary organ/system reported in IRIS or HEAST (i.e., other effects may be associated with a chemical, but may only occur at higher doses). Thus, if all the individual target organ HIs are less than 1.0, the potential for adverse effects is less likely to be of concern; however, this result does not provide an absolute measure of certainty that adverse health effects could not occur.

6.2.2 Quantitation of Non-Cancer Hazards by Pathway

Tables 6-2 and 6-3 provide summaries of total HIs calculated for RME and CT scenarios, respectively. The chemicals contributing at least 10 percent of the total HI for each pathway, and each chemical exceeding risk target criteria (cancer risk of 10^{-6} or HI of 1.0), are identified on Tables 6-4 and 6-5, respectively. The fish consumption pathway exceeded an HI of 1.0 for all recreational receptors for both RME and CT scenarios, ranging from a maximum for the young child RME (HI of 28.3) to the adult CT (HI of about 4.5) recreational scenarios for Onondaga Lake. The RME HIs are less than 1.0 for the other pathways evaluated (see Table 6-1).

For the RME scenario associated with recreational use of Onondaga Lake, total HIs of 18.4 (adult) to 28.7 (young child) across all pathways for all media were calculated (Table 6-2). The majority of the hazard (over 95 percent for children and 99 percent for adults) was associated with ingestion of COPCs, predominantly PCBs and methylmercury, in fish. For adults, the elevated HI for the fish consumption pathway was primarily related to the following:

- Methylmercury: HQ of 3.9.
- PCBs (the total HQ for less chlorinated Aroclors [i.e., Aroclors 1016, 1221, and 1242] and more highly chlorinated Aroclors [i.e., Aroclors 1248, 1254, and 1254/1260] combined): HQ of 12.7.

Based on the assumptions and toxicity values used in this HHRA, these results indicate the potential for non-cancer adverse health effects as a result of long-term exposures via ingestion of lake fish. The fact that methylmercury concentrations in Onondaga Lake fish fillets frequently exceed US Food and Drug Administration (US FDA) action levels (49 percent, or 357 out of 728 fillet samples exceed the US FDA action limit of 1 mg/kg for mercury) is consistent with the finding of concern for adverse health effects from consumption of lake fish. Other than fish consumption, no RME pathways had an HI greater than 1.0.

For the CT scenario, the only pathway that exceeded an HI of 1.0 was fish consumption, which was exceeded for all recreational receptors, ranging from 4.5 for adults to about 7 for young children. For adults, the HI CT estimate for the fish consumption pathway (about 4.5) was primarily based on the following HQs:

- Mercury: HQ of 1.2.
- PCBs: HQ of 2.7 (the sum of the HQs for less chlorinated Aroclors [HQ of 0.52] and highly chlorinated Aroclors [HQ of 2.2]).

6.2.3 Quantitation of Non-Cancer Hazards by Receptor

The highest non-cancer hazards at the site are from the fish ingestion pathway, which represents more than 95 percent of the RME non-cancer hazard to recreational receptors. For example, the RME fish ingestion hazard for the young child recreator is 28.3, and the total hazard across all pathways (fish ingestion and exposure to COPCs in sediment and surface water) is 29.1 (see Table 6-2). The receptor-specific cumulative risk summary tables include the risks and hazards associated with just the sediment, soil, and surface water pathways. In addition to the non-cancer hazards for each pathway, Tables 6-2 and 6-3 show the total HI for each receptor: adult recreational; older child recreational; young child recreational; and construction workers.

The HIs for the three recreational receptor groups exceed 1.0 for both RME and CT scenarios, driven almost entirely by the HIs for fish ingestion (HIs of 18 to 28 for RME and 4.5 to 7 for CT in each case). To illustrate the degree of hazard from other pathways, Tables 6-2 and 6-3 also show the hazard to receptors for all media except fish consumption. The total RME HIs for the other pathways to which each receptor may be exposed (i.e., sediments, dredge spoils, and lake water) ranged from about 0.2 for the adult recreator to about 1.0 (the calculated HI is 0.98) for the older child recreator. These HIs may be high-end estimates, as the HIs for the adult and older child recreational receptors were calculated assuming that each of these receptors is exposed to all seven of the soil/sediment media (i.e., the northern and southern basin sediments; each of the four wetlands; and the near-surface dredge spoils) at the RME frequency.

The RME HI for the construction worker is below 1.0 (about 0.83). The HI for the construction worker may be high for the same reasons presented above for the recreational receptors, as it was calculated assuming that the worker is involved in construction projects in each of the seven soil/sediment media. The construction worker scenario assumed that the construction worker consumes no Onondaga Lake fish.

The CT HI for all recreational receptors is driven by fish consumption, which accounts for over 95 percent of the non-cancer HI for recreational receptors. The CT HIs (also shown on Table 6-3), excluding fish consumption, for all receptors (recreational and construction worker) were well below 1.0, ranging from less than 0.1 for the adult recreator to about 0.3 for the construction worker.

6.3 Summary of Risk Characterization

The HHRA focuses on current and potential future recreational uses of and construction worker exposure to Onondaga Lake, including fish consumption and COPCs in surface water, nearshore sediments, wetland sediments, and dredge spoils. The principal findings of the HHRA are as follows:

- PCBs, PCDD/PCDFs, and PAHs were the primary chemicals contributing to cancer risk estimates for Onondaga Lake media. Methylmercury and PCBs were the primary chemicals contributing to non-cancer hazard estimates for Onondaga Lake media.

- Consumption of fish was the only exposure pathway with a total cancer risk estimate greater than the upper end of the 10^{-6} to 10^{-4} target risk range for carcinogens, due primarily to PCBs and PCDD/PCDFs.
- RME cancer risk estimates were greater than the lower end of the acceptable risk range of 1×10^{-6} for at least one pathway and receptor for lake sediments, wetland sediments, and dredge spoils. The highest RME risk estimate, after fish consumption, was about 2.6×10^{-4} for older child exposure to Wetland SYW-6 sediments, with RME risks greater than 1×10^{-5} for both older and young children's ingestion and dermal contact with southern basin lake sediments, adult recreational exposure to Wetland SYW-6 sediments, older child exposure to Wetland SYW-12 sediments, and for adult and older child exposure to Wetland SYW-19 sediments.
- Consumption of fish had total HIs of greater than 1.0 for PCBs and methylmercury in both RME and CT estimates for adults, older children, and young children. No other pathway evaluated in this HHRA had an HI, either RME or CT, of greater than 1.0. The RME HI for young children's exposure to southern basin nearshore sediments (0.54) represents the next highest non-cancer hazard.
- The finding of elevated risk and hazard estimates for methylmercury and PCBs is consistent with the fact that concentrations of these chemicals in fish tissues collected from Onondaga Lake exceeded FDA action limits.
- Estimates for exposure to COPCs in the surface water of the lake were less than the lower end of the target risk range for carcinogens (i.e., 10^{-6}) and had HIs of less than 1.0, indicating little potential risk associated with recreational exposure to COPCs in water.

7. UNCERTAINTY ASSESSMENT

Because risk characterization serves as a bridge between risk assessment and risk management, it is important that major assumptions, scientific judgments, and estimates of uncertainties be described in the risk assessment. Superfund risk assessment methods are designed to be protective of human health and to address the uncertainties associated with each step in the risk assessment process.

This chapter addresses aspects of this HHRA that are likely to overestimate or underestimate site risks. The risk discussion below is organized according to the three principal areas of uncertainty described in USEPA's (1989) risk assessment guidance (RAGS Part A, Section 8.4):

- Selection of chemical substances included in the characterization. This includes both the initial selection of chemicals for which samples were analyzed, as well as the screening performed to refine the list of substances carried through the quantitative risk assessment.
- Exposure assessment. Uncertainties include both those associated with the data and chemical concentrations (e.g., exposure point concentrations [EPCs]), as well as uncertainties associated with the individual exposures (e.g., exposure frequency, consumption rate).
- Toxicity values. This includes uncertainties associated with the reference doses (RfDs) and cancer slope factors (CSFs) used in the quantitative HHRA, extrapolations from one route to another (e.g., oral to dermal), and the effect of substances which were not included in the quantitative HHRA due to lack of quantitative toxicity data.

7.1 Uncertainties Associated with Selection of Substances

The two areas of uncertainty associated with the selection of substances are the selection of analytical parameters for which samples from each medium (pathway) were analyzed, and the screening of contaminants to reduce the number of chemicals for the quantitative risk characterization.

7.1.1 Selection of Analytical Parameters

The selection of analytical parameters for samples analyzed for the HHRA was based largely on the site history and previous investigations, and focused on data for parameters with a reasonable potential to be present (e.g., mercury/methylmercury, chlorinated benzenes). However, since the full history of each of the exposure areas is not known, many samples were analyzed for a much broader range of contaminants (full organic Target Compound List [TCL] and inorganic Target Analyte List [TAL] contaminants) in order to determine if other analytes are present at concentrations of potential concern.

7.1.1.1 Fish Fillets

Analysis of fish fillets focused on the contaminants that have historically been of concern; specifically, mercury, pesticides, chlorinated benzenes, and polychlorinated biphenyls (PCBs). In recent years, analysis for polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs) has also been conducted on many of the samples. However, only a few of the fillet samples have been analyzed for the full suite of semivolatile organic compounds (SVOCs) (including polycyclic aromatic hydrocarbons [PAHs]) and TAL metals. Only four adult composite samples have been analyzed for volatile organic compounds (VOCs), as these compounds do not tend to bioaccumulate and the presence of VOCs in fish at high-enough concentrations to be of concern is not expected. The limited data support this assumption, as no VOCs were detected at concentrations exceeding the screening criteria.

With the exception of hexachlorobenzene, only four adult composite samples were analyzed for SVOCs (including PAHs). Since less than ten samples were analyzed for these substances, statistical evaluation of the data set (i.e., calculation of the 95 percent upper confidence limit [UCL] on the mean) was not possible. Therefore, for these compounds, the maximum detected concentration was used as the EPC (consistent with risk assessment guidance [USEPA, 1989]). However, bis(2-ethylhexyl)phthalate is the only SVOC affected, as it was the only SVOC compound (other than hexachlorobenzene) for which a detected concentration exceeded the screening criteria, and was, therefore, the only additional SVOC carried through the assessment as a contaminant of potential concern (COPC) for which the EPC was calculated.

Pesticide data are reasonably comprehensive, as there are at least 38 data points for most of the pesticides and over 100 data points for some (e.g., chlordane; DDT and related compounds; mirex/photomirex).

Despite the fact that a large amount of earlier (i.e., 1992) PCB data were considered to be unusable for this HHRA (see Appendix A), there are over 100 PCB analyses used in this HHRA, covering both low molecular weight (less chlorinated) Aroclors (either Aroclor 1016 or Aroclor 1242) and high molecular weight (highly chlorinated) Aroclors (reported as Aroclor 1254/1260). Although there are only seven fillets for which the full suite of individual Aroclors (including Aroclor 1268) were analyzed, the usable analyses conducted provide a sufficient data set for characterization of PCB concentrations in fillets for the HHRA.

There were 30 fish fillets analyzed for PCDD/PCDFs. Although some of the locations from which these samples were collected were biased toward suspected hot spots or source areas, there were sufficient data that a meaningful 95 percent UCL on the mean could be calculated. It was not necessary to use the maximum detected concentration as the EPC.

With the exception of mercury and methylmercury, analysis for metals was conducted on only seven fillets and four adult composite samples. The limited amount of data for TAL metals does make characterization of the EPCs of these constituents difficult. However, there were sufficient data to perform the statistical tests (determination of data distribution type and calculation of the 95 percent UCL on the mean). Meaningful 95 percent UCLs on the mean were calculated for all the TAL metals that exceeded screening criteria; in no case was it necessary to use the maximum detected concentration as the EPC.

7.1.1.2 Lake Sediments – Northern Basin

Northern basin nearshore shallow surface sediments (all intervals in the 0 to 30 cm range at stations in less than 6.5 feet [ft] [2 meters {m}] of water) were analyzed for full TAL inorganics, VOCs, SVOCs (including PAHs), pesticides, and PCBs. There were typically between 22 and 56 data points for each of these fractions (in isolated cases, there were less for an individual analyte within a fraction, due to slight differences in the target analytes reported by a laboratory or analytical methods). Five northern basin samples used in this HHRA were also analyzed for PCDD/PCDFs, and three samples for methylmercury (in addition to the 56 samples analyzed for total mercury). For these last two parameters, the maximum detected concentration was used as the EPC, as there were an insufficient number of data points for meaningful statistical analysis. The northern basin data set is comprehensive and complete, and little, if any, uncertainty is likely due to the type or quantity of analyses performed.

7.1.1.3 Lake Sediments – Southern Basin

Southern basin nearshore shallow surface sediments (all intervals in the 0 to 30 cm range at stations in less than 2 meters [m] of water) were analyzed for full TAL inorganics, VOCs, SVOCs (including PAHs), pesticides, and PCBs. There were typically between about 80 and 100 data points for each of these fractions (in isolated cases, there were less for an individual analyte within a fraction due to slight differences in TCLs or analytical methods). Eighteen southern basin nearshore samples used in this HHRA were also analyzed for PCDD/PCDFs, and 14 samples from seven locations were analyzed for methylmercury (in addition to the 114 samples analyzed for total mercury). The southern basin data set is comprehensive and complete (with the possible exception of methylmercury data), and little, if any, uncertainty is likely due to the type or quantity of analyses performed. Sufficient data are available for meaningful statistical analyses of all parameters, except methylmercury.

7.1.1.4 Wetland Sediments (SYW-6, 10, 12, and 19)

The wetland samples were analyzed for the full suite of TCL/TAL compounds. PCDD/PCDFs were analyzed in all wetlands except SYW-12, as per the Phase 2A Work Plan (Exponent, 2000). However, since each wetland was evaluated as a separate exposure area and there were only eight samples (and four data points, as the sample data were combined from each pair of samples collected from two depths at each of four locations) from Wetlands SYW-10, 12, and 19, no statistical analysis was performed on the wetlands data (a minimum of ten samples was used as the threshold for conducting statistical analysis of data sets for this HHRA); therefore, the detected maximum value of each COPC was used as the EPC. This may result in an overestimate of the EPC concentration; on the other hand, due to the limited number of samples, it is also possible that areas of higher concentration may have been missed in the sampling program.

Data from additional samples collected from Wetland SYW-6 in May 2002 by NYSDEC/TAMS were also used in this HHRA. This data set consisted of samples, which were analyzed for metals and SVOCs only, from two depths (0 to 15 cm and 15 to 30 cm) at each of five locations. As with the data from the

wetlands sampling performed in 2000, the two samples were composited mathematically to generate a single length-weighted average for each location. This increased the total number of data points for Wetland SYW-6 to nine (five from 2002 and four from 2000), which was still too few to perform meaningful statistical analyses; therefore, the maximum value of these nine length-weighted averages was used as the EPC for each COPC in Wetland SYW-6.

However, inspection of the data indicated that there was one location, and in particular one result (the 15 to 30 cm interval sample at 2002 Station W6-3), in which concentrations of PAHs were significantly higher than in other samples collected from this wetland. To assess the possibility that use of this one high value might overestimate the EPC, statistical analyses of the SYW-6 data set were performed on each of the 18 discrete samples (i.e., using each of the two data points at a given location as separate samples, rather than combining them into a single result at each location). The results of this alternative approach to evaluating the SYW-6 data are presented in Table 7-1A. In general, assessing each of the 18 samples as discrete data points results in EPCs which for most, though not all, COPCs are lower than the corresponding length-weighted maxima. Cancer risks and non-cancer hazards, as presented in Table 7-1B, are on the same order-of-magnitude, though lower, using the discrete sample approach. There is no change as a result of the discrete sample approach in which pathways exceed target risk levels, with the exception of the CT cancer risk for the construction worker, which is 1.5×10^{-6} using the maximum of the nine length-weighted average values, but is less than 10^{-6} (7.4×10^{-7}) when using the EPCs calculated from the UCL of the 18 discrete samples.

The data from the Wetland SYW-6 sample with the high PAH concentrations (2002 sample from Station W6-3) were compared to the data from the nearest sample collected in the Phase 2A investigation (Station S375 in 2000). The data from these two samples, which are from locations about 90 ft (27 m) apart, are compared in Table 7-2. As can be seen from the table, the metals data are comparable (most of the data agree within about 30 percent), but the Station W6-3 PAH data are much higher, typically by factors of about 50 to 100. This discrepancy between the PAH concentrations in relatively close samples suggests either that the high value is an anomaly and may be an outlier or that the distribution of contaminants in Wetland SYW-6 is very heterogenous and there may be other areas of high or even higher concentration that have not been sampled.

Wetland sediment samples were not analyzed for methylmercury. For calculation of EPCs and hazards, it was assumed that 1 percent of the total mercury was in the form of methylmercury (the remaining 99 percent was assumed to be inorganic mercury). The implications of this assumption are discussed in Section 7.5.3.1, below.

7.1.1.5 Dredge Spoils Area Soils

The dredge spoils data did not have a depth distribution corresponding to depth intervals preferred for use in HHRA's (nominally, from 0 to 6 inches [0 to 15 cm] for exposure to receptors other than construction workers). As a result, the depth interval used for assessing human recreational exposure to surface soils included data from samples at greater depths, up to 3.5 ft (107 cm). (Note that for the construction worker

receptor, data from all depths down to approximately 12 ft [3.5 m] were used.) The extent of the effect on the data and the potential direction of bias is unknown. However, most of these samples were collected from the relatively uniform cover material above the more-contaminated spoils; thus, no significant bias is expected.

For metals, SVOCs, and PCBs, only eight samples were collected from the depth interval used for the surface soil assessment; thus, the maximum detected value was used as the EPC for this pathway.

A total of about 40 dredge spoil area samples were analyzed (including the eight near-surface samples). None of the samples were analyzed for VOCs, and pesticide analysis was limited to a reduced suite examined in one deeper sample (Station S438). PCDD/PCDF analysis was conducted on about half the samples (20 in all, including four of the eight near-surface samples).

Dredge spoil samples were not analyzed for methylmercury. For calculation of EPCs and hazards, it was assumed that all (100 percent) of the total mercury was in the form of inorganic mercury. The implications of this assumption are discussed in Section 7.5.3.1, below.

7.1.1.6 Surface Water

The extent of available Onondaga Lake surface water data varies both among and within each analytical fraction.

Metals analysis was performed on 33 to 75 samples for 12 of the 23 TAL metals and methylmercury (not all samples were analyzed for the same suite of metals), and 32 samples were analyzed for dimethylmercury, ionic mercury, and elemental mercury. Although no analyses were conducted on any of the surface water samples (from the 0 to 3 m depth interval) for the other TAL metals, including arsenic, antimony, selenium, and thallium, they were not detected, or were detected at low concentrations, in samples collected from greater depths in the lake (6 to 12 m). Although the lake water is reasonably well characterized for the metals that were analyzed in near-surface (0 to 3 m) samples, it is possible that the calculated risks are underestimated for metals excluded based on data from samples collected from greater depths.

Full VOC analysis was performed on 11 of the 48 surface water samples, with a much greater number of analyses (i.e., 48) performed for the volatile contaminants considered most likely to be present – i.e., benzene, toluene, ethylbenzene, xylenes, and chlorobenzene.

SVOC analysis included 37 to 48 samples analyzed for three dichlorobenzene isomers and three trichlorobenzene isomers, plus five samples analyzed for hexachlorobenzene (not all samples were analyzed for the same suite of parameters, and some of the dichlorobenzene data came from the VOC analysis of some samples). No analyses of samples from the 0 to 3 m depth were performed for other SVOC compounds (including PAHs). However, four samples from the 6 to 12 m interval were analyzed, and the only SVOC COPC detected was bis(2-ethylhexyl)phthalate, and only at low concentrations (i.e., at a

maximum concentration of 10 µg/L, which is less than the Region 3 tap water screening criterion.). Therefore, the presence of other SVOC compounds in surface water is considered unlikely, and the quantitation of risks associated with SVOCs is affected minimally, if at all.

None of the surface water samples from the 0 to 3 m interval were analyzed for pesticides, PCBs, or PCDD/PCDFs; therefore, the extent of risk, if any, posed by the potential presence of these compounds was assessed using data from deeper samples (6 to 12 m). No PCBs or pesticides were detected in these deeper samples and are, therefore, not expected to be present in surface water (0 to 3 m) at significant concentrations. Onondaga Lake water samples have not been analyzed for PCDD/PCDFs.

7.1.2 Contaminant Screening

The contaminant screening, which is presented in Chapter 3, poses potential uncertainty only if chemicals that may present risk are inappropriately screened out of the quantitative risk assessment, or if chemicals that are not site-specific contaminants (e.g., naturally occurring substances whose detection in site samples is not a result of discharge or release of contaminants) are included in the quantitative HHRA. Each of these possibilities is discussed below.

7.1.2.1 Uncertainty Associated with Screening Contaminants

The screening was conservative in that the procedures used for exclusion of a compound from the quantitative assessment were rigorous. A compound was included as a COPC for the quantitative HHRA if there was any indication it could have an impact on risk. For all chemicals detected, the concentration used for screening was the maximum concentration detected, although in the quantitative risk characterization the 95 percent UCL on the mean was used as the EPC, where allowable. Non-carcinogenic chemicals were screened against screening criteria representing a hazard index (HI) of 0.1 (one-tenth of the concentration believed to be associated with the potential for toxic effects).

Inorganics (metals) designated as nutrients (calcium, potassium, sodium, and magnesium) were screened out (flagged “NUT” for “nutrient” on RAGS Tables 2.1 through 2.10) and were not assessed in the quantitative HHRA.

Screening criteria used were developed by USEPA Regions 3 and 9 using the same toxicity data as used in this HHRA (i.e., the Integrated Risk Information System [IRIS], the Health Effects Assessment Summary Tables [HEAST], and the National Center for Environmental Assessment [NCEA]). The USEPA Region 9 soil screening criteria include an estimate of dermal exposure so that those criteria address both of the complete pathways identified for the Onondaga Lake site (ingestion and dermal contact). Because of this, the USEPA Region 9 criteria are generally, though not always, more conservative than the USEPA Region 3 criteria. The USEPA Region 3 and Region 9 screening tables, along with text prepared by each region explaining the applicability and derivation of their screening criteria, are provided in Appendix C. To reduce the chance of screening out a contaminant that may contribute to risk, the more conservative of the USEPA Region 3 or Region 9 criteria were applied for each COPC.

The USEPA Region 3 fish ingestion screening criteria are based on a higher consumption rate than used in this HHRA (54 grams per day [g/day], rather than 25 g/day). Therefore, it is unlikely that any contaminants that may pose significant risk through the fish ingestion pathway were screened out of the assessment.

Surface water concentrations were screened against the USEPA Region 3 and Region 9 tap water criteria, which are based on ingestion of water as drinking water at a rate of 2 liters per day. As the ingestion rates assumed for this quantitative HHRA are based on the much lower ingestion rates for incidental ingestion through recreational or construction activities, the screening for ingestion of contaminants in surface water is unlikely to have eliminated significant contaminants. However, the exposure scenarios envisioned for the complete exposure pathways also include dermal contact with water; this pathway is not addressed in the USEPA Region 3 or Region 9 tap water screening criteria. To reduce the chance of screening out chemicals which might be significant contributors to dermal risk, a second screening was also conducted using the dermal criteria in the draft USEPA RAGS Part E (dermal guidance) document (USEPA, 2001a). Based on this further screening, three additional compounds (1,2-dichlorobenzene, 1,2,4-trichlorobenzene, and chromium) were added as COPCs although their concentrations were below the Region 3 and Region 9 screening criteria. Thus, it is unlikely that compounds that were screened out would add to potential risk.

7.1.2.2 Uncertainty Associated with Naturally Occurring Chemicals in Soil/Sediment

Inclusion of chemicals identified in the HHRA as COPCs that are naturally occurring may overestimate the site-specific component of the risk. This is generally not an issue for organic chemicals, as those that are assessed quantitatively in this HHRA are anthropogenic and thus are not naturally occurring, although the attribution of some relatively ubiquitous contaminants such as PAHs to an individual site may be problematic (see the Remedial Investigation [RI] report for further discussion [TAMS, 2002b]).

Many metals and inorganic substances are naturally occurring constituents of soil. In accordance with recent USEPA guidance (M. Sivak, pers. comm., 2002), no inorganics were eliminated from the HHRA based on their concentrations being similar to those in background samples. Metals that were detected in sediments at concentrations similar to background concentrations (defined as the maximum concentration detected being less than two times the average of the Otisco Lake samples) were flagged as "BKG" on the screening tables (Appendix B, RAGS Tables 2.2 through 2.6). Metals exceeding risk-based screening criteria, but detected at concentrations below the background screening concentration, include the following:

- Manganese (all evaluated media).
- Iron (northern basin nearshore sediments; Wetlands SYW-10 and 19).
- Arsenic (northern basin nearshore sediments; Wetland SYW-6).
- Thallium (northern basin nearshore sediments).
- Aluminum (Wetland SYW-10).

As shown in Chapter 6, Tables 6-4 and 6-5, manganese, iron, and arsenic contributed a significant portion (at least 10 percent) of the cancer risk or non-cancer hazard in some of the media in which they were detected at concentrations below the background concentrations. Although this indicates the possibility that the background levels of metals may result in an overestimate of their effect, it is not significant for non-cancer hazards, as the non-cancer hazards for media were well below 1.0. However, arsenic was a major contributor to cancer risks in the northern basin nearshore sediments and, to a lesser extent, Wetland SYW-6. Calculated RME risks to all recreational receptors exceeded 10^{-6} for the northern basin sediments, and calculated RME and CT cancer risks for SYW-6 exceeded 10^{-6} for construction workers and adult and child recreational receptors. For the northern basin sediments, elimination of arsenic from the cancer risk calculation would reduce the adult recreator RME risk to below 10^{-6} (from 1.3×10^{-6} to 6.5×10^{-7}); cancer risks to young and older children would still exceed 10^{-6} but would decrease from slightly less than 4×10^{-6} to slightly over 2×10^{-6} . If the contribution of arsenic to the cancer risk were removed from the calculation for SYW-6, the RME and CT cancer risks for all the receptors would still be greater than 10^{-6} , although the risk would be slightly lower (for example, the adult recreational RME would decrease from 6.5×10^{-5} to 6.4×10^{-5}).

7.2 Uncertainties Associated with Exposure Point Concentrations

Uncertainties associated with the EPCs used in risk calculations may be related to the quality and quantity of the available data, and to the manner in which the data are then processed to generate the EPCs used in the quantitative HHRA.

7.2.1 Uncertainties Related to Calculation Procedure for Exposure Point Concentrations

As discussed in Chapter 4, Section 4.7, the EPCs for the media evaluated in this HHRA were determined, based on guidance from NYSDEC and USEPA Region 2, as the lower of:

- The 95 percent UCL on the arithmetic mean of the site data (for data sets with ten samples or more).
- The maximum detected concentration (for all data sets with less than ten data points, and for larger data sets where the 95 percent UCL is greater than the maximum detected concentration).

Data sets were identified as best fitting either a normal or lognormal distribution (the higher W-statistic for data sets with $n < 50$, and the Y-statistic for data sets closer to zero for $n > 50$, as described in Gilbert, 1987). For data best fitting a normal distribution, the 95 percent UCL on the mean was calculated using the Student's t-statistic. For data identified as best fitting a lognormal distribution, the 95 percent UCL on the mean was calculated using Land's H-statistic. The equations for the UCL calculations are presented in Chapter 4, Section 4.7. The use of the 95 percent UCL on the mean for calculating the EPC, which is designed to be protective (i.e., there is less than a 5 percent chance that the true "population" average exceeds the calculated 95 percent UCL on the mean), may overestimate the actual concentrations to which

individuals would be exposed at Onondaga Lake. However, for robust data sets, the 95 percent UCL on the mean (true population mean) and the arithmetic average converge; this is the case for the Onondaga Lake fish fillet mercury data used in this HHRA, where the 95 percent UCL on the mean used as the EPC (1.08 mg/kg) is only very slightly higher than the arithmetic average (1.05 mg/kg).

Use of the maximum detected concentration when it is lower than the 95 percent UCL on the mean, and use of a maximum value for all data sets with fewer than ten samples (as was done here), are also potential sources of overestimation. Conversely, where there are only a few data points from which the EPC is derived, there is no certainty that the available data are representative of the site or that there may be higher – possibly much higher – COPC concentrations in the area. (This is especially true of Wetland SYW-6, as was previously discussed in Section 7.1.1.4.) Therefore, it is also possible that the use of the highest value of a small data set could underestimate EPCs if samples from more-contaminated parts of the area were not collected.

7.2.2 Uncertainties Related to Assigned Data Distribution Types and Resultant Upper Confidence Limit-Based Exposure Point Concentrations

As discussed in Chapter 4, Section 4.7.3, all data sets (for $n \geq 10$) were assigned to either a normal or lognormal distribution using a “best-fit” approach. However, not all data sets meet the criteria to be classified as either normal or lognormal distributions. To address this issue, USEPA has recently developed software (ProUCL 2.1) to calculate UCLs for these non-parametric data sets (USEPA, 2002c).

As much of the data processing for the calculations of the EPCs was substantially underway at the time the USEPA ProUCL 2.1 software became available, several subsets of the HHRA data were reevaluated using the ProUCL software to see how much of a difference, if any, use of the ProUCL software would make on the EPCs and resultant risk and hazard calculations. The discussion below is a summary of the more detailed evaluation presented in Appendix D.

The data sets selected for this evaluation were those for which the risk associated with the analyte exceeded the target risk level (for this purpose, a cancer risk of 10^{-6} or hazard quotient [HQ] greater than 1.0). The previously assigned data distribution was confirmed in the majority of cases (i.e., 11 of 18). In cases where the previously assigned distribution was not confirmed (i.e., ProUCL indicated that the data were neither normal nor lognormal), the EPCs were calculated using the ProUCL methods and compared to the EPCs utilized for the HHRA. The differences between the UCLs calculated by the two methods were generally not large, although for three of the 18 data sets evaluated the ProUCL-calculated value differed from the default value by more than 50 percent. However, even with this difference, the overall effect on calculated risks was small (see Appendix D, Table D-2), as these three chemicals were not significant contributors to the cumulative risk. As the effect on the EPC calculations and subsequent risk characterization was minimal, the EPCs presented in this HHRA are those using the original assignment of normal or lognormal distribution to each data set. The uncertainty associated with use of the best-fit approach to the data distribution and EPC calculations is considered to be small for the Onondaga Lake HHRA.

7.2.3 Effect of Cooking Loss on PCB and PCDD/PCDF Concentrations in Fillets

The RME calculations presented in the HHRA are based on a fish consumption rate of 25 g/day (this consumption rate is discussed below in Section 7.3), with no reduced fractional intake (FI) relating to the amount of fish consumed from the lake (i.e., FI of 1.0) and no loss of PCBs or PCDD/PCDFs due to cooking or trimming. A 33 percent reduction was assumed in the CT scenario, the rationale for which was discussed in Chapter 4, Section 4.3.1.3. While the literature shows that there are a wide range of estimates in the amount of PCB (and PCDD/PCDF) loss in cooking, the assumption of no loss for the RME scenario is certainly a reasonable one. The 33 percent loss assumed for the CT scenario is also reasonable; while there are some studies showing greater losses, others show less. The “health-protective values” (HPVs) established by the Great Lakes Sport Fish Advisory Task Force (GLSFATF) for Great Lakes fish assume a 50 percent loss of organic contaminants through preparation and cooking; however, the Great Lakes advisories are also based on a higher assumed fish consumption rate of 140 g/day (225 8-ounce meals per year) for “unrestricted” consumption (GLSFATF, 1993). In addition, many of the target species in the Great Lakes, such as chinook salmon (*Oncorhynchus tshawytscha*) and lake trout (*Salvelinus namaycush*), are not present in Onondaga Lake, and the percent reductions in organic contaminants vary by species (Table C-1 of USEPA, 2000b).

Recent USEPA guidance (USEPA, 2000b) presents some data specific to species evaluated for Onondaga Lake. One older study (Skea et al., 1979, as cited in Appendix C of USEPA, 2000b) showed PCB reductions in smallmouth bass (*Micropterus salmoides*) ranging from 16 to 80 percent. However, most of the more recent data (e.g., a number of studies by Zabik in the mid-1990s) show PCB losses in walleye (*Stizostedion vitreum*) ranging from about 15 to 30 percent for Great Lakes fish. Homolog-specific PCB data for five (unspecified) species showed reductions ranging from 15 to 40 percent, with most of the reductions in the 30 to 35 percent range (Zabik and Zabik, 1996; cited in Table C-1 of USEPA, 2000b). It was also noted that “... smoking lake trout reduced pesticides and total PCBs significantly more than other cooking methods, but this cooking method resulted in the formation of PAHs” (Zabik, 1994, as cited in USEPA, 2000b).

7.3 Uncertainties Related to Fish Consumption Rates

In this section, uncertainties related to assumptions made for the fish ingestion rates used in the exposure assessment are discussed. As ingestion of fish was identified as the most significant risk pathway for exposure to Onondaga Lake COPCs, and because assumed fish ingestion rates are sometimes a controversial issue in risk assessments, the issues associated with the approach taken in this HHRA are discussed in depth below.

In the quantitative risk characterization presented in Chapter 6 of this HHRA, the RME calculations were conducted using a fish consumption rate of 25 g/day for adults, an FI of 1.0, and the assumption that COPC concentrations are not reduced by cooking. The recreationally caught fish ingestion rates were estimated as two-thirds of the adult rate for older children (6 to less than 18 years old) and one-third of the

adult rate for young children (up to age six). In this section, these assumptions are evaluated and their potential effect on the calculated risk is discussed.

7.3.1 Fish Consumption Rates for Adults

A comprehensive review of available data on fish consumption was used by USEPA in developing the RME (and CT) recreational angler fish consumption rates presented in the Exposure Factors Handbook (USEPA, 1997a) and utilized in this HHRA. The fish consumption rate of 25 g/day used in the RME risk calculations was derived by USEPA from the 95th percentile fish consumption rates from surveys of anglers on the Great Lakes and in Maine. This rate is lower than the value previously used by USEPA (i.e., 54 g/day; USEPA, 1991b) and the value used in the Onondaga Lake Public Health Consultation (i.e., 32 g/day; NYSDOH and ATSDR, 1995). Because there are no data specific to Onondaga Lake – and the Onondaga Lake fish advisories preclude generation of a reliable estimate – it cannot be determined how well the data from other freshwater bodies predict consumption rates for Onondaga Lake without advisories in place. As the direction of bias in this uncertainty is unknown, the HHRA may overestimate or underestimate exposure and risks.

USEPA (2000c) used a value of 17.5 g/day for recreational fishers (and 142.5 g/day for subsistence fishers) in setting the water quality criteria that are protective of human health (EPA 822-B-00-004; 2000c). This value is based on the 90th, rather than the 95th, percentile of the study data). Inspection of the data used in the Exposure Factors Handbook suggests that the 90th percentile value of the three studies used to derive the recommended default used in this HHRA would be close to the 17.5 g/day value used in deriving the water quality criteria. Therefore, the difference in these values (i.e., between 25 g/day and 17.5 g/day) represents a different policy based on slightly different use of the data, not uncertainty in the underlying data.

The default adult fish ingestion value (54 g/day) presented in the Standard Default Exposure Factors guidance (USEPA, 1991b) is not considered applicable based on more recent data and guidance (e.g., Connelly [1992] and the Exposure Factors Handbook [USEPA, 1997]).

7.3.2 Fish Consumption Rates for Children

Fish ingestion by children was evaluated quantitatively by using an assumed ingestion rate of two-thirds of the adult rate for older children, and one-third of the adult rate for young children. However, as there are limited data on which to establish age-specific ingestion rates for recreationally caught fish for children, this may underestimate or overestimate the actual ingestion rates for children, and thereby may underestimate or overestimate the risk to children.

This HHRA used fish ingestion rates for children recommended by USEPA Region 2. The ingestion rate for young children was assumed to be one-third of the rate for adults, and the rate for older children/adolescents (6 to less than 18 years old) was assumed to be two-thirds of the rate for adults. These rates were also recently used for the Hudson River PCBs site HHRA (TAMS/USEPA, 2000). It

should also be noted that the Hudson River HHRA used a slightly higher RME value for fish ingestion by adults – 31.9 g/day (based on the 90th percentile of the Connelly et al. [1992] study of New York anglers) – than the 25 g/day used for the adult RME for Onondaga Lake (based on the recommended RME value from the Exposure Factors Handbook [USEPA, 1997a]).

Other ingestion rates for children may also be assumed, depending on the study used and the manner in which the data are processed. For example, based on data provided by NYSDOH (Pao, 1982), which are also presented in summary form in USEPA (1997a), some assumed consumption rates for children, relative to adults, can be calculated (see Table 7-3). However, these consumption rates were not developed quantitatively due to the uncertainty of the underlying data and assumptions, and the calculated rates were presented merely to provide the reader with a sense of the range of potential error or underestimation that may be present by not addressing children's fish consumption as a separate receptor population. Consistent with USEPA policy and conventional practice, children were evaluated as two subgroups: young children (under six years old); and older children (ages 6 to less than 18). However, it should be noted that the limited amount of age-specific fish consumption data that are available do not necessarily correspond to the age ranges used in this HHRA.

An extensive literature survey completed recently by the California EPA, Office of Environmental Health Hazard Assessment (OEHHA) notes that, "... in some cases, although not all, the differences in the rates of fish consumption that have been reported are likely to correspond to differences in body weight" (California EPA, OEHHA, 2001). A linear multiplier recommended by USEPA (USEPA, 1994, as cited in OEHHA, 2001; while the version of the USEPA document cited has been superseded by USEPA, Revision 3, November 2000, the approach referenced is unchanged) "... does not account for the higher caloric requirements of young children, and pregnant and nursing women" (OEHHA, 2001). Table 7-3 presents estimates of age-based consumption of fish. No data relative to pregnant or nursing women were found.

The Great Lakes protocol states that it is "... assumed that the meal size will change proportionally with body weight. Most dieticians consider the best predictor of meal size to be the body mass of the individual" (p. 8 in GLSFATF, 1993). As shown on Tables 7-3B and 7-3C, the data from Rupp (1980) and Javitz (1980) suggest that younger children consume fish (finfish and shellfish from commercial and recreational sources, in grams per day) at an average rate of 10 to 30 percent greater than that of adults on a body weight-normalized basis, although both studies suggest that the consumption rate for older children is actually lower than that for adults. The data from Pao (1982), presented on Table 7-3D, suggest a more dramatic increase in consumption by children based on grams per meal. However, this apparent effect is mitigated by a US Department of Agriculture (USDA) study suggesting that the average number of meals per year of fish consumed by children is lower than that for adults (USDA, 1992, as cited in Table 10-46 of USEPA, 1997a). (The presentation of the USDA data makes it impossible to determine if there are a few children eating a lot of fish, or a lot of children eating a small amount of fish, since the data are presented as "percent of population consuming fish in one day." Regardless, the percentage is lower for children; for example, the percent consuming reported for adults is 10.9 percent, whereas for children [males or females] the corresponding value is 6 percent.)

In general, USEPA, by convention, explicitly or implicitly considers “children” to be “young children,” i.e., 0 to 6 years old (USEPA, 1991b, 1999). However, the peer-reviewed Hudson River HHRA (TAMS/USEPA, 2000) did evaluate consumption of fish by “child” and “adolescent” (in addition to adult) anglers. The age ranges evaluated in the Hudson River HHRA were assumed to be under six years old, and 6 to less than 18 years old (the age range was not stated, but was back-calculated based on the body weights used for each age group), and the body weights (15 kg for the young child, 43 kg for the adolescent) and RME exposure durations were consistent with the age ranges. The RME exposure durations were 6 years for the young child, 12 years for the older child/adolescent, and 23 years for the adult. (Summing these RME exposure durations across the age groups results in a total RME exposure duration of 41 years, which is higher than the normal “default” RME exposure duration of 30 years. However, there were detailed county-specific demographic data developed for the Hudson River HHRA, so site-specific, rather than default, exposure durations were used.)

The relative ingestion rates, using those utilized in the quantitative Onondaga lake HHRA as well as those derived from some of the other studies discussed above, are illustrated on Tables 7-3A through 7-3D. A comparison between the risks and hazards calculated using the Onondaga Lake assumptions and the linear estimate (body weight-normalized) assumptions is presented in Table 7-4. Both the cancer risks and the non-cancer hazards for children based on the linear (body weight-normalized) estimates are only slightly lower than the corresponding risks and hazards based on the baseline assumption.

7.3.3 Subsistence Fishers

Subsistence fishers were not evaluated quantitatively, as there is a lack of data regarding whether or not subsistence fishers (or other high-end consumers) exist in the Onondaga Lake vicinity, and a lack of data on fish ingestion rates for subsistence fishers. Based on limited data, USEPA (1997a) suggests an RME (95th percentile) rate of 170 g/day, and a CT (mean) rate of 70 g/day for subsistence fishers. As other factors in the risk calculation would be the same for these receptors, cancer risks and non-cancer hazards would be related linearly to the differences in consumption rates between the subsistence fisher and the adult angler; so the RME risks and hazards would be greater by a factor of about seven (170 g/day divided by 25 g/day), and the CT risks and hazards would be greater by a factor of about nine (70 divided by 8). RME cancer risks, which already exceed 10^{-4} , would increase further (e.g., the adult subsistence fisher risk would be about 5.3×10^{-3} , compared to 7.8×10^{-4} for the adult recreational fisher), and CT cancer risks, which are about 4.5×10^{-5} for all recreational receptors, would be about 4×10^{-4} for the adult subsistence fisher. Non-cancer hazards would also increase similarly (e.g., the adult subsistence fisher RME HI would be about 124, compared to the adult recreational fisher RME HI of 18.2). However, the very limited data also suggest that the ingestion rates, as well as the species consumed, are highly variable and site- or location-specific (see, for example, Table B-5 and section B.3.2 of Appendix B in USEPA, 2000b).

7.3.4 Fractional Intake

An additional factor considered in the uncertainty associated with this risk assessment is the amount of fish collected and consumed from Onondaga Lake versus from other resources. The fish consumption rates

used in the HHRA represent consumption from all freshwater resources fished during the season. As was the case for anglers evaluated in the studies used to derive these intake rates, however, several desirable fishing locations are available to anglers who live near Onondaga Lake. Thus, the RME fish consumption rates may not be representative of consumption from a single water body such as Onondaga Lake. The CT estimate also utilized an FI rate of 1.0 (i.e., assumed that all of an angler's total recreationally caught fish are from Onondaga Lake), but used the lower ingestion rate of 8 g/day.

The use of 1.0 as the fraction ingested for the CT scenario may lead to an overestimation of risk from fish consumption. An FI value of 0.3 would be consistent with two New York State sport-angler surveys, which evaluated the number of fishing locations for individual anglers (Connelly et al., 1990, 1992). The authors indicated that 63 percent of the respondents listed used at least three fishing locations. Use of the 0.3 FI value in this uncertainty assessment is also similar to the 0.25 FI value used by USEPA (USEPA, 1993b) in assessing upper-bound risks associated with consumption of fish in the Buffalo (New York) River, although empirically Onondaga Lake is a much more attractive fishing location and has been cited in publications as a highly desirable bass fishing spot. If the CT FI was reduced from 1.0 to 0.3, the associated risk would decrease accordingly (e.g., the adult fish ingestion CT HI would decrease from about 4.5 to about 1.3).

7.4 Uncertainties Associated with Exposure Assessment Assumptions

In the paragraphs below, uncertainties related to assumptions made for the exposure assessment are discussed. These include assumptions regarding ingestion (i.e., sediment and water, excluding ingestion of COPCs in fish, which was discussed above); exposure frequency (i.e., how many days per year an individual might be exposed to contaminated media); and similar issues. The identification of receptor populations with regard to the age of the receptors (young child, older child, or adult) is also discussed where appropriate.

For the most part, the rationale and basis for the assumptions made in the exposure assessment were presented in Chapter 4, Section 4.3.2 of this HHRA. This section is not intended to reiterate that material, but rather to assess qualitatively or quantitatively the effect of those assumptions on the risk characterization presented in Chapter 6.

7.4.1 Dermal Absorption Pathway Assumptions

There are a number of factors and assumptions which are particular to the dermal absorption pathway, including:

- Soil-to-skin adherence factors (SSAFs), addressed in Section 7.4.1.1.
- Dermal absorption factors (DAFs), addressed in Section 7.4.1.2.
- Route-to-route extrapolation factors, addressed in Section 7.4.1.3.

The field of dermal risk assessment has undergone significant change in the last few years; accordingly, USEPA guidance for dermal risk assessment has been revised and updated several times (USEPA, 1992c, 1999, 2001a). For the most part, the approach taken in this HHRA, including both the equations and the default assumptions, are consistent with the recently issued RAGS Part E (USEPA, 2001a).

7.4.1.1 Soil-Skin Adherence Factor

The SSAF is a measure of how much of the soil an individual is in contact with remains on the skin, thus serving as a source of contaminants that can be absorbed by the body. The SSAF is a function of both the type of activity in which the individual is engaged and the body part in contact exposed to the soil. A number of studies of SSAFs have been conducted for or reviewed by USEPA. The SSAFs used in this HHRA, referred to as the “consensus” SSAFs, are the result of several discussions among NYSDEC and their consultant TAMS; NYSDOH; USEPA; and consultants for Onondaga Lake National Priorities List (NPL) site Potentially Responsible Parties (PRPs). While these consensus SSAFs were established prior to the release of the current USEPA dermal guidance document, they are generally consistent with that guidance.

The specifics of the rationale for the various SSAFs used in this document are discussed in Chapter 4, Section 4.4.2 and are not repeated here. While the most recent USEPA guidance has been reviewed and utilized in this HHRA, it is noted that there are a wide range of SSAFs reported in the literature. Although extreme cases (e.g., the “kids-in-mud” scenario as reported in USEPA, 2001a and elsewhere) have not been used for establishing the SSAFs used in the quantitative HHRA, they do indicate the range of values that may exist. The values used for the RME assessment were selected to be both protective and reasonable; and the CT values were those thought, based on professional judgment, to most likely represent the typical individual of a given receptor type (e.g., adult or child recreator; construction worker). However, the true SSAF cannot be determined with precision and the values used in this HHRA may overestimate, or underestimate, the adherence of contaminated soil to potential receptor populations.

7.4.1.2 Uncertainties Related to Dermal Absorption Factors

This HHRA has used the relative absorption factors recommended by USEPA (listed in Chapter 4, Table 4-1) for both dermal and oral exposures to reflect the dermal and oral bioavailability of the constituents evaluated (i.e., the extent to which the constituents contacted or ingested are capable of being absorbed and available to act metabolically). Some of those absorption factors may overestimate the risks posed by dermal exposure, although the absence of DAFs for other contaminants (e.g., many of the metals) may underestimate exposure.

Experimental conditions from which the USEPA DAFs absorption factors were determined may differ from those which exist in Onondaga Lake, so the “true” site-specific DAFs may differ from USEPA’s experimentally determined factors used in this HHRA. For example, the DAF for PCBs is 0.14 (14 percent), based on a study by Wester et al. (1993) in which dermal absorption was measured following application of freshly spiked PCB soil/sand to the skin of monkeys; the application of PCBs from sand with

a low (less than 0.9 percent) organic carbon content would lead to greater dermal absorption than soils containing a higher fraction of organic carbon. Moreover, the Wester et al. study methodology assumed that dirt would remain pressed to the skin for 24 hours and not be washed off, which is an unrealistic scenario for most people and likely a source of risk overestimation for this factor. On the other hand, most of the experimental determinations of absorption factors have been conducted at loadings higher than those necessary to completely cover skin; consequently, the actual absorption factors "... could be larger than experimentally determined" (p. 3-22 of USEPA, 2001a), and the risks would be underestimated.

A default DAF of 0.1 was used for SVOCs (other than PAHs), based on USEPA's assessment of dermal absorption data for other organic chemicals. However, no default absorption factor was recommended for VOCs or metals other than arsenic or cadmium. VOCs and other metals are not, therefore, included in the dermal pathway in this HHRA. This is further discussed in Section 7.4.1.6, below.

7.4.1.3 Route-to-Route Extrapolation

Nearly all available toxicity data are based on pathways other than dermal absorption – i.e., ingestion or inhalation. Therefore, an assumption or extrapolation from toxicity data from one of the other routes – generally ingestion – must be made in order to assess the dermal absorbed dose, relative to that from the pathway used in the study(ies) from which the toxicity data (RfD or CSF) were derived.

In general, if the gastrointestinal (GI) absorption of a contaminant is relatively high – 50 percent or greater – no adjustment is made to the dermal route. However, studies have shown some substances, mostly metals, to be poorly absorbed from the GI tract. For these chemicals, an adjustment factor is applied to reflect the poor absorption in the oral RfD study. This factor is shown as the "oral to dermal adjustment factor" on RAGS Tables 5.1 and 6.1 (Appendix B).

There is obviously some uncertainty with regard to the route-to-route extrapolation. While no adjustment was made for any organic chemicals (since the GI absorption of organics is typically 50 percent or more, in accordance with RAGS Part E), the toxicity of the dermal absorbed dose may be underestimated to the extent that the GI absorption is less than 100 percent. There is also some uncertainty in the oral-to-dermal adjustment factors used for metals, although the direction of the uncertainty (bias) is unknown.

7.4.1.4 Dermal Absorption of Contaminants from Water

The contaminants absorbed from water are calculated differently than those from soil. For water, a chemical-specific permeability constant (K_p) controls the migration of contaminants from the water across the skin and into the body. While the available K_p values may not be precise, the risks and hazards associated with dermal absorption of contaminants from surface water were not found to be significant (i.e., non-cancer hazards were well below 1.0 [0.01 for RME] and cancer risks were below 10^{-6} [10^{-9}] for all receptors). Therefore, uncertainties associated with this pathway have little or no measurable impact on the overall risks.

7.4.1.5 Skin Surface Area Available for Dermal Contact

The values used in this HHRA are generally those recommended in USEPA RAGS Part E, which are, in turn, based on assumptions about the types of clothing likely to be worn by the receptor, coupled with studies of the surface areas of various body parts. The 50th percentile values of the skin surface areas were used. While the relationship is not strictly linear, a higher percentile of exposed skin area is typically associated with greater body mass; e.g., an individual at the high end of the skin surface area distribution would be expected to also be at the high end of the body mass distribution. Therefore, the ratio of skin surface area to body mass does not vary greatly, so the effect of using the 50th percentile for these variables (as opposed to a 90th percentile value) in the RME calculation is limited.

The assumptions regarding body parts exposed are less certain; some potential activities, such as children playing along the shoreline in the park areas (e.g., northern basin shoreline sediments) could result in close to 100 percent of the skin being in contact with the sediments. On the other hand, other activities such as older children looking for frogs or turtles in the wetlands, might involve much lower fractions of the body being in contact with the sediments (possibly just hands, and maybe feet). There is less uncertainty associated with estimates of the total skin surface area potentially in contact with Onondaga Lake surface water for high-contact recreational activities; for example, activities such as swimming or waterskiing would result in 100 percent of the total skin area being in contact with water.

7.4.1.6 Contaminants of Potential Concern Evaluated in the Dermal Pathway

Due to the general lack of data on dermal and GI absorption, USEPA (RAGS Part E) recommends quantitative analysis of the dermal pathway for only a relatively small number of COPCs (SVOCs, PCBs, PCDD/PCDFs, some pesticides, and two metals). USEPA (2001a) recommends no quantitative assessment of VOCs in soil; the rationale for this is that VOCs tend to volatilize and are accounted for by the inhalation pathway, which was not considered to be a complete pathway for the Onondaga Lake HHRA (see Chapter 4, Section 4.2.5). For inorganics other than arsenic and cadmium, USEPA indicates that absorption of metals is highly dependent upon speciation of the metals; as a result, there are too little data to recommend default absorption factors (USEPA, 2001a). As a result, risks associated with dermal absorption of some chemicals may be underestimated.

7.4.2 Uncertainties Associated with Ingestion of Soil/Sediment and Surface Water

Uncertainties associated with calculated intakes of soil/sediment and surface water are discussed below.

7.4.2.1 Soil/Sediment Ingestion Rates

The fact that USEPA recommendations for soil/sediment ingestion rates have changed over time suggests that there is some uncertainty associated with these estimates. USEPA (1991b) recommended default soil ingestion rates of 100 mg/day and 200 mg/day for adults and young children, respectively; no estimates were provided for older children or construction workers. The more recent Exposure Factors Handbook

(USEPA, 1997a) recommended a 50 mg/day ingestion rate for adults in residential or commercial/industrial settings and 100 mg/day as the mean ingestion rate for children.

Risk estimates in this HHRA used soil/sediment ingestion rates of 100 mg/day (RME) and 50 mg/day (CT) for adults and older children and 200 mg/day and 100 mg/day for young child RME and CT, respectively. (The Exposure Factors Handbook notes that although children might ingest up to 400 mg/day, there were insufficient data to recommend a 95th percentile soil ingestion rate for children.) While not explicitly making a recommendation for construction worker ingestion, the Exposure Factors Handbook does cite a study by Hawley (1985) which suggested a 480 mg/day rate. This rate (480 mg/day) has been used in other Onondaga Lake subsite risk assessment documents (e.g., LCP Bridge Street HHRA [NYSDEC/TAMS, 1998a]); however, USEPA has reevaluated the study on which this rate was based and currently recommends a construction worker ingestion rate of 330 mg/day (Exhibit 5-1 in USEPA, 2001c). It is acknowledged that there is an extremely limited data set for these assumptions, especially for the construction worker scenario; however, the direction of the bias is unknown.

7.4.2.2 Fraction Ingested from Contaminated Site

The soil/sediment ingestion rates used were applied on the assumption that the individuals' total daily intake of soil came from the Onondaga Lake site – in other words, the FI term in the intake equation is assumed to be 1.0. For recreational users of the site, who would be expected to be present at the site for only a limited time on any given day, this assumption is conservative although not unrealistic. While it is likely that, for such users, at least some portion of their total daily intake of soil would come from other sources that are uncontaminated, such as other outdoor areas and/or indoor dust, it is reasonable that incidental soil ingestion is likely to occur at much higher rates during recreational activities than during other, typically more passive, activities. Although Stanek and Calabrese (1992) found that nearly 50 percent of the total daily soil ingestion of 64 preschool children originated from indoor dust, it is not known to what extent the other activities engaged in by those children are representative of activities (and hence ingestion) likely to occur at the shore, wetlands, or the dredge spoils area adjacent to Onondaga Lake. Thus, the assumed FI of 100 percent of soil/sediment ingestion from the contaminated site, as used in this HHRA, may overestimate actual exposures to site soils and sediments via incidental ingestion, although it is possible that the default ingestion rate could underestimate ingestion as well.

7.4.2.3 Surface Water Ingestion Rates

The USEPA default assumption of 50 mL/hour (about 2 ounces/hour) ingestion while swimming was used for assumed high-contact recreational activities such as swimming and waterskiing (Exhibit 6-13 in USEPA, 1989); this value was used for both the RME and CT scenarios. The uncertainty associated with this rate is unknown.

7.4.2.4 Duration of Swimming Events

The total intake of COPCs during an “event” is also dependent of the length (duration) of the event. RAGS Part A cites an average of 2.6 hours per swimming event, based on US Department of Interior (USDOI) data; however, the Exposure Factors Handbook cites other data showing an average of one hour per swimming event. Due to the discrepancy between the two values, the higher number (2.6 hours) was used in the RME calculation, and the lower (1 hour) was used in the CT calculations. These values were used for calculation of COPC intakes resulting from both ingestion of and dermal contact with surface water. Although there is some uncertainty in these values, it is unlikely that many receptors would have a significantly higher exposure than the RME value assumed.

7.4.3 Body Weight

The 50th percentile body weight was used for young children and older children, based on the data in the Exposure Factors Handbook. Although recent data show 71.8 kg (rather than 70 kg) as the 50th percentile body weight for adults, the USEPA default value of 70 kg is used in this HHRA. The 70 kg adult body weight is used both by convention and due to the fact that many of the toxicity values (RfDs and CSFs) are based on the assumption of a 70 kg body weight. The body weight is a less sensitive parameter (varying by less than a factor of two between the 5th percentile and 95th percentile for both male and female adults) than some of the other variables in the risk calculations, so the 50th percentile body weight used for all scenarios does not have a major impact on the uncertainty in this HHRA. The error introduced by this is negligible, and the uncertainty introduced is insignificant in comparison to the uncertainty associated with some of the other parameters (e.g., estimates of exposure frequency or ingestion rates).

7.4.4 Exposure Duration and Frequency

The assumed exposure durations used in this HHRA were 30 years for the adult RME and nine years for the adult CT. These default values were established in the Exposure Factors Handbook (as cited in USEPA RAGS Part A, 1989), based on national demographics (30 years being the 90th percentile value, and nine years the 50th percentile) on the length of time individuals live at a single residence. (More recent studies summarized in the Exposure Factors Handbook [USEPA, 1997a] provided estimates very similar to the default values recommended in 1989 and used in this HHRA.) These default assumptions may underestimate exposure of individuals in situations where exposure may continue even if a person moves but continues to be exposed to the site; for example, a move within Onondaga County. Since some people live in a single location or single area for periods longer than 30 years (e.g., 70 years or more), the cancer risks, although not the non-cancer hazards, would increase proportionally for such persons.

In the HHRA conducted for the Hudson River PCBs site (TAMS/USEPA, 2000), a detailed evaluation of the exposure duration for fish ingestion (based on the number of years an individual fishes in the Hudson River site) was conducted. This evaluation was based on three factors:

- The age an individual begins fishing.
- When the individual stops fishing.
- The length of time the person resides in the five-county area adjacent to the 34-mi (54.7-km) length of Hudson River under consideration.

Despite the fact that a significantly larger area was under consideration, the Hudson River-specific exposure durations were only about one-third greater than the standard defaults assumed for the one-county Onondaga Lake HHRA area. Specifically, the 95th percentile exposure duration for the Hudson River HHRA was about 40 years (compared to the 30-year RME exposure duration for this Onondaga Lake HHRA), and the 50th percentile exposure duration for the Hudson River was 12 years (compared to the nine years used as the CT exposure duration for the Onondaga Lake HHRA). The exposure durations calculated for the Hudson River suggest that the USEPA defaults used for the Onondaga HHRA are reasonable and are not likely to significantly understate the exposure to the vast majority of receptors. However, exposure may be underestimated for those individuals who live in the same residence their entire life, or who change residences but remain near Onondaga Lake.

In accordance with the risk characterization equations provided in RAGS Part A (USEPA, 1989), an averaging time of 25,550 days (70 years \times 365 days/year) was used for calculations of cancer risk. The more recent data in the Exposure Factors Handbook (USEPA, 1997a) indicates that the current average life span is 75 years. However, the 70-year (25,550-day) period was used as the averaging time for cancer calculations, as the derivation of CSFs is based on an average lifetime of 70 years. As the averaging time appears in the denominator of the risk calculation, a higher averaging time of 75 years would effectively decrease the calculated cancer risk by about 7 percent.

For non-cancer hazard calculations, the averaging time is equal to the exposure duration multiplied by 365 days/year. As the exposure duration values cancel each other out in the non-cancer hazard calculation (the exposure duration is in the numerator, and the averaging time is in the denominator), there is no uncertainty associated with the non-cancer averaging time.

The exposure frequency is not an uncertainty issue for fish ingestion, as the data used are a daily average ingestion rate, so a duration of 365 days is necessary to balance the equation. Issues related to the uncertainty of the fish ingestion rate were discussed previously in this chapter.

For northern basin sediments, including the two northern basin wetlands, SYW-6 and SYW-10, 44 days/year was used for the RME frequency and 32 for the CT. By comparison, the Hudson River HHRA used an assumed recreational exposure frequency of 26 days for the RME and 13 days for the CT for children, and 13 days for the RME and seven days for the CT for adults. However, acknowledging that there might be a subpopulation of highly exposed recreators for the Hudson River, an "avid recreator" subpopulation was also evaluated. For the Hudson River avid recreator, the assumed exposure frequencies for adults and adolescents were 104 days for the RME and 52 days for the CT (children below the age

of 12 were not assumed to be avid recreators). The exposure frequencies used for the northern basin of Onondaga Lake are within the range used for the Hudson River recreator and are approximately half of the frequencies used for the Hudson River avid recreator, and as such are reasonable for a typical recreator. However, the exposure frequency used in this HHRA may not capture all highly exposed individuals (i.e., avid recreators) and thereby may underestimate risks to such persons.

In order to assess the hypothetical avid recreator, site-specific high-end exposure frequencies have also been estimated using the following assumptions:

- The exposed population is older children (age 6 to 18 years old).
- Exposure occurs only when the daily maximum temperature is 70°F or higher (May 19 to September 21, based on 1971 to 2000 Syracuse weather data) (NOAA, 2002).
- Exposure occurs five days/week during summer vacation (assumed to be ten weeks, from June 23 to September 1).
- Exposure occurs two days/week (i.e., on weekends) during the school year (May 19 to June 23, and September 2 to September 21) for a total of eight weeks.

Based on the assumptions listed above, the avid recreator exposure frequency would be 66 days/year $[(5 \times 10) + (2 \times 8)]$. This hypothetical avid recreator exposure (66 days/year) is 50 percent higher than the RME value (44 days/year) assumed for the northern basin sediments. Cancer risks and non-cancer hazards would also increase correspondingly (i.e., by 50 percent) for the avid recreator. However, this would not increase the HI to a value greater than 1.0, nor would it change the cancer risk category for the northern basin sediments or Wetlands SYW-6 or SYW-10.

The exposure frequency for southern basin sediments, including Wetlands SYW-12 and SYW-19, was assumed to be 14 days/year for the RME and five days/year for the CT. This exposure frequency is much lower than that assumed for the northern basin, due to the relative lack of potential points of contact, as the landside borders of the southern basin are dominated by industrial and commercial properties, while the northern basin shores have large amounts of parkland. It was, therefore, considered unlikely that exposures would be as high to the southern basin sediments as to the northern basin sediments, even under future scenarios. However, as with the northern basin sediments, an exposure frequency as high as 66 days per year could be used as the RME, assuming visits to the southern basin using the same assumptions as described above for the northern basin hypothetical avid recreator. Under this hypothetical avid recreator scenario, RME exposure, and, consequently, the associated risks and hazards, would increase by a factor of about 4.7 (66 divided by 14). Under this scenario, the RME cancer risk for the older child exposure to Wetland SYW-19 sediments would increase to over 10^{-4} (from about 4.9×10^{-5} to about 2.3×10^{-4}); other risk and hazard categories would be unchanged. The appropriateness of the professional judgment

estimates of exposure frequency is uncertain for the southern basin, and it is certainly possible that higher or lower estimates might better represent the likelihood of exposure.

For the construction worker scenario, the exposure frequency was assumed to be 25 days (five weeks) for the RME, and ten days (two weeks) for the CT; in each case, the exposure duration was assumed to be two years. These estimates are based on the assumption that the nature of the construction activity will be sewer line installation, utility repair, or similar work, but these exposure frequencies would probably be low for a construction period for erecting a structure (e.g., a warehouse or marina). Due to the fact that much of the lake area includes regulated wetlands or parkland, large-scale construction projects in areas covered by this HHRA (lake sediments, wetlands, and the dredge spoils area) were considered unlikely. However, to the extent that such a larger project may occur, this HHRA may underestimate risks or hazards to construction workers. For example, if a more generic default assumption of 250 days (i.e., the construction worker default in USEPA, 2001c) for a larger construction project is applied, the RME risks and hazards for the construction worker would increase by a factor of 10 (250 days divided by 25 days). This would result in the RME HI for the construction worker increasing to greater than 1.0 for southern basin sediments (HI of 2.2), Wetlands SYW-12 (HI of 1.4) and SYW-19 (HI of 1.6), and the dredge spoils area soils (HI just over 1.0). RME cancer risks would increase to greater than 10^{-6} (from about 6×10^{-7} to about 6×10^{-6}); other risk and hazard categories would be unchanged (i.e., cancer risks would still be less than 10^{-6} , and HIs would still be less than 1.0).

7.5 Uncertainties Related to Available Toxicity Data

The toxicity data discussed in this section are the quantitative values used in the risk characterization presented in Chapter 6 of this HHRA; specifically, the non-cancer RfDs and the CSFs used to calculate risks and hazards. It is important to note that the toxicity data developed by USEPA are designed to be plausible upper-bound estimates; i.e., the values are intended to be protective. From that perspective, some of the toxicity values used may be considered to be "conservative." With that in mind, the three main issues related to the uncertainty of the toxicity data used in this HHRA are:

- Accuracy of toxicity data (RfDs, CSFs) for the COPCs (including chemical "surrogates" approved by NCEA, such as pyrene for some non-cancer PAHs and lindane [gamma-HCH] for delta-HCH).
- Effect of lack of quantitative data for chemicals that have been detected. The issue of chemicals that may be present but were not analyzed, or at least not consistently analyzed, is addressed in Section 7.1.1.
- Toxicity data assumptions for chemicals for which the specific form was not analyzed, including:
 - Fish: Mercury and arsenic forms.
 - All media: Chromium (trivalent and hexavalent) and mercury forms.

It should also be noted that NYSDOH has developed quantitative risk factors for a few chemicals for which USEPA has also published values or, in a few cases, has recommendations for assessment of COPCs for which USEPA does not have published toxicity values. Onondaga Lake-specific COPCs for which NYSDOH has developed toxicity values (i.e., CSFs or RfDs) include the following:

- Cadmium.
- Benzene.
- Benzo(a)pyrene.
- DDE.
- delta-HCH.
- Chlordane.

Although tending to be slightly more conservative, the NYSDOH toxicity values for these chemicals are generally similar to those developed by USEPA and are not for the major COPCs that contribute to the cancer risks or non-cancer hazards in this HHRA. The exception to this is dioxins/furans (PCDD/PCDFs), for which NYSDOH recommends a quantitative assessment for non-cancer effects. A semi-quantitative assessment of potential non-cancer hazards from dioxin-like compounds is presented in Section 7.5.1.3.

7.5.1 Accuracy of Quantitative Cancer and Non-Cancer Toxicity Values

7.5.1.1 Polychlorinated Biphenyls

Issues related to both the cancer and non-cancer toxicity data are discussed below.

Cancer Slope Factor for PCBs

The CSF is an upper-bound estimate of the carcinogenic potency of a chemical used to calculate cancer risk from exposure to carcinogens by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over that lifetime. IRIS, the online database of USEPA's consensus review of toxicity data, provides both upper-bound and central estimate CSFs for three different tiers of PCB mixtures, as described in Chapter 5, Section 5.2.6.1. These values are based on USEPA's reassessment of the carcinogenic potency toxicity data for PCBs (USEPA, 1996a), and were derived using the proposed revisions to the USEPA Guidelines for Carcinogen Risk Assessment (USEPA, 1996b). A range of potency estimates was determined using studies for a range of mixtures, rather than focusing on the highest-potency mixture. USEPA's reassessment concludes that "uncertainty around the CSF estimate extends in both directions." However, the overall CSFs developed by USEPA represent plausible upper-bound estimates, indicating that there is reasonable confidence that the actual cancer risk will not exceed the cancer risk calculated using the CSF.

Reference Dose for Non-Cancer Hazards of PCBs

The non-cancer hazards for PCBs are based on the low molecular weight (Aroclor 1016) and high molecular weight (Aroclor 1254) oral RfDs in IRIS, as summarized in Chapter 5, Section 5.2.6.2 of this HHRA. The confidence level for both the RfDs is characterized by USEPA in IRIS as medium, indicating a moderate amount of uncertainty with regard to the numerical values. The combined uncertainty and modifying factors used in the development of these factors are 100 for Aroclor 1016 and 300 for Aroclor 1254; these uncertainty factors are lower than those for the majority of the other organic COPCs assessed in this HHRA (see Appendix B, RAGS Table 5.1).

It is noted that there are numerous studies, both human and animal, of the health effects of PCBs, some of which have been published since the last significant revision to the IRIS file. As noted in the Hudson River PCBs Site HHRA (TAMS/USEPA, 2000), USEPA is currently performing an evaluation of these studies as part of the ongoing IRIS process. In addition to carcinogenicity, these recent studies have focused on non-cancer effects including the developmental, neurotoxic, thyroid, immunological, and reproductive effects of PCB exposure.

Assessing both exposure to and effects of PCBs *in utero* to fetuses, to nursing infants, and to the children of exposed individuals (i.e., those with higher-than-normal body burdens of PCBs) is even more complex. PCB transfer from the placenta and from breast milk can result in significant exposures *in utero* and to nursing infants (DeKoning and Karmaus, 2000, as cited in the Hudson River PCBs Site HHRA [TAMS/USEPA, 2000]). However, the means for assessing the effects of such exposure quantitatively do not yet exist. Therefore, fetuses, nursing infants, and children of exposed individuals may constitute an additional highly exposed subpopulation on whom the effects of PCB exposure cannot be quantified.

Dioxin-Like PCBs

Twelve PCB congeners (non-ortho and mono-ortho substituted) have been identified as “dioxin-like PCBs” (Van den Berg, et al., 1998), and toxicity equivalence factors (TEFs) have been calculated for these congeners. As no congener-specific PCB analyses were performed for the Onondaga Lake investigation, the specific cancer potential of these congeners is not assessed. However, the Aroclor CSFs were developed using commercial Aroclors which contain these congeners and, therefore, the cancer potential of these congeners is included in the CSF for PCBs. However, to the extent that the distribution of congeners in the environment differs from that of the commercial Aroclors used to develop the CSFs, the associated risks could be either higher or lower than those presented in this HHRA.

7.5.1.2 Methylmercury Non-Cancer Reference Dose

The methylmercury RfD of 0.0001 mg/kg-day (1×10^{-4} mg/kg-day, or 0.1 µg/kg-day) is published in IRIS, and USEPA notes that confidence in this value is high. The RfD was originally derived in 1995 from data on delayed walking reported in Iraqi infants whose mothers were accidentally exposed to relatively high levels of alkylmercury in grain (Marsh et al., 1987).

In 1999, the National Research Council (NRC) was directed by Congress to review the USEPA-derived RfD and to focus on review of new studies published since USEPA established the RfD. The NRC report, *Toxicological Effects of Methylmercury*, was published in 2000; the current RfD was derived from the findings of that report and confirms the RfD from IRIS (1×10^{-4} mg/kg-day). A benchmark dose approach (BMD) was used, rather than a no observed adverse effect level/lowest observed adverse effect level (NOAEL/LOAEL) approach, as the response variable for analyzing the neurological effects in children.

The adverse effect of methylmercury at the lowest observed dose is neurotoxicity (i.e., the brain is the most sensitive organ), particularly among developing organisms. Among the extensive array of peer-reviewed data from low-dose exposure to methylmercury, USEPA and NRC identified three longitudinal developmental studies of the human populations consuming mercury-contaminated marine mammals. The three studies determined to be suitable for use in establishing quantitative risk assessment are the Seychelles child development study, ongoing studies of children in the Faeroe Islands, and the study of children in New Zealand. Although the Seychelles study found no evidence of impairment related to methylmercury exposure, the Faeroe and New Zealand studies found dose-related adverse effects on a number of endpoints. In the establishment of the RfD, emphasis was placed on the results of the Faeroe Islands study, as it is the larger of the two studies that identified methylmercury-related developmental neurotoxicity. Supporting evidence from the New Zealand study provides assurance that this focus is the appropriate strategy for protecting public health. In addition, an integrative analysis of all three studies was also performed.

Benchmark dose analyses were performed for a number of endpoints from all three studies (Chapter 7 of NRC, 2000). NRC estimated a CT measure, equivalent to a BMD, across all three studies for all endpoints that were identified as significant in the Faeroe Islands and New Zealand studies, and for all endpoints at 5.5 years of age in the Seychelles study. NRC also determined a lower limit based on a theoretical distribution of BMDs, which is the logical equivalent of a BMDL (benchmark dose limit). NRC also used a hierarchical random-effect model to reduce random variation in the estimate for these same endpoints from all three studies (Table 7-5, pp. 290-294 in NRC, 2000). Additionally, this analysis was used in calculating BMDs and BMDLs for the most sensitive and median endpoints from both the Faeroe Islands and New Zealand studies (Table 7-6, p. 294 in NRC, 2000). This approach also allowed an integrative analysis of data from all three studies.

The IRIS file for methylmercury presents BMDL_{05s} (BMDLs at the 5 percent significance level) from a number of endpoints in terms of cord-blood mercury (see Table 2 of IRIS file, included in Appendix E of this HHRA). These tests are all indications of neuropsychological processes involved in a child's ability to learn and process information. The BMDLs for these scores are all within a relatively close range. They were converted using a one-compartment model to an ingested dose of methylmercury that would result in the cord-blood level. The last column shows the corresponding RfD from application of an uncertainty factor (UF) of 10 (discussed in the following paragraph). The calculated RfD values converge at the same point: 0.1 µg/kg-day. USEPA also calculated geometric means from the four endpoints from the Faeroe Islands study; RfDs were 0.1 µg/kg-day based on these calculations. For the New Zealand study, both the median value and the results of the McCarthy Perceived Performance test yielded RfDs of 0.05 µg/kg-day,

and the McCarthy Motor Test yielded an RfD of 0.1 µg/kg-day. Based on the integrative analysis of all three studies, the RfD would be 0.1 µg/kg-day.

Due to the availability of human epidemiological studies, a relatively low composite uncertainty factor (combination of the modifying factor and uncertainty factor) of 10 was applied in the development of the methylmercury RfD. This choice was made to account for the following factors:

- Pharmacokinetic variability and uncertainty in estimating an ingested mercury dose from cord-blood mercury concentration: a factor of 3 was applied.
- Pharmacodynamic variability and uncertainty: a factor of 3 was applied.

Due to the availability of adequate data and peer-reviewed and government-reviewed studies, and the convergence of the RfD values calculated or derived from different studies and different endpoints, confidence in the RfD for methylmercury is considered high and there is little uncertainty associated with it.

7.5.1.3 Dioxins and Furans

Substantial uncertainties exist in the quantitative toxicity assessment for PCDD/PCDFs. These uncertainties result from both the numerical CSFs and TEFs used to calculate risks associated with these compounds, and the absence of numerical toxicity data to quantitatively estimate non-cancer hazards associated with this compound class.

It should be noted that USEPA is conducting an extensive dioxin reassessment; preliminary drafts of some of the documentation generated by that reassessment have been released for review (USEPA, 2000a). While this reassessment has not been completed or peer-reviewed, tentative or preliminary conclusions from it are noted where applicable.

Cancer Slope Factors for Dioxins and Furans

The primary issues of debate focus on the appropriate interpretation of the available data. Regulatory agencies and others in the US and elsewhere have calculated quantitative estimates of TCDD carcinogenicity that span more than three orders of magnitude. These differences arise as a result of alternative assumptions used when interpreting the underlying animal bioassay data, changes that have occurred in standard procedures for interpreting and extrapolating from such data to predict human health risks (e.g., scaling factors for relating animal data to human populations), and, most importantly, assumptions regarding the mechanism of action by which 2,3,7,8-TCDD exerts carcinogenic effects (threshold or non-threshold). The range of CSF values (carcinogenicity estimates) suggest there may be considerable uncertainty associated with the CSF used in this HHRA for PCDD/PCDFs.

As described in Chapter 5, Section 5.2.7, the cancer risks associated with PCDD/PCDFs were assessed by converting the 2,3,7,8-substituted congeners to a TEQ of 2,3,7,8-TCDD through the use of TEFs published by the WHO (van den Berg et al., 1998). A CSF for 2,3,7,8-TCDD was then applied to the resulting TEQs. USEPA does not list a CSF for 2,3,7,8-TCDD on IRIS; the value used in this HHRA is from HEAST (USEPA, 1997). In the past, a wide range of CSFs have been proposed for 2,3,7,8-TCDD based on a rat study by Kociba et al. (1978). For this HHRA, a CSF of 1.5×10^5 (mg/kg-day)⁻¹, which is at the upper end of this range, was used, which is the value listed by USEPA on HEAST.

The TEF system for converting other PCDD/PCDF congeners to a 2,3,7,8-TCDD equivalence (i.e., TEQ) is generally accepted both nationally and internationally by cancer researchers and regulatory agencies.

A reassessment of this study, along with data from three human studies, led USEPA in the draft dioxin reassessment to suggest “the use of 1×10^{-3} per pg/TEQ/kgBW/day as an estimator of upper-bound cancer risk for both background intakes and incremental intakes above background” (p. 105 of USEPA, 2000a). This converts to a CSF of 1.0×10^6 (mg/kg-day)⁻¹. If the final version of the dioxin reassessment confirms this value, the dioxin-related cancer risks presented in this HHRA would increase by a factor of about seven, resulting in an upper-bound RME cancer risk estimate of 3×10^{-3} for the adult fish ingestion pathway.

Non-Cancer Toxicity of Dioxins and Furans

Non-carcinogenic risks from exposure to PCDD/PCDFs were not estimated in this risk assessment because USEPA does not currently recommend an RfD for non-cancer hazards. (The preliminary reassessment being conducted by USEPA does not recommend establishing an RfD for dioxin and related compounds due to the relatively high background compared to effect levels [p. 108 of USEPA, 2000a]. The current estimated average dose to the US population, about 1 picogram per kilogram per day (pg/kg-day), is greater than the RfD/reference concentration (RfC) values that would be calculated given the data reviewed in the reassessment, and, therefore, toxicity-based RfD or RfC values would be uninformative for evaluation of non-cancer health risks [p. 122 of USEPA, 2000a].) Non-cancer risk estimates are presented to address this uncertainty.

Specifically, an HQ can be calculated by comparing the non-carcinogenic RME estimate of PCDD/PCDFs intake from fish (7×10^{-9} mg/kg-day) with the minimum risk level (MRL) published by ATSDR of 1 picogram TCDD TEQ/kg-day, or 1×10^{-9} mg/kg-day (ATSDR, 1999; however, USEPA’s dioxin reassessment indicates that there are some questions about the ATSDR MRL, due to concerns with the lower bounds of effects data for non-cancer endpoints [p. 109 of USEPA, 2000a]). The ATSDR MRL is analogous to a USEPA RfD in that it is the level of exposure below which the agency believes is without an appreciable risk for non-cancer health effects. Use of this MRL as an RfD would result in an HQ for PCDD/PCDFs of 7 in the RME adult fish consumption exposure scenario (based on the non-cancer intake of 7×10^{-9} mg/kg-day) and 1.5 in the CT fish consumption scenario (based on the non-cancer CT chronic daily intake [CDI] of 1.5×10^{-9} mg/kg-day), which would indicate that there is concern for adverse non-cancer effects related to exposure to PCDD/PCDFs in fish. These HQs would also correspondingly

increase the total HIs for the recreational angler through fish consumption (summarized in Chapter 6, Table 6-1).

More recently, the WHO has cited intakes in the range of 1 to 4 pg/kg-day ($1 \text{ to } 4 \times 10^{-9} \text{ mg/kg-day}$) as a “tolerable daily intake” (WHO, 2000, as cited in USEPA, 2000a). The RME estimate of intake from fish exceeds the high end of the WHO range; the CT intake estimate is within this range, although it exceeds the low end.

The draft dioxin reassessment indicates that USEPA is taking a margin-of-exposure (MOE) approach for non-cancer endpoints to inform risk management decisions. The MOE is the dimensionless ratio of a low-end effect level, such as an LOAEL, in the comparison species, to calculated or observed CDI. Higher MOE values are associated with the assumption of decreased risk, such as the decreased likelihood of adverse health effects occurring. Based on the USEPA estimated average (background) dose to the US population (about 1 pg/kg-day), USEPA indicates that MOEs range from less than 1 to as high as 15, depending on the study, species, and effect monitored (p. 122 of USEPA, 2000a) for establishing the LOAEL or effective dose. NYSDOH staff support the MOE approach and suggested using several studies of reproductive effects in rhesus monkeys to establish a lowest observed effect level (LOEL) of 0.13 ng/kg-day ($130 \times 10^{-9} \text{ mg/kg-day}$) (NYSDOH, 2002c; 2002, pers. comm., August 5). Using this LOAEL and the RME intake of $7 \times 10^{-9} \text{ mg/kg-day}$ would result in an MOE of about 20.

Although the available toxicity data show that exposure to dioxin-related compounds may result in a measurable increase in the non-cancer hazards, inclusion of the results discussed immediately above in the quantitative risk estimates would not change the overall conclusions in this HHRA regarding the site because the excess cancer risk estimate for PCDD/PCDFs for the fish consumption pathway was 4.5×10^{-4} and the total HI for fish consumption was already above 1.0.

7.5.2 Lack of Quantitative Toxicity Values for Detected Chemicals

7.5.2.1 Use of Surrogate Toxicity Data for Polycyclic Aromatic Hydrocarbons

As discussed in Chapter 5, Section 5.2.4, there are specific toxicity data available for only a few of the PAHs detected in soil/sediment samples used in the Onondaga Lake HHRA. Both cancer and non-cancer toxicity data were extrapolated to other PAH compounds for the quantitative cancer risk and non-cancer hazard calculations.

Cancer Slope Factors for PAHs

Benzo(a)pyrene is the only PAH compound for which a specific CSF has been developed. As noted in Chapter 5, Section 5.2.4.1, the cancer risk associated with other PAH compounds classified as B2 carcinogens has been estimated quantitatively using relative provisional carcinogenic potency factors published in 1993. As these factors were only considered to represent order-of-magnitude accuracy at the time they were first published, and they were provisional and have not been updated or finalized in nearly

ten years, there may be a fair amount of uncertainty associated with the cancer risks associated with these PAHs, although the direction of the uncertainty is unknown.

Dermal contact with PAHs was evaluated in this risk assessment using the oral CSF and RfD. There is a fair amount of uncertainty on this issue due to uncertainties related to derivation of toxicity values for dermal exposures for chemicals having site-of-contact effects, such as PAHs (USEPA, 1989b). The most recent USEPA dermal guidance recommends that PAHs be assessed quantitatively for systemic effects (non-cancer hazards) and qualitatively for carcinogenicity (RAGS Part E, Section 5.2.3). Therefore, there is a fairly high degree of uncertainty associated with the quantitative estimate of cancer risk associated with dermal contact with carcinogenic PAHs.

Reference Doses for Non-Cancer Effects of PAHs

As with carcinogenic PAHs, USEPA has non-cancer toxicity data for only a few PAH compounds. However, USEPA NCEA has reviewed studies for various PAHs and has provided recommended values for several non-carcinogenic PAHs for which there are no published values in IRIS or HEAST (see Chapter 5, Section 5.2.4.2 in this HHRA). While these values cannot be considered to have the same level of certainty as IRIS values, USEPA has specifically reviewed these values for use in the Onondaga Lake HHRA. Therefore, although there is some uncertainty associated with the non-cancer hazard calculations for these compounds, recent review has confirmed that these values are adequate for quantitative risk assessment.

The USEPA NCEA has only reviewed and approved the RfDs for the non-carcinogenic PAH COPCs. NYSDOH has noted that the carcinogenic PAHs may also have non-cancer health effects, and has suggested that the RfD for pyrene be utilized for assessing non-cancer hazards associated with PAH compounds that are assessed for cancer risks. The potential impact of this was assessed by reviewing the data for the southern basin sediments, a medium with high PAH concentrations. If these carcinogenic PAH compounds were also evaluated for non-cancer toxicity in the quantitative HHRA, the increase in RME hazards associated with these compounds would be insignificant (i.e., HIs for the individual PAHs would increase on the order of 10^{-4} to 10^{-5}). For example, applying the pyrene RfD to the southern basin sediment RME hazard for the young child would increase the calculated HI slightly to 0.54 from 0.535, and the older child RME hazard would increase to 0.256 from 0.253.

7.5.2.2 Surrogate Toxicity Data for Other Contaminants of Potential Concern

USEPA NCEA has indicated that the toxicity data for gamma-BHC (lindane) is appropriate for assessing risks associated with the Onondaga Lake COPC delta-BHC. Lindane is a relatively well studied compound due to its use as a commercial pesticide. As it is unlikely that the related compound delta-BHC is more toxic than lindane, and the calculated risks associated with this compound are not significant (i.e., do not contribute as much as ten percent of the risk or hazard by any pathway), any error or uncertainty introduced by this assumption does not materially affect the conclusions of this HHRA.

7.5.2.3 Contaminants of Potential Concern Without Quantitative Toxicity Data

Concentrations of two chemicals, lead and 2,4'-DDE, exceeded screening level concentrations and these chemicals were, therefore, identified as COPCs in one or more pathways. As sufficient quantitative toxicity data for lead and 2,4'-DDE were not available, these compounds were not included in the quantitative risk calculations. The potential impact of not having toxicity data for them is discussed below.

Lead

An analysis for lead was conducted in all site media. Lead in sediment and dredge spoils was screened against the USEPA residential soil screening criterion of 400 mg/kg, and lead in water was screened against the federal drinking water action level of 15 µg/L (40 CFR, Part 141). No screening values were available for lead in fish tissue. IRIS classifies lead as B2, probable human carcinogen, but no quantitative CSFs or RfDs were available in IRIS.

Lead was detected in 8 of 11 fish tissue samples at a maximum concentration of 0.29 mg/kg (290 µg/kg). Lead was not identified as a COPC in fish tissue, as there were no toxicity data for screening or risk quantitation. While the concentrations of lead detected are low on a qualitative basis (i.e., the maximum detected lead concentration was lower than the maximum detected concentration of almost every other metal in fish tissue; see Appendix B, RAGS Table 2.1), the potential impact of not being able to quantitate risks or hazards associated with lead cannot be determined.

Lead was identified as a COPC in southern basin sediments (lead concentrations were below the screening criteria in other sediment and dredge spoil samples), where it was detected in all 100 samples at a maximum concentration of 2,050 mg/kg. However, the medium-specific EPC (i.e., the 95 percent UCL on the mean) was 235 mg/kg (see Appendix B, RAGS Table 3.3), which is lower than the screening criterion of 400 mg/kg. Considering also that the potential for exposure to the southern basin sediments is lower than the exposure potential assumed by USEPA in deriving the residential screening criterion for lead, it is likely that lead does not contribute significantly to cancer risk or non-cancer hazard in the southern basin (or in other soil/sediment media) of Onondaga Lake.

Lead was detected in 12 of the 48 surface water samples from Onondaga Lake at a maximum concentration of 7.7 µg/L, about half the screening criterion of 15 µg/L (see Appendix B, RAGS Table 2.10). Lead was, therefore, screened out and was not considered a COPC in surface water. The lack of quantitative toxicity data for lead did not affect the calculation of risks for the surface water pathway.

2,4'-DDE

2,4'-DDE was identified as a COPC in fish tissue; samples were not analyzed for 2,4'-DDE in other media (surface water, sediments, or dredge spoil soils). 2,4'-DDE is chemically related to 4,4'-DDE and DDT, and was screened against the USEPA Region 3 fish ingestion criteria for "DDE" – the specific isomer (i.e., 2,4'- or 4,4'-) is not specified for the DDT-related compounds (which also include DDE and DDD).

Analysis for the 2,4' isomers of DDT-related compounds was conducted on 38 of the 134 fish tissue samples (about 28 percent of the total) for which pesticide analysis was conducted. 2,4'-DDE was detected in 7 of the 38 samples in which it was analyzed, at a maximum concentration of 24 µg/kg (Appendix B, RAGS Table 2.1), and the medium-specific EPC (95 percent UCL on the mean) was determined to be 4.1 µg/kg (Appendix B, RAGS Table 3.1); this EPC is substantially lower than the EPCs for the 4,4' isomers of the three DDT-related compounds (ranging from less than half to about one-eighth of the values). The non-cancer HQs associated with the 4,4'-DDT compounds were all less than 0.01 (Appendix B, RAGS Table 7.1); therefore, even assuming similar toxicity for 2,4'-DDE, it would not cause a significant increase in the non-cancer hazard for fish ingestion. For cancer risks, if the CSF for 4,4'-DDE were applied to 2,4'-DDE, the resultant RME risk would be about 2×10^{-8} , using the cancer intakes and CSFs as shown in Appendix B, RAGS Table 8.1. This would not result in a noticeable increase in the RME cancer risk for fish ingestion.

7.5.3 Lack of Analytical Data on the Specific Form of Contaminants of Potential Concern

The toxicity of some chemicals is affected by the form in which it exists, such as the structural or positional isomer (for organics) or the specific compound or valence state (for inorganics). For the Onondaga Lake HHRA, there are not significant uncertainties for most of the organic chemicals which contributed to risks or hazards. For the Onondaga Lake HHRA, the four COPCs for which there are form- and toxicity-related uncertainties are metals and PCBs, specifically:

- Mercury, especially in fish. The non-cancer toxicity varies depending on whether the mercury exists as organic (methylmercury) or inorganic mercury.
- Arsenic in fish. Inorganic arsenic is much more toxic than organic arsenic; however, much, if not most, of the arsenic in fish may be in the organic form.
- Chromium in all media. Hexavalent chromium (Cr^{+6}) is much more toxic than trivalent chromium (Cr^{+3}), although most of the chromium detected in environmental samples is usually in the trivalent form.
- PCBs in all media. Uncertainties exist with regard to analytical reporting and dioxin-like toxicity.

The specific assumptions used in the HHRA for each of these chemicals, and the potential impact of these assumptions on the HHRA, are discussed below.

7.5.3.1 Mercury and Methylmercury

Due to the different oral toxicity of inorganic mercury and methylmercury (methylmercury is considered to be three times as toxic as inorganic mercury, based on the oral RfDs published in IRIS), it was necessary to determine the fraction of mercury in each medium or pathway which was in the inorganic form. For some

media (i.e., fish tissue, lake sediments, and surface water) there are site-specific data for both forms of mercury and the assumptions made are based on the available data. For other media (i.e., wetlands sediments and dredge spoils) there are no site-specific data and therefore estimates of the fraction of the total mercury which is present as methylmercury are based on data from nearby sites, literature reviews, and professional judgment. The basis for the assumptions for each of the five media, as well as the potential impact on risk calculations of other assumptions (i.e., assumptions of either higher or lower percentages of methylmercury) are presented below.

A more detailed discussion of the literature and data used for the assumptions made for both the Onondaga Lake Baseline Ecological Risk Assessment (BERA) and HHRA regarding the methylation of mercury is presented in Chapter 6, Section 6.3.1.1 of the BERA (TAMS, 2002a).

Fish Tissue

For the fish samples, the assumption that all the mercury detected is in the form of methylmercury is likely to cause very little error. There are some site-specific data which support the assumption that the vast majority of the mercury detected in fish samples is methylmercury. Inorganic mercury is also toxic; however, it is less toxic by a factor of three and has an oral RfD corresponding to its lesser toxicity. (If, for example, 10 percent of the mercury in the fish were inorganic, then the assumption that 100 percent of the mercury is methylmercury would overestimate the hazard by about 7 percent. Fish ingestion hazards would still be substantially greater than 1.0.) The predominance of the mercury as organic mercury in fish tissue is attributed to the validated assumption for the Onondaga Lake site that most of the mercury in fish is methylmercury or organic mercury.

Lake Bottom Sediment

For lake sediments, the methylmercury concentration is based on data from the 17 northern and southern basin nearshore sediment samples analyzed for total and methylmercury, in which the detected concentrations of methylmercury were typically two to three orders-of-magnitude lower than the concentrations of total mercury, and averaged about 0.2 percent of the total mercury concentration. This same ratio (methylmercury about 0.2 percent of total mercury) was also observed in 14 samples from the greater depths in Onondaga Lake which were not used to calculate EPCs in the HHRA (as they were collected from depths of 3 m or more). These data are shown on Table 7-5. Although the data sets are consistent with each other, as well as within the range of concentrations expected based on the literature, the methylmercury concentrations, especially in the northern basin (from which only three samples used in the HHRA were collected), are based on a relatively small number of data points and represent a "snapshot," as all the samples were collected during a single sampling event (summer 2000).

For the lake sediments, the actual concentrations of both methylmercury and mercury (evaluated as inorganic mercury) were used quantitatively in the HHRA. For northern and southern basin sediments, hazards associated with both total (as inorganic) mercury and methylmercury were calculated separately, based on the data available. For the northern basin sediments, the EPC for methylmercury (2×10^{-3} mg/kg)

is about 0.1 percent of the EPC for total (assumed inorganic) mercury (1.9 mg/kg). If it were assumed that the methylmercury concentration is 1 percent, rather than about 0.1 percent, of the total mercury value, the methylmercury HQ would not change enough to affect the overall non-cancer hazard to receptors exposed to northern basin sediments. The methylmercury HQ for the most affected receptor – the young child – would increase from about 3×10^{-5} to 3×10^{-4} ; the difference would not be noticeable in the total HI of 0.221.

Wetlands Sediment

For the four wetlands evaluated in this HHRA, the total mercury concentration (which was the only analysis conducted) was assumed to be 1 percent methylmercury (and 99 percent inorganic mercury); this is the same assumption made for the BERA. Although no wetland samples were analyzed for methylmercury for the Onondaga Lake HHRA, 29 samples from the nearby LCP Bridge Street site were analyzed for mercury and methylmercury. These LCP Bridge Street site samples include some from regulated Wetland SYW-14 and other samples from near-surface wet or moist soils which are the best available data for estimating methylmercury concentrations in the Onondaga Lake wetlands. The fraction of methylmercury in these 29 samples ranged from 0.003 percent to 2.2 percent (this high value was from a sample described as “lined ditch” and may not be comparable to the wetland sediments), and averaged about 0.25 percent (see BERA Chapter 6, Table 6-3 [TAMS, 2002a]), supporting the assumption that the mercury in the wetlands is principally in the inorganic form.

As discussed in Chapter 6, Section 6.3.1.1, of the Onondaga Lake BERA (TAMS, 2002a), submitted concurrently with this HHRA, the BERA includes an assumption that 1 percent of the mercury in wetland sediments is organic (i.e., methylmercury). This assumption was applied to the wetlands sediments only, as there are specific mercury and methylmercury data for the lake sediments, and the methylation of inorganic to organic mercury was assumed to be negligible in other media such as the dredge spoils area soils. The RfD for methylmercury is three times higher than the RfD used for inorganic mercury; therefore, if 1 percent methylmercury was assumed for the wetlands sediments, mercury-related non-cancer toxicity would increase by a factor of 1.02 (2 percent).

A US Geological Survey (USGS) survey cited in the BERA (Krabbenhoft et al., 1999) indicates that methylation rates ranging from about 1 to 10 percent of total mercury have been detected in sediments of aquatic ecosystems (e.g., from streams and/or wetlands sediments in mixed agricultural/forest areas, abandoned mines, urban areas, etc.) in the US. Although the conditions favorable to a rate this high (e.g., low pH, anaerobic environment) are not considered to be present at Onondaga Lake (see BERA Chapter 6, Section 6.3.1.1), the potential for increased risk from a higher fraction of organic mercury in the wetland sediments was evaluated, as there are no methylmercury data from the four wetland areas included in this HHRA. The construction worker RME exposure to Wetland SYW-19 sediment has the highest HQ associated with exposure to mercury (0.046, the sum of the HQs for inorganic mercury and methylmercury); as shown in Appendix B, RAGS Table 7.23); therefore, this scenario would be most impacted by changes in the assumed form of mercury to which the receptor is exposed.

If the fraction of methylmercury in the Wetland SYW-19 sediments were assumed to be 10 percent instead of 1 percent, the HQ from mercury exposure would increase from 0.046 to about 0.055, and the total HI would increase only slightly, from 0.156 to 0.165. Therefore, even for the most exposed receptor, changes in the assumption of the form of mercury present do not significantly change overall hazard estimates. (If, on the other hand, the actual fraction of methylmercury is zero, then the HQ and HI would both show an insignificant decrease of only 0.001.) It is noted that Wetland SYW-19 is now part of the Wastedbed B/Harbor Brook site and additional sampling will be conducted in this wetland during the RI, including analysis of methylmercury in addition to other contaminants. Risks will be recalculated in the Wastedbed B/Harbor Brook HHRA based on these new data.

Dredge Spoils Area

For the dredge spoils area, the total mercury (which was the only analysis conducted) was assumed to be inorganic mercury, which is the same assumption made for the BERA. Although there are no site-specific data from a matrix similar to the dredge spoils, the dredge spoil samples were collected above the water table in an oxic environment that is not ordinarily favorable to the methylation process. There are insufficient data in the literature to estimate the percent of methylmercury that may be formed under these conditions, but methylation rates are thought to be substantially lower than in wetlands.

In order to assess the potential impact of methylmercury on the calculated hazards from dredge spoils, the potential hazard to the most impacted receptor (construction worker RME to surface and subsurface dredge spoils) was evaluated using the high-end assumption of 10 percent methylmercury. If 10 percent of the total mercury is assumed to be methylmercury (the remaining 90 percent is inorganic), then the total mercury-related hazard (the sum of the HQs from inorganic mercury and methylmercury) would increase from 0.052 to 0.062, and the total RME HI for the construction worker would increase from 0.126 to 0.136. Therefore, the RME hazard would not change significantly, even under high-end or worst-case assumptions of methylmercury concentration.

Surface Water

There were more extensive data on mercury speciation in the surface water data set used in this HHRA. Of the 75 surface water samples analyzed for total mercury, 32 of the samples were also analyzed for methylmercury, dimethylmercury, and ionic mercury. There were sufficient data to enable the methylmercury in surface water to be evaluated separately from the "total" (interpreted as inorganic) mercury in surface water. Dimethylmercury was not detected in any of the 32 samples in which it was analyzed. Ionic mercury was detected in all 32 samples, although at lower concentrations (ranging from 0.15 to 3.3 ng/L) than either total mercury or methylmercury. Ionic mercury data, where available, were screened separately as inorganic mercury; due to the low concentrations of ionic mercury detected, ionic mercury was screened out (excluded from the quantitative HHRA). Due to the low concentrations detected, the treatment of the ionic mercury has little effect on the results of this HHRA.

7.5.3.2 Arsenic

In the HHRA, it was assumed that 10 percent of the arsenic detected in fish tissue was inorganic arsenic, which is the more toxic form. It is generally understood that most of the arsenic in fish is in the relatively non-toxic organic form called arsenobetain (Oregon Health Department and Washington [State] Department of Health [OHD/WDOH], 1996). Inorganic arsenic, which is considerably more toxic, makes up a small amount of the total arsenic in fish and is most relevant for assessing health impacts from consumption of contaminated fish. However, there are no adequate data or other bases on which to quantify the fraction of arsenic in the inorganic form in Onondaga Lake fish fillets.

In an attempt to quantify or estimate the fraction of inorganic arsenic in fish, staff from the OHD and WDOH were contacted, based on their involvement in the risk assessment for the Columbia River in Oregon and Washington, where arsenic was a contaminant of concern. The consensus was that there is not a good means of making an estimate, as the fraction of arsenic may vary significantly due to a variety of factors, including the fish species, the water body, and characteristics of the individual fish analyzed (K. Kauffman, pers. comm., 2002; J. White, pers. comm., 2002).

Studies show an organic arsenic range of 1 to 25 or 30 percent of the total arsenic (K. Kauffman, pers. comm., 2002), although default assumptions of 1 or 10 percent are typically used (J. White, pers. comm., 2002). Subsequent data obtained by TAMS from USEPA Region 10 (R. Lorenzana, pers. comm., 2002; Tetra Tech, 1996; EVS, 2000) confirmed this range. These data are tabulated on Tables 7-6A and 7-6B and show a range from none detected to about 26 percent inorganic arsenic in the 42 freshwater fish samples in which arsenic was detected, with an arithmetic mean of about 6.4 percent, with an upper-bound 95th percentile of 10.7 percent (calculated using the non-parametric Chebyshev test in the USEPA ProUCL software). These data are useful to support the general observation that only a relatively small fraction of the total arsenic in fish is in the toxic inorganic form.

Although it is difficult to use the West Coast data to generate a quantitative estimate for use in the Onondaga Lake HHRA due to differences in location and water body (river versus lake) and species (the West Coast data are, for the most part from species not present in Onondaga Lake – e.g., salmon, sturgeon), they support the use of the default assumption of 10 percent of the total arsenic detected in fish samples in the inorganic form. While not enough data were obtained to perform a rigorous analysis, inspection of the Columbia River data suggests that the fraction of arsenic existing in the inorganic form may also be species-dependent.

The effect of this uncertainty is not great in terms of affecting the overall risk or hazard estimates for the fish consumption pathway. With the assumption of 10 percent inorganic arsenic, arsenic contributes slightly less than 2 percent of the adult RME cancer risk (1.8×10^{-5} , out of a total ingestion pathway risk of 7.8×10^{-4}). For a worst-case assumption of 100 percent inorganic arsenic, the total RME cancer risk would increase to about 9.4×10^{-4} . On the other hand, the RME cancer risk from arsenic would be reduced to about 7.6×10^{-4} , based on inorganic arsenic percentages of 1 percent. The contribution of arsenic to the non-cancer hazard associated with fish ingestion is lower, with arsenic contributing about 0.5 percent of the adult RME

total non-cancer ingestion hazard (contributing an HQ of about 0.1 to a total adult RME HI of about 18). The total non-cancer adult RME hazard associated with fish ingestion would increase slightly (from 18.2 to 19) if the worst-case assumption of 100 percent inorganic arsenic were made, and decrease slightly (from 18.2 to 18.1) if only 1 percent of the total arsenic were assumed to be inorganic.

7.5.3.3 Chromium

Chromium typically exists in two valence states in the environment: Cr(III) (Cr^{+3} , or trivalent chromium), and Cr(VI) (Cr^{+6} , or hexavalent chromium). Normally the bulk of the chromium is in the less-toxic trivalent form. For example, the USEPA Region 9 soil-screening concentrations are based on the assumption of a six-to-one ratio of Cr(III) to Cr(VI).

As the available data for chromium were reported as "total chromium" (no speciation between Cr[III] and Cr[VI] was performed), this HHRA used the conservative assumption that all chromium detected was in the form of the more-toxic hexavalent form. While chromium did contribute a significant fraction of the non-cancer hazard in Wetlands SYW-6 and SYW-12 and in surface water, the total HI for all receptors in each of these media was very low, as summarized on Table 6-5. Therefore, although the hazard associated with chromium may be overestimated, it does not have a material impact on the conclusions of this HHRA.

7.5.3.4 Polychlorinated Biphenyls

As discussed previously, data for PCBs were reported as Aroclors, with the particular suite of Aroclors reported varying depending on the medium, the sampling organization and analytical laboratory, and when the samples were analyzed. These factors do not contribute materially to the calculation of PCB EPCs or the assignment of appropriate toxicity factors (RfDs or CSFs).

One area of uncertainty regarding PCBs is their potential for dioxin-like toxicity. As with many of the 2,3,7,8-substituted PCDD/PCDF compounds, certain PCB congeners have been found to have, or are assumed to have, dioxin-like toxicity. TEFs have been developed to estimate the dioxin-like toxicity. However, as there were no analyses of PCB congeners conducted on Onondaga Lake samples, no estimate of the dioxin-like toxicity of PCBs is presented in this HHRA.

7. UNCERTAINTY ASSESSMENT

Because risk characterization serves as a bridge between risk assessment and risk management, it is important that major assumptions, scientific judgments, and estimates of uncertainties be described in the risk assessment. Superfund risk assessment methods are designed to be protective of human health and to address the uncertainties associated with each step in the risk assessment process.

This chapter addresses aspects of this HHRA that are likely to overestimate or underestimate site risks. The risk discussion below is organized according to the three principal areas of uncertainty described in USEPA's (1989) risk assessment guidance (RAGS Part A, Section 8.4):

- Selection of chemical substances included in the characterization. This includes both the initial selection of chemicals for which samples were analyzed, as well as the screening performed to refine the list of substances carried through the quantitative risk assessment.
- Exposure assessment. Uncertainties include both those associated with the data and chemical concentrations (e.g., exposure point concentrations [EPCs]), as well as uncertainties associated with the individual exposures (e.g., exposure frequency, consumption rate).
- Toxicity values. This includes uncertainties associated with the reference doses (RfDs) and cancer slope factors (CSFs) used in the quantitative HHRA, extrapolations from one route to another (e.g., oral to dermal), and the effect of substances which were not included in the quantitative HHRA due to lack of quantitative toxicity data.

7.1 Uncertainties Associated with Selection of Substances

The two areas of uncertainty associated with the selection of substances are the selection of analytical parameters for which samples from each medium (pathway) were analyzed, and the screening of contaminants to reduce the number of chemicals for the quantitative risk characterization.

7.1.1 Selection of Analytical Parameters

The selection of analytical parameters for samples analyzed for the HHRA was based largely on the site history and previous investigations, and focused on data for parameters with a reasonable potential to be present (e.g., mercury/methylmercury, chlorinated benzenes). However, since the full history of each of the exposure areas is not known, many samples were analyzed for a much broader range of contaminants (full organic Target Compound List [TCL] and inorganic Target Analyte List [TAL] contaminants) in order to determine if other analytes are present at concentrations of potential concern.

7.1.1.1 Fish Fillets

Analysis of fish fillets focused on the contaminants that have historically been of concern; specifically, mercury, pesticides, chlorinated benzenes, and polychlorinated biphenyls (PCBs). In recent years, analysis for polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs) has also been conducted on many of the samples. However, only a few of the fillet samples have been analyzed for the full suite of semivolatile organic compounds (SVOCs) (including polycyclic aromatic hydrocarbons [PAHs]) and TAL metals. Only four adult composite samples have been analyzed for volatile organic compounds (VOCs), as these compounds do not tend to bioaccumulate and the presence of VOCs in fish at high-enough concentrations to be of concern is not expected. The limited data support this assumption, as no VOCs were detected at concentrations exceeding the screening criteria.

With the exception of hexachlorobenzene, only four adult composite samples were analyzed for SVOCs (including PAHs). Since less than ten samples were analyzed for these substances, statistical evaluation of the data set (i.e., calculation of the 95 percent upper confidence limit [UCL] on the mean) was not possible. Therefore, for these compounds, the maximum detected concentration was used as the EPC (consistent with risk assessment guidance [USEPA, 1989]). However, bis(2-ethylhexyl)phthalate is the only SVOC affected, as it was the only SVOC compound (other than hexachlorobenzene) for which a detected concentration exceeded the screening criteria, and was, therefore, the only additional SVOC carried through the assessment as a contaminant of potential concern (COPC) for which the EPC was calculated.

Pesticide data are reasonably comprehensive, as there are at least 38 data points for most of the pesticides and over 100 data points for some (e.g., chlordane; DDT and related compounds; mirex/photomirex).

Despite the fact that a large amount of earlier (i.e., 1992) PCB data were considered to be unusable for this HHRA (see Appendix A), there are over 100 PCB analyses used in this HHRA, covering both low molecular weight (less chlorinated) Aroclors (either Aroclor 1016 or Aroclor 1242) and high molecular weight (highly chlorinated) Aroclors (reported as Aroclor 1254/1260). Although there are only seven fillets for which the full suite of individual Aroclors (including Aroclor 1268) were analyzed, the usable analyses conducted provide a sufficient data set for characterization of PCB concentrations in fillets for the HHRA.

There were 30 fish fillets analyzed for PCDD/PCDFs. Although some of the locations from which these samples were collected were biased toward suspected hot spots or source areas, there were sufficient data that a meaningful 95 percent UCL on the mean could be calculated. It was not necessary to use the maximum detected concentration as the EPC.

With the exception of mercury and methylmercury, analysis for metals was conducted on only seven fillets and four adult composite samples. The limited amount of data for TAL metals does make characterization of the EPCs of these constituents difficult. However, there were sufficient data to perform the statistical tests (determination of data distribution type and calculation of the 95 percent UCL on the mean). Meaningful 95 percent UCLs on the mean were calculated for all the TAL metals that exceeded screening criteria; in no case was it necessary to use the maximum detected concentration as the EPC.

7.1.1.2 Lake Sediments – Northern Basin

Northern basin nearshore shallow surface sediments (all intervals in the 0 to 30 cm range at stations in less than 6.5 feet [ft] [2 meters {m}] of water) were analyzed for full TAL inorganics, VOCs, SVOCs (including PAHs), pesticides, and PCBs. There were typically between 22 and 56 data points for each of these fractions (in isolated cases, there were less for an individual analyte within a fraction, due to slight differences in the target analytes reported by a laboratory or analytical methods). Five northern basin samples used in this HHRA were also analyzed for PCDD/PCDFs, and three samples for methylmercury (in addition to the 56 samples analyzed for total mercury). For these last two parameters, the maximum detected concentration was used as the EPC, as there were an insufficient number of data points for meaningful statistical analysis. The northern basin data set is comprehensive and complete, and little, if any, uncertainty is likely due to the type or quantity of analyses performed.

7.1.1.3 Lake Sediments – Southern Basin

Southern basin nearshore shallow surface sediments (all intervals in the 0 to 30 cm range at stations in less than 2 meters [m] of water) were analyzed for full TAL inorganics, VOCs, SVOCs (including PAHs), pesticides, and PCBs. There were typically between about 80 and 100 data points for each of these fractions (in isolated cases, there were less for an individual analyte within a fraction due to slight differences in TCLs or analytical methods). Eighteen southern basin nearshore samples used in this HHRA were also analyzed for PCDD/PCDFs, and 14 samples from seven locations were analyzed for methylmercury (in addition to the 114 samples analyzed for total mercury). The southern basin data set is comprehensive and complete (with the possible exception of methylmercury data), and little, if any, uncertainty is likely due to the type or quantity of analyses performed. Sufficient data are available for meaningful statistical analyses of all parameters, except methylmercury.

7.1.1.4 Wetland Sediments (SYW-6, 10, 12, and 19)

The wetland samples were analyzed for the full suite of TCL/TAL compounds. PCDD/PCDFs were analyzed in all wetlands except SYW-12, as per the Phase 2A Work Plan (Exponent, 2000). However, since each wetland was evaluated as a separate exposure area and there were only eight samples (and four data points, as the sample data were combined from each pair of samples collected from two depths at each of four locations) from Wetlands SYW-10, 12, and 19, no statistical analysis was performed on the wetlands data (a minimum of ten samples was used as the threshold for conducting statistical analysis of data sets for this HHRA); therefore, the detected maximum value of each COPC was used as the EPC. This may result in an overestimate of the EPC concentration; on the other hand, due to the limited number of samples, it is also possible that areas of higher concentration may have been missed in the sampling program.

Data from additional samples collected from Wetland SYW-6 in May 2002 by NYSDEC/TAMS were also used in this HHRA. This data set consisted of samples, which were analyzed for metals and SVOCs only, from two depths (0 to 15 cm and 15 to 30 cm) at each of five locations. As with the data from the

wetlands sampling performed in 2000, the two samples were composited mathematically to generate a single length-weighted average for each location. This increased the total number of data points for Wetland SYW-6 to nine (five from 2002 and four from 2000), which was still too few to perform meaningful statistical analyses; therefore, the maximum value of these nine length-weighted averages was used as the EPC for each COPC in Wetland SYW-6.

However, inspection of the data indicated that there was one location, and in particular one result (the 15 to 30 cm interval sample at 2002 Station W6-3), in which concentrations of PAHs were significantly higher than in other samples collected from this wetland. To assess the possibility that use of this one high value might overestimate the EPC, statistical analyses of the SYW-6 data set were performed on each of the 18 discrete samples (i.e., using each of the two data points at a given location as separate samples, rather than combining them into a single result at each location). The results of this alternative approach to evaluating the SYW-6 data are presented in Table 7-1A. In general, assessing each of the 18 samples as discrete data points results in EPCs which for most, though not all, COPCs are lower than the corresponding length-weighted maxima. Cancer risks and non-cancer hazards, as presented in Table 7-1B, are on the same order-of-magnitude, though lower, using the discrete sample approach. There is no change as a result of the discrete sample approach in which pathways exceed target risk levels, with the exception of the CT cancer risk for the construction worker, which is 1.5×10^{-6} using the maximum of the nine length-weighted average values, but is less than 10^{-6} (7.4×10^{-7}) when using the EPCs calculated from the UCL of the 18 discrete samples.

The data from the Wetland SYW-6 sample with the high PAH concentrations (2002 sample from Station W6-3) were compared to the data from the nearest sample collected in the Phase 2A investigation (Station S375 in 2000). The data from these two samples, which are from locations about 90 ft (27 m) apart, are compared in Table 7-2. As can be seen from the table, the metals data are comparable (most of the data agree within about 30 percent), but the Station W6-3 PAH data are much higher, typically by factors of about 50 to 100. This discrepancy between the PAH concentrations in relatively close samples suggests either that the high value is an anomaly and may be an outlier or that the distribution of contaminants in Wetland SYW-6 is very heterogenous and there may be other areas of high or even higher concentration that have not been sampled.

Wetland sediment samples were not analyzed for methylmercury. For calculation of EPCs and hazards, it was assumed that 1 percent of the total mercury was in the form of methylmercury (the remaining 99 percent was assumed to be inorganic mercury). The implications of this assumption are discussed in Section 7.5.3.1, below.

7.1.1.5 Dredge Spoils Area Soils

The dredge spoils data did not have a depth distribution corresponding to depth intervals preferred for use in HHRA's (nominally, from 0 to 6 inches [0 to 15 cm] for exposure to receptors other than construction workers). As a result, the depth interval used for assessing human recreational exposure to surface soils included data from samples at greater depths, up to 3.5 ft (107 cm). (Note that for the construction worker

receptor, data from all depths down to approximately 12 ft [3.5 m] were used.) The extent of the effect on the data and the potential direction of bias is unknown. However, most of these samples were collected from the relatively uniform cover material above the more-contaminated spoils; thus, no significant bias is expected.

For metals, SVOCs, and PCBs, only eight samples were collected from the depth interval used for the surface soil assessment; thus, the maximum detected value was used as the EPC for this pathway.

A total of about 40 dredge spoil area samples were analyzed (including the eight near-surface samples). None of the samples were analyzed for VOCs, and pesticide analysis was limited to a reduced suite examined in one deeper sample (Station S438). PCDD/PCDF analysis was conducted on about half the samples (20 in all, including four of the eight near-surface samples).

Dredge spoil samples were not analyzed for methylmercury. For calculation of EPCs and hazards, it was assumed that all (100 percent) of the total mercury was in the form of inorganic mercury. The implications of this assumption are discussed in Section 7.5.3.1, below.

7.1.1.6 Surface Water

The extent of available Onondaga Lake surface water data varies both among and within each analytical fraction.

Metals analysis was performed on 33 to 75 samples for 12 of the 23 TAL metals and methylmercury (not all samples were analyzed for the same suite of metals), and 32 samples were analyzed for dimethylmercury, ionic mercury, and elemental mercury. Although no analyses were conducted on any of the surface water samples (from the 0 to 3 m depth interval) for the other TAL metals, including arsenic, antimony, selenium, and thallium, they were not detected, or were detected at low concentrations, in samples collected from greater depths in the lake (6 to 12 m). Although the lake water is reasonably well characterized for the metals that were analyzed in near-surface (0 to 3 m) samples, it is possible that the calculated risks are underestimated for metals excluded based on data from samples collected from greater depths.

Full VOC analysis was performed on 11 of the 48 surface water samples, with a much greater number of analyses (i.e., 48) performed for the volatile contaminants considered most likely to be present – i.e., benzene, toluene, ethylbenzene, xylenes, and chlorobenzene.

SVOC analysis included 37 to 48 samples analyzed for three dichlorobenzene isomers and three trichlorobenzene isomers, plus five samples analyzed for hexachlorobenzene (not all samples were analyzed for the same suite of parameters, and some of the dichlorobenzene data came from the VOC analysis of some samples). No analyses of samples from the 0 to 3 m depth were performed for other SVOC compounds (including PAHs). However, four samples from the 6 to 12 m interval were analyzed, and the only SVOC COPC detected was bis(2-ethylhexyl)phthalate, and only at low concentrations (i.e., at a

maximum concentration of 10 µg/L, which is less than the Region 3 tap water screening criterion.). Therefore, the presence of other SVOC compounds in surface water is considered unlikely, and the quantitation of risks associated with SVOCs is affected minimally, if at all.

None of the surface water samples from the 0 to 3 m interval were analyzed for pesticides, PCBs, or PCDD/PCDFs; therefore, the extent of risk, if any, posed by the potential presence of these compounds was assessed using data from deeper samples (6 to 12 m). No PCBs or pesticides were detected in these deeper samples and are, therefore, not expected to be present in surface water (0 to 3 m) at significant concentrations. Onondaga Lake water samples have not been analyzed for PCDD/PCDFs.

7.1.2 Contaminant Screening

The contaminant screening, which is presented in Chapter 3, poses potential uncertainty only if chemicals that may present risk are inappropriately screened out of the quantitative risk assessment, or if chemicals that are not site-specific contaminants (e.g., naturally occurring substances whose detection in site samples is not a result of discharge or release of contaminants) are included in the quantitative HHRA. Each of these possibilities is discussed below.

7.1.2.1 Uncertainty Associated with Screening Contaminants

The screening was conservative in that the procedures used for exclusion of a compound from the quantitative assessment were rigorous. A compound was included as a COPC for the quantitative HHRA if there was any indication it could have an impact on risk. For all chemicals detected, the concentration used for screening was the maximum concentration detected, although in the quantitative risk characterization the 95 percent UCL on the mean was used as the EPC, where allowable. Non-carcinogenic chemicals were screened against screening criteria representing a hazard index (HI) of 0.1 (one-tenth of the concentration believed to be associated with the potential for toxic effects).

Inorganics (metals) designated as nutrients (calcium, potassium, sodium, and magnesium) were screened out (flagged “NUT” for “nutrient” on RAGS Tables 2.1 through 2.10) and were not assessed in the quantitative HHRA.

Screening criteria used were developed by USEPA Regions 3 and 9 using the same toxicity data as used in this HHRA (i.e., the Integrated Risk Information System [IRIS], the Health Effects Assessment Summary Tables [HEAST], and the National Center for Environmental Assessment [NCEA]). The USEPA Region 9 soil screening criteria include an estimate of dermal exposure so that those criteria address both of the complete pathways identified for the Onondaga Lake site (ingestion and dermal contact). Because of this, the USEPA Region 9 criteria are generally, though not always, more conservative than the USEPA Region 3 criteria. The USEPA Region 3 and Region 9 screening tables, along with text prepared by each region explaining the applicability and derivation of their screening criteria, are provided in Appendix C. To reduce the chance of screening out a contaminant that may contribute to risk, the more conservative of the USEPA Region 3 or Region 9 criteria were applied for each COPC.

The USEPA Region 3 fish ingestion screening criteria are based on a higher consumption rate than used in this HHRA (54 grams per day [g/day], rather than 25 g/day). Therefore, it is unlikely that any contaminants that may pose significant risk through the fish ingestion pathway were screened out of the assessment.

Surface water concentrations were screened against the USEPA Region 3 and Region 9 tap water criteria, which are based on ingestion of water as drinking water at a rate of 2 liters per day. As the ingestion rates assumed for this quantitative HHRA are based on the much lower ingestion rates for incidental ingestion through recreational or construction activities, the screening for ingestion of contaminants in surface water is unlikely to have eliminated significant contaminants. However, the exposure scenarios envisioned for the complete exposure pathways also include dermal contact with water; this pathway is not addressed in the USEPA Region 3 or Region 9 tap water screening criteria. To reduce the chance of screening out chemicals which might be significant contributors to dermal risk, a second screening was also conducted using the dermal criteria in the draft USEPA RAGS Part E (dermal guidance) document (USEPA, 2001a). Based on this further screening, three additional compounds (1,2-dichlorobenzene, 1,2,4-trichlorobenzene, and chromium) were added as COPCs although their concentrations were below the Region 3 and Region 9 screening criteria. Thus, it is unlikely that compounds that were screened out would add to potential risk.

7.1.2.2 Uncertainty Associated with Naturally Occurring Chemicals in Soil/Sediment

Inclusion of chemicals identified in the HHRA as COPCs that are naturally occurring may overestimate the site-specific component of the risk. This is generally not an issue for organic chemicals, as those that are assessed quantitatively in this HHRA are anthropogenic and thus are not naturally occurring, although the attribution of some relatively ubiquitous contaminants such as PAHs to an individual site may be problematic (see the Remedial Investigation [RI] report for further discussion [TAMS, 2002b]).

Many metals and inorganic substances are naturally occurring constituents of soil. In accordance with recent USEPA guidance (M. Sivak, pers. comm., 2002), no inorganics were eliminated from the HHRA based on their concentrations being similar to those in background samples. Metals that were detected in sediments at concentrations similar to background concentrations (defined as the maximum concentration detected being less than two times the average of the Otisco Lake samples) were flagged as "BKG" on the screening tables (Appendix B, RAGS Tables 2.2 through 2.6). Metals exceeding risk-based screening criteria, but detected at concentrations below the background screening concentration, include the following:

- Manganese (all evaluated media).
- Iron (northern basin nearshore sediments; Wetlands SYW-10 and 19).
- Arsenic (northern basin nearshore sediments; Wetland SYW-6).
- Thallium (northern basin nearshore sediments).
- Aluminum (Wetland SYW-10).

As shown in Chapter 6, Tables 6-4 and 6-5, manganese, iron, and arsenic contributed a significant portion (at least 10 percent) of the cancer risk or non-cancer hazard in some of the media in which they were detected at concentrations below the background concentrations. Although this indicates the possibility that the background levels of metals may result in an overestimate of their effect, it is not significant for non-cancer hazards, as the non-cancer hazards for media were well below 1.0. However, arsenic was a major contributor to cancer risks in the northern basin nearshore sediments and, to a lesser extent, Wetland SYW-6. Calculated RME risks to all recreational receptors exceeded 10^{-6} for the northern basin sediments, and calculated RME and CT cancer risks for SYW-6 exceeded 10^{-6} for construction workers and adult and child recreational receptors. For the northern basin sediments, elimination of arsenic from the cancer risk calculation would reduce the adult recreator RME risk to below 10^{-6} (from 1.3×10^{-6} to 6.5×10^{-7}); cancer risks to young and older children would still exceed 10^{-6} but would decrease from slightly less than 4×10^{-6} to slightly over 2×10^{-6} . If the contribution of arsenic to the cancer risk were removed from the calculation for SYW-6, the RME and CT cancer risks for all the receptors would still be greater than 10^{-6} , although the risk would be slightly lower (for example, the adult recreational RME would decrease from 6.5×10^{-5} to 6.4×10^{-5}).

7.2 Uncertainties Associated with Exposure Point Concentrations

Uncertainties associated with the EPCs used in risk calculations may be related to the quality and quantity of the available data, and to the manner in which the data are then processed to generate the EPCs used in the quantitative HHRA.

7.2.1 Uncertainties Related to Calculation Procedure for Exposure Point Concentrations

As discussed in Chapter 4, Section 4.7, the EPCs for the media evaluated in this HHRA were determined, based on guidance from NYSDEC and USEPA Region 2, as the lower of:

- The 95 percent UCL on the arithmetic mean of the site data (for data sets with ten samples or more).
- The maximum detected concentration (for all data sets with less than ten data points, and for larger data sets where the 95 percent UCL is greater than the maximum detected concentration).

Data sets were identified as best fitting either a normal or lognormal distribution (the higher W-statistic for data sets with $n < 50$, and the Y-statistic for data sets closer to zero for $n > 50$, as described in Gilbert, 1987). For data best fitting a normal distribution, the 95 percent UCL on the mean was calculated using the Student's t-statistic. For data identified as best fitting a lognormal distribution, the 95 percent UCL on the mean was calculated using Land's H-statistic. The equations for the UCL calculations are presented in Chapter 4, Section 4.7. The use of the 95 percent UCL on the mean for calculating the EPC, which is designed to be protective (i.e., there is less than a 5 percent chance that the true "population" average exceeds the calculated 95 percent UCL on the mean), may overestimate the actual concentrations to which

individuals would be exposed at Onondaga Lake. However, for robust data sets, the 95 percent UCL on the mean (true population mean) and the arithmetic average converge; this is the case for the Onondaga Lake fish fillet mercury data used in this HHRA, where the 95 percent UCL on the mean used as the EPC (1.08 mg/kg) is only very slightly higher than the arithmetic average (1.05 mg/kg).

Use of the maximum detected concentration when it is lower than the 95 percent UCL on the mean, and use of a maximum value for all data sets with fewer than ten samples (as was done here), are also potential sources of overestimation. Conversely, where there are only a few data points from which the EPC is derived, there is no certainty that the available data are representative of the site or that there may be higher – possibly much higher – COPC concentrations in the area. (This is especially true of Wetland SYW-6, as was previously discussed in Section 7.1.1.4.) Therefore, it is also possible that the use of the highest value of a small data set could underestimate EPCs if samples from more-contaminated parts of the area were not collected.

7.2.2 Uncertainties Related to Assigned Data Distribution Types and Resultant Upper Confidence Limit-Based Exposure Point Concentrations

As discussed in Chapter 4, Section 4.7.3, all data sets (for $n \geq 10$) were assigned to either a normal or lognormal distribution using a “best-fit” approach. However, not all data sets meet the criteria to be classified as either normal or lognormal distributions. To address this issue, USEPA has recently developed software (ProUCL 2.1) to calculate UCLs for these non-parametric data sets (USEPA, 2002c).

As much of the data processing for the calculations of the EPCs was substantially underway at the time the USEPA ProUCL 2.1 software became available, several subsets of the HHRA data were reevaluated using the ProUCL software to see how much of a difference, if any, use of the ProUCL software would make on the EPCs and resultant risk and hazard calculations. The discussion below is a summary of the more detailed evaluation presented in Appendix D.

The data sets selected for this evaluation were those for which the risk associated with the analyte exceeded the target risk level (for this purpose, a cancer risk of 10^{-6} or hazard quotient [HQ] greater than 1.0). The previously assigned data distribution was confirmed in the majority of cases (i.e., 11 of 18). In cases where the previously assigned distribution was not confirmed (i.e., ProUCL indicated that the data were neither normal nor lognormal), the EPCs were calculated using the ProUCL methods and compared to the EPCs utilized for the HHRA. The differences between the UCLs calculated by the two methods were generally not large, although for three of the 18 data sets evaluated the ProUCL-calculated value differed from the default value by more than 50 percent. However, even with this difference, the overall effect on calculated risks was small (see Appendix D, Table D-2), as these three chemicals were not significant contributors to the cumulative risk. As the effect on the EPC calculations and subsequent risk characterization was minimal, the EPCs presented in this HHRA are those using the original assignment of normal or lognormal distribution to each data set. The uncertainty associated with use of the best-fit approach to the data distribution and EPC calculations is considered to be small for the Onondaga Lake HHRA.

7.2.3 Effect of Cooking Loss on PCB and PCDD/PCDF Concentrations in Fillets

The RME calculations presented in the HHRA are based on a fish consumption rate of 25 g/day (this consumption rate is discussed below in Section 7.3), with no reduced fractional intake (FI) relating to the amount of fish consumed from the lake (i.e., FI of 1.0) and no loss of PCBs or PCDD/PCDFs due to cooking or trimming. A 33 percent reduction was assumed in the CT scenario, the rationale for which was discussed in Chapter 4, Section 4.3.1.3. While the literature shows that there are a wide range of estimates in the amount of PCB (and PCDD/PCDF) loss in cooking, the assumption of no loss for the RME scenario is certainly a reasonable one. The 33 percent loss assumed for the CT scenario is also reasonable; while there are some studies showing greater losses, others show less. The “health-protective values” (HPVs) established by the Great Lakes Sport Fish Advisory Task Force (GLSFATF) for Great Lakes fish assume a 50 percent loss of organic contaminants through preparation and cooking; however, the Great Lakes advisories are also based on a higher assumed fish consumption rate of 140 g/day (225 8-ounce meals per year) for “unrestricted” consumption (GLSFATF, 1993). In addition, many of the target species in the Great Lakes, such as chinook salmon (*Oncorhynchus tshawytscha*) and lake trout (*Salvelinus namaycush*), are not present in Onondaga Lake, and the percent reductions in organic contaminants vary by species (Table C-1 of USEPA, 2000b).

Recent USEPA guidance (USEPA, 2000b) presents some data specific to species evaluated for Onondaga Lake. One older study (Skea et al., 1979, as cited in Appendix C of USEPA, 2000b) showed PCB reductions in smallmouth bass (*Micropterus salmoides*) ranging from 16 to 80 percent. However, most of the more recent data (e.g., a number of studies by Zabik in the mid-1990s) show PCB losses in walleye (*Stizostedion vitreum*) ranging from about 15 to 30 percent for Great Lakes fish. Homolog-specific PCB data for five (unspecified) species showed reductions ranging from 15 to 40 percent, with most of the reductions in the 30 to 35 percent range (Zabik and Zabik, 1996; cited in Table C-1 of USEPA, 2000b). It was also noted that “. . . smoking lake trout reduced pesticides and total PCBs significantly more than other cooking methods, but this cooking method resulted in the formation of PAHs” (Zabik, 1994, as cited in USEPA, 2000b).

7.3 Uncertainties Related to Fish Consumption Rates

In this section, uncertainties related to assumptions made for the fish ingestion rates used in the exposure assessment are discussed. As ingestion of fish was identified as the most significant risk pathway for exposure to Onondaga Lake COPCs, and because assumed fish ingestion rates are sometimes a controversial issue in risk assessments, the issues associated with the approach taken in this HHRA are discussed in depth below.

In the quantitative risk characterization presented in Chapter 6 of this HHRA, the RME calculations were conducted using a fish consumption rate of 25 g/day for adults, an FI of 1.0, and the assumption that COPC concentrations are not reduced by cooking. The recreationally caught fish ingestion rates were estimated as two-thirds of the adult rate for older children (6 to less than 18 years old) and one-third of the

adult rate for young children (up to age six). In this section, these assumptions are evaluated and their potential effect on the calculated risk is discussed.

7.3.1 Fish Consumption Rates for Adults

A comprehensive review of available data on fish consumption was used by USEPA in developing the RME (and CT) recreational angler fish consumption rates presented in the Exposure Factors Handbook (USEPA, 1997a) and utilized in this HHRA. The fish consumption rate of 25 g/day used in the RME risk calculations was derived by USEPA from the 95th percentile fish consumption rates from surveys of anglers on the Great Lakes and in Maine. This rate is lower than the value previously used by USEPA (i.e., 54 g/day; USEPA, 1991b) and the value used in the Onondaga Lake Public Health Consultation (i.e., 32 g/day; NYSDOH and ATSDR, 1995). Because there are no data specific to Onondaga Lake – and the Onondaga Lake fish advisories preclude generation of a reliable estimate – it cannot be determined how well the data from other freshwater bodies predict consumption rates for Onondaga Lake without advisories in place. As the direction of bias in this uncertainty is unknown, the HHRA may overestimate or underestimate exposure and risks.

USEPA (2000c) used a value of 17.5 g/day for recreational fishers (and 142.5 g/day for subsistence fishers) in setting the water quality criteria that are protective of human health (EPA 822-B-00-004; 2000c). This value is based on the 90th, rather than the 95th, percentile of the study data). Inspection of the data used in the Exposure Factors Handbook suggests that the 90th percentile value of the three studies used to derive the recommended default used in this HHRA would be close to the 17.5 g/day value used in deriving the water quality criteria. Therefore, the difference in these values (i.e., between 25 g/day and 17.5 g/day) represents a different policy based on slightly different use of the data, not uncertainty in the underlying data.

The default adult fish ingestion value (54 g/day) presented in the Standard Default Exposure Factors guidance (USEPA, 1991b) is not considered applicable based on more recent data and guidance (e.g., Connelly [1992] and the Exposure Factors Handbook [USEPA, 1997]).

7.3.2 Fish Consumption Rates for Children

Fish ingestion by children was evaluated quantitatively by using an assumed ingestion rate of two-thirds of the adult rate for older children, and one-third of the adult rate for young children. However, as there are limited data on which to establish age-specific ingestion rates for recreationally caught fish for children, this may underestimate or overestimate the actual ingestion rates for children, and thereby may underestimate or overestimate the risk to children.

This HHRA used fish ingestion rates for children recommended by USEPA Region 2. The ingestion rate for young children was assumed to be one-third of the rate for adults, and the rate for older children/adolescents (6 to less than 18 years old) was assumed to be two-thirds of the rate for adults. These rates were also recently used for the Hudson River PCBs site HHRA (TAMS/USEPA, 2000). It

should also be noted that the Hudson River HHRA used a slightly higher RME value for fish ingestion by adults – 31.9 g/day (based on the 90th percentile of the Connelly et al. [1992] study of New York anglers) – than the 25 g/day used for the adult RME for Onondaga Lake (based on the recommended RME value from the Exposure Factors Handbook [USEPA, 1997a]).

Other ingestion rates for children may also be assumed, depending on the study used and the manner in which the data are processed. For example, based on data provided by NYSDOH (Pao, 1982), which are also presented in summary form in USEPA (1997a), some assumed consumption rates for children, relative to adults, can be calculated (see Table 7-3). However, these consumption rates were not developed quantitatively due to the uncertainty of the underlying data and assumptions, and the calculated rates were presented merely to provide the reader with a sense of the range of potential error or underestimation that may be present by not addressing children's fish consumption as a separate receptor population. Consistent with USEPA policy and conventional practice, children were evaluated as two subgroups: young children (under six years old); and older children (ages 6 to less than 18). However, it should be noted that the limited amount of age-specific fish consumption data that are available do not necessarily correspond to the age ranges used in this HHRA.

An extensive literature survey completed recently by the California EPA, Office of Environmental Health Hazard Assessment (OEHHA) notes that, "... in some cases, although not all, the differences in the rates of fish consumption that have been reported are likely to correspond to differences in body weight" (California EPA, OEHHA, 2001). A linear multiplier recommended by USEPA (USEPA, 1994, as cited in OEHHA, 2001; while the version of the USEPA document cited has been superseded by USEPA, Revision 3, November 2000, the approach referenced is unchanged) "... does not account for the higher caloric requirements of young children, and pregnant and nursing women" (OEHHA, 2001). Table 7-3 presents estimates of age-based consumption of fish. No data relative to pregnant or nursing women were found.

The Great Lakes protocol states that it is "... assumed that the meal size will change proportionally with body weight. Most dieticians consider the best predictor of meal size to be the body mass of the individual" (p. 8 in GLSFATF, 1993). As shown on Tables 7-3B and 7-3C, the data from Rupp (1980) and Javitz (1980) suggest that younger children consume fish (finfish and shellfish from commercial and recreational sources, in grams per day) at an average rate of 10 to 30 percent greater than that of adults on a body weight-normalized basis, although both studies suggest that the consumption rate for older children is actually lower than that for adults. The data from Pao (1982), presented on Table 7-3D, suggest a more dramatic increase in consumption by children based on grams per meal. However, this apparent effect is mitigated by a US Department of Agriculture (USDA) study suggesting that the average number of meals per year of fish consumed by children is lower than that for adults (USDA, 1992, as cited in Table 10-46 of USEPA, 1997a). (The presentation of the USDA data makes it impossible to determine if there are a few children eating a lot of fish, or a lot of children eating a small amount of fish, since the data are presented as "percent of population consuming fish in one day." Regardless, the percentage is lower for children; for example, the percent consuming reported for adults is 10.9 percent, whereas for children [males or females] the corresponding value is 6 percent.)

In general, USEPA, by convention, explicitly or implicitly considers “children” to be “young children,” i.e., 0 to 6 years old (USEPA, 1991b, 1999). However, the peer-reviewed Hudson River HHRA (TAMS/USEPA, 2000) did evaluate consumption of fish by “child” and “adolescent” (in addition to adult) anglers. The age ranges evaluated in the Hudson River HHRA were assumed to be under six years old, and 6 to less than 18 years old (the age range was not stated, but was back-calculated based on the body weights used for each age group), and the body weights (15 kg for the young child, 43 kg for the adolescent) and RME exposure durations were consistent with the age ranges. The RME exposure durations were 6 years for the young child, 12 years for the older child/adolescent, and 23 years for the adult. (Summing these RME exposure durations across the age groups results in a total RME exposure duration of 41 years, which is higher than the normal “default” RME exposure duration of 30 years. However, there were detailed county-specific demographic data developed for the Hudson River HHRA, so site-specific, rather than default, exposure durations were used.)

The relative ingestion rates, using those utilized in the quantitative Onondaga lake HHRA as well as those derived from some of the other studies discussed above, are illustrated on Tables 7-3A through 7-3D. A comparison between the risks and hazards calculated using the Onondaga Lake assumptions and the linear estimate (body weight-normalized) assumptions is presented in Table 7-4. Both the cancer risks and the non-cancer hazards for children based on the linear (body weight-normalized) estimates are only slightly lower than the corresponding risks and hazards based on the baseline assumption.

7.3.3 Subsistence Fishers

Subsistence fishers were not evaluated quantitatively, as there is a lack of data regarding whether or not subsistence fishers (or other high-end consumers) exist in the Onondaga Lake vicinity, and a lack of data on fish ingestion rates for subsistence fishers. Based on limited data, USEPA (1997a) suggests an RME (95th percentile) rate of 170 g/day, and a CT (mean) rate of 70 g/day for subsistence fishers. As other factors in the risk calculation would be the same for these receptors, cancer risks and non-cancer hazards would be related linearly to the differences in consumption rates between the subsistence fisher and the adult angler; so the RME risks and hazards would be greater by a factor of about seven (170 g/day divided by 25 g/day), and the CT risks and hazards would be greater by a factor of about nine (70 divided by 8). RME cancer risks, which already exceed 10^{-4} , would increase further (e.g., the adult subsistence fisher risk would be about 5.3×10^{-3} , compared to 7.8×10^{-4} for the adult recreational fisher), and CT cancer risks, which are about 4.5×10^{-5} for all recreational receptors, would be about 4×10^{-4} for the adult subsistence fisher. Non-cancer hazards would also increase similarly (e.g., the adult subsistence fisher RME HI would be about 124, compared to the adult recreational fisher RME HI of 18.2). However, the very limited data also suggest that the ingestion rates, as well as the species consumed, are highly variable and site- or location-specific (see, for example, Table B-5 and section B.3.2 of Appendix B in USEPA, 2000b).

7.3.4 Fractional Intake

An additional factor considered in the uncertainty associated with this risk assessment is the amount of fish collected and consumed from Onondaga Lake versus from other resources. The fish consumption rates

used in the HHRA represent consumption from all freshwater resources fished during the season. As was the case for anglers evaluated in the studies used to derive these intake rates, however, several desirable fishing locations are available to anglers who live near Onondaga Lake. Thus, the RME fish consumption rates may not be representative of consumption from a single water body such as Onondaga Lake. The CT estimate also utilized an FI rate of 1.0 (i.e., assumed that all of an angler's total recreationally caught fish are from Onondaga Lake), but used the lower ingestion rate of 8 g/day.

The use of 1.0 as the fraction ingested for the CT scenario may lead to an overestimation of risk from fish consumption. An FI value of 0.3 would be consistent with two New York State sport-angler surveys, which evaluated the number of fishing locations for individual anglers (Connelly et al., 1990, 1992). The authors indicated that 63 percent of the respondents listed used at least three fishing locations. Use of the 0.3 FI value in this uncertainty assessment is also similar to the 0.25 FI value used by USEPA (USEPA, 1993b) in assessing upper-bound risks associated with consumption of fish in the Buffalo (New York) River, although empirically Onondaga Lake is a much more attractive fishing location and has been cited in publications as a highly desirable bass fishing spot. If the CT FI was reduced from 1.0 to 0.3, the associated risk would decrease accordingly (e.g., the adult fish ingestion CT HI would decrease from about 4.5 to about 1.3).

7.4 Uncertainties Associated with Exposure Assessment Assumptions

In the paragraphs below, uncertainties related to assumptions made for the exposure assessment are discussed. These include assumptions regarding ingestion (i.e., sediment and water, excluding ingestion of COPCs in fish, which was discussed above); exposure frequency (i.e., how many days per year an individual might be exposed to contaminated media); and similar issues. The identification of receptor populations with regard to the age of the receptors (young child, older child, or adult) is also discussed where appropriate.

For the most part, the rationale and basis for the assumptions made in the exposure assessment were presented in Chapter 4, Section 4.3.2 of this HHRA. This section is not intended to reiterate that material, but rather to assess qualitatively or quantitatively the effect of those assumptions on the risk characterization presented in Chapter 6.

7.4.1 Dermal Absorption Pathway Assumptions

There are a number of factors and assumptions which are particular to the dermal absorption pathway, including:

- Soil-to-skin adherence factors (SSAFs), addressed in Section 7.4.1.1.
- Dermal absorption factors (DAFs), addressed in Section 7.4.1.2.
- Route-to-route extrapolation factors, addressed in Section 7.4.1.3.

The field of dermal risk assessment has undergone significant change in the last few years; accordingly, USEPA guidance for dermal risk assessment has been revised and updated several times (USEPA, 1992c, 1999, 2001a). For the most part, the approach taken in this HHRA, including both the equations and the default assumptions, are consistent with the recently issued RAGS Part E (USEPA, 2001a).

7.4.1.1 Soil-Skin Adherence Factor

The SSAF is a measure of how much of the soil an individual is in contact with remains on the skin, thus serving as a source of contaminants that can be absorbed by the body. The SSAF is a function of both the type of activity in which the individual is engaged and the body part in contact exposed to the soil. A number of studies of SSAFs have been conducted for or reviewed by USEPA. The SSAFs used in this HHRA, referred to as the “consensus” SSAFs, are the result of several discussions among NYSDEC and their consultant TAMS; NYSDOH; USEPA; and consultants for Onondaga Lake National Priorities List (NPL) site Potentially Responsible Parties (PRPs). While these consensus SSAFs were established prior to the release of the current USEPA dermal guidance document, they are generally consistent with that guidance.

The specifics of the rationale for the various SSAFs used in this document are discussed in Chapter 4, Section 4.4.2 and are not repeated here. While the most recent USEPA guidance has been reviewed and utilized in this HHRA, it is noted that there are a wide range of SSAFs reported in the literature. Although extreme cases (e.g., the “kids-in-mud” scenario as reported in USEPA, 2001a and elsewhere) have not been used for establishing the SSAFs used in the quantitative HHRA, they do indicate the range of values that may exist. The values used for the RME assessment were selected to be both protective and reasonable; and the CT values were those thought, based on professional judgment, to most likely represent the typical individual of a given receptor type (e.g., adult or child recreator; construction worker). However, the true SSAF cannot be determined with precision and the values used in this HHRA may overestimate, or underestimate, the adherence of contaminated soil to potential receptor populations.

7.4.1.2 Uncertainties Related to Dermal Absorption Factors

This HHRA has used the relative absorption factors recommended by USEPA (listed in Chapter 4, Table 4-1) for both dermal and oral exposures to reflect the dermal and oral bioavailability of the constituents evaluated (i.e., the extent to which the constituents contacted or ingested are capable of being absorbed and available to act metabolically). Some of those absorption factors may overestimate the risks posed by dermal exposure, although the absence of DAFs for other contaminants (e.g., many of the metals) may underestimate exposure.

Experimental conditions from which the USEPA DAFs absorption factors were determined may differ from those which exist in Onondaga Lake, so the “true” site-specific DAFs may differ from USEPA’s experimentally determined factors used in this HHRA. For example, the DAF for PCBs is 0.14 (14 percent), based on a study by Wester et al. (1993) in which dermal absorption was measured following application of freshly spiked PCB soil/sand to the skin of monkeys; the application of PCBs from sand with

a low (less than 0.9 percent) organic carbon content would lead to greater dermal absorption than soils containing a higher fraction of organic carbon. Moreover, the Wester et al. study methodology assumed that dirt would remain pressed to the skin for 24 hours and not be washed off, which is an unrealistic scenario for most people and likely a source of risk overestimation for this factor. On the other hand, most of the experimental determinations of absorption factors have been conducted at loadings higher than those necessary to completely cover skin; consequently, the actual absorption factors "... could be larger than experimentally determined" (p. 3-22 of USEPA, 2001a), and the risks would be underestimated.

A default DAF of 0.1 was used for SVOCs (other than PAHs), based on USEPA's assessment of dermal absorption data for other organic chemicals. However, no default absorption factor was recommended for VOCs or metals other than arsenic or cadmium. VOCs and other metals are not, therefore, included in the dermal pathway in this HHRA. This is further discussed in Section 7.4.1.6, below.

7.4.1.3 Route-to-Route Extrapolation

Nearly all available toxicity data are based on pathways other than dermal absorption – i.e., ingestion or inhalation. Therefore, an assumption or extrapolation from toxicity data from one of the other routes – generally ingestion – must be made in order to assess the dermal absorbed dose, relative to that from the pathway used in the study(ies) from which the toxicity data (RfD or CSF) were derived.

In general, if the gastrointestinal (GI) absorption of a contaminant is relatively high – 50 percent or greater – no adjustment is made to the dermal route. However, studies have shown some substances, mostly metals, to be poorly absorbed from the GI tract. For these chemicals, an adjustment factor is applied to reflect the poor absorption in the oral RfD study. This factor is shown as the "oral to dermal adjustment factor" on RAGS Tables 5.1 and 6.1 (Appendix B).

There is obviously some uncertainty with regard to the route-to-route extrapolation. While no adjustment was made for any organic chemicals (since the GI absorption of organics is typically 50 percent or more, in accordance with RAGS Part E), the toxicity of the dermal absorbed dose may be underestimated to the extent that the GI absorption is less than 100 percent. There is also some uncertainty in the oral-to-dermal adjustment factors used for metals, although the direction of the uncertainty (bias) is unknown.

7.4.1.4 Dermal Absorption of Contaminants from Water

The contaminants absorbed from water are calculated differently than those from soil. For water, a chemical-specific permeability constant (K_p) controls the migration of contaminants from the water across the skin and into the body. While the available K_p values may not be precise, the risks and hazards associated with dermal absorption of contaminants from surface water were not found to be significant (i.e., non-cancer hazards were well below 1.0 [0.01 for RME] and cancer risks were below 10^{-6} [10^{-9}] for all receptors). Therefore, uncertainties associated with this pathway have little or no measurable impact on the overall risks.

7.4.1.5 Skin Surface Area Available for Dermal Contact

The values used in this HHRA are generally those recommended in USEPA RAGS Part E, which are, in turn, based on assumptions about the types of clothing likely to be worn by the receptor, coupled with studies of the surface areas of various body parts. The 50th percentile values of the skin surface areas were used. While the relationship is not strictly linear, a higher percentile of exposed skin area is typically associated with greater body mass; e.g., an individual at the high end of the skin surface area distribution would be expected to also be at the high end of the body mass distribution. Therefore, the ratio of skin surface area to body mass does not vary greatly, so the effect of using the 50th percentile for these variables (as opposed to a 90th percentile value) in the RME calculation is limited.

The assumptions regarding body parts exposed are less certain; some potential activities, such as children playing along the shoreline in the park areas (e.g., northern basin shoreline sediments) could result in close to 100 percent of the skin being in contact with the sediments. On the other hand, other activities such as older children looking for frogs or turtles in the wetlands, might involve much lower fractions of the body being in contact with the sediments (possibly just hands, and maybe feet). There is less uncertainty associated with estimates of the total skin surface area potentially in contact with Onondaga Lake surface water for high-contact recreational activities; for example, activities such as swimming or waterskiing would result in 100 percent of the total skin area being in contact with water.

7.4.1.6 Contaminants of Potential Concern Evaluated in the Dermal Pathway

Due to the general lack of data on dermal and GI absorption, USEPA (RAGS Part E) recommends quantitative analysis of the dermal pathway for only a relatively small number of COPCs (SVOCs, PCBs, PCDD/PCDFs, some pesticides, and two metals). USEPA (2001a) recommends no quantitative assessment of VOCs in soil; the rationale for this is that VOCs tend to volatilize and are accounted for by the inhalation pathway, which was not considered to be a complete pathway for the Onondaga Lake HHRA (see Chapter 4, Section 4.2.5). For inorganics other than arsenic and cadmium, USEPA indicates that absorption of metals is highly dependent upon speciation of the metals; as a result, there are too little data to recommend default absorption factors (USEPA, 2001a). As a result, risks associated with dermal absorption of some chemicals may be underestimated.

7.4.2 Uncertainties Associated with Ingestion of Soil/Sediment and Surface Water

Uncertainties associated with calculated intakes of soil/sediment and surface water are discussed below.

7.4.2.1 Soil/Sediment Ingestion Rates

The fact that USEPA recommendations for soil/sediment ingestion rates have changed over time suggests that there is some uncertainty associated with these estimates. USEPA (1991b) recommended default soil ingestion rates of 100 mg/day and 200 mg/day for adults and young children, respectively; no estimates were provided for older children or construction workers. The more recent Exposure Factors Handbook

(USEPA, 1997a) recommended a 50 mg/day ingestion rate for adults in residential or commercial/industrial settings and 100 mg/day as the mean ingestion rate for children.

Risk estimates in this HHRA used soil/sediment ingestion rates of 100 mg/day (RME) and 50 mg/day (CT) for adults and older children and 200 mg/day and 100 mg/day for young child RME and CT, respectively. (The Exposure Factors Handbook notes that although children might ingest up to 400 mg/day, there were insufficient data to recommend a 95th percentile soil ingestion rate for children.) While not explicitly making a recommendation for construction worker ingestion, the Exposure Factors Handbook does cite a study by Hawley (1985) which suggested a 480 mg/day rate. This rate (480 mg/day) has been used in other Onondaga Lake subsite risk assessment documents (e.g., LCP Bridge Street HHRA [NYSDEC/TAMS, 1998a]); however, USEPA has reevaluated the study on which this rate was based and currently recommends a construction worker ingestion rate of 330 mg/day (Exhibit 5-1 in USEPA, 2001c). It is acknowledged that there is an extremely limited data set for these assumptions, especially for the construction worker scenario; however, the direction of the bias is unknown.

7.4.2.2 Fraction Ingested from Contaminated Site

The soil/sediment ingestion rates used were applied on the assumption that the individuals' total daily intake of soil came from the Onondaga Lake site – in other words, the FI term in the intake equation is assumed to be 1.0. For recreational users of the site, who would be expected to be present at the site for only a limited time on any given day, this assumption is conservative although not unrealistic. While it is likely that, for such users, at least some portion of their total daily intake of soil would come from other sources that are uncontaminated, such as other outdoor areas and/or indoor dust, it is reasonable that incidental soil ingestion is likely to occur at much higher rates during recreational activities than during other, typically more passive, activities. Although Stanek and Calabrese (1992) found that nearly 50 percent of the total daily soil ingestion of 64 preschool children originated from indoor dust, it is not known to what extent the other activities engaged in by those children are representative of activities (and hence ingestion) likely to occur at the shore, wetlands, or the dredge spoils area adjacent to Onondaga Lake. Thus, the assumed FI of 100 percent of soil/sediment ingestion from the contaminated site, as used in this HHRA, may overestimate actual exposures to site soils and sediments via incidental ingestion, although it is possible that the default ingestion rate could underestimate ingestion as well.

7.4.2.3 Surface Water Ingestion Rates

The USEPA default assumption of 50 mL/hour (about 2 ounces/hour) ingestion while swimming was used for assumed high-contact recreational activities such as swimming and waterskiing (Exhibit 6-13 in USEPA, 1989); this value was used for both the RME and CT scenarios. The uncertainty associated with this rate is unknown.

7.4.2.4 Duration of Swimming Events

The total intake of COPCs during an “event” is also dependent of the length (duration) of the event. RAGS Part A cites an average of 2.6 hours per swimming event, based on US Department of Interior (USDOI) data; however, the Exposure Factors Handbook cites other data showing an average of one hour per swimming event. Due to the discrepancy between the two values, the higher number (2.6 hours) was used in the RME calculation, and the lower (1 hour) was used in the CT calculations. These values were used for calculation of COPC intakes resulting from both ingestion of and dermal contact with surface water. Although there is some uncertainty in these values, it is unlikely that many receptors would have a significantly higher exposure than the RME value assumed.

7.4.3 Body Weight

The 50th percentile body weight was used for young children and older children, based on the data in the Exposure Factors Handbook. Although recent data show 71.8 kg (rather than 70 kg) as the 50th percentile body weight for adults, the USEPA default value of 70 kg is used in this HHRA. The 70 kg adult body weight is used both by convention and due to the fact that many of the toxicity values (RfDs and CSFs) are based on the assumption of a 70 kg body weight. The body weight is a less sensitive parameter (varying by less than a factor of two between the 5th percentile and 95th percentile for both male and female adults) than some of the other variables in the risk calculations, so the 50th percentile body weight used for all scenarios does not have a major impact on the uncertainty in this HHRA. The error introduced by this is negligible, and the uncertainty introduced is insignificant in comparison to the uncertainty associated with some of the other parameters (e.g., estimates of exposure frequency or ingestion rates).

7.4.4 Exposure Duration and Frequency

The assumed exposure durations used in this HHRA were 30 years for the adult RME and nine years for the adult CT. These default values were established in the Exposure Factors Handbook (as cited in USEPA RAGS Part A, 1989), based on national demographics (30 years being the 90th percentile value, and nine years the 50th percentile) on the length of time individuals live at a single residence. (More recent studies summarized in the Exposure Factors Handbook [USEPA, 1997a] provided estimates very similar to the default values recommended in 1989 and used in this HHRA.) These default assumptions may underestimate exposure of individuals in situations where exposure may continue even if a person moves but continues to be exposed to the site; for example, a move within Onondaga County. Since some people live in a single location or single area for periods longer than 30 years (e.g., 70 years or more), the cancer risks, although not the non-cancer hazards, would increase proportionally for such persons.

In the HHRA conducted for the Hudson River PCBs site (TAMS/USEPA, 2000), a detailed evaluation of the exposure duration for fish ingestion (based on the number of years an individual fishes in the Hudson River site) was conducted. This evaluation was based on three factors:

- The age an individual begins fishing.
- When the individual stops fishing.
- The length of time the person resides in the five-county area adjacent to the 34-mi (54.7-km) length of Hudson River under consideration.

Despite the fact that a significantly larger area was under consideration, the Hudson River-specific exposure durations were only about one-third greater than the standard defaults assumed for the one-county Onondaga Lake HHRA area. Specifically, the 95th percentile exposure duration for the Hudson River HHRA was about 40 years (compared to the 30-year RME exposure duration for this Onondaga Lake HHRA), and the 50th percentile exposure duration for the Hudson River was 12 years (compared to the nine years used as the CT exposure duration for the Onondaga Lake HHRA). The exposure durations calculated for the Hudson River suggest that the USEPA defaults used for the Onondaga HHRA are reasonable and are not likely to significantly understate the exposure to the vast majority of receptors. However, exposure may be underestimated for those individuals who live in the same residence their entire life, or who change residences but remain near Onondaga Lake.

In accordance with the risk characterization equations provided in RAGS Part A (USEPA, 1989), an averaging time of 25,550 days (70 years \times 365 days/year) was used for calculations of cancer risk. The more recent data in the Exposure Factors Handbook (USEPA, 1997a) indicates that the current average life span is 75 years. However, the 70-year (25,550-day) period was used as the averaging time for cancer calculations, as the derivation of CSFs is based on an average lifetime of 70 years. As the averaging time appears in the denominator of the risk calculation, a higher averaging time of 75 years would effectively decrease the calculated cancer risk by about 7 percent.

For non-cancer hazard calculations, the averaging time is equal to the exposure duration multiplied by 365 days/year. As the exposure duration values cancel each other out in the non-cancer hazard calculation (the exposure duration is in the numerator, and the averaging time is in the denominator), there is no uncertainty associated with the non-cancer averaging time.

The exposure frequency is not an uncertainty issue for fish ingestion, as the data used are a daily average ingestion rate, so a duration of 365 days is necessary to balance the equation. Issues related to the uncertainty of the fish ingestion rate were discussed previously in this chapter.

For northern basin sediments, including the two northern basin wetlands, SYW-6 and SYW-10, 44 days/year was used for the RME frequency and 32 for the CT. By comparison, the Hudson River HHRA used an assumed recreational exposure frequency of 26 days for the RME and 13 days for the CT for children, and 13 days for the RME and seven days for the CT for adults. However, acknowledging that there might be a subpopulation of highly exposed recreators for the Hudson River, an "avid recreator" subpopulation was also evaluated. For the Hudson River avid recreator, the assumed exposure frequencies for adults and adolescents were 104 days for the RME and 52 days for the CT (children below the age

of 12 were not assumed to be avid recreators). The exposure frequencies used for the northern basin of Onondaga Lake are within the range used for the Hudson River recreator and are approximately half of the frequencies used for the Hudson River avid recreator, and as such are reasonable for a typical recreator. However, the exposure frequency used in this HHRA may not capture all highly exposed individuals (i.e., avid recreators) and thereby may underestimate risks to such persons.

In order to assess the hypothetical avid recreator, site-specific high-end exposure frequencies have also been estimated using the following assumptions:

- The exposed population is older children (age 6 to 18 years old).
- Exposure occurs only when the daily maximum temperature is 70°F or higher (May 19 to September 21, based on 1971 to 2000 Syracuse weather data) (NOAA, 2002).
- Exposure occurs five days/week during summer vacation (assumed to be ten weeks, from June 23 to September 1).
- Exposure occurs two days/week (i.e., on weekends) during the school year (May 19 to June 23, and September 2 to September 21) for a total of eight weeks.

Based on the assumptions listed above, the avid recreator exposure frequency would be 66 days/year $[(5 \times 10) + (2 \times 8)]$. This hypothetical avid recreator exposure (66 days/year) is 50 percent higher than the RME value (44 days/year) assumed for the northern basin sediments. Cancer risks and non-cancer hazards would also increase correspondingly (i.e., by 50 percent) for the avid recreator. However, this would not increase the HI to a value greater than 1.0, nor would it change the cancer risk category for the northern basin sediments or Wetlands SYW-6 or SYW-10.

The exposure frequency for southern basin sediments, including Wetlands SYW-12 and SYW-19, was assumed to be 14 days/year for the RME and five days/year for the CT. This exposure frequency is much lower than that assumed for the northern basin, due to the relative lack of potential points of contact, as the landside borders of the southern basin are dominated by industrial and commercial properties, while the northern basin shores have large amounts of parkland. It was, therefore, considered unlikely that exposures would be as high to the southern basin sediments as to the northern basin sediments, even under future scenarios. However, as with the northern basin sediments, an exposure frequency as high as 66 days per year could be used as the RME, assuming visits to the southern basin using the same assumptions as described above for the northern basin hypothetical avid recreator. Under this hypothetical avid recreator scenario, RME exposure, and, consequently, the associated risks and hazards, would increase by a factor of about 4.7 (66 divided by 14). Under this scenario, the RME cancer risk for the older child exposure to Wetland SYW-19 sediments would increase to over 10^{-4} (from about 4.9×10^{-5} to about 2.3×10^{-4}); other risk and hazard categories would be unchanged. The appropriateness of the professional judgment

estimates of exposure frequency is uncertain for the southern basin, and it is certainly possible that higher or lower estimates might better represent the likelihood of exposure.

For the construction worker scenario, the exposure frequency was assumed to be 25 days (five weeks) for the RME, and ten days (two weeks) for the CT; in each case, the exposure duration was assumed to be two years. These estimates are based on the assumption that the nature of the construction activity will be sewer line installation, utility repair, or similar work, but these exposure frequencies would probably be low for a construction period for erecting a structure (e.g., a warehouse or marina). Due to the fact that much of the lake area includes regulated wetlands or parkland, large-scale construction projects in areas covered by this HHRA (lake sediments, wetlands, and the dredge spoils area) were considered unlikely. However, to the extent that such a larger project may occur, this HHRA may underestimate risks or hazards to construction workers. For example, if a more generic default assumption of 250 days (i.e., the construction worker default in USEPA, 2001c) for a larger construction project is applied, the RME risks and hazards for the construction worker would increase by a factor of 10 (250 days divided by 25 days). This would result in the RME HI for the construction worker increasing to greater than 1.0 for southern basin sediments (HI of 2.2), Wetlands SYW-12 (HI of 1.4) and SYW-19 (HI of 1.6), and the dredge spoils area soils (HI just over 1.0). RME cancer risks would increase to greater than 10^{-6} (from about 6×10^{-7} to about 6×10^{-6}); other risk and hazard categories would be unchanged (i.e., cancer risks would still be less than 10^{-6} , and HIs would still be less than 1.0).

7.5 Uncertainties Related to Available Toxicity Data

The toxicity data discussed in this section are the quantitative values used in the risk characterization presented in Chapter 6 of this HHRA; specifically, the non-cancer RfDs and the CSFs used to calculate risks and hazards. It is important to note that the toxicity data developed by USEPA are designed to be plausible upper-bound estimates; i.e., the values are intended to be protective. From that perspective, some of the toxicity values used may be considered to be "conservative." With that in mind, the three main issues related to the uncertainty of the toxicity data used in this HHRA are:

- Accuracy of toxicity data (RfDs, CSFs) for the COPCs (including chemical "surrogates" approved by NCEA, such as pyrene for some non-cancer PAHs and lindane [gamma-HCH] for delta-HCH).
- Effect of lack of quantitative data for chemicals that have been detected. The issue of chemicals that may be present but were not analyzed, or at least not consistently analyzed, is addressed in Section 7.1.1.
- Toxicity data assumptions for chemicals for which the specific form was not analyzed, including:
 - Fish: Mercury and arsenic forms.
 - All media: Chromium (trivalent and hexavalent) and mercury forms.

It should also be noted that NYSDOH has developed quantitative risk factors for a few chemicals for which USEPA has also published values or, in a few cases, has recommendations for assessment of COPCs for which USEPA does not have published toxicity values. Onondaga Lake-specific COPCs for which NYSDOH has developed toxicity values (i.e., CSFs or RfDs) include the following:

- Cadmium.
- Benzene.
- Benzo(a)pyrene.
- DDE.
- delta-HCH.
- Chlordane.

Although tending to be slightly more conservative, the NYSDOH toxicity values for these chemicals are generally similar to those developed by USEPA and are not for the major COPCs that contribute to the cancer risks or non-cancer hazards in this HHRA. The exception to this is dioxins/furans (PCDD/PCDFs), for which NYSDOH recommends a quantitative assessment for non-cancer effects. A semi-quantitative assessment of potential non-cancer hazards from dioxin-like compounds is presented in Section 7.5.1.3.

7.5.1 Accuracy of Quantitative Cancer and Non-Cancer Toxicity Values

7.5.1.1 Polychlorinated Biphenyls

Issues related to both the cancer and non-cancer toxicity data are discussed below.

Cancer Slope Factor for PCBs

The CSF is an upper-bound estimate of the carcinogenic potency of a chemical used to calculate cancer risk from exposure to carcinogens by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over that lifetime. IRIS, the online database of USEPA's consensus review of toxicity data, provides both upper-bound and central estimate CSFs for three different tiers of PCB mixtures, as described in Chapter 5, Section 5.2.6.1. These values are based on USEPA's reassessment of the carcinogenic potency toxicity data for PCBs (USEPA, 1996a), and were derived using the proposed revisions to the USEPA Guidelines for Carcinogen Risk Assessment (USEPA, 1996b). A range of potency estimates was determined using studies for a range of mixtures, rather than focusing on the highest-potency mixture. USEPA's reassessment concludes that "uncertainty around the CSF estimate extends in both directions." However, the overall CSFs developed by USEPA represent plausible upper-bound estimates, indicating that there is reasonable confidence that the actual cancer risk will not exceed the cancer risk calculated using the CSF.

Reference Dose for Non-Cancer Hazards of PCBs

The non-cancer hazards for PCBs are based on the low molecular weight (Aroclor 1016) and high molecular weight (Aroclor 1254) oral RfDs in IRIS, as summarized in Chapter 5, Section 5.2.6.2 of this HHRA. The confidence level for both the RfDs is characterized by USEPA in IRIS as medium, indicating a moderate amount of uncertainty with regard to the numerical values. The combined uncertainty and modifying factors used in the development of these factors are 100 for Aroclor 1016 and 300 for Aroclor 1254; these uncertainty factors are lower than those for the majority of the other organic COPCs assessed in this HHRA (see Appendix B, RAGS Table 5.1).

It is noted that there are numerous studies, both human and animal, of the health effects of PCBs, some of which have been published since the last significant revision to the IRIS file. As noted in the Hudson River PCBs Site HHRA (TAMS/USEPA, 2000), USEPA is currently performing an evaluation of these studies as part of the ongoing IRIS process. In addition to carcinogenicity, these recent studies have focused on non-cancer effects including the developmental, neurotoxic, thyroid, immunological, and reproductive effects of PCB exposure.

Assessing both exposure to and effects of PCBs *in utero* to fetuses, to nursing infants, and to the children of exposed individuals (i.e., those with higher-than-normal body burdens of PCBs) is even more complex. PCB transfer from the placenta and from breast milk can result in significant exposures *in utero* and to nursing infants (DeKoning and Karmaus, 2000, as cited in the Hudson River PCBs Site HHRA [TAMS/USEPA, 2000]). However, the means for assessing the effects of such exposure quantitatively do not yet exist. Therefore, fetuses, nursing infants, and children of exposed individuals may constitute an additional highly exposed subpopulation on whom the effects of PCB exposure cannot be quantified.

Dioxin-Like PCBs

Twelve PCB congeners (non-ortho and mono-ortho substituted) have been identified as “dioxin-like PCBs” (Van den Berg, et al., 1998), and toxicity equivalence factors (TEFs) have been calculated for these congeners. As no congener-specific PCB analyses were performed for the Onondaga Lake investigation, the specific cancer potential of these congeners is not assessed. However, the Aroclor CSFs were developed using commercial Aroclors which contain these congeners and, therefore, the cancer potential of these congeners is included in the CSF for PCBs. However, to the extent that the distribution of congeners in the environment differs from that of the commercial Aroclors used to develop the CSFs, the associated risks could be either higher or lower than those presented in this HHRA.

7.5.1.2 Methylmercury Non-Cancer Reference Dose

The methylmercury RfD of 0.0001 mg/kg-day (1×10^{-4} mg/kg-day, or 0.1 µg/kg-day) is published in IRIS, and USEPA notes that confidence in this value is high. The RfD was originally derived in 1995 from data on delayed walking reported in Iraqi infants whose mothers were accidentally exposed to relatively high levels of alkylmercury in grain (Marsh et al., 1987).

In 1999, the National Research Council (NRC) was directed by Congress to review the USEPA-derived RfD and to focus on review of new studies published since USEPA established the RfD. The NRC report, *Toxicological Effects of Methylmercury*, was published in 2000; the current RfD was derived from the findings of that report and confirms the RfD from IRIS (1×10^{-4} mg/kg-day). A benchmark dose approach (BMD) was used, rather than a no observed adverse effect level/lowest observed adverse effect level (NOAEL/LOAEL) approach, as the response variable for analyzing the neurological effects in children.

The adverse effect of methylmercury at the lowest observed dose is neurotoxicity (i.e., the brain is the most sensitive organ), particularly among developing organisms. Among the extensive array of peer-reviewed data from low-dose exposure to methylmercury, USEPA and NRC identified three longitudinal developmental studies of the human populations consuming mercury-contaminated marine mammals. The three studies determined to be suitable for use in establishing quantitative risk assessment are the Seychelles child development study, ongoing studies of children in the Faeroe Islands, and the study of children in New Zealand. Although the Seychelles study found no evidence of impairment related to methylmercury exposure, the Faeroe and New Zealand studies found dose-related adverse effects on a number of endpoints. In the establishment of the RfD, emphasis was placed on the results of the Faeroe Islands study, as it is the larger of the two studies that identified methylmercury-related developmental neurotoxicity. Supporting evidence from the New Zealand study provides assurance that this focus is the appropriate strategy for protecting public health. In addition, an integrative analysis of all three studies was also performed.

Benchmark dose analyses were performed for a number of endpoints from all three studies (Chapter 7 of NRC, 2000). NRC estimated a CT measure, equivalent to a BMD, across all three studies for all endpoints that were identified as significant in the Faeroe Islands and New Zealand studies, and for all endpoints at 5.5 years of age in the Seychelles study. NRC also determined a lower limit based on a theoretical distribution of BMDs, which is the logical equivalent of a BMDL (benchmark dose limit). NRC also used a hierarchical random-effect model to reduce random variation in the estimate for these same endpoints from all three studies (Table 7-5, pp. 290-294 in NRC, 2000). Additionally, this analysis was used in calculating BMDs and BMDLs for the most sensitive and median endpoints from both the Faeroe Islands and New Zealand studies (Table 7-6, p. 294 in NRC, 2000). This approach also allowed an integrative analysis of data from all three studies.

The IRIS file for methylmercury presents BMDL_{05s} (BMDLs at the 5 percent significance level) from a number of endpoints in terms of cord-blood mercury (see Table 2 of IRIS file, included in Appendix E of this HHRA). These tests are all indications of neuropsychological processes involved in a child's ability to learn and process information. The BMDLs for these scores are all within a relatively close range. They were converted using a one-compartment model to an ingested dose of methylmercury that would result in the cord-blood level. The last column shows the corresponding RfD from application of an uncertainty factor (UF) of 10 (discussed in the following paragraph). The calculated RfD values converge at the same point: 0.1 µg/kg-day. USEPA also calculated geometric means from the four endpoints from the Faeroe Islands study; RfDs were 0.1 µg/kg-day based on these calculations. For the New Zealand study, both the median value and the results of the McCarthy Perceived Performance test yielded RfDs of 0.05 µg/kg-day,

and the McCarthy Motor Test yielded an RfD of 0.1 µg/kg-day. Based on the integrative analysis of all three studies, the RfD would be 0.1 µg/kg-day.

Due to the availability of human epidemiological studies, a relatively low composite uncertainty factor (combination of the modifying factor and uncertainty factor) of 10 was applied in the development of the methylmercury RfD. This choice was made to account for the following factors:

- Pharmacokinetic variability and uncertainty in estimating an ingested mercury dose from cord-blood mercury concentration: a factor of 3 was applied.
- Pharmacodynamic variability and uncertainty: a factor of 3 was applied.

Due to the availability of adequate data and peer-reviewed and government-reviewed studies, and the convergence of the RfD values calculated or derived from different studies and different endpoints, confidence in the RfD for methylmercury is considered high and there is little uncertainty associated with it.

7.5.1.3 Dioxins and Furans

Substantial uncertainties exist in the quantitative toxicity assessment for PCDD/PCDFs. These uncertainties result from both the numerical CSFs and TEFs used to calculate risks associated with these compounds, and the absence of numerical toxicity data to quantitatively estimate non-cancer hazards associated with this compound class.

It should be noted that USEPA is conducting an extensive dioxin reassessment; preliminary drafts of some of the documentation generated by that reassessment have been released for review (USEPA, 2000a). While this reassessment has not been completed or peer-reviewed, tentative or preliminary conclusions from it are noted where applicable.

Cancer Slope Factors for Dioxins and Furans

The primary issues of debate focus on the appropriate interpretation of the available data. Regulatory agencies and others in the US and elsewhere have calculated quantitative estimates of TCDD carcinogenicity that span more than three orders of magnitude. These differences arise as a result of alternative assumptions used when interpreting the underlying animal bioassay data, changes that have occurred in standard procedures for interpreting and extrapolating from such data to predict human health risks (e.g., scaling factors for relating animal data to human populations), and, most importantly, assumptions regarding the mechanism of action by which 2,3,7,8-TCDD exerts carcinogenic effects (threshold or non-threshold). The range of CSF values (carcinogenicity estimates) suggest there may be considerable uncertainty associated with the CSF used in this HHRA for PCDD/PCDFs.

As described in Chapter 5, Section 5.2.7, the cancer risks associated with PCDD/PCDFs were assessed by converting the 2,3,7,8-substituted congeners to a TEQ of 2,3,7,8-TCDD through the use of TEFs published by the WHO (van den Berg et al., 1998). A CSF for 2,3,7,8-TCDD was then applied to the resulting TEQs. USEPA does not list a CSF for 2,3,7,8-TCDD on IRIS; the value used in this HHRA is from HEAST (USEPA, 1997). In the past, a wide range of CSFs have been proposed for 2,3,7,8-TCDD based on a rat study by Kociba et al. (1978). For this HHRA, a CSF of 1.5×10^5 (mg/kg-day)⁻¹, which is at the upper end of this range, was used, which is the value listed by USEPA on HEAST.

The TEF system for converting other PCDD/PCDF congeners to a 2,3,7,8-TCDD equivalence (i.e., TEQ) is generally accepted both nationally and internationally by cancer researchers and regulatory agencies.

A reassessment of this study, along with data from three human studies, led USEPA in the draft dioxin reassessment to suggest “the use of 1×10^{-3} per pg/TEQ/kgBW/day as an estimator of upper-bound cancer risk for both background intakes and incremental intakes above background” (p. 105 of USEPA, 2000a). This converts to a CSF of 1.0×10^6 (mg/kg-day)⁻¹. If the final version of the dioxin reassessment confirms this value, the dioxin-related cancer risks presented in this HHRA would increase by a factor of about seven, resulting in an upper-bound RME cancer risk estimate of 3×10^{-3} for the adult fish ingestion pathway.

Non-Cancer Toxicity of Dioxins and Furans

Non-carcinogenic risks from exposure to PCDD/PCDFs were not estimated in this risk assessment because USEPA does not currently recommend an RfD for non-cancer hazards. (The preliminary reassessment being conducted by USEPA does not recommend establishing an RfD for dioxin and related compounds due to the relatively high background compared to effect levels [p. 108 of USEPA, 2000a]. The current estimated average dose to the US population, about 1 picogram per kilogram per day (pg/kg-day), is greater than the RfD/reference concentration (RfC) values that would be calculated given the data reviewed in the reassessment, and, therefore, toxicity-based RfD or RfC values would be uninformative for evaluation of non-cancer health risks [p. 122 of USEPA, 2000a].) Non-cancer risk estimates are presented to address this uncertainty.

Specifically, an HQ can be calculated by comparing the non-carcinogenic RME estimate of PCDD/PCDFs intake from fish (7×10^{-9} mg/kg-day) with the minimum risk level (MRL) published by ATSDR of 1 picogram TCDD TEQ/kg-day, or 1×10^{-9} mg/kg-day (ATSDR, 1999; however, USEPA’s dioxin reassessment indicates that there are some questions about the ATSDR MRL, due to concerns with the lower bounds of effects data for non-cancer endpoints [p. 109 of USEPA, 2000a]). The ATSDR MRL is analogous to a USEPA RfD in that it is the level of exposure below which the agency believes is without an appreciable risk for non-cancer health effects. Use of this MRL as an RfD would result in an HQ for PCDD/PCDFs of 7 in the RME adult fish consumption exposure scenario (based on the non-cancer intake of 7×10^{-9} mg/kg-day) and 1.5 in the CT fish consumption scenario (based on the non-cancer CT chronic daily intake [CDI] of 1.5×10^{-9} mg/kg-day), which would indicate that there is concern for adverse non-cancer effects related to exposure to PCDD/PCDFs in fish. These HQs would also correspondingly

increase the total HIs for the recreational angler through fish consumption (summarized in Chapter 6, Table 6-1).

More recently, the WHO has cited intakes in the range of 1 to 4 pg/kg-day ($1 \text{ to } 4 \times 10^{-9} \text{ mg/kg-day}$) as a “tolerable daily intake” (WHO, 2000, as cited in USEPA, 2000a). The RME estimate of intake from fish exceeds the high end of the WHO range; the CT intake estimate is within this range, although it exceeds the low end.

The draft dioxin reassessment indicates that USEPA is taking a margin-of-exposure (MOE) approach for non-cancer endpoints to inform risk management decisions. The MOE is the dimensionless ratio of a low-end effect level, such as an LOAEL, in the comparison species, to calculated or observed CDI. Higher MOE values are associated with the assumption of decreased risk, such as the decreased likelihood of adverse health effects occurring. Based on the USEPA estimated average (background) dose to the US population (about 1 pg/kg-day), USEPA indicates that MOEs range from less than 1 to as high as 15, depending on the study, species, and effect monitored (p. 122 of USEPA, 2000a) for establishing the LOAEL or effective dose. NYSDOH staff support the MOE approach and suggested using several studies of reproductive effects in rhesus monkeys to establish a lowest observed effect level (LOEL) of 0.13 ng/kg-day ($130 \times 10^{-9} \text{ mg/kg-day}$) (NYSDOH, 2002c; 2002, pers. comm., August 5). Using this LOAEL and the RME intake of $7 \times 10^{-9} \text{ mg/kg-day}$ would result in an MOE of about 20.

Although the available toxicity data show that exposure to dioxin-related compounds may result in a measurable increase in the non-cancer hazards, inclusion of the results discussed immediately above in the quantitative risk estimates would not change the overall conclusions in this HHRA regarding the site because the excess cancer risk estimate for PCDD/PCDFs for the fish consumption pathway was 4.5×10^{-4} and the total HI for fish consumption was already above 1.0.

7.5.2 Lack of Quantitative Toxicity Values for Detected Chemicals

7.5.2.1 Use of Surrogate Toxicity Data for Polycyclic Aromatic Hydrocarbons

As discussed in Chapter 5, Section 5.2.4, there are specific toxicity data available for only a few of the PAHs detected in soil/sediment samples used in the Onondaga Lake HHRA. Both cancer and non-cancer toxicity data were extrapolated to other PAH compounds for the quantitative cancer risk and non-cancer hazard calculations.

Cancer Slope Factors for PAHs

Benzo(a)pyrene is the only PAH compound for which a specific CSF has been developed. As noted in Chapter 5, Section 5.2.4.1, the cancer risk associated with other PAH compounds classified as B2 carcinogens has been estimated quantitatively using relative provisional carcinogenic potency factors published in 1993. As these factors were only considered to represent order-of-magnitude accuracy at the time they were first published, and they were provisional and have not been updated or finalized in nearly

ten years, there may be a fair amount of uncertainty associated with the cancer risks associated with these PAHs, although the direction of the uncertainty is unknown.

Dermal contact with PAHs was evaluated in this risk assessment using the oral CSF and RfD. There is a fair amount of uncertainty on this issue due to uncertainties related to derivation of toxicity values for dermal exposures for chemicals having site-of-contact effects, such as PAHs (USEPA, 1989b). The most recent USEPA dermal guidance recommends that PAHs be assessed quantitatively for systemic effects (non-cancer hazards) and qualitatively for carcinogenicity (RAGS Part E, Section 5.2.3). Therefore, there is a fairly high degree of uncertainty associated with the quantitative estimate of cancer risk associated with dermal contact with carcinogenic PAHs.

Reference Doses for Non-Cancer Effects of PAHs

As with carcinogenic PAHs, USEPA has non-cancer toxicity data for only a few PAH compounds. However, USEPA NCEA has reviewed studies for various PAHs and has provided recommended values for several non-carcinogenic PAHs for which there are no published values in IRIS or HEAST (see Chapter 5, Section 5.2.4.2 in this HHRA). While these values cannot be considered to have the same level of certainty as IRIS values, USEPA has specifically reviewed these values for use in the Onondaga Lake HHRA. Therefore, although there is some uncertainty associated with the non-cancer hazard calculations for these compounds, recent review has confirmed that these values are adequate for quantitative risk assessment.

The USEPA NCEA has only reviewed and approved the RfDs for the non-carcinogenic PAH COPCs. NYSDOH has noted that the carcinogenic PAHs may also have non-cancer health effects, and has suggested that the RfD for pyrene be utilized for assessing non-cancer hazards associated with PAH compounds that are assessed for cancer risks. The potential impact of this was assessed by reviewing the data for the southern basin sediments, a medium with high PAH concentrations. If these carcinogenic PAH compounds were also evaluated for non-cancer toxicity in the quantitative HHRA, the increase in RME hazards associated with these compounds would be insignificant (i.e., HIs for the individual PAHs would increase on the order of 10^{-4} to 10^{-5}). For example, applying the pyrene RfD to the southern basin sediment RME hazard for the young child would increase the calculated HI slightly to 0.54 from 0.535, and the older child RME hazard would increase to 0.256 from 0.253.

7.5.2.2 Surrogate Toxicity Data for Other Contaminants of Potential Concern

USEPA NCEA has indicated that the toxicity data for gamma-BHC (lindane) is appropriate for assessing risks associated with the Onondaga Lake COPC delta-BHC. Lindane is a relatively well studied compound due to its use as a commercial pesticide. As it is unlikely that the related compound delta-BHC is more toxic than lindane, and the calculated risks associated with this compound are not significant (i.e., do not contribute as much as ten percent of the risk or hazard by any pathway), any error or uncertainty introduced by this assumption does not materially affect the conclusions of this HHRA.

7.5.2.3 Contaminants of Potential Concern Without Quantitative Toxicity Data

Concentrations of two chemicals, lead and 2,4'-DDE, exceeded screening level concentrations and these chemicals were, therefore, identified as COPCs in one or more pathways. As sufficient quantitative toxicity data for lead and 2,4'-DDE were not available, these compounds were not included in the quantitative risk calculations. The potential impact of not having toxicity data for them is discussed below.

Lead

An analysis for lead was conducted in all site media. Lead in sediment and dredge spoils was screened against the USEPA residential soil screening criterion of 400 mg/kg, and lead in water was screened against the federal drinking water action level of 15 µg/L (40 CFR, Part 141). No screening values were available for lead in fish tissue. IRIS classifies lead as B2, probable human carcinogen, but no quantitative CSFs or RfDs were available in IRIS.

Lead was detected in 8 of 11 fish tissue samples at a maximum concentration of 0.29 mg/kg (290 µg/kg). Lead was not identified as a COPC in fish tissue, as there were no toxicity data for screening or risk quantitation. While the concentrations of lead detected are low on a qualitative basis (i.e., the maximum detected lead concentration was lower than the maximum detected concentration of almost every other metal in fish tissue; see Appendix B, RAGS Table 2.1), the potential impact of not being able to quantitate risks or hazards associated with lead cannot be determined.

Lead was identified as a COPC in southern basin sediments (lead concentrations were below the screening criteria in other sediment and dredge spoil samples), where it was detected in all 100 samples at a maximum concentration of 2,050 mg/kg. However, the medium-specific EPC (i.e., the 95 percent UCL on the mean) was 235 mg/kg (see Appendix B, RAGS Table 3.3), which is lower than the screening criterion of 400 mg/kg. Considering also that the potential for exposure to the southern basin sediments is lower than the exposure potential assumed by USEPA in deriving the residential screening criterion for lead, it is likely that lead does not contribute significantly to cancer risk or non-cancer hazard in the southern basin (or in other soil/sediment media) of Onondaga Lake.

Lead was detected in 12 of the 48 surface water samples from Onondaga Lake at a maximum concentration of 7.7 µg/L, about half the screening criterion of 15 µg/L (see Appendix B, RAGS Table 2.10). Lead was, therefore, screened out and was not considered a COPC in surface water. The lack of quantitative toxicity data for lead did not affect the calculation of risks for the surface water pathway.

2,4'-DDE

2,4'-DDE was identified as a COPC in fish tissue; samples were not analyzed for 2,4'-DDE in other media (surface water, sediments, or dredge spoil soils). 2,4'-DDE is chemically related to 4,4'-DDE and DDT, and was screened against the USEPA Region 3 fish ingestion criteria for "DDE" – the specific isomer (i.e., 2,4'- or 4,4'-) is not specified for the DDT-related compounds (which also include DDE and DDD).

Analysis for the 2,4' isomers of DDT-related compounds was conducted on 38 of the 134 fish tissue samples (about 28 percent of the total) for which pesticide analysis was conducted. 2,4'-DDE was detected in 7 of the 38 samples in which it was analyzed, at a maximum concentration of 24 µg/kg (Appendix B, RAGS Table 2.1), and the medium-specific EPC (95 percent UCL on the mean) was determined to be 4.1 µg/kg (Appendix B, RAGS Table 3.1); this EPC is substantially lower than the EPCs for the 4,4' isomers of the three DDT-related compounds (ranging from less than half to about one-eighth of the values). The non-cancer HQs associated with the 4,4'-DDT compounds were all less than 0.01 (Appendix B, RAGS Table 7.1); therefore, even assuming similar toxicity for 2,4'-DDE, it would not cause a significant increase in the non-cancer hazard for fish ingestion. For cancer risks, if the CSF for 4,4'-DDE were applied to 2,4'-DDE, the resultant RME risk would be about 2×10^{-8} , using the cancer intakes and CSFs as shown in Appendix B, RAGS Table 8.1. This would not result in a noticeable increase in the RME cancer risk for fish ingestion.

7.5.3 Lack of Analytical Data on the Specific Form of Contaminants of Potential Concern

The toxicity of some chemicals is affected by the form in which it exists, such as the structural or positional isomer (for organics) or the specific compound or valence state (for inorganics). For the Onondaga Lake HHRA, there are not significant uncertainties for most of the organic chemicals which contributed to risks or hazards. For the Onondaga Lake HHRA, the four COPCs for which there are form- and toxicity-related uncertainties are metals and PCBs, specifically:

- Mercury, especially in fish. The non-cancer toxicity varies depending on whether the mercury exists as organic (methylmercury) or inorganic mercury.
- Arsenic in fish. Inorganic arsenic is much more toxic than organic arsenic; however, much, if not most, of the arsenic in fish may be in the organic form.
- Chromium in all media. Hexavalent chromium (Cr^{+6}) is much more toxic than trivalent chromium (Cr^{+3}), although most of the chromium detected in environmental samples is usually in the trivalent form.
- PCBs in all media. Uncertainties exist with regard to analytical reporting and dioxin-like toxicity.

The specific assumptions used in the HHRA for each of these chemicals, and the potential impact of these assumptions on the HHRA, are discussed below.

7.5.3.1 Mercury and Methylmercury

Due to the different oral toxicity of inorganic mercury and methylmercury (methylmercury is considered to be three times as toxic as inorganic mercury, based on the oral RfDs published in IRIS), it was necessary to determine the fraction of mercury in each medium or pathway which was in the inorganic form. For some

media (i.e., fish tissue, lake sediments, and surface water) there are site-specific data for both forms of mercury and the assumptions made are based on the available data. For other media (i.e., wetlands sediments and dredge spoils) there are no site-specific data and therefore estimates of the fraction of the total mercury which is present as methylmercury are based on data from nearby sites, literature reviews, and professional judgment. The basis for the assumptions for each of the five media, as well as the potential impact on risk calculations of other assumptions (i.e., assumptions of either higher or lower percentages of methylmercury) are presented below.

A more detailed discussion of the literature and data used for the assumptions made for both the Onondaga Lake Baseline Ecological Risk Assessment (BERA) and HHRA regarding the methylation of mercury is presented in Chapter 6, Section 6.3.1.1 of the BERA (TAMS, 2002a).

Fish Tissue

For the fish samples, the assumption that all the mercury detected is in the form of methylmercury is likely to cause very little error. There are some site-specific data which support the assumption that the vast majority of the mercury detected in fish samples is methylmercury. Inorganic mercury is also toxic; however, it is less toxic by a factor of three and has an oral RfD corresponding to its lesser toxicity. (If, for example, 10 percent of the mercury in the fish were inorganic, then the assumption that 100 percent of the mercury is methylmercury would overestimate the hazard by about 7 percent. Fish ingestion hazards would still be substantially greater than 1.0.) The predominance of the mercury as organic mercury in fish tissue is attributed to the validated assumption for the Onondaga Lake site that most of the mercury in fish is methylmercury or organic mercury.

Lake Bottom Sediment

For lake sediments, the methylmercury concentration is based on data from the 17 northern and southern basin nearshore sediment samples analyzed for total and methylmercury, in which the detected concentrations of methylmercury were typically two to three orders-of-magnitude lower than the concentrations of total mercury, and averaged about 0.2 percent of the total mercury concentration. This same ratio (methylmercury about 0.2 percent of total mercury) was also observed in 14 samples from the greater depths in Onondaga Lake which were not used to calculate EPCs in the HHRA (as they were collected from depths of 3 m or more). These data are shown on Table 7-5. Although the data sets are consistent with each other, as well as within the range of concentrations expected based on the literature, the methylmercury concentrations, especially in the northern basin (from which only three samples used in the HHRA were collected), are based on a relatively small number of data points and represent a "snapshot," as all the samples were collected during a single sampling event (summer 2000).

For the lake sediments, the actual concentrations of both methylmercury and mercury (evaluated as inorganic mercury) were used quantitatively in the HHRA. For northern and southern basin sediments, hazards associated with both total (as inorganic) mercury and methylmercury were calculated separately, based on the data available. For the northern basin sediments, the EPC for methylmercury (2×10^{-3} mg/kg)

is about 0.1 percent of the EPC for total (assumed inorganic) mercury (1.9 mg/kg). If it were assumed that the methylmercury concentration is 1 percent, rather than about 0.1 percent, of the total mercury value, the methylmercury HQ would not change enough to affect the overall non-cancer hazard to receptors exposed to northern basin sediments. The methylmercury HQ for the most affected receptor – the young child – would increase from about 3×10^{-5} to 3×10^{-4} ; the difference would not be noticeable in the total HI of 0.221.

Wetlands Sediment

For the four wetlands evaluated in this HHRA, the total mercury concentration (which was the only analysis conducted) was assumed to be 1 percent methylmercury (and 99 percent inorganic mercury); this is the same assumption made for the BERA. Although no wetland samples were analyzed for methylmercury for the Onondaga Lake HHRA, 29 samples from the nearby LCP Bridge Street site were analyzed for mercury and methylmercury. These LCP Bridge Street site samples include some from regulated Wetland SYW-14 and other samples from near-surface wet or moist soils which are the best available data for estimating methylmercury concentrations in the Onondaga Lake wetlands. The fraction of methylmercury in these 29 samples ranged from 0.003 percent to 2.2 percent (this high value was from a sample described as “lined ditch” and may not be comparable to the wetland sediments), and averaged about 0.25 percent (see BERA Chapter 6, Table 6-3 [TAMS, 2002a]), supporting the assumption that the mercury in the wetlands is principally in the inorganic form.

As discussed in Chapter 6, Section 6.3.1.1, of the Onondaga Lake BERA (TAMS, 2002a), submitted concurrently with this HHRA, the BERA includes an assumption that 1 percent of the mercury in wetland sediments is organic (i.e., methylmercury). This assumption was applied to the wetlands sediments only, as there are specific mercury and methylmercury data for the lake sediments, and the methylation of inorganic to organic mercury was assumed to be negligible in other media such as the dredge spoils area soils. The RfD for methylmercury is three times higher than the RfD used for inorganic mercury; therefore, if 1 percent methylmercury was assumed for the wetlands sediments, mercury-related non-cancer toxicity would increase by a factor of 1.02 (2 percent).

A US Geological Survey (USGS) survey cited in the BERA (Krabbenhoft et al., 1999) indicates that methylation rates ranging from about 1 to 10 percent of total mercury have been detected in sediments of aquatic ecosystems (e.g., from streams and/or wetlands sediments in mixed agricultural/forest areas, abandoned mines, urban areas, etc.) in the US. Although the conditions favorable to a rate this high (e.g., low pH, anaerobic environment) are not considered to be present at Onondaga Lake (see BERA Chapter 6, Section 6.3.1.1), the potential for increased risk from a higher fraction of organic mercury in the wetland sediments was evaluated, as there are no methylmercury data from the four wetland areas included in this HHRA. The construction worker RME exposure to Wetland SYW-19 sediment has the highest HQ associated with exposure to mercury (0.046, the sum of the HQs for inorganic mercury and methylmercury); as shown in Appendix B, RAGS Table 7.23); therefore, this scenario would be most impacted by changes in the assumed form of mercury to which the receptor is exposed.

If the fraction of methylmercury in the Wetland SYW-19 sediments were assumed to be 10 percent instead of 1 percent, the HQ from mercury exposure would increase from 0.046 to about 0.055, and the total HI would increase only slightly, from 0.156 to 0.165. Therefore, even for the most exposed receptor, changes in the assumption of the form of mercury present do not significantly change overall hazard estimates. (If, on the other hand, the actual fraction of methylmercury is zero, then the HQ and HI would both show an insignificant decrease of only 0.001.) It is noted that Wetland SYW-19 is now part of the Wastedbed B/Harbor Brook site and additional sampling will be conducted in this wetland during the RI, including analysis of methylmercury in addition to other contaminants. Risks will be recalculated in the Wastedbed B/Harbor Brook HHRA based on these new data.

Dredge Spoils Area

For the dredge spoils area, the total mercury (which was the only analysis conducted) was assumed to be inorganic mercury, which is the same assumption made for the BERA. Although there are no site-specific data from a matrix similar to the dredge spoils, the dredge spoil samples were collected above the water table in an oxic environment that is not ordinarily favorable to the methylation process. There are insufficient data in the literature to estimate the percent of methylmercury that may be formed under these conditions, but methylation rates are thought to be substantially lower than in wetlands.

In order to assess the potential impact of methylmercury on the calculated hazards from dredge spoils, the potential hazard to the most impacted receptor (construction worker RME to surface and subsurface dredge spoils) was evaluated using the high-end assumption of 10 percent methylmercury. If 10 percent of the total mercury is assumed to be methylmercury (the remaining 90 percent is inorganic), then the total mercury-related hazard (the sum of the HQs from inorganic mercury and methylmercury) would increase from 0.052 to 0.062, and the total RME HI for the construction worker would increase from 0.126 to 0.136. Therefore, the RME hazard would not change significantly, even under high-end or worst-case assumptions of methylmercury concentration.

Surface Water

There were more extensive data on mercury speciation in the surface water data set used in this HHRA. Of the 75 surface water samples analyzed for total mercury, 32 of the samples were also analyzed for methylmercury, dimethylmercury, and ionic mercury. There were sufficient data to enable the methylmercury in surface water to be evaluated separately from the "total" (interpreted as inorganic) mercury in surface water. Dimethylmercury was not detected in any of the 32 samples in which it was analyzed. Ionic mercury was detected in all 32 samples, although at lower concentrations (ranging from 0.15 to 3.3 ng/L) than either total mercury or methylmercury. Ionic mercury data, where available, were screened separately as inorganic mercury; due to the low concentrations of ionic mercury detected, ionic mercury was screened out (excluded from the quantitative HHRA). Due to the low concentrations detected, the treatment of the ionic mercury has little effect on the results of this HHRA.

7.5.3.2 Arsenic

In the HHRA, it was assumed that 10 percent of the arsenic detected in fish tissue was inorganic arsenic, which is the more toxic form. It is generally understood that most of the arsenic in fish is in the relatively non-toxic organic form called arsenobetain (Oregon Health Department and Washington [State] Department of Health [OHD/WDOH], 1996). Inorganic arsenic, which is considerably more toxic, makes up a small amount of the total arsenic in fish and is most relevant for assessing health impacts from consumption of contaminated fish. However, there are no adequate data or other bases on which to quantify the fraction of arsenic in the inorganic form in Onondaga Lake fish fillets.

In an attempt to quantify or estimate the fraction of inorganic arsenic in fish, staff from the OHD and WDOH were contacted, based on their involvement in the risk assessment for the Columbia River in Oregon and Washington, where arsenic was a contaminant of concern. The consensus was that there is not a good means of making an estimate, as the fraction of arsenic may vary significantly due to a variety of factors, including the fish species, the water body, and characteristics of the individual fish analyzed (K. Kauffman, pers. comm., 2002; J. White, pers. comm., 2002).

Studies show an organic arsenic range of 1 to 25 or 30 percent of the total arsenic (K. Kauffman, pers. comm., 2002), although default assumptions of 1 or 10 percent are typically used (J. White, pers. comm., 2002). Subsequent data obtained by TAMS from USEPA Region 10 (R. Lorenzana, pers. comm., 2002; Tetra Tech, 1996; EVS, 2000) confirmed this range. These data are tabulated on Tables 7-6A and 7-6B and show a range from none detected to about 26 percent inorganic arsenic in the 42 freshwater fish samples in which arsenic was detected, with an arithmetic mean of about 6.4 percent, with an upper-bound 95th percentile of 10.7 percent (calculated using the non-parametric Chebyshev test in the USEPA ProUCL software). These data are useful to support the general observation that only a relatively small fraction of the total arsenic in fish is in the toxic inorganic form.

Although it is difficult to use the West Coast data to generate a quantitative estimate for use in the Onondaga Lake HHRA due to differences in location and water body (river versus lake) and species (the West Coast data are, for the most part from species not present in Onondaga Lake – e.g., salmon, sturgeon), they support the use of the default assumption of 10 percent of the total arsenic detected in fish samples in the inorganic form. While not enough data were obtained to perform a rigorous analysis, inspection of the Columbia River data suggests that the fraction of arsenic existing in the inorganic form may also be species-dependent.

The effect of this uncertainty is not great in terms of affecting the overall risk or hazard estimates for the fish consumption pathway. With the assumption of 10 percent inorganic arsenic, arsenic contributes slightly less than 2 percent of the adult RME cancer risk (1.8×10^{-5} , out of a total ingestion pathway risk of 7.8×10^{-4}). For a worst-case assumption of 100 percent inorganic arsenic, the total RME cancer risk would increase to about 9.4×10^{-4} . On the other hand, the RME cancer risk from arsenic would be reduced to about 7.6×10^{-4} , based on inorganic arsenic percentages of 1 percent. The contribution of arsenic to the non-cancer hazard associated with fish ingestion is lower, with arsenic contributing about 0.5 percent of the adult RME

total non-cancer ingestion hazard (contributing an HQ of about 0.1 to a total adult RME HI of about 18). The total non-cancer adult RME hazard associated with fish ingestion would increase slightly (from 18.2 to 19) if the worst-case assumption of 100 percent inorganic arsenic were made, and decrease slightly (from 18.2 to 18.1) if only 1 percent of the total arsenic were assumed to be inorganic.

7.5.3.3 Chromium

Chromium typically exists in two valence states in the environment: Cr(III) (Cr^{+3} , or trivalent chromium), and Cr(VI) (Cr^{+6} , or hexavalent chromium). Normally the bulk of the chromium is in the less-toxic trivalent form. For example, the USEPA Region 9 soil-screening concentrations are based on the assumption of a six-to-one ratio of Cr(III) to Cr(VI).

As the available data for chromium were reported as "total chromium" (no speciation between Cr[III] and Cr[VI] was performed), this HHRA used the conservative assumption that all chromium detected was in the form of the more-toxic hexavalent form. While chromium did contribute a significant fraction of the non-cancer hazard in Wetlands SYW-6 and SYW-12 and in surface water, the total HI for all receptors in each of these media was very low, as summarized on Table 6-5. Therefore, although the hazard associated with chromium may be overestimated, it does not have a material impact on the conclusions of this HHRA.

7.5.3.4 Polychlorinated Biphenyls

As discussed previously, data for PCBs were reported as Aroclors, with the particular suite of Aroclors reported varying depending on the medium, the sampling organization and analytical laboratory, and when the samples were analyzed. These factors do not contribute materially to the calculation of PCB EPCs or the assignment of appropriate toxicity factors (RfDs or CSFs).

One area of uncertainty regarding PCBs is their potential for dioxin-like toxicity. As with many of the 2,3,7,8-substituted PCDD/PCDF compounds, certain PCB congeners have been found to have, or are assumed to have, dioxin-like toxicity. TEFs have been developed to estimate the dioxin-like toxicity. However, as there were no analyses of PCB congeners conducted on Onondaga Lake samples, no estimate of the dioxin-like toxicity of PCBs is presented in this HHRA.

8. CONCLUSIONS

The objective of this HHRA was to evaluate the potential for adverse human health effects associated with current and future exposures to chemicals present in Onondaga Lake surface water, fish, certain nearshore sediments, wetlands sediments, and dredge spoils soils in the absence of any action to control or mitigate those chemicals. Under this “no remedial action” assumption, the HHRA focused on lake conditions, assuming its unrestricted recreational use and the absence of a specific, restrictive fish consumption advisory.

8.1 Exposure Pathways

The potential exposure pathways evaluated, and the rationale for their inclusion or exclusion, are summarized in Appendix B, RAGS Table 1. In addition to fish ingestion, other potential pathways considered to be complete and evaluated quantitatively in this HHRA included:

- Dermal exposure to and ingestion of sediments from nearshore sediments.
- Dermal exposure to and ingestion of sediments from four wetland areas.
- Dermal exposure to and ingestion of soils from the dredge spoils area adjacent to the lake.
- Dermal contact with and ingestion of lake surface water.

The likely exposure scenarios were based on recreational use (which includes trespassers on, for example, Wetland SYW-19 on Honeywell property) and relatively short-term construction projects such as utility work to assess potential construction worker exposure. Residential and industrial/commercial exposures were not evaluated, based on current and reasonably foreseeable future site uses. Similarly, risks associated with the ingestion of groundwater or surface water as a potable water source were not addressed. As noted in Chapter 4, Section 4.2.5, an initial preliminary site assessment (PSA) conducted for Onondaga Lake by NYSDEC (NYSDEC, 1989a, as cited in PTI, 1991) concluded that there was little potential for releases of contaminants to air. The data for volatile organic compounds (VOCs) in surface water and near-surface soils were reviewed as part of this HHRA, and the initial conclusion by NYSDEC is considered to still be appropriate for recreational users and nearby residents.

In addition, there are currently no structures on the site nor are any likely to be built, due to regulatory restrictions (e.g., zoning and wetlands) and the nature of the area (e.g., much of the lake shoreline area is owned by or under the jurisdiction of the Onondaga County Parks Department [OCPD], and the wetlands areas are generally unsuitable for construction, even absent regulatory restrictions). Therefore, the inhalation pathway was considered to be incomplete for all media and was not assessed further in this report.

8.2 Contaminant Screening and Identification of Contaminants of Potential Concern

Contaminants of potential concern (COPCs) were identified after screening all contaminants detected in site media against risk-based screening criteria. This process is discussed in Chapter 3 and presented in Appendix B, RAGS Tables 2.1 through 2.10. As a result of this screening, a total of about 60 COPCs or groups of COPCs were retained for further analysis in the HHRA.

After the screening of COPCs, exposure point concentrations (EPCs) were calculated for each COPC in each medium. The EPC is a statistically derived number representing a value that has a 95 percent likelihood of not being less than the “true” arithmetic average for that medium (the 95 percent upper confidence limit [UCL] on the mean). Where the statistically calculated UCL exceeds the maximum concentration actually detected in that medium, or where there are less than ten data points (a data set that is assumed to be the minimum for which statistical calculations are valid), the maximum detected concentration is used as the EPC. The EPC calculations are presented in Appendix B, RAGS Tables 3.1 through 3.10.

In order to assess exposure to COPCs, which is referred to as “intake” or “dermally absorbed dose” in risk assessment terminology, other assumptions must be made about human behavior, such as how much recreationally caught fish an individual might eat, how often an individual might be exposed to contaminated sediments, and similar factors. Consistent with USEPA guidance, reasonable maximum exposure (RME) and central tendency (CT; sometimes referred to as “typical”) exposure scenarios were evaluated for a future fish consumption pathway, assuming an RME consumption rate of 25 grams per day (approximately 40 eight-ounce meals of fish per year) from the lake. The various assumptions used in calculating the intake (exposure) of various receptors (recreational users and construction workers) are presented in Appendix B, RAGS Tables 4.1 through 4.31. As some of the values used are assumptions or based on professional judgment, there is a degree of uncertainty associated with such values. A qualitative discussion of the relative uncertainty of many of these values is presented in Chapter 7, Uncertainty Assessment, along with a discussion of the potential effect of such uncertainty on the quantitative results of the HHRA.

8.3 Toxicity Assessment

USEPA sources were researched to obtain toxicity data for the approximately 60 COPCs and the 31 identified exposure pathways in Onondaga Lake. Toxicity data (reference doses [RfDs] for non-cancer effects, and cancer slope factors [CSFs] for carcinogens) used in the calculations are discussed in Chapter 5, Toxicity Assessment, and presented in Appendix B, RAGS Tables 5 and 6 for the chemicals assessed quantitatively. Most of the toxicity data used in this quantitative HHRA are from USEPA’s peer-reviewed database, the Integrated Risk Information System (IRIS). All of the other toxicity data are from USEPA sources (e.g., Health Effects Assessment Summary Tables [HEAST], National Center for Environmental Assessment [NCEA]) and have been reviewed and approved by USEPA for use in this risk assessment.

8.4 Risk Characterization

The results of the risk calculations are presented in Chapter 6, Risk Characterization and Appendix B, RAGS Tables 7 through 10. The highest cancer risks and non-cancer hazards are associated with the ingestion of fish from Onondaga Lake. Cancer risks and non-cancer hazards were also calculated for the sediment, dredge spoil, and surface water pathways. Total cancer risks and non-cancer hazards were also calculated for each receptor; e.g., the cumulative (total) risk or hazard from exposure to multiple contaminated media (i.e., fish, sediments, dredge spoils, and surface water).

8.4.1 Fish Ingestion

RME cancer risk estimates for recreationally caught fish ingestion by adults and children exceeded the upper end of USEPA's acceptable risk range (i.e., RME risk estimates exceeded 10^{-4} , ranging from 2.4×10^{-4} to 7.8×10^{-4}), primarily due to total polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs), and, to a lesser extent, arsenic. CT cancer risks were about 4.5×10^{-5} for all recreational receptors.

For non-cancer hazards, the RME hazard index (HI) was calculated to be 18 for adults and 28 and 20 for young and older children, respectively. Non-cancer health hazards are primarily due to PCBs and methylmercury, while total CT HIs ranged from 4.5 for adults to 7.0 for young children for the fish consumption pathway.

Fish ingestion was not explicitly evaluated for the construction worker.

8.4.2 Sediment/Soil Pathways

Seven sediment and soil pathways were evaluated, including northern and southern basin sediments; sediments in the four wetlands areas; and the dredge spoils area soils.

None of the RME and CT HIs for soil or sediment pathways for the receptors evaluated (adults, young children, and older children) exceeded the target level of 1.0. The highest HI was about 0.5, for young child exposure to southern basin sediments.

RME cancer risks exceeded 10^{-4} for Wetland SYW-6 for the older child recreational scenario. RME risks did not exceed 10^{-4} for any of the other sediment or soil pathways; however, cancer risks exceeded 10^{-6} for at least two receptors in each of the seven sediment/soil media.

None of the CT cancer risks associated with the sediment and soil exceeded 10^{-4} . The CT risk for the older child exposure to Wetland SYW-6 sediments was the highest CT risk at 1.4×10^{-5} , and adult exposure to Wetland SYW-6 sediments was the second highest CT risk at 7.1×10^{-6} . CT risks to one or two receptors also exceeded 10^{-6} for the southern basin sediments, Wetland SYW-10, and Wetland SYW-19.

None of the CT risks associated with exposure to northern basin sediments, Wetland SYW-12 sediments, or dredge spoils area soils exceeded 10^{-6} .

8.4.3 Surface Water Pathway

No unacceptable cancer risks or non-cancer hazards were associated with exposure to COPCs in Onondaga Lake surface water under the scenarios evaluated in this HHRA. CT and RME HIs for all receptors were less than 0.04, and all RME and CT cancer risks were less than 10^{-7} for all receptors.

8.4.4 Receptor-Specific Risks and Hazards Across Pathways

As evident from the pathway-specific discussion above (Sections 8.4.1 through 8.4.3), the highest site risks and hazards are from the fish ingestion pathway. The fish ingestion pathway represents more than 95 percent of the RME non-cancer hazard for recreational receptors, and between 47 and 88 percent of the RME cancer risk to recreational receptors. For example, the RME fish ingestion hazard for the young child recreator is 28.3, and the total hazard across all pathways (fish ingestion and exposure to COPCs in sediment and surface water) is 29.1 (see Chapter 6, Table 6-2). The receptor-specific cumulative risk summary tables include the risks and hazards associated with just the sediment, soil, and surface water pathways.

The total HI for the older child recreators for all pathways other than fish ingestion adds up to 0.98 for the RME scenario, although, as noted in Chapter 7, this may overestimate the older child's exposure, as it may be unrealistic to assume that an older child is exposed to all possible media at the RME frequency.

RME cancer risks for pathways other than fish ingestion were generally below 10^{-4} ; however, the total RME risks for the older child recreator (assuming RME exposure to all possible media) were about 3.8×10^{-4} without fish ingestion and 7.2×10^{-4} with fish ingestion. RME total cancer risks for the other receptors (for pathways other than fish ingestion) were in the range of 2×10^{-5} to 1×10^{-4} , with many of the pathway-specific receptor risks exceeding 10^{-6} .

8.5 Uncertainty Assessment

USEPA's risk assessment methods are designed to be protective of human health. Thus, when the uncertainties associated with use of these methods are accounted for, "true" site risks for most receptors are likely to be less than the RME risks presented for this HHRA. However, as indicated in the uncertainty assessment (Chapter 7), many of the aspects of the exposure assumptions applied here are based on professional judgment, default values, or estimates; therefore, the actual risks to any particular individual could be higher or lower than those presented in this HHRA.

Chapter 7, Uncertainty Assessment, provides a discussion of alternative values that were considered, but not applied, for this HHRA. For example, several studies (or extrapolation of data from them) suggest that the fish consumption rate of young children is higher, on a body weight-normalized basis, than that of adults.

For this HHRA, it was assumed that older children (age six to 18) consumed two-thirds as much recreationally caught fish as adults, and that young children (under age six) consumed one-third as much as adults. However, there are limited quantitative data from which develop a child-specific ingestion rate; therefore, the risks/hazards to children could be higher or lower than those presented in this HHRA to the extent that children consume Onondaga Lake fish at rates higher or lower than those assumed. Similarly, the potential susceptibility of children to developmental and other effects that may not have been explicitly assessed in the development of toxicity data used to quantify risk may also result in children being at greater risk than the quantitative estimates presented in this report.

For non-cancer hazards related to systemic toxicants such as methylmercury, the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) states that “acceptable exposure levels shall represent concentration levels to which the human population . . . may be exposed without adverse effect” [40 CFR 300.430 (e)(2)]. The non-cancer HQ is based on the assumption that there is a level of exposure to a COPC (i.e., RfD) below which it is unlikely that even sensitive populations will experience adverse health effects. If the HQ exceeds unity (1.0), there is the potential for non-cancer effects, and the greater the value is above unity, the greater the level of concern. However, the HQ (or sum of HQs, or HI) is not a statistical probability (e.g., an HQ or HI of 0.1 does not mean there is a one-in-ten chance of adverse health effects). Similarly, the level of concern does not increase linearly as the RfD is approached or exceeded.

Other uncertainties discussed in Chapter 7, most of which could result in either an overestimate or underestimate of risks and hazards, include:

- Adequacy of the data – discusses the quality, nature, and quantity of the data (numbers of samples and contaminants analyzed) for characterization of the exposure media
- Calculation of EPCs – discusses the mathematical and statistical procedures utilized for estimate COPC concentrations to which individuals (receptors) may be exposed, and what effect different ways of processing the data may change the calculations of risk and hazard.
- Exposure assumptions – the other exposure assumptions (e.g., frequency of exposure, duration of exposure, sediment and surface water ingestion rates, etc.) are discussed with regard to the extent to which they are both reasonable and protective of human health.
- Toxicity data – discusses the availability and reliability of the toxicity values used for calculating risks and hazards for all the major COPCs identified at the site.

8.6 Summary of Risks and Hazards Exceeding Target Levels

For cancer risks, the target risk levels range from an upper bound of risks to an individual of 1×10^{-4} to 1×10^{-6} , with 10^{-6} as the point of departure for determination of remedial goals (as specified in the NCP, 40 § CFR 300.430[e][2][A][2]). For non-cancer hazards, the target hazard level is an HQ of 1.0 or less, the level below which adverse health effects are considered to be unlikely.

Table 8-1 provides a list of all 31 complete pathways and the risks and hazard levels exceeded for each pathway. Cancer risk exceedances are noted on the table for three risk levels of 10^{-4} , 10^{-5} , and 10^{-6} . Table 8-2 provides a summary of receptor-cumulative cancer risk or non-cancer hazard exceedances for each of the four receptor populations (adults, young children, older children, and construction workers). As the cancer risk and non-cancer hazards associated with fish ingestion are so high as to mask the risks and hazards associated with other contaminated site media (sediments, dredge spoils soils, and surface water), the receptor risks and hazards are shown both including and excluding the fish ingestion pathway.

Cancer risks and non-cancer hazards calculated for the RME scenario for consumption of Onondaga Lake fish exceeded the upper end of the target risk levels (see Executive Summary, Table ES-6). For the RME scenario, the calculated cancer risk to adults and children (ranging from 2.4×10^{-4} to 7.8×10^{-4}) exceeded the high end of the target risk range (10^{-4}), and exceeded the low end of the target cancer risk (10^{-6}) by more than two orders-of-magnitude. The non-cancer HIs (ranging from 18 to 28) exceeded the target RME non-cancer HI (1.0) by a factor of almost 20 or more. The calculated CT non-cancer HIs (4.5 to 7 for adults and children) also exceeded the target, although the CT fish ingestion cancer risk (about 4.5×10^{-5} for all recreational receptors) was below the upper end of the target range. RME cancer risks for 21 of the 28 pathways other than fish ingestion equaled or exceeded the low end of the target risk range of 1×10^{-6} , with the highest of these being about 2.6×10^{-4} for older child exposure to Wetland SYW-6 sediments. For the CT cancer risk calculations, the low end of the 10^{-6} target range was equaled or exceeded in eight of the 28 pathways other than fish ingestion, with a maximum CT risk of about 1.4×10^{-5} for older child exposure to Wetland SYW-6 sediments.

Cumulative risks and hazards were calculated for receptors who may be exposed to COPCs in multiple site media – for example, eating contaminated fish and being exposed to contaminated sediments. The receptors evaluated were adult recreators, young child recreators, older child recreators, and construction workers. For all cumulative risk and hazard calculations including fish ingestion, the cumulative risk or hazard was essentially the same as that associated with the fish ingestion pathway alone. Therefore, to assess the cumulative risks associated with pathways other than fish ingestion (i.e., exposure to lake sediment, wetlands sediment, dredge spoils soil, and lake surface water), the cumulative risk for each receptor was also calculated excluding the fish ingestion pathway.

Cumulative RME cancer risks for adults (excluding fish ingestion) were calculated as 1×10^{-4} ; about 3.5×10^{-5} for younger children; about 3.8×10^{-4} for older children; and 2×10^{-5} for construction workers. It should be noted that these estimates may be more conservative than under typical exposure scenarios, especially for the adult and older child recreational receptors, as the cumulative risk calculation assumes

RME frequencies to each of the exposure media (i.e., sediments, wetlands, and dredge spoils soils). Cumulative RME HIs calculated in the same manner (excluding fish ingestion) generally did not exceed 1.0, although some approached 1.0; i.e, the calculated value is 0.98 for the older child recreator and 0.79 for the young child, and 0.83 for the construction worker.

9. REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological profile for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. PB/89/214522/AS. US Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1992. Public Health Statement for Antimony.

ATSDR. 1993a. 'ToxFAQ®' for Di(ethylhexyl)phthalate.

ATSDR. 1993b. 'ToxFAQ®' for Aldrin and Dieldrin.

ATSDR. 1993c. 'ToxFAQ®' for Heptachlor/Heptachlor Epoxide.

ATSDR. 1994. Toxicological profile for mercury (update). US Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1995a. 'ToxFAQ®' for Antimony.

ATSDR. 1995b. 'ToxFAQ®' for Barium.

ATSDR. 1995c. 'ToxFAQ®' for Thallium.

ATSDR. 1995d. 'ToxFAQ®' for Vanadium.

ATSDR. 1995e. 'ToxFAQ®' for Zinc.

ATSDR. 1995f. 'ToxFAQ®' for Chlordane.

ATSDR. 1995g. 'ToxFAQ®' for DDT, DDE, and DDD.

ATSDR. 1995h. 'ToxFAQ®' for Chlorodibenzofurans (CDFs).

ATSDR. 1996a. 'ToxFAQ®' for Xylene.

ATSDR. 1996b. 'ToxFAQ®' for Polycyclic Aromatic Hydrocarbons.

ATSDR. 1996c. 'ToxFAQ®' for Naphthalene.

ATSDR. 1997. Toxicological profile for polychlorinated biphenyls – update. PB98-101173. Agency for Toxic Substances and Disease Registry, Atlanta, Georgia, and US Department of Health and Human Services, Public Health Service.

ATSDR. 1997a. 'ToxFAQ®' for Nickel.

ATSDR. 1997b. 'ToxFAQ®' for Selenium.

ATSDR. 1997c. 'ToxFAQ®' for Benzene.

ATSDR. 1997d. 'ToxFAQ®' for Hexachlorobenzene.

ATSDR. 1997e. 'ToxFAQ®' for Chloroform.

ATSDR. 1999. 'ToxFAQ®' for Mercury.

ATSDR. 1999a. 'ToxFAQ®' for Aluminum.

ATSDR. 1999b. Public Health Statement for Aluminum.

ATSDR. 1999c. 'ToxFAQ®' for Cadmium.

ATSDR. 1999d. 'ToxFAQ®' for Copper.

ATSDR. 1999e. 'ToxFAQ®' for Lead.

ATSDR. 1999f. 'ToxFAQ®' for Bromodichloromethane.

ATSDR. 1999g. 'ToxFAQ®' for Chlorobenzene.

ATSDR. 1999h. 'ToxFAQ®' for Chloroform.

ATSDR. 1999i. 'ToxFAQ®' for 1,4-Dichlorobenzene.

ATSDR. 1999j. 'ToxFAQ®' for Hexachlorocyclohexanes.

ATSDR. 1999k. 'ToxFAQ®' for Chlorinated Dibenzo-p-Dioxins (CDDs).

ATSDR. 2000. Minimal risk level for methylmercury (final). Agency for Toxic Substances and Disease Registry website.

ATSDR. 2001a. 'ToxFAQ®' for Arsenic.

ATSDR. 2001b. 'ToxFAQ®' for Chromium.

ATSDR. 2001c. 'ToxFAQ®' for Manganese.

ATSDR. 2001d. 'ToxFAQ®' for Methylene Chloride.

ATSDR. 2001e. 'ToxFAQ®' for Polychlorinated Biphenyls (PCBs).

Blasland & Bouck. 1989. Hydrogeologic assessment of the Allied waste beds in the Syracuse area. Volume 1 of 2. Prepared for AlliedSignal Inc., Syracuse, NY. Blasland & Bouck Engineers, P.C., Syracuse, NY.

Calabrese, E.J., R. Barnes, E.J. Stanek, H. Pastides, C.E. Gilbert, P. Veneman, X.R. Wang, A. Lasztity, and P.T. Kostecki. 1989. How much soil do young children ingest: An epidemiologic study. *Regul. Toxicol. Pharmacol.* (2):123–137.

California EPA, Office of Environmental Health Hazard Assessment (OEHHA). 2001. Chemicals in Fish: Consumption of Fish and Shellfish in California and the United States. October.

Connelly, N.A., T.L. Brown, and B.A. Knuth. 1990. New York statewide angler survey, 1988. New York State Department of Environmental Conservation, Albany, NY.

Connelly, N.A., B.A. Knuth, and C.A. Bisogni. 1992. Effects of the health advisory and advisory changes on fishing habits and fish consumption in New York sport fisheries. Report for the New York Sea Grant Institute Project No. R/FHD-2-PD. Human Dimensions Research Unit, Cornell University, Ithaca, NY.

Connelly, N.A., B.A. Knuth, and T.L. Brown. 1996. Sportfish consumption patterns of Lake Ontario anglers and the relationship to health advisories. *N. Am. J. Fish. Manage.* 16:90–101.

Eallonardo, John, Director of Planning, Onondaga County Parks Department (OCPD). 2002. Personal communication via telephone with A. Burton, TAMS Consultants, Inc. October 1.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. *N. Am. J. Fish. Manage.* 13:737–745.

Effler, S.W. and C.T. Driscoll. 1986. A chloride budget for Onondaga Lake, New York, USA. *Water Air Soil Pollut.* 27:29–44, as cited in OLMC, 1989.

Effler, S.W. (ed). 1996. *Limnological and engineering analysis of a polluted urban lake.* Springer-Verlag, New York, NY.

EVS Environment Consultants, Inc. 2000. Prepared for Oregon Department of Environmental Quality, Portland, OR. Human Health Risk Assessment of Chemical Contaminants in Four Fish Species from the Middle Willamette River, Oregon. November.

Exponent. 2000. Onondaga Lake Remedial Investigation/Feasibility Study. Supplemental Data Phase 2A Work Plan and Dredged Material Disposal Area Addendum. Prepared for Honeywell, East Syracuse, NY. Exponent, Bellevue, WA. June.

Exponent. 2001a. Onondaga Lake Remedial Investigation/Feasibility Study. Revised Draft Human Health Risk Assessment. Prepared for Honeywell, East Syracuse, NY. Exponent, Bellevue, WA. March.

Exponent. 2001b. Geddes Brook/Ninemile Creek Remedial Investigation/Feasibility Study. Revised Draft Human Health Risk Assessment. Under Revision. Prepared for Honeywell, East Syracuse, NY. Exponent, Bellevue, WA. November.

Game and Fish Magazine. 2002. Web site <http://gameandfish.about.com/library/weekly/aa073601b.htm>. Accessed May and June.

Geraghty & Miller. 1982. Ground-water quality conditions at the former Willis Avenue plant. Geraghty & Miller, Inc., Syosset, NY.

Gilbert, R.O. 1987. Statistical Methods for Environmental Pollution Monitoring. Van Nostrand Reinhold. New York.

Great Lakes Sport Fish Advisory Task Force (GLSFATF). 1993. Protocol for a Uniform Great Lakes Sport Fish Advisory. September.

Hawley, Gessner G. (ed.). 1981. The Condensed Chemical Dictionary, Tenth Edition. Van Nostrand Reinhold Company, New York

Integrated Risk Information System (IRIS). USEPA. 1997a. IRIS substance file for Free Cyanide.

IRIS. 1997b. IRIS substance file for Calcium Cyanide.

IRIS. 1997c. IRIS substance file for Sodium Cyanide.

IRIS. 1997d. IRIS substance file for Lead.

IRIS. 1997e. IRIS substance file for Manganese.

IRIS. 1997f. IRIS substance file for Mercury – Elemental.

IRIS. 1997g. IRIS substance file for Mercuric Chloride.

IRIS. 1997h. IRIS substance file for Selenium.

IRIS. 1997i. IRIS substance file for Selenium Sulfide.

IRIS. 1997j. IRIS substance file for Thallium Chloride.

IRIS. 1997k. IRIS substance file for Thallium Sulfate.

IRIS. 1997l. IRIS substance file for Thallium Carbonate.

IRIS. 1997m. IRIS substance file for Vanadium Pentoxide.

IRIS. 1997n. IRIS substance file for Bromodichloromethane.

IRIS. 1997o. IRIS substance file for Chlorobenzene.

IRIS. 1997p. IRIS substance file for Dichloromethane.

IRIS. 1997q. IRIS substance file for Hexachlorobenzene.

IRIS. 1997r. IRIS substance file for 1,2,4-Trichlorobenzene.

IRIS. 1997s. IRIS substance file for Benz[a]anthracene.

IRIS. 1997t. IRIS substance file for Benzo[b]fluoranthene.

IRIS. 1997u. IRIS substance file for Benzo[k]fluoranthene.

IRIS. 1997v. IRIS substance file for Chrysene.

IRIS. 1997w. IRIS substance file for Dibenz[a,h]anthracene.

IRIS. 1997x. IRIS substance file for Indeno[1,2,3-cd]pyrene.

IRIS. 1997y. IRIS substance file for Acenaphthylene.

IRIS. 1997z. IRIS substance file for Benzo[g,h,i]perylene.

IRIS. 1997aa. IRIS substance file for Fluoranthene.

IRIS. 1997bb. IRIS substance file for Phenanthrene.

IRIS. 1997cc. IRIS substance file for Aldrin.

IRIS. 1997dd. IRIS substance file for delta-Hexachlorocyclohexane (delta-HCH).

IRIS. 1997ee. IRIS substance file for p,p'-Dichlorodiphenyl Dichloroethane (DDD).

IRIS. 1997ff. IRIS substance file for p,p'-Dichlorodiphenyldichloroethylene (DDE).

IRIS. 1997gg. IRIS substance file for p,p'-Dichlorodiphenyltrichloroethane (DDT).

IRIS. 1997hh. IRIS substance file for Dieldrin.

IRIS. 1997ii. IRIS substance file for Heptachlor Epoxide.

IRIS. 1997jj. IRIS substance file for Aroclor 1016.

IRIS. 1997kk. IRIS substance file for Aroclor 1248.

IRIS. 1997ll. IRIS substance file for Aroclor 1254.

IRIS. 1997mm. IRIS substance file for alpha-Hexachlorocyclohexane.

IRIS. 1997nn. IRIS substance file for beta-Hexachlorocyclohexane.

IRIS. 1997oo. IRIS substance file for epsilon-Hexachlorocyclohexane.

IRIS. 1997pp. IRIS substance file for technical Hexachlorocyclohexane.

IRIS. 1997qq. IRIS substance file for Pyrene.

IRIS. 1998a. IRIS substance file for Cadmium.

IRIS. 1998b. IRIS substance file for Chromium(III).

IRIS. 1998c. IRIS substance file for Chromium(VI).

IRIS. 1998d. IRIS substance file for Copper.

IRIS. 1998e. IRIS substance file for Nickel (Soluble Salts).

IRIS. 1998f. IRIS substance file for Zinc.

IRIS. 1998g. IRIS substance file for Xylene.

IRIS. 1998h. IRIS substance file for Di(ethylhexyl)phthalate.

IRIS. 1998i. IRIS substance file for Benzo[a]pyrene.

IRIS. 1998j. IRIS substance file for Chlordane.

IRIS. 1998k. IRIS substance file for Polychlorinated biphenyls (PCBs).

IRIS. 1999. IRIS substance file for Barium.

IRIS. 2000a. IRIS substance file for Antimony.

IRIS. 2000b. IRIS substance file for Arsenic.

IRIS. 2000c. IRIS substance file for Dibenzofuran.

IRIS. 2000d. IRIS substance file for 1,2-Dichlorobenzene.

IRIS. 2000e. IRIS substance file for 1,3-Dichlorobenzene.

IRIS. 2000f. IRIS substance file for 1,4-Dichlorobenzene.

IRIS. 2001a. IRIS substance file for Methyl Mercury.

IRIS. 2001b. IRIS substance file for Benzene.

IRIS. 2002a. IRIS substance file for Chloroform.

IRIS. 2002b. IRIS substance file for Naphthalene.

IRIS. 2002c. IRIS substance file for gamma-Hexachlorocyclohexane (gamma-HCH).

Javitz, H. 1980. Seafood consumption data analysis. SRI International. Final report prepared for USEPA Office of Water Regulations and Standards. EPA Contract 68-01-3887.

Kauffman, Ken, Oregon Health Division (OHD). 2002. Personal communication via telephone with A. Burton, TAMS Consultants, Inc., July 24.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. Toxicol. Appl. Pharmacol. 46:279-303.

Krabbenhoft, D.P., J.G. Wiener, W.G. Brumbaugh, M.L. Olson, J.F. DeWild, and T. J. Sabin. 1999. A National Pilot Study of Mercury Contamination of Aquatic Ecosystems along Multiple Gradients. USGS Toxic Substances Hydrology Program. Proceedings of the Technical Meetings. Charleston, SC. Water Res. Invest. Report. 99-4018 B. pp. 147-160.

Lakefront Development Corporation. 2001. Sidewalk Talk: The Syracuse Lakefront: A Sidewalk Community. Syracuse, New York. Fall.

Lorenzana, Roseanne, Ph.D., Science Liaison, USEPA Region 10. 2002. Personal communication with A. Burton, TAMS Consultants, Inc., August 5.

Marsh, D.O., T.W. Clarkson, G. Myers, C. Cox, E. Cernichiari, M.A. Tanner, W. Lednar, C. Shamlaye, O. Choisy, C. Horareau, and M. Berlin. 1995. The Seychelles study of fetal methylmercury exposure and child development: Introduction. *Neurotoxicology* 16:583-596.

Martin, Karen L., USEPA, Office of Solid Waste and Emergency Response, Community Involvement and Outreach. 2002. Personal communication via e-mail with A. Burton, TAMS Consultants, Inc., regarding RAGS Part D Update. June 10.

McBride, Dave, Washington Department of Health (WDOH). Personal communication via telephone with A. Burton, TAMS Consultants, Inc., July 24.

Moore, C. 1991. Personal communication via telephone conversation with T. Michelson, PTI Environmental Services, Bellevue, WA. Onondaga County Department of Parks and Recreation, Liverpool, NY. March 25.

Murphy, C.B. 1978. Onondaga Lake. In: *Lakes of Western New York State*. J. Bloomfield (ed). Academic Press, New York, NY. pp. 223-365.

National Center for Environmental Assessment (NCEA). 2002a. Dr. Harlal Choudhury/Director to Mike Sivak, USEPA Region 2: Requested toxicity information on Benzene, Copper, DDD, DDE, Dibenzofuran, 1,3-Dichlorobenzene, and 1,4-Dichlorobenzene (Onondaga Lake). June 28.

NCEA. 2002b. Dr. Harlal Choudhury/Director to Mike Sivak, USEPA Region 2: Requested toxicity information on 2-Methylnaphthalene (Onondaga Lake). July 2.

NCEA. 2002c. Personal communication via e-mail with M. Sivak, USEPA Region 2, transmitting the NCEA, 2002a files. NCEA e-mail addresses vanadium guidance. June 28.

NCEA. 2002d. A. Parker e-mail to M. Sivak, USEPA Region 2. Forwarded to A. Burton at TAMS Consultants, Inc. by M. Sivak on June 29, 2002. NCEA e-mail addresses chloroform. June 28.

National Oceanic and Atmospheric Administration (NOAA). 1993. Local climatological data; 1993 annual summary with comparative data, Syracuse, New York. National Oceanic and Atmospheric Administration, Asheville, NC.

NOAA. 2002. National Weather Service. Binghamton Field Office. Unofficial climate data for Syracuse. Web site: www.erh.noaa.gov/er/bgm/cli/syrcli. Accessed October.

National Research Council (NRC). 2000. Toxicological effects of methylmercury. National Research Council, Washington, DC.

New York State Department of Environmental Conservation (NYSDEC). 1989. Engineering Investigations at Inactive Hazardous Waste Sites: Phase II Investigation, Mercury Sediments – Onondaga Lake, Onondaga County. New York State Department of Environmental Conservation, Division of Hazardous Waste Remediation, Albany, NY.

NYSDEC. 1992. Annual monitoring data for Onondaga Lake fish. New York State Department of Environmental Conservation, Albany, NY.

NYSDEC. 2000. Geddes Brook/Ninemile Creek Remedial Investigation and Feasibility Study: Work Plan. Prepared by New York State Department of Environmental Conservation, Division of Environmental Remediation, Albany, NY, based on the original version prepared by Exponent, Bellevue, WA for Honeywell, Solvay, NY.

NYSDEC and New York State Department of Law (NYSDOL). 1998a. Comments dated October 2, 1998, on the Onondaga Lake RI/FS: Draft Human Health Risk Assessment Report. New York State Department of Environmental Conservation and New York State Department of Law, Albany, NY.

NYSDEC and NYSDOL. 1998b. Comments dated December 31, 1998, on the Onondaga Lake RI/FS: Draft Human Health Risk Assessment Report. New York State Department of Environmental Conservation and New York State Department of Law, Albany, NY.

NYSDEC and NYSDOL. 2000. Comments dated November 17, 2000, on the Geddes Brook/Ninemile Creek RI/FS: Draft Remedial Investigation Report. New York State Department of Environmental Conservation and New York State Department of Law, Albany, NY.

NYSDEC and TAMS Consultants, Inc. (TAMS). 1998a. New York State Revision of the Remedial Investigation Report and HHRA, LCP Bridge Street Site, Solvay, New York. New York State Department of Environmental Conservation, Albany, NY, and TAMS Consultants, New York, NY.

NYSDEC/TAMS. 1998b. New York State's Revision of the Onondaga Lake Mercury Modeling Report. Prepared for New York State Department of Environmental Conservation, Superfund Standby Program, Albany, NY. TAMS Consultants, Inc., Bloomfield, NJ.

New York State Department of Health (NYSDOH). 2002a. Health Advisories – Chemicals in Sportfish and Game 2001-2002, July 2, 2001. Web site: <http://www.health.state.ny.us/nysdoh/environ/fish/htm>. Accessed May.

NYSDOH. 2002b. Personal communication from Dr. Tom Johnson, NYSDOH, to A. Burton, TAMS Consultants, Inc., regarding NYSDOH toxicity data and risk assessment. August.

NYSDOH and ATSDR. 1995. Public health assessment: Onondaga Lake. Prepared by the New York State Department of Health, Albany, NY, under a cooperative agreement with the US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

New York State Department of Transportation (NYSDOT). No date. Onondaga Lake vicinity map, Centralized Local Accident Surveillance System (CLASS) files. 1:25,000 scale quadrangle. New York State Department of Transportation, Albany, NY.

O'Brien & Gere. 1991. Remedial Investigation: Semet Residue Ponds, Geddes, New York. Draft Report. Prepared for Allied-Signal, Inc. O'Brien & Gere Engineers, Inc., Syracuse, NY.

O'Brien & Gere. 1996a. Laboratory reports for AlliedSignal, Inc., Solvay, New York. Volume 1 of 1, Metals – Solids, and Volume 1 of 4, Organic Analyses. O'Brien & Gere Laboratories, Inc.

O'Brien & Gere. 1996b. Laboratory reports for AlliedSignal, Inc., Solvay, New York. Volume 1 of 1, Metals – Waters, and Volume 1 of 7, Organic Analyses. O'Brien & Gere Laboratories, Inc.

O'Brien & Gere. 2002. Willis Avenue Chlorobenzene Site: Remedial Investigation, Geddes, New York. Under Review. Prepared for AlliedSignal, Inc., Solvay, NY. O'Brien & Gere, Syracuse, NY.

Onondaga County Department of Water Environment Protection (OCDWEP). 2002. Web site: <http://www.lake.onondaga.ny.us/ol14.htm>. Accessed November 22.

Onondaga County Parks Department (OCPD). 2002. Map of Onondaga Lake Park. Web site: <http://www.ongov.net/parks>. Accessed September.

Onondaga Lake Management Conference (OLMC). 1989. State of Onondaga Lake Report. Preliminary Draft. Onondaga Lake Management Conference, Syracuse, NY.

OLMC. 1993. Onondaga Lake: a plan for action. Onondaga Lake Management Conference, Syracuse, NY.

Onondaga Lake Partnership (OLP). 2002. Web site: <http://www.onlakepartners.org/outreach.faq.htm>. Accessed May.

Oregon Department of Human Services (DHS), Oregon Public Health Services (OPHS), CEHS Environmental Services and Consultation (ESC). 2002. Health Analysis of Chemical Contaminants in the Lower Columbia River; Section 3.4, Arsenic. Web site: <http://www.ohd.hr.state.or.us/esc/lcolmriv/arsenic.htm>. Accessed July 23.

Oregon Health Division (OHD) and Washington Department of Health (WDOH). 1996. Health Analysis of Chemical Contaminants in Lower Columbia River Fish. April. Web site: <http://www.ohd.hr.state.or.us/programs.htm>. Accessed July 24, 2002.

Pao, E.M., K.H. Fleming, P.M. Guenther, S.J. Mickel. 1982. Foods commonly eaten by individuals: amount per day and per eating occasion. US Department of Agriculture. Home Economics Report No. 44.

PTI Environmental Services (PTI). 1991. Onondaga Lake RI/FS Work Plan. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Bellevue, WA.

PTI. 1992. Onondaga Lake RI/FS Site History Report. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Waltham, MA.

PTI. 1993a. Onondaga Lake RI/FS Bioaccumulation Investigation Data Report. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Waltham, MA.

PTI. 1993b. Onondaga Lake RI/FS Mercury and Calcite Mass Balance Investigation Data Report. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Waltham, MA.

PTI. 1993c. Onondaga Lake RI/FS Substance Distribution Investigation Data Report. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Waltham, MA.

PTI. 1994. Supplemental Sediment Sampling at Onondaga Lake-East Flume. Prepared for AlliedSignal, Inc., Solvay, NY. PTI Environmental Services, Waltham, MA.

PTI. 1995a. Unpublished Fish Data Collected by New York State Department of Environmental Conservation in 1994. PTI Environmental Services, Bellevue, WA.

PTI. 1995b. Unpublished Fish Data Collected by New York State Department of Environmental Conservation in 1995. PTI Environmental Services, Bellevue, WA.

Reimann-Buechner Partnership. 1991. Onondaga Lake Development Plan.

Rupp, E.M., et al. 1980. Age dependent values of dietary intake for assessing human exposures to environmental pollutants, and Some results of recent surveys of fish and shellfish consumption by age and region of US residents. Health Physics 39:151-175.

Sittig, Marshall. 1991. Handbook of Toxic and Hazardous Chemicals and Carcinogens, Volumes 1 and 2, Third Edition. Noyes Publications, Park Ridge, NJ.

Standen, Anthony (executive editor). 1967. Kirk-Othmer Encyclopedia of Chemical Technology, Volume 12, Second Edition. Interscience Publishers division of John Wiley and Sons, New York.

Stanek, E.J. and E. Calabrese. 1992. Soil ingestion in children: Outdoor soil or indoor dust? J. Soil Contam. 1(1):1-28.

Syracuse Department of Water (SDW), City of Syracuse. 2000. Water newsletter home page. Web site: www.syracuse.ny.us/syrmayor/Services/Departments/waterreport.html. Accessed May.

SDW. 2001. Drinking Water Quality Report for 2000. Web site: <http://www.syracuse.ny.us/deptWater/>. Accessed May 7, 2002.

TAMS Consultants, Inc. (TAMS)/USEPA. 2000. Human Health Risk Assessment for the Hudson River PCBs Site. Prepared by TAMS and Gradient for USEPA and US Army Corps of Engineers. TAMS Consultants, Inc., Bloomfield, NJ.

TAMS. 2002a. Onondaga Lake Baseline Ecological Risk Assessment. Original document prepared by Exponent, Bellevue, Washington, for Honeywell, East Syracuse, New York. Revision prepared by TAMS, New York, New York and YEC, Valley Cottage, New York, for New York State Department of Environmental Conservation, Albany, New York. December.

TAMS. 2002b. Onondaga Lake Remedial Investigation Report. Original document prepared by Exponent, Bellevue, Washington, for Honeywell, East Syracuse, New York. Revision prepared by TAMS, New York, New York and YEC, Valley Cottage, New York, for New York State Department of Environmental Conservation, Albany, New York. December.

Tetra Tech. 1996. Assessing Human Health Risks from Chemically Contaminated Fish in the Lower Columbia River. Final Report TC 9968-05. May.

US Census Bureau. 2000. United States Census Bureau home page. Web site: <http://www.census.gov/>. Accessed October 2002.

US Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund. Volume 1: Human Health Evaluation Manual (Part A). Interim Final Report. EPA 540/1-89/002. US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 1990. Guidance for Data Usability in Risk Assessment. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1991a. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals). Interim Report. US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 1991b. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual Supplemental Guidance. Standard Default Exposure Factors. Interim Final. OSWER Directive 9285.6-03. US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 1991c. Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Memorandum from Don R. Clay, Assistant Administrator, to Regional Offices. OSWER Directive 9355.0-30. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1992a. Supplemental Guidance to Rags: Calculating the Concentration Term. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1992b. Internal memorandum dated February 26, 1992, from F.H. Habicht II, Deputy Administrator to Assistant and Regional Administrators, regarding guidance on risk characterization for risk managers and risk assessors. US Environmental Protection Agency, Washington, DC.

USEPA. 1992c. Dermal Exposure Assessment: Principles and Applications. EPA/600/8-91/011B. Interim Report. US Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC.

USEPA. 1993a. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. ECAO-CIN-842. US Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC.

USEPA. 1993b. Baseline Human Health Risk Assessment: Buffalo River, New York, Area of Concern. Assessment and Remediation of Contaminated Sediments (ARCS) Program. EPA 905-R93-008. US Environmental Protection Agency, Great Lakes National Program Office, Chicago, IL.

USEPA. 1994. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. US Environmental Protection Agency, Office of Solid Waste and Emergency Response Directive 9355.4-12.

USEPA. 1996a. PCBs: Cancer dose-response assessment and application to environmental mixtures. EPA/600/P-96/001F. US Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC.

USEPA. 1996b. Proposed Guidelines for Carcinogen Risk Assessment. EPA/600/P-92/003C. USEPA Office of Research and Development, Washington DC. April.

USEPA. 1997a. Exposure Factors Handbook. EPA/600/P-95/002F. US Environmental Protection Agency, Office of Research and Development, Washington, DC.

USEPA. 1997b. Health Effects Assessment Summary Tables. EPA 540/R-93/058. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1997c. An SAB Report: Review of the EPA Draft Mercury Study Report to Congress. EPA-SAB-EC-98-001. US Environmental Protection Agency, Science Advisory Board, Washington, DC.

USEPA. 1998. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments). Interim Report. EPA 540-R-97-033. US Environmental Protection Agency, Office of Emergency Response and Remedial Response, Washington, DC.

USEPA. 1999. Risk Assessment Guidance for Superfund Supplemental Guidance: Dermal Risk Assessment. Interim Guidance (Internal Draft). US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 2000a. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds, Part III: Integrated Risk Summary and Risk Characterization for 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds, EPA/600/P-00/001Bg, September 2000 – Preliminary Review Draft.

USEPA. 2000b. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume 2–Risk Assessment and Fish Consumption Limits. Third Edition. EPA-823-B-00-008. US Environmental Protection Agency, Office of Water. November.

USEPA. 2000c. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000). EPA-822-B-004. US Environmental Protection Agency, Office of Water, Office of Science and Technology. October.

USEPA. 2001a. RAGS Volume I: Human Health Evaluation Manual, Part E–Supplemental Guidance for Dermal Risk Assessment for Public Comment. Interim Draft. USEPA Office of Emergency and Remedial Response. EPA/540/R/99/005. September.

USEPA. 2001b. Risk Assessment Guidance for Superfund (RAGS) 3, Part A–Process for Conducting Probabilistic Risk Assessment. Exhibit 3-2, Definitions for Chapter 3. December 31.

USEPA. 2001c. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, March 2001, Peer Review Draft; OSWER 9355.4-24.

USEPA. 2001d. RAGS Volume I – Human Health Evaluation Manual, Part D – Standardized Planning, Reporting and Review of Superfund Risk Assessments. Final. December.

USEPA. 2002a. USEPA Region 3 Risk-Based Concentration (RBC) Table. US Environmental Protection Agency Region 3, Philadelphia, PA. Web site: <http://www.epa.gov/reg3hwmd/risk/index.htm>. Accessed April.

USEPA. 2002b. USEPA Region 9 Preliminary Remediation Goals (PRGs) Table. US Environmental Protection Agency Region 9, San Francisco, CA. Web site: <http://www.epa.gov/region09>. Accessed October.

USEPA. 2002c. ProUCL Version 2.1. Software and User's Guide. Prepared for USEPA Region 3 by Lockheed-Martin Environmental Systems and distributed by USEPA National Exposure Research Laboratory, Las Vegas, NV. July.

Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. van Leeuwen, A.K. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environ. Health Perspect.* 106:775–792.

West, P.C., J.M. Fly, R. Marans, and F. Larkin. 1989. Michigan Sport Anglers Fish Consumption Survey Report to the Michigan Toxic Substance Control Commission. Michigan Department of Management and Budget Contract No. 87-20141.

West, P.C., J.M. Fly, R. Marans, F. Larkin, and D. Rosenblatt. 1993. 1991–92 Michigan Sport Anglers Fish Consumption Study. Technical Report No. 6. Prepared for the Michigan Department of Natural Resources, Ann Arbor, MI. University of Michigan, School of Natural Resources.

Wester, R.C., H.I. Maibach, L. Sedik, and J. Melendres. 1993. Percutaneous absorption of PCBs from soil: *In vivo* rhesus monkey, *in vitro* human skin, and binding to powdered human *stratum corneum*. *J. Toxicol. Environ. Health* 39:375–382.

White, Jim, Ph.D., Washington Department of Health (WDOH). 2002. Personal communication via telephone with A. Burton, TAMS Consultants, Inc. August 1.

Wilson, N.D., N.M. Shear, D.J. Paustenbach, and P.S. Price. 1998. The effects of cooking practices on the concentration of DDT and PCB compounds in the edible tissue of fish. *J. Exp. Anal. Environ. Epidemiol.* 8(3):423–436.

World Health Organization (WHO). 1998. Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI). WHO Consultation. Geneva, Switzerland. May.

Zabik, M.E. and M.J. Zabik. 1996. Influence of processing on environmental contaminants in food. Food Technol. May:225–229.