

# Ecological Screening-Level Risk Assessment of the Lower Ottawa River

*Prepared for*

**Limno-Tech, Inc.**  
501 Avis Drive  
Ann Arbor, Michigan 48108

*Prepared by*

**Parametrix**  
5808 Lake Washington Blvd. NE, Suite 200  
Kirkland, WA 98033-7350  
(425) 822-8880  
[www.parametrix.com](http://www.parametrix.com)

---

October 2001

Project No. 555-3763-001 (01/03)

## TABLE OF CONTENTS

<b>ACRONYMS .....</b>	<b><i>iv</i></b>
<b>GLOSSARY .....</b>	<b><i>vi</i></b>
<b>EXECUTIVE SUMMARY .....</b>	<b><i>ix</i></b>
<b>1. INTRODUCTION .....</b>	<b>1-1</b>
1.1 PURPOSE AND SCOPE.....	1-1
1.2 OBJECTIVES.....	1-1
1.3 REPORT ORGANIZATION.....	1-1
<b>2. PROBLEM FORMULATION .....</b>	<b>2-1</b>
2.1 OVERVIEW OF STUDY SITE .....	2-1
2.2 CHEMICAL SOURCES .....	2-1
2.3 RECEPTORS OF CONCERN .....	2-3
2.4 ASSESSMENT AND MEASUREMENT ENDPOINTS.....	2-4
2.5 CONCEPTUAL SITE MODEL .....	2-5
<b>3. EXPOSURE CHARACTERIZATION.....</b>	<b>3-1</b>
3.1 CHEMICAL CONCENTRATIONS .....	3-1
3.1.1 Measured Concentrations .....	3-1
3.1.2 Estimated Concentrations .....	3-2
3.2 EXPOSURE QUANTIFICATION.....	3-2
3.2.1 Wildlife.....	3-3
3.2.2 Aquatic Life.....	3-6
<b>4. EFFECTS CHARACTERIZATION .....</b>	<b>4-1</b>
4.1 WILDLIFE .....	4-1
4.1.1 Toxicity Thresholds.....	4-1
4.1.2 Water Quality Criteria for Wildlife .....	4-1
4.2 AQUATIC LIFE.....	4-2
4.2.1 Surface water .....	4-2
4.2.2 Sediment.....	4-3
4.2.3 Tissue Residues .....	4-4
<b>5. RISK CHARACTERIZATION .....</b>	<b>5-1</b>
5.1 WILDLIFE .....	5-1
5.1.1 Dose-Based Hazard Quotients.....	5-1
5.1.2 Water Quality Criteria-Based Hazard Quotients .....	5-9
5.2 AQUATIC LIFE.....	5-9
5.2.1 Hazard Quotients .....	5-9
5.2.2 Bioassays .....	5-18
5.2.3 Biological Criteria .....	5-20

## TABLE OF CONTENTS (Continued)

5.3	UNCERTAINTIES.....	5-25
<b>6.</b>	<b>CONCLUSIONS .....</b>	<b>6-1</b>
<b>7.</b>	<b>REFERENCES .....</b>	<b>7-1</b>

### LIST OF FIGURES

E-1	Ottawa River Site.....	x
E-2	Risk Characterization Approach.....	xiv
E-3	Ecological Hazard Quotient Comparison by River Segment .....	xv
1-1	Ottawa River Site.....	1-2
1-2	Framework for Ecological Risk Assessment.....	1-3
2-1	Right Bank of Ottawa River at River Mile 5.0.....	2-2
2-2	The Dura Avenue Landfill.....	2-2
2-3	Ottawa River Adjacent to Dura Avenue Landfill .....	2-3
2-4	Conceptual Model for Wildlife.....	2-7
2-5	Conceptual Model for Aquatic Life.....	2-8
5-1	Risk Characterization Approach.....	5-2
5-2	Chronic Hazard Quotients for Bald Eagles Feeding in the Ottawa River .....	5-4
5-3	Chronic Hazard Quotients for Common Terns Feeding in the Ottawa River.....	5-5
5-4	Chronic Hazard Quotients for Spotted Sandpipers Feeding in the Ottawa River.....	5-7
5-5	Chronic Hazard Quotients for Mink Feeding in the Ottawa River .....	5-8
5-6	Sediment HQs > 1.0 Based on ERMs and PELs Using 1998 Surface (< 24") Sediment Data.....	5-15
5-7	Comparison of Sediment HQs Based on 1998 Surface (< 24") and Core (> 24") Sediment Data.....	5-16
5-8	Sediment HQs > 1.0 Based on ERMs and PELs Using 2000 Surface Sediment Data .....	5-17
5-9	Chronic Hazard Quotients for Fish in the Ottawa River Using Tissue-Based TRVs.....	5-19
5-10	Deformed carp caught in the Lower Ottawa River .....	5-24
6-1	Ecological Hazard Quotient Comparison by River Segment .....	6-3

### LIST OF TABLES

E-1	Chemicals with Chronic HQs > 1.0 by Ecological Receptor and River Segment.....	xiii
2-1	Ecological Receptors to be Evaluated in the SLRA and Their Routes of Exposure .....	2-4

## TABLE OF CONTENTS (Continued)

2-2	Ecological Assessment and Measurement Endpoints Used in the Lower Ottawa River SLRA .....	2-5
3-1	Summary of Chemistry Data Used in Wildlife and Aquatic Life SLRAs .....	3-2
3-2	Ingestion Rate and Body Weight Values Used for Avian and Mammalian Receptors .....	3-4
4-1	Ohio EPA Water Quality Criteria for Wildlife .....	4-2
5-1	Chemicals with Individual Acute HQs Exceeding 1.0 .....	5-11
5-2	Chemicals with Individual Chronic HQs Exceeding 1.0 .....	5-12
5-3	$\Sigma$ HQs for Divalent Metals in Surface Water .....	5-14
5-4	Location and Dates of Whole Sediment Toxicity Bioassays .....	5-20
5-5	Comparison of Sediment Chemistry from Bioassay Sample Site 09 to Sediment Quality Guidelines .....	5-21
5-6	Summary Indices for Benthic Macroinvertebrates and Fish .....	5-22
5-7	Summary of Chronic HQs for Surface Water and Invertebrate and Fish Biotic Indices .....	5-23
5-8	Sediment HQs and Invertebrate and Fish Biotic Indices .....	5-25
6-1	Chemicals with chronic HQs > 1.0 by ecological receptor and river segment .....	6-2

### APPENDICES

A	Exposure Data
B	Toxicity Data
C	Hazard Quotients
D	Sediment HQ Maps

## ACRONYMS

ACR	Acute-Chronic Ratio
ANOVA	Analysis of Variance
AWQC	Acute Water Quality Criteria
AVS	Acid-Volatile Sulphide
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BMF	Biomagnification Factor
BSAF	Biota-Sediment Accumulation Factor
COPC	Chemical of Potential Concern
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethylene
DDT	Dichlorodiphenyltrichloroethane
DELT	Deformities, Fin Erosions, Lesions/Ulcers, and Tumors
DOC	Dissolved Organic Carbon
EC10	Effects Concentration
EEC	Expected Environmental Concentration
EPA	Environmental Protection Agency
EPT	Ephemeroptera Plecoptera Trichoptera
EqP	Equilibrium Partitioning
ERL	Effects Range Low
ERM	Effects Range Median
HQ	Hazard Quotient
IBI	Index of Biotic Integrity
IR	Ingestion Rate
IWB2	Index of Well Being
Kow	Octanol-Water Partition Coefficient
LC50	Lethal Concentration
LD50	Lethal Dose
LICI	Lacustrary Invertebrate Community Index
LOAEL	Lowest Observed Adverse Effects Level
LTI	Limno-Tech, Inc.
NOAEL	No Observable Adverse Effects Level

## ACRONYMS (Continued)

OEPA	Ohio Environmental Protection Agency
PAH	Polycyclic Aromatic Hydrocarbon
PCB	Polychlorinated Biphenyl
PEL	Probable Effect Level
RM	River Mile
SEM	Simultaneously Extracted Metals
SLRA	Screening Level Risk Assessment
SVOC	Semi-Volatile Organic Compound
TEL	Threshold Effect Level
TRV	Toxicity Reference Value
TU	Toxic Unit
UCL	Upper Confidence Limit
U.S. EPA	United States Environmental Protection Agency
U.S. FWS	United States Fish and Wildlife Service
WQC	Water Quality Criteria
WQS	Water Quality Standard

## GLOSSARY

<i>Acute Exposure</i>	One dose or multiple doses occurring within a short time (1 to 7 days).
<i>Acute Toxicity</i>	Significant probability of mortality or other effects from short-term (often 96 hours), relatively high-concentration exposure to toxic chemicals (Rand 1995).
<i>Acute-Chronic Ratio (ACR)</i>	The ratio of a chemical's acute toxicity to its chronic toxicity for the same species (Rand 1995).
<i>Additive effects</i>	The potential for adverse effects on health due to the combined action of two or more chemicals which have a similar mode of action. It assumes that the combined effect of the subthreshold effects of several chemicals could result in an adverse effect.
<i>Assessment Endpoint</i>	Explicit expressions of the actual environmental or societal value to be protected, or the undesired effect whose probability of occurrence is estimated in a risk assessment. Examples include extinction of an endangered species, eutrophication of a lake, or the damage to a fishery by water pollution (Parkhurst et al. 1996).
<i>Background</i>	Chemical concentrations or intakes originating from chemical concentrations in local environmental media unimpacted by human activity.
<i>Bioaccumulation</i>	The amount of chemical taken up by the organism attributable to both bioconcentration and dietary accumulation (Rand 1995).
<i>Bioavailability</i>	The degree to which a chemical is available to the target organism or tissue.
<i>Biomagnification</i>	The process by which the tissue concentration of a bioaccumulated chemical increases as it passes up the food chain through at least two trophic levels (minimum of three involved) (Rand 1995).
<i>Chemicals of Potential Concern (COPCs)</i>	Chemicals that have been identified by the balance of available evidence as posing potential risks to aquatic and wildlife receptors.
<i>Chronic Exposure</i>	Multiple exposures occurring over an extended period, or a significant fraction of the animal's or the individual's lifetime, up to the entire duration of life.
<i>Chronic Toxicity</i>	Significant probability of effects on growth, yield, reproduction, or survival from long-term exposure to toxic chemicals (Rand 1995).
<i>Community</i>	An assemblage of populations of plants, animals, bacteria, and fungi that live in an environment and interact with one another, forming a distinctive living system with its own composition, structure, environmental relations, development, and function.
<i>Conceptual Model</i>	A written description and visual representation of predicted relationships between ecological entities and the chemicals that they may be exposed to.

## GLOSSARY (Continued)

<i>Dose</i>	The mass of a substance given to an organism and in contact with an exchange boundary (e.g., gastrointestinal tract) per unit body weight per unit time (e.g., mg/kg-day).
<i>Ecosystem</i>	The biotic community and abiotic factors that interact within a specified location in space and time.
<i>Effects Characterization</i>	The process for quantitatively defining the adverse effects on individuals, populations, and communities elicited from exposure (Parkhurst et al. 1996).
<i>Expected Environmental Concentration</i>	The estimated concentration of chemicals in surface water, sediments, and the food of fish and wildlife (Parkhurst et al. 1996).
<i>Exposure</i>	The contact or co-occurrence of a chemical with a receptor.
<i>Exposure Assessment</i>	The determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, and route of exposure to chemicals in environmental media.
<i>Exposure Characterization</i>	The process for quantitatively defining the expected environmental concentrations/doses (EECs and EEDs) and pathways to which the receptors are exposed (Parkhurst et al. 1996).
<i>Exposure Pathway</i>	The course a chemical or physical agent takes from a source to an exposed organism. An exposure pathway describes a unique mechanism by which an individual or population is exposed to chemicals or physical agents at or originating from a site. Each exposure pathway includes a source, or release from a source, an exposure point, and an exposure route. If the exposure point differs from the source, a transport/exposure medium (e.g., air) or media (in cases of intermedia transfer) is/are also included.
<i>Exposure Route</i>	The mechanism by which a chemical or physical agent comes in contact with an organism (i.e., by ingestion, inhalation, dermal contact).
<i>Hazard Quotient (HQ)</i>	The ratio of the concentration or dose of a chemical over the concentration or dose at which no adverse effects of any kind are expected. When HQs are less than one, negligible risks are expected.
<i>LC50</i>	Median lethal concentration; the concentration causing death to 50 percent of the test organisms (Rand 1995).
<i>LD50</i>	Median lethal dose; the dose causing death to 50 percent of the test organisms (Rand 1995).
<i>Lipophilic Chemical</i>	Chemicals that have a propensity to partition into lipids (i.e., fatty tissue), rather than water.
<i>Lowest Observed Adverse Effect Level (LOAEL)</i>	In dose-response experiments, the lowest exposure level at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group.

## GLOSSARY (Continued)

<i>Measurement Endpoint</i>	An expression of an observed or measured response to a hazard; it is a measurable environmental characteristic that is related to the valued characteristic chosen as the assessment endpoint (Parkhurst et al. 1996).
<i>No Observed Adverse Effect Level (NOAEL)</i>	In dose-response experiments, an exposure level at which there are no statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered to be adverse, nor precursors to specific adverse effects. In an experiment with more than one NOAEL, the regulatory focus is primarily on the highest one, leading to the common usage of the term NOAEL to mean the highest exposure level without adverse effect.
<i>Off-River Water Bodies</i>	Tie channels, oxbow lakes, blocked valley lakes, swamps, lagoons, periodically flooded forest, and any other body of water that is directly affected by the river but not part of the main stream.
<i>Population</i>	A potentially interbreeding group of individuals of a single species.
<i>Problem Formulation</i>	The step where goals of the risk assessment are defined and exposure routes for stressors (chemicals) are identified.
<i>Receptor</i>	Wildlife species for which risk from chemical exposure is being evaluated.
<i>Risk</i>	The likelihood of a prescribed undesired effect, such as injury, disease or death, resulting from human actions or a natural catastrophe (Parkhurst et al. 1996).
<i>Risk Characterization</i>	The process that defines the potential for or probability of adverse effects to the receptor population given exposure to a range of expected environmental concentrations (EECs).
<i>Screening</i>	A risk assessment process in which conservative estimates of exposure and toxicity are used to identify chemicals that pose negligible risks. Chemicals so identified are referred to as being “screened out”.
<i>Toxicity Reference Value (TRV)</i>	Toxicity threshold for aquatic or wildlife receptor.
<i>Trophic Levels</i>	A functional classification of taxa within a community that is based on feeding relationships.
<i>Upper Bound</i>	An estimate of the plausible upper limit to the true value of the quantity. This is usually not a statistical confidence limit.

## EXECUTIVE SUMMARY

An ecological screening-level risk assessment (SLRA) of the lower Ottawa River was conducted as an initial effort to prioritize chemical hot spots for possible remediation. The SLRA followed the U.S. EPA's standard ecological risk assessment guidelines and included the following sections: (1) Problem Formulation; (2) Exposure Characterization; (3) Effects Characterization; and (4) Risk Characterization. The Problem Formulation stated the goals of the SLRA, described the study area, and identified the ecological receptors evaluated. The Exposure Characterization described the chemistry data available and the methods for quantifying exposure of ecological receptors to chemicals. The Effects Characterization presented the Toxicity Reference Values (TRVs) used for comparison to the chemical exposure levels. Lastly, the Risk Characterization compared the Exposure and Effects Characterization results to estimate whether ecological receptors are at risk from chemicals in the lower Ottawa. Multiple lines of evidence were included in the Risk Characterization to provide an overall weight-of-evidence. The following summarizes each of these SLRA sections.

### PROBLEM FORMULATION

Potential chemical risks in the lower 9 miles of the Ottawa River were assessed (Figure E-1). Chemical sources to this reach of the Ottawa include various industries, landfills, textile producers, and fertilizer manufacturers. Chemicals of interest from historical investigations include polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and metals. For consistency with Ohio EPA management goals, potential risks were estimated separately for five different segments of the lower Ottawa River: river miles (RMs) 0 to 3.2, 3.2 to 4.9, 4.9 to 6.5, 6.5 to 8.8, and > 8.8. Wildlife receptors evaluated included the bald eagle (*Haliaeetus leucocephalus*), common tern (*Sterna hirundo*), spotted sandpiper (*Actitis macularia*), and mink (*Mustela vison*). Potential risks to the bald eagle were also evaluated in North Maumee Bay since there have been reports of unsuccessful breeding in this area. In addition, potential risks to the aquatic community (fish, invertebrates) were also evaluated. These receptors were selected because they are known to use the lower Ottawa River as habitat and/or for feeding and they represent a range of exposure routes (e.g., fish-eaters, invertebrate-feeding probers, direct contact with water and sediment).

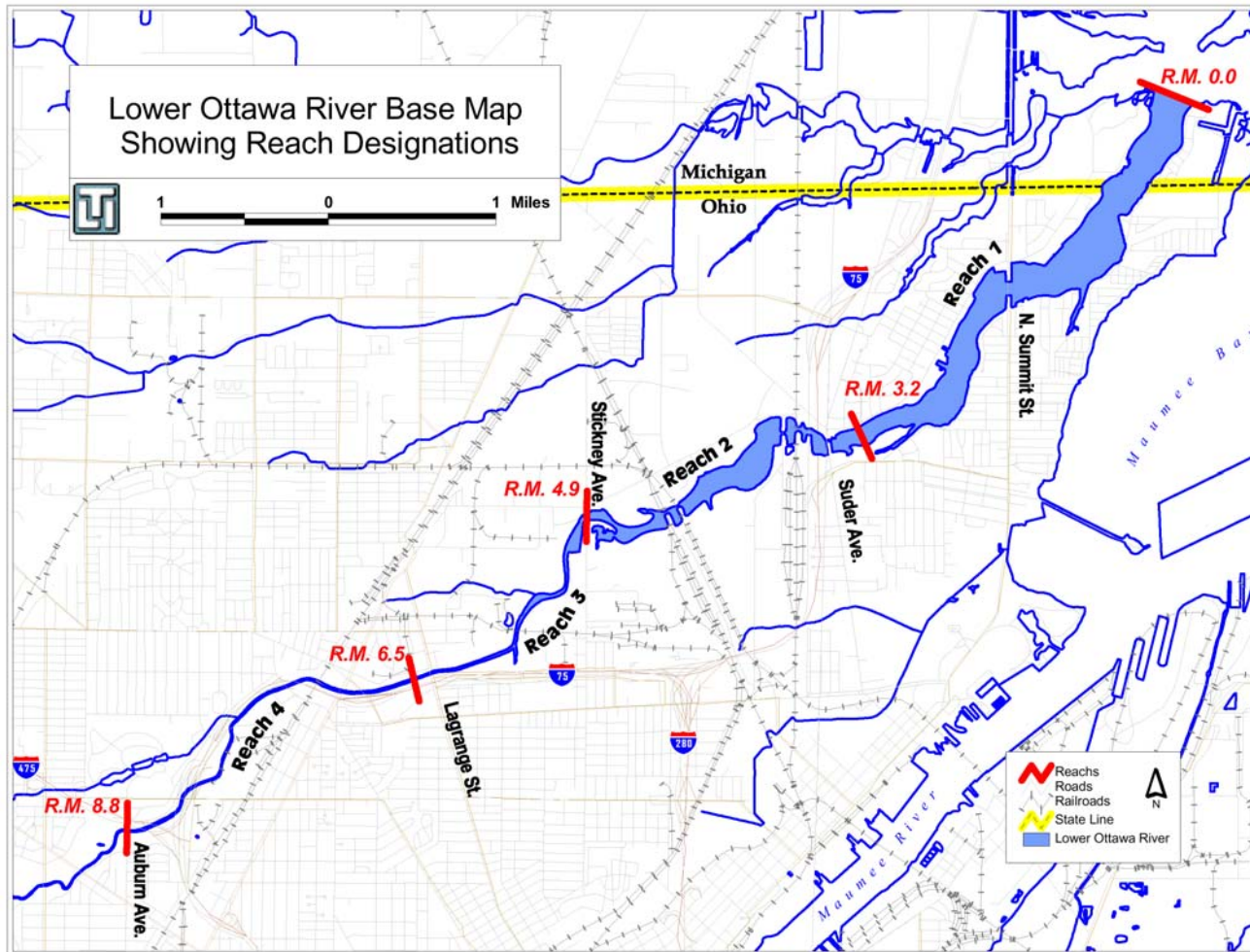
### EXPOSURE CHARACTERIZATION

Chemistry data for sediment, fish tissue, and surface water samples collected during summer of 2000 were used to quantify exposure of all ecological receptors. In addition, sediment data collected in 1998 from various depths were also included in the aquatic life assessment (wildlife were considered to receive combined exposures through sediment, tissue, and water ingestion, so only the temporally co-located data from 2000 were used for these receptors). Both acute (short-term) and chronic (long-term) chemical exposures were evaluated in each river segment. Conservative estimates of mean<sup>1</sup> and upper bound<sup>2</sup> chemical concentrations were used to approximate chronic and acute exposure concentrations, respectively.

For aquatic life, chemical concentrations in sediment, tissue, and surface water were used directly to estimate exposure. Wildlife exposures were estimated by calculating species-specific doses. Chemical-specific doses for wildlife were estimated as milligram chemical per kilogram body weight per day

<sup>1</sup> The 95 percent upper confidence limit (UCL) on the mean.

<sup>2</sup> The 95<sup>th</sup> percentile of all the data.



(mg/kg/d). The doses were calculated using the chemistry data and wildlife receptor body weights and ingestion rates (food, sediment, and water). It was conservatively assumed that wildlife receptors could receive 100 percent of their chemical dose from an individual river segment, and that all chemicals in each medium were completely absorbed following ingestion. Bald eagles, common terns, and mink were assumed to feed exclusively on fish. Both of these assumptions are more likely to over- rather than underpredict risk potential. For sandpipers, which feed predominantly on macroinvertebrates in the sediment, chemical concentrations in invertebrates were estimated from sediment concentrations where recognized methods exist.<sup>3</sup>

## EFFECTS CHARACTERIZATION

Toxicity data were identified for comparison to the estimated exposure levels calculated in the Exposure Characterization. For wildlife, acute and chronic toxicity data (expressed as doses) were compiled. These toxicity data were generally based on surrogate test animals (e.g., rats, mallards), but mink toxicity data for some chemicals were available. The acute toxicity data were based on mortality, while chronic toxicity data were based on mortality or sublethal endpoints such as reproduction, growth, and development.

Sediment and tissue- and water-based toxicity data were all compiled for aquatic life. Sediment toxicity data were compiled from numerous sources, including the scientific literature and government agencies (e.g., Environment Canada, Ontario Ministry of the Environment). Multiple sediment guidelines for individual chemicals were often compiled, when available. This provides a weight of evidence when evaluating sediment risks since site-specific factors can have substantial impacts on chemical bioavailability in sediment. Tissue-based toxicity data were also compiled for chemicals detected in fish tissue. These toxicity data are quite limited compared to water-based toxicity data, but provide a useful approach for assessing potential risks to fish for chemicals that are hydrophobic and/or tend to be passed through dietary pathways rather than directly from the water column (e.g., PCBs). Lastly, water-based toxicity data were compiled for comparison to water chemistry data. When available, Ohio EPA water quality criteria (WQC) or U.S. EPA WQC were used. These WQC are designed to be protective of aquatic communities. If WQC were not available, the lowest chemical-specific toxicity data identified for individual species were used to assess water column risks. Both acute and chronic water-based toxicity data were compiled (only chronic sediment and tissue-based toxicity data were compiled because concentrations in these media tend to represent long-term accumulation).

## RISK CHARACTERIZATION

Potential risks to ecological receptors were estimated by comparing the exposure levels calculated in the Exposure Characterization to the effects levels identified in the Effects Characterization. Thus, for wildlife, calculated exposure doses for avian and mammalian receptors were compared to their respective TRVs. For aquatic life, mean and upper bound exposure concentrations in sediment, tissue, or water were compared directly to their respective media-specific TRVs. These ratios of exposure levels to toxicity levels, for both wildlife and aquatic life, are termed hazard quotients (HQs). An HQ less than 1.0 suggested that a receptor was *not* at risk, while an HQ greater than 1.0 suggested a receptor *may* be at

---

<sup>3</sup> Benthic tissue concentrations were estimated only for non-polar, lipophilic organics.

risk<sup>4</sup>. Given the conservative assumptions used in the SLRA, chemicals with HQs greater than 1.0 should be further evaluated in a detailed assessment with additional site data to determine whether they are truly of concern. Accordingly, these were referred to as chemicals of potential concern (COPCs) in the SLRA. The COPCs for wildlife and aquatic life are discussed separately below. The risk characterization approach is summarized in Figure E-2.

## Wildlife

### Chemistry Data

Acute HQs for wildlife were almost always less than 1.0. The only exceptions were a lead HQ of 3.9 for RMs 4.9 to 6.5 and a zinc HQ of 1.1 for RMs 6.5 to 8.8. These HQs were calculated for the spotted sandpiper and were influenced by incidental sediment ingestion (the spotted sandpiper has a high sediment ingestion rate since it feeds by probing the sediment for food). Zinc is likely not of concern for the sandpiper given that the HQ only slightly exceeded 1.0. However, lead appears to be a COPC within RMs 4.9 to 6.5 because it is highly affected by a single sediment sample with an elevated lead concentration of 13,000 mg/kg-wet (parts per million) at RM 5.5. Although acute HQs for other metals did not exceed 1.0 for this reach, it was noted that maximum concentrations of several other metals within this river segment were found in the same sediment sample. Accordingly, this portion of the river represents a possible hot spot of metal contamination.

Chronic HQs for lead and PCBs exceeded 1.0 in at least one river segment for all wildlife receptors (i.e., bald eagle, common tern, spotted sandpiper, and mink). Like the acute assessment, the largest chronic HQs for lead, ranging from 2.6 (bald eagle) to 254 (mink), occurred between RMs 4.9 and 6.5. Lead chronic HQs between 1.0 and 6.0 were calculated for mink for the other river segments as well, but it is likely that lead is not actually of concern in these segments since the HQs only slightly exceeded 1.0 and because it was conservatively assumed that mink fed exclusively in any one of the individual river segments. PCB chronic HQs exceeded 1.0 for all receptors between RMs 3.2 to 4.9, 4.9 to 6.5, and 6.5 to 8.8. The HQs were largest for fish-eating receptors (bald eagle [1.3 to 4.4], common tern [6.7 to 22], and mink [0.6 to 1.9]), likely reflecting the potential for PCBs to biomagnify (i.e., increase in concentration up the food chain). The PCB HQs increase slightly from downstream to upstream, but were quite similar in magnitude, suggesting that PCB contamination is not substantially elevated in any single river segment. Chronic HQs for the pesticide DDT also exceeded 1.0 for the eagle, tern, and sandpiper, with HQ ranges of 0.15 to 1.7, 0.75 to 8.7, and 0.65 to 1.7, respectively. The largest HQs for DDT occurred above RM 8.8 and decreased to less than 1.0 in the lower river miles.

To identify river segments posing the greatest potential risk, all chemical HQs greater than 1.0 (i.e., the risk drivers) were summed by river segment and wildlife receptor (Figure E-3). As shown, Segment 3 (RM 4.9 to 6.5) poses the greatest potential to all wildlife receptors. Potential risks were also identified in the other river segments, but at lower levels. In general, the lowest potential risk for wildlife receptors was identified in Segment 1 at the mouth of the Ottawa River (RM 0 to 3.2). The chemicals with HQs greater than 1.0 by river segment and wildlife receptor are shown in Table E-1. However, it should be emphasized that the chemicals listed in Table E-1 do not contribute equally to potential risk for a given

---

<sup>4</sup> It is not possible to reasonably differentiate chemicals as posing low, moderate, or high potential risk for those with HQs greater than 1.0, because the conservatism in the data and assumptions used are chemical-specific. As a general guideline, however, HQs greater than 10 likely suggest a high potential risk.

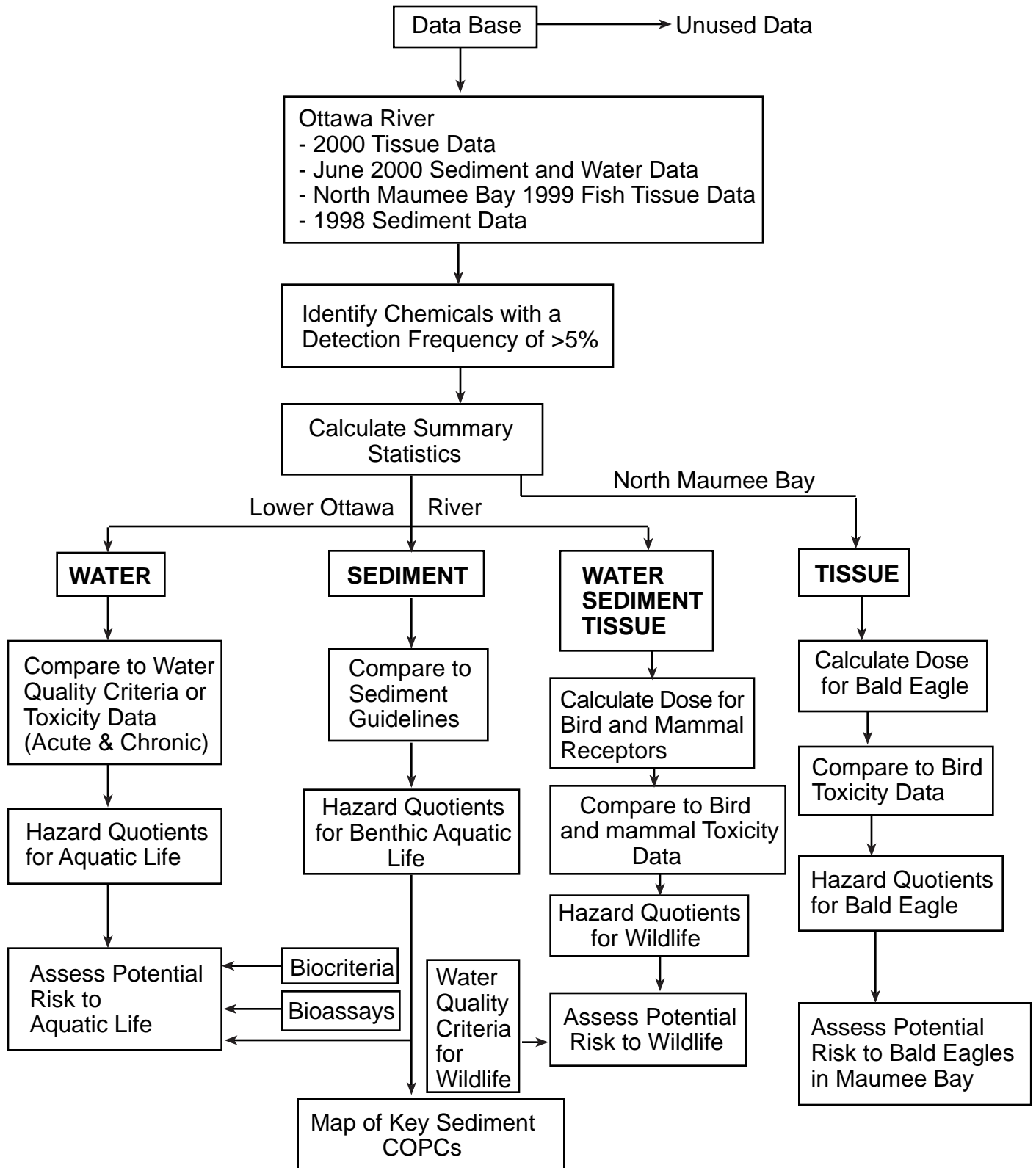
wildlife receptor and river segment. As shown by the magnitude of the HQs (Table E-1), lead and PCBs appear to be the risk drivers for wildlife.

Using fish tissue concentration data from North Maumee Bay, potential risks to the bald eagle were also estimated. Like the lower Ottawa River assessment, the chronic PCB HQ (2.8) exceeded 1.0. The HQs did not exceed 1.0 for any other chemicals measured. These results suggest that potential PCB risks to the bald eagle (and similar higher-trophic fish-eating wildlife) are fairly widespread in the lower Ottawa River region. Accordingly, even if it were more realistically assumed that the eagles feed throughout a much larger area than that assumed in the SLRA, potential risks would still likely be estimated.

**Table E-1. Chemicals with Chronic HQs > 1.0 by Ecological Receptor and River Segment.**

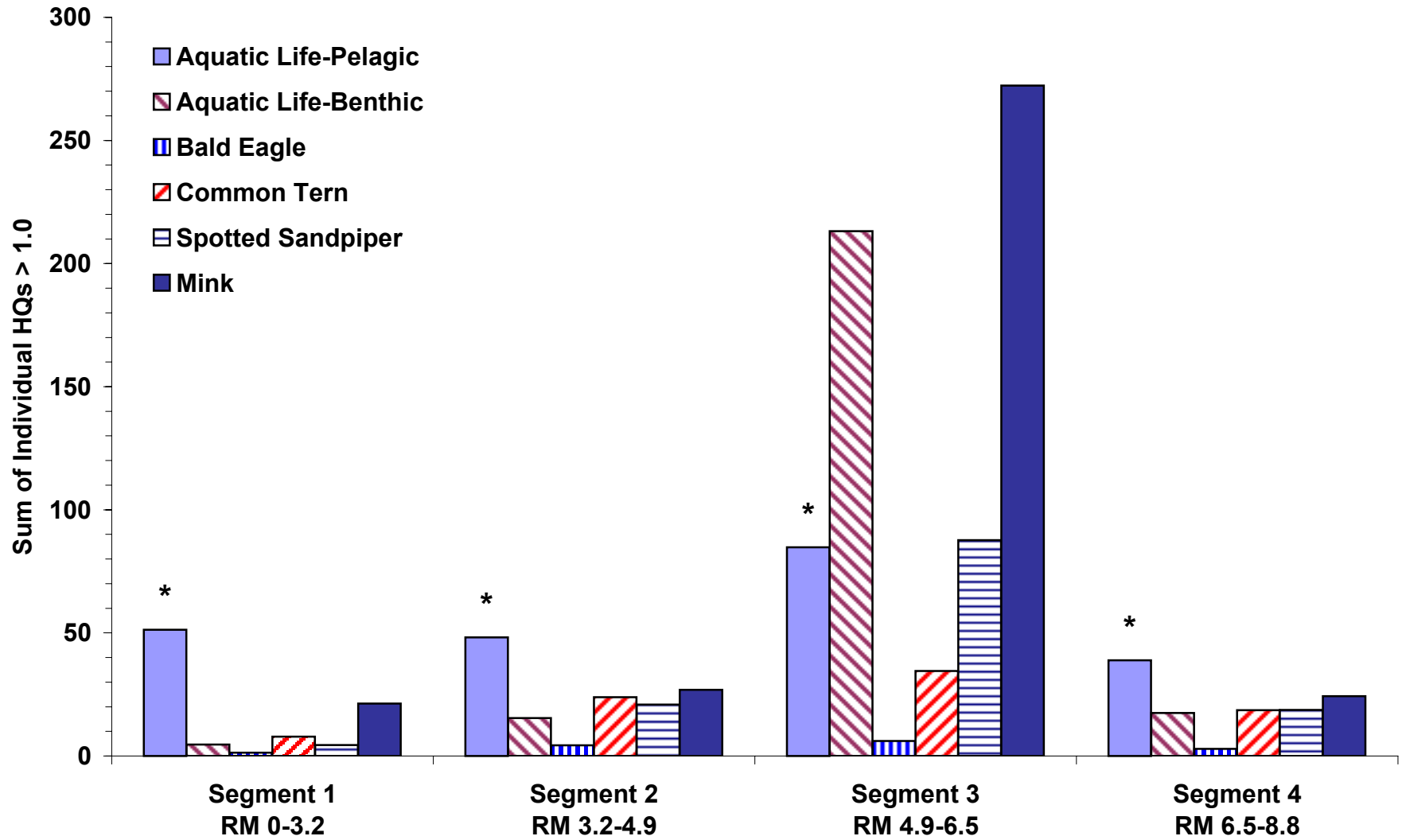
<b>Receptor</b>	<b>Segment 1 (RM 0-3.2)</b>	<b>Segment 2 (RM 3.2-4.9)</b>	<b>Segment 3 (RM 4.9-6.5)</b>	<b>Segment 4 (RM 6.5-8.8)</b>
Aquatic Life - Pelagic	Aluminum (30) <sup>a</sup>	Aluminum (25)	Aluminum (14)	Aluminum (13)
	Iron (4.3)	Iron (4.2)	Iron (2.8)	Iron (1.9)
	Manganese (17)	Manganese (19)	Manganese (68)	Manganese (24)
Aquatic Life - Benthic	Lead (1.2)	Lead (3.2)	Cadmium (1.2)	Chromium (1.1)
	Nickel (1.3)	Nickel (1.1)	Chromium (1.2)	Copper (1.0)
	PCBs (2.2)	PCBs (11)	Lead (199)	Lead (3.6)
			Nickel (1.2)	Zinc (1.1)
		PCBs (11)	PCBs (11)	
Bald Eagle	PCBs (1.3)	PCBs (4.3)	Lead (2.6)	PCBs (2.9)
			PCBs (3.5)	
Common Tern	Selenium (1.1)	PCBs (22)	Lead (13)	PCBs (15)
	PCBs (6.8)	DDT (1.9)	Selenium (1.2)	DDT (4.0)
			PCBs (18)	DDT (2.4)
Spotted Sandpiper	Aluminum (1.7)	Aluminum (1.9)	Aluminum (1.6)	Aluminum (1.6)
	PCBs (2.7)	Chromium (1.0)	Chromium (2.3)	Chromium (2.3)
		Lead (1.0)	Lead (71)	Lead (1.4)
		PCBs (17)	PCBs (11)	Cyanide (1.2)
			DDT (1.6)	PCBs (11)
			DDT (1.7)	
Mink	Aluminum (16)	Aluminum (18)	Aluminum (15)	Aluminum (15)
	Lead (1.7)	Iron (1.0)	Lead (254)	Lead (6.0)
	Selenium (1.6)	Lead (4.1)	Selenium (1.6)	Selenium (1.1)
	Thallium (1.7)	Thallium (2.1)	Thallium (1.5)	Thallium (2.7)
			PCBs (1.9)	

<sup>a</sup> Value in parentheses is the chronic HQ.



**Figure E-2  
Risk Characterization Approach**

### Ecological Hazard Quotient Comparison by River Segment



\* Pelagic aquatic life HQs driven by ubiquitous metals and are likely extremely conservative.

## ***Other Lines of Evidence for Wildlife Risk Potential***

In addition to the dose-based HQs described above, potential risks to wildlife were estimated using Ohio EPA WQC for wildlife. Mercury was the only chemical detected in surface water for which a wildlife criterion was available. The mercury HQs using this line of evidence were very large, ranging from 113 to 177. These results are not consistent with the dose-based HQs above, where none of the mercury HQs exceeded 1.0. Accordingly, potential risks posed by mercury were uncertain. The wildlife criterion for mercury is based on mercury bioaccumulation from water into aquatic prey items, while the dose-based HQs are calculated using mercury concentrations measured in Ottawa River fish tissue. It may be that the wildlife criterion greatly overestimated mercury's bioaccumulation potential in the Ottawa River.

## ***Aquatic Life***

Potential risks to aquatic life were considered by separately evaluating HQs based on chemistry data for surface water, tissue, and sediment. Sediment bioassay results and biological monitoring data were used as additional lines of evidence for comparison to the HQs.

## ***Surface Water***

Acute HQs exceeded 1.0 for aluminum (1.5 to 4.6) and manganese (3.8 to 21.2), while chronic HQs exceeded 1.0 for aluminum (12.7 to 30), manganese (17 to 68), iron (1.9 to 4.3), and selenium (1.3 to 3.0). The significance of the HQs for all of these metals is uncertain for various reasons. For example, aluminum, manganese, and iron are fairly ubiquitous metals that can exist in particulate forms that are non-bioavailable to aquatic biota. Since total metal concentrations were measured in the surface water, non-bioavailable forms were likely included in the chemical analysis, and it is likely that the HQs for these metals are largely overestimated. The chronic HQ for selenium is uncertain because the potential for selenium to pose risk to aquatic life is highly site-specific. Selenium toxicity is manifested via dietary exposure routes rather than through the water, and the amount of selenium in the diet depends on site-specific conditions. A better way to assess potential selenium risks is through the use of tissue residue data for fish, which is described further in the next section. Several acute and chronic HQs also exceeded 1.0 for some organic chemicals, and polycyclic aromatic hydrocarbons (PAHs) in particular. However, these chemicals were rarely detected (detection limits were well above PAH WQC) and, when they were, only at concentrations equal to the detection limit. Accordingly, the exposure estimates for these chemicals are heavily influenced by the detection limits for these chemicals. Regardless, given the hydrophobicity of these chemicals, they are more appropriately addressed through the direct evaluation of sediment chemistry data rather than water quality data.

## ***Tissue***

As mentioned above, fish tissue chemistry data were also used to assess potential risks to aquatic life through direct comparison to toxicity data based on tissue residue. Consistent with the general pattern observed for wildlife receptors, HQs for lead and PCBs exceeded 1.0, with HQ ranges of 0.7 to 4.4 and 0.7 to 2.1, respectively. The lead HQs were largest above RM 8.8 and followed a generally decreasing pattern from upstream to downstream. Since fish are mobile, it is not possible to confidently link the fish concentrations to sources, but the data do provide confirmation that lead and PCBs appear to pose a potential for widespread risk in the lower Ottawa. The selenium concentrations in fish tissue were below

tissue-based toxicity thresholds, suggesting that the chronic HQs calculated for surface water were overestimating the potential risk to aquatic life posed by selenium.

## **Sediment**

Chemical concentrations in sediment were compared to sediment quality guidelines from various sources. Sediment data collected in both 1998 and 2000 were used (separately) to assess potential risks to aquatic life. The 1998 sampling was more widespread than the 2000 sampling, and also included the collection of core samples. The biological relevance of these data is uncertain since they reflect concentrations from depths greater than the biologically active zone. While fewer samples were collected in 2000, the data reflect conditions in the biologically active top 10 cm of sediment. Accordingly, the 2000 data represent more recent and realistic exposure conditions. Based on the 1998 surface data (depths less than 24"), the key COPCs identified from RMs 0 to 8.8 were lead and PCBs. Cadmium and chromium were also identified as COPCs between RMs 3.2 to 4.9. These four chemicals were all considered COPCs because they exceeded at least one sediment guideline based on a high probability for effects. The true risks posed by these chemicals are unknown without further study because there are a number of factors in sediment that can influence chemical bioavailability. Hazard quotients for PAHs reached as high as 0.5 between RMs 6.5 to 8.8 using recently derived guidance. Although the highest HQ for PAHs was less than 1.0, recent studies suggest that the number of PAHs present and potentially contributing to toxicity is often more than the number typically measured. Accordingly, if the full suite of PAHs was measured, it is possible that the HQ would exceed 1.0. The HQs for sediment COPCs tended to decrease at depths greater than 24", probably as a result of the larger compositing volumes.

Using the 2000 sediment data, lead and PCBs were again identified as COPCs. Additional COPCs identified in one or more river segments included other metals (e.g., cadmium, chromium, and nickel) and organochlorine pesticides (e.g., DDD, DDE, dieldrin, and heptachlor epoxide). All of these COPCs (metals, PCBs, and organochlorine pesticides) are very persistent in sediment and thus may reflect both current and historical sources to the river. Acid volatile sulfide-simultaneously extracted metal (AVS:SEM) data were available to assess whether cationic metals in sediment would be bioavailable to aquatic biota (AVS is the primary partitioning phase controlling cationic metal activity and toxicity in the sediment-pore water system). If the simultaneously extracted sum of molar concentrations of cadmium, copper, lead, nickel, silver, and zinc are less than the molar AVS concentrations, toxicity will not be observed. Of the 19 AVS:SEM samples, the molar ratio of SEM (based on the sum of cadmium, copper, lead, nickel, silver, and zinc) to AVS was always greater than 1.0 (mean, minimum, and maximum were 1,664, 1.1, and 8,562, respectively). Accordingly, the AVS:SEM data could not be used to support an absence of metal toxicity in sediment.

The sum of sediment-based HQs greater than 1.0 for aquatic life are plotted in Figure E-3 by river segment. Consistent with the wildlife receptors, the highest potential risk to benthic aquatic life was identified in Segment 3 (RM 4.9 to 6.5). The driver chemical was lead in this river segment, with other heavy metals (cadmium, chromium, nickel) and PCBs also contributing to the potential risk (Table E-1). As mentioned previously, it should be noted that the chemicals listed in Table E-1 do not contribute equally to the potential risk for a given river segment (the HQs associated with each chemical are noted in parentheses next to its respective chemical). Potential risks to benthic aquatic life were also identified in the other river segments, but at lower levels. The lowest potential risk, as for wildlife, was identified at the river mouth (Segment 1, RM 0 to 3.2) (Figure E-3).

## ***Other Lines of Evidence for Aquatic Risk Potential***

As mentioned above, sediment bioassays and biological monitoring results were also used as additional lines of evidence for evaluating potential risks to aquatic life. A total of ten sediment bioassays were conducted with either the amphipod *Hyalella azteca* or the oligochaete worm *Lumbriculus variegatus*. All bioassays were short-term (i.e., acute toxicity) tests, and no toxicity was observed in any test. In one sediment sample, chemistry data were also collected. Although some concentrations exceeded sediment guidelines, the absence of toxicity from the acute bioassays does not necessarily suggest that the guidelines are overly conservative for the site. It is possible that toxicity would have been observed if long-term (i.e., chronic) bioassays were conducted, because these are based on more sensitive toxicity endpoints (growth and reproduction rather than just survival).

The second additional line of evidence used in assessing potential risks to aquatic life was biological monitoring data. These data include indices of fish and macroinvertebrate abundance and species richness. The river segments with the highest predicted HQs did not always correspond with the locations having the lowest (poorest) biological index scores. Nevertheless, this line of evidence confirmed that fish and macroinvertebrate communities are being impacted in the lower Ottawa River.

## **CONCLUSIONS**

Lead and PCBs were consistently identified as COPCs for both wildlife receptors and aquatic life. Lead was primarily identified as a COPC due to its concentrations in sediment, but concentrations in fish tissue also reached levels that have been shown in the laboratory to be directly toxic to fish. Based on the year 2000 sediment data, the largest lead concentration was measured at RM 5.5. This sample also contained maximum concentrations of other metals between RMs 4.9 to 6.5. Accordingly, the river segment from which this sample was collected (Segment 3) was identifying as posing the highest risk to both wildlife and aquatic life (Figure E-3). The PCB HQs for wildlife were influenced by PCB concentrations in fish tissue, which were fairly similar in magnitude along multiple river segments. Similarly, sediment-based PCB HQs for aquatic life were comparable in magnitude across multiple river segments. Accordingly, potential PCB risks to wildlife and aquatic life are fairly evenly distributed throughout the lower Ottawa River. The risks posed by PAHs to bottom-dwelling aquatic organisms are more uncertain. Although the maximum HQ calculated was 0.5, it is possible that HQs of greater than 1.0 would be calculated if all PAHs and their substituted derivatives were measured. The biological monitoring results support that fish and macroinvertebrate communities are being impacted. Additional data that could improve hot spot delineation include temporally and spatially co-located chemistry sampling, with bioassays using more sensitive (chronic) effect endpoints.

# 1. INTRODUCTION

## 1.1 PURPOSE AND SCOPE

The Ottawa River is an urban waterway that drains into North Maumee Bay, Lake Erie (Figure 1-1). There are numerous potential sources of chemicals to the river, including several landfills. Being a tributary to Maumee Bay, the Ottawa River is part of the Maumee Area of Concern, designated in 1985 by the International Joint Commission.<sup>5</sup> In 1991, the Ohio Department of Health issued a fish consumption/contact advisory for the lower 8.8 miles of the river (see Figure 1-1). This advisory was based on polychlorinated biphenyl (PCB) concentrations in sediment and fish tissue. In addition to PCBs, concentrations of heavy metals, pesticides, and other chemicals were also known to be elevated in Ottawa River sediment and tissue. The purpose of this ecological screening-level risk assessment (SLRA) is to assist in prioritizing areas of the lower Ottawa River for possible remediation.

## 1.2 OBJECTIVES

Based on the purpose and scope of the SLRA, three main objectives were identified. The first objective was to identify the nature and magnitude of potential risks to wildlife and aquatic life under recent conditions within the Ottawa River. The subsequent second and third objectives were to identify chemicals of potential concern and prioritize locations within the Ottawa River for possible remediation action.

## 1.3 REPORT ORGANIZATION

The general methods used in the ecological SLRA follow U.S. EPA guidelines for conducting ecological risk assessments at Superfund sites (U.S. EPA 1997). Accordingly, this ecological SLRA consists of four primary components: (1) Problem Formulation; (2) Exposure Characterization; (3) Effects Characterization; and (4) Risk Characterization (Figure 1-2). The Problem Formulation focuses on defining site issues and management goals and how these goals are incorporated in the SLRA process. As such, the Problem Formulation section of this document describes the study area and summarizes the key risk assessment issues and how they are evaluated to help meet the overall management goals. This information is also used to support the ecological receptors selected and evaluated in the Exposure Characterization, and how their exposure is estimated. The Exposure Characterization describes in detail how chemical exposures to the ecological receptors are quantified. The Effects Characterization presents the toxicity thresholds or guideline values that are compared to the exposure estimates. Lastly, the Risk Characterization methods describe how screening potential risks are quantified by integrating the Exposure and Effects Characterizations, as well as by considering other lines of evidence.

---

<sup>5</sup> The Maumee Area of Concern stretches from the lower 23 miles of the Maumee River to Maumee Bay and Lake Erie. The Area of Concern includes 3,942 miles of several streams, including the Ottawa River.



Figure

## 1-2 Framework for Ecological Risk Assessment

## 2. PROBLEM FORMULATION

Ecological risk assessments are designed and conducted to provide information on ecological risk potential to risk managers to assist in identifying management decisions appropriate to the site (U.S. EPA 1998a). Accordingly, understanding the management goals is critical and influences how issues are addressed in the risk assessment. A primary management goal for the Ottawa River SLRA is to identify locations of chemical hot spots in the lower portion of the river and to prioritize these areas for possible future remediation or additional risk assessment. The remainder of this section discusses how the river was divided into segments and identifies the ecological receptors that were evaluated.

### 2.1 OVERVIEW OF STUDY SITE

The lower 9 miles of the Ottawa River represent the critical reaches of the river to be evaluated in the SLRA (specifically, the river mouth to river mile [RM] 8.8) (see Figure 1-1). The river flows through a primarily industrialized area in the city of Toledo, but there is limited development along the banks, and significant riparian zones are present (OEPA 2000). LTI (2000) summarized the distinct zones over this stretch of the river. From RMs 8.8 to 7.0, the river is relatively free-flowing with unidirectional flow. The river flows through a transitional zone from RMs 7.0 to 2.5. In this zone, flow velocities are considerably slower and flow reversals occur in some areas. Bathymetry data suggest that there are two “basins” in this reach, above and below approximately RM 4.0. Downstream of RM 2.5, the magnitudes of flows and frequencies of flow reversals increase.

Based primarily on field observations of human and wildlife uses of the river, the OEPA provided additional characterization of the river (Williams 2000, personal communication). For example, between river RMs 6.5 to 8.8, children have been observed fishing and wading in the river. Between RMs 4.9 to 6.5, there are signs of human use, including dirt bike trails and public access where fishing may occur. Wildlife, including turtles and waterfowl, have also been observed between these river miles which includes wetland habitat. Between RMs 3.2 to 4.9, a depositional zone exists with large areas of exposed sediment under low flow conditions. Turtles and waterfowl have also been observed in this area of the river, as well as children walking along the river banks and fishing at RM 3.6. Lastly, from RM 0 to 3.2, the river widens and experiences high recreational use and frequent flow reversals. Wildlife, including waterfowl, have also been observed between these river miles.

Thus, the lower Ottawa River contains a variety of habitats and potential chemical exposure pathways for ecological receptors. The Ottawa River was delineated into five river segments based on the above considerations: RMs 0 to 3.2 (Segment 1 [mouth]), RMs 3.2 to 4.9 (Segment 2), RMs 4.9 to 6.5 (Segment 3), RMs 6.5 to 8.8 (Segment 4), and > RM 8.8 (Segment 5). Figures 2-1, 2-2, and 2-3 show the urban nature of the river.

### 2.2 CHEMICAL SOURCES

Numerous potential sources of chemical contaminants were identified in the Ottawa River Geographic Initiative Work Plan (OEPA 2000). These sources include mostly industries and landfills, but also include textile producers and fertilizer manufacturers. Many of these sources are shown in Figure 1-1. Several landfills, such as the Tyler Street Landfill, Stickney Avenue Landfill, and Dura Avenue Landfill, have been chemical sources to the Ottawa River, including heavy metals, semi-volatile organics, volatile organics, and PCBs (OEPA 2000). Many of the potential source sites identified no longer have ongoing operations, and several have undergone remediation. The Tyler Street, Stickney Avenue, and Dura Avenue landfills, for example, have all been capped.



**Figure 2-1 Right Bank of Ottawa River at River Mile 5.0**



**Figure 2-2 The Dura Avenue Landfill**



**Figure 2-3 Ottawa River Adjacent to Dura Avenue Landfill**

### **2.3 RECEPTORS OF CONCERN**

Several receptors were evaluated in the SLRA. The avian and mammalian receptors selected for evaluation were: (1) bald eagle (*Haliaeetus leucocephalus*), (2) common tern (*Sterna hirundo*), (3) spotted sandpiper (*Actitis macularia*), and (4) mink (*Mustela vison*). The bald eagle was selected because it feeds on fish and has been reported to feed in the area. Furthermore, there have been reports of unsuccessful breeding in North Maumee Bay (DeVault 2000, personal communication). The common tern was identified as a second fish-eating bird receptor because it is expected to feed throughout the lower Ottawa River (Shieldcastle 2000 personal communication) and has a higher ingestion rate relative to body weight than the bald eagle, and thus is a more highly exposed receptor. The spotted sandpiper was selected as a representative invertebrate-eating bird and is common in reaches of the river with appropriate habitat (Shieldcastle 2000 personal communication). The exposure potential of the sandpiper is quite different from bald eagles or terns because invertebrates tend to bioaccumulate chemicals differently than fish. In addition, being a probing bird, they tend to have higher sediment ingestion rates than many other fish-eating birds. Lastly, the spotted sandpiper has a smaller home range than bald eagles (U.S. EPA 1993a). Thus, it is likely to receive a greater portion of its diet from the Ottawa River. In summary, bird receptors were selected that are most appropriate for chemicals that bioaccumulate in high levels in fish tissue (eagle, tern) and that are most appropriate for chemicals that are elevated in sediment (sandpiper).

There are limited fish-eating mammals that use the Ottawa River (Shieldcastle 2000 personal communication). The river otter is the only mammal likely to exclusively eat fish, but is unlikely to inhabit the lower Ottawa River (Shieldcastle 2000 personal communication). Although the mink is also unlikely to permanently inhabit the lower Ottawa, it is possible that they may feed along the river on occasion as they move between streams which provide better habitat (Shieldcastle 2000 personal communication). In addition, it is known to be very sensitive to one of the key chemicals of concern in the river (PCBs) (Eisler 1986). Accordingly, mink was chosen as the representative mammalian receptor. Mink feed on both fish and macroinvertebrates (e.g., crayfish), as well as birds and mammals (U.S. EPA 1993a). In this SLRA, it was conservatively assumed that they feed solely on fish from the Ottawa River.

The snapping turtle (*Chelydra serpentina*) is an additional wildlife receptor in the lower Ottawa for which data are available. Being a reptile, its sensitivity to chemical contaminants compared to the other wildlife receptors discussed above is uncertain. However, although PCB congener concentrations in turtle eggs are available in turtle tissues, it is difficult to screen risks to reptiles due to the limited toxicological effects data available for this class of organisms. Accordingly, the PCB data for turtle eggs are indicative of exposure, but whether the measured egg levels are toxic is unknown. As discussed in a recent review of reptile toxicology (Hopkins 2000), ecotoxicological studies on reptiles often document tissue concentrations of chemicals, but seldom provide adequate insight on the biological significance of the tissue concentrations measured. Moreover, it is difficult to screen risks based on dietary intake estimations because reptile responses to chemical concentrations are unknown (Hopkins 2000).

Additional ecological receptors evaluated include fish and aquatic invertebrates in the river (surface water and sediment). In addition to evaluating the overall aquatic community as a whole, piscivorous fish (e.g., smallmouth and largemouth bass) were identified as ecological receptors based on their potentially different sensitivity compared to birds and mammals. Moreover, water quality standards (WQS) or criteria (WQC) for protection of biota in surface water tend to be based on water-only exposures (i.e., gill uptake). Evaluation of piscivorous fish allowed screening risks to be estimated for these receptors using tissue residue data, which reflect gill uptake and dietary exposures and are particularly important for hydrophobic organic compounds such as PCBs.

Based on the above discussions, the receptors selected for evaluation in the ecological SLRA, and the exposure pathways for each, are summarized in Table 2-1 below.

**Table 2-1. Ecological Receptors to be Evaluated in the SLRA and Their Routes of Exposure**

<b>Ecological Receptor</b>	<b>Exposure Pathway</b>
Bald eagle	Tissue, water, and sediment ingestion
Common tern	Tissue, water, and sediment ingestion
Spotted sandpiper	Tissue, water, and sediment ingestion
Mink	Tissue, water, and sediment ingestion
Piscivorous fish	Tissue ingestion
Aquatic community	Direct contact with surface water and sediment

## **2.4 ASSESSMENT AND MEASUREMENT ENDPOINTS**

Assessment endpoints are explicit statements of the environmental values to be protected at the site (U.S. EPA 1997). The assessment endpoints are defined not only in terms of environmental entities (e.g., fish community) and properties of those entities (e.g., species richness), but also identify the level of effect on

those properties that should be detected or estimated (Cook et al. 1999). Cook et al. (1999), for example, used 20 percent reduction in one of the endpoint properties in the field or a 20 percent reduction in survivorship, growth, or reproduction in a toxicity test as potentially significant. In this SLRA, toxicity data were based on survival, growth, or reproductive effects that were not significantly different than control organisms. When such data were not available, low effects levels were estimated as described in the Effects Characterization. This level is the lowest level of effects that standard field and laboratory techniques can detect with conventionally acceptable confidence.

Measurement endpoints are the measurable characteristics that are related to the valued characteristic chosen as the assessment endpoint (U.S. EPA 1997). In other words, the measurement endpoints represent the types of data (exposure or toxicity; site-specific or literature-based) that are used to determine whether there is an affect on the assessment endpoints. In this SLRA, measurement endpoints include literature-derived chemical toxicity data and site-specific data on species richness and diversity; bioassay tests for aquatic organisms; and sediment, water, and tissue concentration data. Multiple measurement endpoints (i.e., lines of evidence) were evaluated, when possible, because it provides more accurate estimates of effects and more reliable estimates about causation than exclusive reliance on modeling risks from concentrations of chemicals in ambient media (Suter 1993). Consideration of multiple lines of evidence in a “weight-of-evidence analysis” was the approach used in the Ottawa River SLRA. Table 2-2 summarizes the assessment and measurement endpoints of the Ottawa River SLRA.

**Table 2-2. Ecological Assessment and Measurement Endpoints Used in the Lower Ottawa River SLRA**

<b>Receptor</b>	<b>Assessment Endpoint</b>	<b>Receptor Type</b>	<b>Measurement Endpoints</b>
Aquatic wildlife	Reduction in abundance or production of piscivorous wildlife populations resulting from toxicity	Bald eagle population Common tern population Spotted sandpiper population Mink population	Receptor toxicity data (literature) Water concentrations (site) Sediment concentrations (site) Tissue concentrations (site)
Aquatic biota, pelagic	Reduction in species richness or abundance resulting from toxicity	Aquatic community	Biological survey data (site) Receptor toxicity data (literature) Water concentrations (site) Tissue concentrations (site)
Aquatic biota, sediment	Reduction in species richness or abundance in benthic communities resulting from toxicity	Benthic community	Biological survey data (site) Receptor toxicity data (literature) Bioassay data (site) Sediment concentrations (site)

## 2.5 CONCEPTUAL SITE MODEL

The above information on chemical sources, ecological receptors evaluated, and assessment/measurement endpoints were integrated into a conceptual site model. Thus, the conceptual model graphically depicts the relationships between site-specific assessment endpoints and exposure scenarios. The conceptual models for wildlife and aquatic life are provided in Figures 2-4 and 2-5, respectively.

As shown in the figures, chemicals enter the Ottawa River from a variety of sources, including leachate, surface runoff, permitted discharges, and groundwater. Once in the system, chemicals may enter the water column or sediments through resuspension/deposition and absorption/desorption. Biota may then accumulate chemicals via exposure to either sediment or surface water.

As shown in Figure 2-4, food ingestion is a significant pathway for all wildlife receptors evaluated. For the spotted sandpiper, sediment ingestion also represents a significant exposure pathway since they are probing feeders; for the remaining wildlife, sediment ingestion is expected to be a complete exposure pathway, but insignificant (see Figure 1-1). Lastly, for all wildlife receptors, surface water ingestion is considered an insignificant exposure pathway.

As shown in Figure 2-5, aquatic life are primarily exposed to chemicals in surface water via their gills. Aquatic life exposure to chemicals in tissue and sediment occurs most significantly via ingestion pathways (see Figure 2-5).

Figure

**2-4 Conceptual Model for Wildlife**

Figure

## 2-5 Conceptual Model for Aquatic Life

## 3. EXPOSURE CHARACTERIZATION

This section describes how the exposure of ecological receptors to chemicals in various environmental matrices (i.e., water, sediment, tissue) was quantified. The section begins with a description of the environmental concentrations used, followed by a description of the approach used for quantifying exposure to each receptor.

### 3.1 CHEMICAL CONCENTRATIONS

#### 3.1.1 Measured Concentrations

Chemistry data for water, sediment, and tissue were available for various reaches of the lower Ottawa River (LTI 2001). The major classes of chemicals that have been measured include metals, polychlorinated biphenyls (PCBs), organochlorine pesticides, and polycyclic aromatic hydrocarbons (PAHs). To address the management goals (i.e., hot spot delineation) mentioned in the Problem Formulation, both the acute and chronic exposure potential of ecological receptors were evaluated.

The chemistry data were used to estimate both acute and chronic exposure concentrations. The sediment, tissue, and water chemistry data used in the SLRA were derived from a variety of studies conducted over a period of several years. Media data were reviewed to determine which data were most representative for determining organism exposure concentrations. Some of the sediment data were quite old (e.g., from 1994), while some tissue data were based only on fillets and were thus not representative of wildlife exposures.

Sediment chemistry has been the most extensively sampled, with recent large sampling events occurring in 1998 and 2000. None of the sediment data from these two studies were combined in estimating exposure concentrations because they were temporally and spatially different (i.e., collected at different times and from a variety of different depths). The sediment data from 2000 were deemed the most appropriate to the SLRA since they most accurately reflect current conditions and represent surface sediments (i.e., the biologically active zone). However, the sediment data from 1998 were also used to screen risks to aquatic life. The 1998 sediment data were composited over a variety of different depths. For the purposes of the SLRA, the sediment composited over a depth of 0-24" was considered surficial sediment, although it must be emphasized biota would not be exposed to sediment up to a depth of 24". Sediment data from depths greater than 24" were also used as an estimation of potential exposure concentrations if overlying sediment was removed from remediation activities. The 1998 sediment data were not used for estimating exposure of wildlife receptors since their exposure represents a concurrent combination of sediment, water, and tissue concentrations that are all in equilibrium. Accordingly, only the sediment, water, and tissue data collected concurrently were used to screen wildlife risks. Fish tissue data were collected in 1999 and 2000 in the Ottawa River, and in 1999 in North Maumee Bay. The 1999 data for the Ottawa River were not used in the wildlife SLRA since only fillets were analyzed and may underestimate the whole body concentrations to which wildlife are exposed. The 2000 data, in addition to being the most current data, also measured whole body concentrations. The 1999 North Maumee Bay data were used to screen risks to bald eagles, as requested by the U.S. FWS. Lastly, chemistry data for surface water were available from one sampling event in 2000. These data were used in both the wildlife and aquatic life SLRAs. A summary of the chemistry data used in the SLRA is provided in Table 3-1.

**Table 3-1. Summary of Chemistry Data Used in Wildlife and Aquatic Life SLRAs<sup>a</sup>**

<b>Media</b>	<b>Year</b>	<b>Location</b>	<b>Chemicals</b>	<b>Receptor Evaluated</b>
Sediment	1998	Ottawa River (RMs 0.1-12.5)	SVOCs, Metals	Aquatic Life
	2000	Ottawa River	SVOCs, Metals	Aquatic Life, Wildlife
Fish Tissue	1999	North Maumee Bay	Pesticides, PCBs	Bald Eagle
	2000	Ottawa River	Pesticides, PCBs, Metals	Bald Eagle, Common Tern, Mink
Water	2000	Ottawa River	SVOCs, Metals	Aquatic Life, Wildlife

<sup>a</sup> See LTI (2001) for a detailed description of the chemistry database used in the SLRA.

Consistent with U.S. EPA risk assessment guidance (U.S. EPA 1998a), data were first screened using frequency of detection to identify which chemicals should be evaluated. On a medium-specific basis, only those chemicals detected in more than 5 percent of the samples were evaluated in the SLRA. For example, if a chemical was detected in sediment in more than 5 percent of the samples, but never in surface water, ecological receptor exposures to that chemical were evaluated in sediment but not in surface water. For those chemicals detected in more than 5 percent of the samples, chronic exposure concentrations were estimated by river segment, using the 95 percent upper confidence limit (UCL) on the mean. This is a typical approach used by the U.S. EPA (Suter et al. 1999). For acute exposure, the 95<sup>th</sup> percentile of the data set for a river segment was used to estimate a short-term upper bound exposure. When a chemical was not detected in an individual sample, but was detected in more than 5 percent of the samples, one-half the detection limit was used in the statistics. This is a common approach for handling non-detect data (Suter et al. 1999). The statistical equations used to calculate acute and chronic exposure concentrations are provided in Appendix A. Summary statistics for tissue, sediment, and surface water are also provided in Appendix A.

### **3.1.2 Estimated Concentrations**

As summarized above, concentration data were available for a variety of chemical classes and environmental media. However, to thoroughly evaluate exposure of all receptors to the chemicals likely to be present in the Ottawa River, it was necessary to estimate the concentrations of some chemicals in tissue. For example, no tissue chemistry data were available for macroinvertebrates in the lower Ottawa River; however, these data were necessary for estimating potential risks to the spotted sandpiper. Because benthos are primarily exposed to chemicals associated with sediment (e.g., pore water, detritus), chemical concentrations in invertebrate tissue were estimated from sediment concentrations using a biota-sediment accumulation factor (BSAF). This approach is applicable to lipophilic organic chemicals (i.e., chemicals that tend to partition into lipids rather than water) (Tracey and Hanson 1996). Concentrations of inorganics in benthos could not be estimated using an analogous approach because site-specific factors that influence metal bioavailability in sediment are highly variable (Ankley et al. 1996) and would impart unreasonable uncertainty in the exposure estimates. BSAFs and the equation for estimating invertebrate tissue concentrations from sediment are presented in Section A.2 of Appendix A.

## **3.2 EXPOSURE QUANTIFICATION**

The chemistry data discussed in Section 3.1 were used to quantify exposures of wildlife and aquatic life receptors as discussed in the following sections.

### 3.2.1 Wildlife

Exposure of avian and mammalian receptors to chemicals in the Ottawa River were estimated using tissue (measured and estimated), sediment, and water data. Chemical exposure of these organisms was expected to occur primarily through food consumption, although they may also receive significant exposure via incidental sediment ingestion (particularly for probing feeders, such as the spotted sandpiper). Water exposures were expected to be relatively insignificant compared to dietary and sediment exposures, but were also evaluated for completeness. Dermal and inhalation exposure pathways were not evaluated in the SLRA. It is expected that the fur or feathers of wildlife receptors will minimize the dermal uptake of chemicals, while inhalation is expected to be insignificant for the relatively non-volatile chemicals being evaluated in the lower Ottawa River.

Chemical exposure of avian and mammalian wildlife receptors was evaluated by estimating daily oral doses. These doses were expressed as milligram chemical per kilogram body weight per day (mg/kg/d). Accordingly, estimates of receptor ingestion rates (food, sediment, and water) and body weights were required. Conservative (i.e., worst-case) ingestion rate and body weight assumptions were used where possible due to the screening nature of the assessment. Furthermore, it was conservatively estimated that receptors may feed exclusively within a given river segment. The following describes the assumed ingestion rates and body weights for each receptor.

#### Bald Eagle

Bald eagles are primarily carrion feeders, but will also catch live fish (U.S. EPA 1993a). In addition, they feed opportunistically on birds and mammals that are easily scavenged or captured (U.S. EPA 1993a). In the SLRA, it was conservatively assumed that eagles feed exclusively on fish (no bird or mammalian tissue data were available for this study). The chemical concentrations in fish were based on samples from Maumee Bay in 1999 and the lower Ottawa River in 2000.

Body weights of adult bald eagles were identified in the literature (Dunning 1993; Stalmaster 1987), and assumed to represent the body weights of eagles in the study area. Mean body weights reported in these studies ranged from 4.13 to 4.33 kg for males and 5.35 to 5.27 for females. However, no data were identified on the differences in food ingestion rates between males and females. According to independent studies reported in U.S. EPA (1993a) and Stalmaster (1987), the daily food ingestion rate of adult eagles is equivalent to approximately 12 percent of their body weight on a wet-weight basis. The mean body weight for females (5.31 kg) and food ingestion rate of 0.64 kg/day (12 percent of body weight) were used in this SLRA.<sup>6</sup>

No data were identified on sediment ingestion rates for eagles, although it is likely they will ingest some sediment when scavenging along shorelines. In this assessment, it was assumed that the sediment ingestion rate is equal to 1 percent of the eagle food diet. No data on sediment ingestion rates for the bald eagle were identified. An ingestion rate of 1 percent, on a wet-weight basis, was estimated given the sediment ingestion rates of other birds reported in U.S. EPA (1993). Data were also not identified on bald eagle water ingestion rates. Thus, an allometric equation based on body weight was used (U.S. EPA 1993a). The water ingestion rate was estimated as shown in Equation 1:

---

<sup>6</sup> Regardless of the body weight assumed, the ingestion rate used to estimate exposure is based on 12 percent of the body weight. Accordingly, it is irrelevant which body weight is assumed.

$$IR_{\text{water}} = 0.059 \times BW^{0.67} \quad (1)$$

Where:  $IR_{\text{water}}$  = Water ingestion rate (L/day)  
 $BW$  = Body weight (kg)

The bald eagle exposure assumptions that were used are summarized in Table 3-2.

**Table 3-2. Ingestion Rate and Body Weight Values Used for Avian and Mammalian Receptors**

Receptor	Exposure Parameter	Value	Units	Reference
Bald eagle	Food IR	0.64 (12% of BW)	kg/day-wet	U.S. EPA 1993a; Stalmaster 1987
	Sediment IR	0.0064 (1% of food IR)	kg/day-wet	Professional judgement
	Water IR	0.18	L/day	U.S. EPA 1993a
	Body Weight	5.31	kg	Dunning 1993; Stalmaster 1987
Common tern	Food IR	0.0732	kg/day-wet	U.S. EPA 1993a
	Sediment IR	0.000732 (1% of food IR)	kg/day-wet	Professional judgement
	Water IR	0.014	L/day	U.S. EPA 1993a
	Body Weight	0.120	kg	Dunning 1993
Spotted sandpiper	Food IR	0.035	kg/day-wet	U.S. EPA 1993a
	Sediment IR	0.0012	kg/day-wet	U.S. EPA 1993a
	Water IR	0.0066	L/day	U.S. EPA 1993a
	Body Weight	0.0379	kg	U.S. EPA 1993a
Mink	Food IR	0.229	kg/day-wet	U.S. EPA 1993a
	Sediment IR	0.00458	kg/day-wet	U.S. EPA 1993a
	Water IR	0.10	L/day	U.S. EPA 1993a
	Body Weight	1.040	kg	U.S. EPA 1993a

IR = Ingestion rate.

### Common Tern

Body weights for the common tern average approximately 0.120 kg (Dunning 1993). The common tern food ingestion rate was estimated using an allometric equation dependent on body weight (U.S. EPA 1993a). The dry-weight ingestion rates calculated by this equation were converted to wet weights to ensure conformity with other data used in estimating common tern risks. The wet-weight ingestion rate was estimated based on the percent moisture in tern food items (approximately 80 percent in fish). The allometric relationship shown in Equation 2 was used:

$$IR_{\text{food}} = (0.0582 \times BW^{0.651}) \times \frac{1 \text{ kg wet matter}}{0.2 \text{ kg dry matter}} \quad (2)$$

Where:  $IR_{\text{food}}$  = Food ingestion rate (kg/day-wet)  
 $BW$  = Body weight (kg)

No data were identified on sediment ingestion rates for the tern. However, given their feeding habits, sediment ingestion is expected to be minimal. A sediment ingestion rate equal to 1 percent of its food ingestion rate was assumed. The water ingestion rate for the common tern was estimated using the allometric equation presented previously for the bald eagle (Equation 1). The exposure parameter values for the common tern are summarized in Table 3-2.

### Spotted Sandpiper

Body weights of male and female spotted sandpipers were identified in the scientific literature and, as for the bald eagles, were substantially different between sexes (0.0379 kg for males and 0.0471 kg for females) (U.S. EPA 1993a). The spotted sandpiper food ingestion rate was estimated using the same allometric equation used for the common tern (Equation 2). The wet-weight ingestion rate was again estimated based on the percent moisture in sandpiper food items (assumed to be 80 percent). Because birds with a lower body weight tend to have a higher ingestion rate to body weight ratio (as demonstrated by this allometric equation), chemical exposures to the smaller males were conservatively estimated.

Due to their probing feeding habits, spotted sandpipers were assumed to have a significant sediment ingestion rate. While spotted sandpiper sediment ingestion rates were not identified, they were available for the semipalmated, Western, stilt, and least sandpipers (U.S. EPA 1993a). Sediment ingestion rates for these four sandpipers, estimated as the percent soil in the diet on a dry-weight basis, average 18 percent. The water ingestion rate was estimated using the same equation used for the bald eagle (Equation 1). The sandpiper exposure values used are summarized in Table 3-2.

### Mink

Mink will feed on both fish and aquatic invertebrates, as well as birds and mammals (U.S. EPA 1993a). According to a study of mink diets in a Michigan stream, 85 percent of their diet (year-round) was comprised of fish (U.S. EPA 1993a). The remainder of their diet included crustaceans, amphibians, birds, and mammals. Based on the available chemistry data for the lower Ottawa River, it was assumed that fish comprise 100 percent of the mink diet.

Mink body weights can be highly variable depending on their range (U.S. EPA 1993a). Body weights for males in Montana have been observed to range from 1.040 to 1.233 kg for adults and 0.777 to 0.952 kg for juveniles, depending on season (U.S. EPA 1993a). In the same study, body weights for females ranged from 0.550 to 0.586 for adults and 0.533 to 0.582 for juveniles. The estimated year-round food ingestion rate of 22 percent of body weight for adult males reported in U.S. EPA (1993a) was used to estimate dietary exposures. Using the adult summer body weight of 1.0 kg results in an estimated dietary intake of 0.229 kg/day wet weight.

No data were identified on sediment ingestion rates for mink; a sediment ingestion rate of 2 percent of their dietary intake was assumed. Data were also not identified on water ingestion rates for the mink.

Using an allometric equation for mammals based on body weight, the water ingestion rate was estimated using the relationship shown in Equation 3 (U.S. EPA 1993a):

$$IR_{\text{water}} = 0.099 \times BW^{0.90} \quad (3)$$

Where:  $IR_{\text{water}}$  = Water ingestion rate (L/day)  
 $BW$  = Body weight (kg)

The final body weight and food, sediment, and water ingestion rates that were used to evaluate the mink are summarized in Table 3-2.

### 3.2.1.1 Wildlife Dose Calculation

Using the ingestion rates and body weights identified above, and the chemistry data for receptor food items, sediment, and water, chemical doses to wildlife receptors were estimated as shown in Equation 4:

$$\text{Chemical Dose (mg/kg/d)} = \frac{(C_{\text{food}} \times IR_{\text{food}}) + (C_{\text{sed}} \times IR_{\text{sed}}) + (C_{\text{water}} \times IR_{\text{water}})}{BW} \quad (4)$$

Where:  $C_{\text{food}}$  = Chemical concentration in food (mg/kg wet weight)  
 $IR_{\text{food}}$  = Food ingestion rate (kg/day wet)  
 $C_{\text{sed}}$  = Chemical concentration in sediment (mg/kg wet weight)  
 $IR_{\text{sed}}$  = Sediment ingestion rate (kg/day wet)  
 $C_{\text{water}}$  = Chemical concentration in water (mg/L)  
 $IR_{\text{water}}$  = Water ingestion rate (L/day)  
 $BW$  = Body weight (kg)

As discussed previously, acute exposures were evaluated using upper bound estimates of the concentration data (to facilitate identification of potential hot spots), and chronic exposures were estimated using conservative estimates of the mean exposure concentration. These exposure estimates tend to be conservative because they assume that the chemicals in the food and adsorbed to the ingested sediment are 100 percent bioavailable for uptake. Use of worst-case exposure estimates is the typical approach used in SLRAs, with the main goal being to eliminate chemicals that are clearly not of concern. As discussed in later sections of this document, wildlife doses were compared to wildlife toxicity threshold doses to estimate the potential for risk.

### 3.2.2 Aquatic Life

Potential risks to aquatic life were evaluated at the community level through direct exposure to chemicals in surface water or sediment. This is the typical approach used for deriving water and sediment quality criteria (Stephan et al. 1985; Di Toro and McGrath 2000). Thus, exposure was simply estimated by directly using the surface water and sediment chemistry data (specifically the 95 percent UCL on the mean for chronic exposures and the 95<sup>th</sup> percentile of the data set for acute exposures). Although aquatic organisms will have highly variable mobility in a system, it is reasonable that many species will have relatively small home ranges, particularly benthos (i.e., sediment-dwelling organisms). Therefore, both acute and chronic exposure concentrations for aquatic communities were estimated by river segment for consistency with the management goals summarized in Section 2.

Although less emphasis has historically been placed on the dietary uptake of chemicals by fish, it has become increasingly realized that this pathway is more important than the water exposure typically addressed by WQS for a variety of species and types of chemicals (Suedel et al. 1994). For example, piscivorous fish are likely to have much greater exposure to PCBs through the dietary pathway than through other pathways. The importance of the dietary route for metals has also received greater attention (Szebedinszky et al. 2001) and was evaluated where data were available. Given the difficulties in estimating chemical doses to fish, due to the lack of data, exposure was estimated by considering tissue residue concentrations. These were compared to tissue residue toxicity thresholds, as discussed later.

## 4. EFFECTS CHARACTERIZATION

This section presents and discusses the toxicity thresholds (hereafter referred to as Toxicity Reference Values, or TRVs) for wildlife and aquatic life receptors. These TRVs were compared to the estimated doses and exposure concentrations described in Section 3 to estimate potential risks to ecological receptors.

### 4.1 WILDLIFE

#### 4.1.1 Toxicity Thresholds

To evaluate a chemical's toxicity (i.e., direct toxicity and/or food chain effects) to wildlife receptors, acute and chronic toxicological effects data were obtained from the scientific literature. The acute toxicity data used were reported as LD50s (i.e., the dose lethal to 50 percent of the organisms tested). Using the U.S. EPA's approach for aquatic life LC50s (Stephan et al. 1985), LD50s were divided by two to estimate a dose that would affect much fewer than 50 percent of the organisms.

The chronic toxicity data used were No Observed Adverse Effect Levels (NOAELs). A NOAEL is the highest concentration tested in a toxicity study that did not result in statistically significant effects when compared to the controls. If a NOAEL was not available, a Lowest Observed Adverse Effect Level (LOAEL) (i.e., the lowest concentration tested resulting in a statistically significant effect) was used with a safety factor of 10 applied to estimate the NOAEL (per U.S. EPA 1997). The NOAELs or LOAELs used were generally based on adverse effects on reproduction, growth, and development, consistent with the toxicity endpoints traditionally evaluated (U.S. EPA 1997). The acute and chronic TRVs are provided in Tables B-1 and B-2 of Appendix B, respectively.

With the exception of the limited toxicity data available for mink, toxicity data were not available for the site-specific receptors being evaluated. Consequently, toxicity data for surrogate species (e.g., rat, chicken, quail) were used. For mammals, scaling the toxicity dose based on the body weight of the test and site-specific receptor species is recommended (Travis and White 1988; Travis et al. 1990; U.S. EPA 1992). Research has demonstrated that numerous physiological functions, such as metabolic rates and responses to toxic chemicals, are functions of body size (Sample et al. 1996). Differences in metabolic rates can lead to more resistance to toxic chemicals because of the rate of detoxification through metabolism and excretion of the chemical (Sample et al. 1996). However, body weight scaling is not considered appropriate for birds (Fischer and Hancock 1997). For birds, differences in toxicological reactions appear to be more a factor of whether the species is passerine or non-passerine (Fischer and Hancock 1997), although no relationships are available on the relative sensitivities of passerine and non-passerine birds. The equation for body weight scaling of mammalian TRVs is provided in Appendix B.

#### 4.1.2 Water Quality Criteria for Wildlife

The Ohio EPA has developed WQC for wildlife for a limited number of chemicals, typically those substances with a propensity to biomagnify in food chains, such as DDT, mercury, and PCBs (OEPA 3745-1) (Table 4-1). These values were developed by back-calculating water concentrations using dietary toxicity thresholds, bioaccumulation factors (BAFs), biomagnification factors (BMFs), and assumptions on wildlife receptor food ingestion rates and body weights. These WQC values were used as an additional line of evidence in characterizing risks to wildlife. It must be emphasized that the criteria represent very low water concentrations, in many cases below the detection limits achieved in the Ottawa River 2000 water sampling event. Accordingly, it is uncertain whether undetected levels of these chemicals are of concern.

**Table 4-1. Ohio EPA Water Quality Criteria for Wildlife**

<b>Chemical</b>	<b>Wildlife Criterion (µg/L)</b>
Mercury	0.0013
PCBs	0.00012
DDT (and its metabolites)	0.000011

## **4.2 AQUATIC LIFE**

### **4.2.1 Surface water**

A hierarchy of sources was used for identifying water-based acute and chronic toxicity thresholds for aquatic life:

1. Ohio Environmental Protection Agency (OEPA 3745-1)
2. U.S. Environmental Protection Agency (U.S. EPA)
3. The general scientific literature.

WQC from both the OEPA and the U.S. EPA are intended to protect at least 95 percent of the species in a generic aquatic community. For many of the metals, the WQC is hardness-dependent. Generally speaking, a decrease in water hardness results in an increase in the bioavailability and, subsequently, the toxicity of certain divalent metals. Thus, the lower the hardness, the lower the WQC. In order to ensure protection of aquatic life at a range of hardness values at the site, a conservatively low hardness based on the lower 95 percent confidence limit on the mean hardness for the river was used (on a segment-by-segment basis).

WQC for two other chemicals were also based on water quality parameters. The acute WQC for ammonia and the acute and chronic WQC for pentachlorophenol are dependent upon the pH. Additionally, the chronic WQC for ammonia is dependent upon both pH and temperature. Ammonia exists in two forms in the environment: ionized ammonia and un-ionized ammonia. Un-ionized ammonia is much more toxic to aquatic life than the ionized form (U.S. EPA 1998b). The equilibrium between these two increasingly favors un-ionized ammonia as the pH and temperature increases. As pH and temperature increase, the equilibrium between ionized and un-ionized ammonia shifts, increasing the un-ionized fraction. Because ammonia is most toxic in the un-ionized form, conditions of elevated pH and temperature, which cause an increase in the un-ionized fraction, correspond with a lower ammonia criterion. In this SLRA, a conservatively high pH and temperature, based on the upper 95 percent confidence limit on the means of pH and temperature, were used.

For pentachlorophenol, the pH relationship is the reverse of that for ammonia. As the pH decreases, the toxicity increases and the criterion decreases. For this SLRA, a conservatively low pH based on the lower 95 percent confidence limit on the mean pH was used.

When neither OEPA nor U.S. EPA WQC were available for a chemical, a toxicity threshold was identified from the scientific literature, generally using the U.S. EPA's AQUIRE database. Acceptable studies were identified using U.S. EPA guidelines (Stephan et al. 1985). If chronic toxicity data were lacking, either completely or for sensitive species, a chronic toxicity threshold was estimated using a

chemical specific acute-chronic ratio (ACR)<sup>7</sup> or generic ACR of 10 (U.S. EPA 1991a). The acute and chronic TRVs used to characterize aquatic effects are presented in Table B-3 of Appendix B.

#### 4.2.2 Sediment

A variety of sources have published freshwater sediment quality guidelines, including:

- Ontario Ministry of the Environment and Energy (1993)
- Environment Canada (1995)
- Ingersoll et al. (1996)
- Di Toro and McGrath (2000)

The sediment guidelines from these sources are presented in Table B-4 of Appendix B. The Ingersoll et al. (1996) guidelines identify four levels of protection. These guidelines provide sediment concentrations where there is a low likelihood of effects (Effects Range Low [ER-L] and Threshold Effect Levels [TEL]) as well as concentrations where effects are more likely to occur (Effects Range Median [ER-M] and Probable Effect Levels [PEL]). When a sediment concentration falls below ER-L and TEL values, effects are rarely observed. In contrast, the probability of effects is more frequent (generally greater than 50 percent) when concentrations exceed ER-M and PEL values (Ingersoll et al. 1996; Long et al. 1998). It should be noted that an exceedance of any one of these sediment guidelines does not necessarily mean that aquatic life are at risk. This is because the sediment guidelines are not site-specific, are conservative, and do not always indicate an effect will actually occur when exceeded (Long et al. 1998). Much of the toxicity data used to develop such guidelines are based on whether effects were observed in bioassays of field-collected samples. Accordingly, if effects were observed, the toxic effect level is assumed to be related to the concentration of an individual chemical in the sample, when in fact, it is likely that a variety of chemicals contributed to the observed toxicity. With these facts in mind, an approach was developed for predicting potential risks to benthos using these guidelines and is discussed in the Risk Characterization (Section 5).

For PAHs, only the guidelines derived by Di Toro and McGrath (2000) were used. According to Di Toro and McGrath, the empirical guidelines (e.g., ER-L, ER-M values) for individual PAHs are one to two orders of magnitude smaller than the narcotic concentrations that are known to cause mortality, growth, or reproduction effects and therefore are not reflective of actual effects concentrations for these endpoints. This likely occurs due to the presence of multiple chemicals in the toxicity studies used to drive the guidelines for individual chemicals (see above). Moreover, for sediment PAH guidelines that are not organic carbon-normalized (Ingersoll et al. and Environment Canada are not), there is a one order of magnitude uncertainty due to the variation in organic carbon concentrations in sediment that they do not account for (Di Toro and McGrath 2000). Use of the methodology presented in Di Toro and McGrath (2000) also reflects the current state of the science by the U.S. EPA in deriving sediment quality criteria for chemicals (U.S. EPA 2000a). Use of these guidelines also presents a method for evaluating PAH mixtures, since many individual PAHs typically co-occur. This method, as well as the technical basis for these guidelines, is presented in Appendix B.

---

<sup>7</sup> As the name implies, an acute-chronic ratio is the ratio of the acute toxicity value for an organism to the chronic toxicity value for the same organism. The acute and chronic values should be developed as part of the same study (Stephan et al. 1985).

### **4.2.3 Tissue Residues**

As discussed, potential risks based on tissue residue data were also evaluated for fish. Accordingly, tissue residue toxicity thresholds were identified for fish where available. Because neither the OEPA nor U.S. EPA has developed tissue-based criteria for fish, all toxicity thresholds were identified from the scientific literature. Tissue-based toxicity data were largely compiled using the recent thorough data summary of Jarvinen and Ankley (1999). The residue-based toxicity thresholds for fish are summarized in Table B-4 of Appendix B.

## 5. RISK CHARACTERIZATION

The Risk Characterization integrates the Exposure and Effects Characterizations to assess whether chemical concentrations are sufficiently high to pose unacceptable risks to ecological receptors. It should be emphasized that this SLRA, where possible, incorporated conservatism where uncertainties were apparent, which is typical for a screening analysis (i.e., risks are likely to be overestimated rather than underestimated). This allows for chemicals posing negligible risk to be confidently removed from further evaluations. The risk characterization approach is summarized in Figure 5-1. The chemicals identified as being of potential concern (i.e., COPCs) may be evaluated further in more detailed site-specific assessment to further characterize the risks they pose. The following sections present the risk characterizations for wildlife and aquatic life, respectively.

### 5.1 WILDLIFE

Potential risks to wildlife receptors were evaluated using two lines of evidence: (1) comparison of estimated chemical doses to TRVs; and (2) comparison of surface water concentrations to Ohio EPA WQC for wildlife. For each line of evidence, a quotient approach (U.S. EPA 1997) was used.

#### 5.1.1 Dose-Based Hazard Quotients

A dose-based quotient represents the ratio of the estimated chemical dose to a receptor to chemical- and receptor-specific TRVs. In this SLRA, this ratio was termed a hazard quotient (HQ) and was derived as shown in Equation 5:

$$HQ_{\text{dose}} = \frac{\text{Chemical Dose (mg/kg/d)}}{\text{Toxicity Reference Value (mg/kg/d)}} \quad (5)$$

An HQ value greater than 1.0 suggests that a chemical *potentially* poses unacceptable risk, while an HQ value of less than 1.0 suggests negligible risks (due to the conservative assumptions that were used in the Exposure and Effects Characterizations)<sup>8</sup>. This approach is useful as an efficient means of identifying high or low risk situations (U.S. EPA 1998a). As such, it is a useful tool for addressing the management goals of identifying chemical hot spots and prioritizing portions of the lower Ottawa River for possible additional assessment or remediation activities. Additional evaluations may be necessary to further delineate these possible remediation areas since HQs provide limited information on the incremental quantification of risks (i.e., an HQ of 30 does not represent “twice the risk” as an HQ of 15). The HQs for the bald eagle, common tern, spotted sandpiper, and mink are summarized and discussed separately below.

##### 5.1.1.1 Bald Eagle

As discussed in Section 3, potential exposures (acute and chronic) of bald eagles to chemicals in the Ottawa River and North Maumee Bay were evaluated. In the Ottawa River, potential chemical exposures based on tissue, sediment, and surface water data were evaluated, while in Maumee Bay, only concentrations in fish tissue were evaluated given the available data. Acute and chronic HQs by river segment are provided in Appendix C, Tables C-1 and C-2, respectively.

<sup>8</sup> It is not possible to reasonably differentiate chemicals as posing low, moderate, or high potential risk for those with HQs greater than 1.0, because the conservatism in the data and assumptions used are chemical-specific. As a general guideline, however, HQs greater than 10 likely suggest a high potential risk.

Figure

## 5-1 Risk Characterization Approach

Comparison of acute exposures to acute TRVs did not result in any HQs greater than 1.0. Comparison of chronic exposure estimates to chronic TRVs did result in chronic HQs greater than 1.0 for lead, PCBs, and DDT. The HQs for these chemicals are plotted by river segment in Figure 5-2.

A lead HQ of approximately 2.5 occurs in the river segment encompassing RMs 4.9 to 6.5; lead HQs are less than 1.0 in the other river segments. This HQ is highly influenced by lead concentrations in the sediment of this river segment, which range from 43 to 13,130 mg/kg-wet (a range greater than two orders of magnitude). More specifically, the lead HQ is highly affected by the sample with the highest measured lead concentration. The next highest concentration measured was 71 mg/kg-wet. The sample with the lead concentration of 13,130 mg/kg-wet may reflect a hot spot of metals concentrations. Although not at the same magnitude, maximum concentrations of cadmium, chromium, copper, mercury, nickel, silver, vanadium, and zinc were all measured in this same sample, which suggests that the elevated lead levels were not an artificial circumstance of the sample, such as from lead shot.

PCB HQs exceeded 1.0 in all river segments except upstream of RM 8.8. The highest HQ, approximately 4.4, was calculated in the river segment encompassing RMs 3.2 to 4.9. All PCB HQs were influenced by Aroclor 1242 PCB levels measured in fish tissue. The fish tissue samples resulting in the highest HQ were collected near the Hoffman Road landfill at RM 3.4.

A DDT HQ of approximately 1.7 occurs upstream of RM 8.8; DDT HQs are less than 1.0 in all other river segments. All DDT HQs were influenced by concentration in the eagle diet (i.e., fish). The maximum concentration measured above RM 8.8 was 33.5 µg/kg-wet in green sunfish (upstream of the University of Toledo dam). DDT HQs successively decline from above RM 8.8 toward RM 0.

It should be reemphasized that these HQs (lead, PCBs, and DDT) are conservative because they assumed that eagles fed exclusively in any individual river segment over a chronic exposure duration. Actual risks from lead are probably significantly lower and could have been below risk thresholds had feeding ranges been considered in the analysis. Nevertheless, these results, though conservative, provide information on locations of possible concern that were compared with other lines of evidence in later sections.

#### **5.1.1.2 Common Tern**

Acute HQ values for the common tern never exceeded 1.0. However, chronic HQs for lead, selenium, PCBs, and DDT exceeded 1.0 in at least one river segment (Figure 5-3). These additional COPCs were identified for the tern (and not for the bald eagle) because of its greater ingestion rate to body weight ratio. The spatial patterns of HQs exceeding 1.0 for lead, PCBs, and DDT by river segment are similar to those observed for the bald eagle, although the HQs for the tern are larger given its larger ingestion rate to body weight ratio (i.e., higher exposure potential).

Selenium HQs slightly exceeded 1.0 within the river segments encompassing RMs 0 to 3.2 and RMs 4.9 to 6.5. These HQs were influenced by selenium concentrations in fish tissue, which ranged from 0.09 to 1.21 mg/kg-wet between RMs 0 to 3.2 and 0.09 to 1.65 mg/kg-wet between RMs 4.9 to 6.5. The maximum wet-weight concentrations in each river segment correspond to dry-weight selenium concentrations of approximately 4.8 and 6.6 mg/kg. Hazard quotients of approximately 1.0 are consistent with the dietary EC10 of 5 mg/kg-dry derived by Fairbrother et al. (2000). According to Skorupa et al. (1996), background selenium concentrations in fish range from less than 1 to 4 mg/kg-dry. Accordingly, the difference between background selenium concentrations and those that may cause effects in birds is quite small.

Figure

**5-2 Chronic Hazard Quotients for Bald Eagles Feeding in the Ottawa River**

Figure

**5-3 Chronic Hazard Quotients for Common Terns Feeding in the Ottawa River**

Overall, there is no consistent pattern in HQs between chemicals. In general, PCB HQs decrease from the river mouth to upstream river segments, while DDT HQs decline from upstream to downstream. The highest HQ for lead occurs in the middle river segment.

#### **5.1.1.3 Spotted Sandpiper**

Potential exposures of spotted sandpipers to chemicals in the lower Ottawa River are quite different than those of bald eagles and common terns. This is due to differences in food items (benthos instead of fish) and because they are likely to ingest a greater amount of sediment since they are probing feeders. The chemicals with HQs greater than 1.0 in at least one river segment were aluminum, chromium, lead, cyanide, PCBs (Aroclor 1242), DDT, and bis(2-ethylhexyl)phthalate (Figure 5-4). The HQs for aluminum, chromium, and lead were influenced by incidental sediment ingestion, while those for the organic compounds were influenced by dietary exposures. Recall that dietary concentrations were estimated from sediment concentrations using BSAFs for lipophilic organic compounds. Accordingly, all sandpiper HQs were directly linked to the ingestion of sediment.

Aluminum HQs were between 1.0 and 2.0 in the lower four river segments. However, since background aluminum concentrations may be elevated and no background data is available, the potential risk associated with these HQs is uncertain and may reflect background aluminum concentrations in the river. The highest lead HQ was approximately 71, occurring between RMs 4.9 and 6.5. Chromium HQs were between 1.0 and 2.5, occurring in the middle three river segments. An HQ of 1.2 occurred for cyanide between RMs 6.5 and 8.8. Cyanide HQs did not exceed 1.0 for any other river segment. The HQs for the three organic COPCs generally decline from upstream to downstream for the spotted sandpiper (see Figure 5-4). The maximum bis(2-ethylhexyl)phthalate HQ of approximately 3 was also influenced by sediment concentrations at RM 8.3.

#### **5.1.1.4 Mink**

No acute HQs exceeded 1.0 for the mink. Chronic HQs exceeded 1.0 for aluminum, iron, lead, selenium, thallium, and PCBs for at least one river segment (Figure 5-5). Hazard quotients for aluminum, lead, and thallium were influenced by incidental sediment ingestion exposures, while HQs for selenium and PCBs were influenced by dietary exposures. The issues associated with aluminum, lead, selenium, and PCBs have been discussed for other receptors. Iron and thallium were not identified as COPCs for avian receptors due to the lack of toxicity TRVs for birds. However, HQs were between 1.0 and 2.0 for the mink. Given the conservative nature of SLRA, however, HQs of 1.0 to 2.0 are probably not of concern.

#### **5.1.1.5 Snapping Turtle**

As discussed earlier, there are currently insufficient tissue-based toxicity data with which to calculate HQs for reptiles or reptile eggs; the latter has limited data available for PCB congeners. Furthermore, given the unique physiology of reptiles, it is not plausible to assume that the sensitivity of reptiles is similar to that of other classes, such as mammals or birds. PCB congener concentrations in snapping turtle eggs ranged from 0.000049 mg/kg-wet (Congener 129) to 0.741 mg/kg-wet (Congener 66), with a corresponding total PCB concentration of 3.683 mg/kg-wet. Accordingly, the data demonstrate that the turtles are being exposed to PCBs, but the impact of these PCB levels in eggs on reproductive success of the turtles is unknown.

Figure

**5-4 Chronic Hazard Quotients for Spotted Sandpipers Feeding in the Ottawa River**

Figure

**5-5 Chronic Hazard Quotients for Mink Feeding in the Ottawa River**

### 5.1.2 Water Quality Criteria-Based Hazard Quotients

A second line of evidence using WQC was evaluated for wildlife. HQ values using WQC were calculated as shown in Equation 6:

$$HQ_{WQC} = \frac{EEC_{water}}{WQC_{wildlife}} \quad (6)$$

Where:  $EEC_{water}$  = Expected environmental concentration in water  
 $WQC_{wildlife}$  = Water quality criterion for wildlife

Mercury was the only chemical detected in surface water with a wildlife criterion available. The HQs ranged from 113 to 177 across the lower four river segments. These HQs considered alone suggest mercury is posing a substantial risk to wildlife in the Ottawa River. However, compared to other lines of evidence, these HQs appear to be overly conservative. For example, the U.S. EPA criterion for protection of human health is 0.012 µg/L. Use of this criterion would result in HQs ten times lower, i.e., between approximately 10 and 20. Furthermore, the mercury HQs based on the wildlife criterion are much higher than those calculated for individual receptors, which were always less than 1.0. These HQs were based on measured concentrations in fish tissue, 100 percent of which was assumed to be present as methyl mercury<sup>9</sup>. Accordingly, the accumulation-based wildlife criterion may be overestimating mercury's bioaccumulation potential in the Ottawa. Given the discrepancies in the lines of evidence for mercury, potential risks to wildlife receptors from mercury may be considered uncertain. However, it does appear that the extreme HQs based on the Ohio EPA wildlife criterion are overly conservative.

## 5.2 AQUATIC LIFE

Potential risks to aquatic life were also characterized using multiple lines of evidence. These include HQs, the results of field studies using Ohio EPA biological criteria, and bioassays. The methods on how each was used are discussed below.

### 5.2.1 Hazard Quotients

Similar to the approach for wildlife shown above, water- and sediment-based HQs for aquatic life were calculated as shown in Equations 7 and 8, respectively:

$$HQ_{water} = \frac{EEC_{water}}{WQC_{aquatic\ life}} \quad (7)$$

$$HQ_{sediment} = \frac{EEC_{sediment}}{\text{Sediment Guideline}} \quad (8)$$

<sup>9</sup> In aquatic systems, mercury is generally present as inorganic mercury and methyl mercury. Methyl mercury is the predominant form in fish tissue (Grieb et al. 1990).

### 5.2.1.1 Surface Water

Like the wildlife HQs, a surface water HQ for aquatic life greater than a value of 1.0 suggests that the chemical may be present at a sufficiently high concentration to adversely affect aquatic communities. However, an HQ greater than 1.0 does not mean that a chemical is definitely adversely affecting the aquatic community, only that it may *potentially* be affecting the community and should be evaluated further. An HQ less than 1.0 suggests negligible risks to the aquatic community.

The chemicals with individual acute and chronic HQs exceeding 1.0 are provided in Tables 5-1 and 5-2, respectively. The HQs for all of the organic chemicals listed in the tables are highly uncertain because these chemicals were rarely detected (usually in one of 19 samples, and never in more than three of 19 samples). For some organics, such as atrazine, the chemical was detected once in a sample with a lower detection limit than the majority of the samples. Consistent with common risk assessment practice (Gliet 1985; Porter et al. 1988; U.S. EPA 1991b), a value of one-half the detection limit was used to calculate risk when a chemical was not detected in a sample. Accordingly, the exposure concentration is likely biased high using one-half the detection limit. It must be noted that the detected concentration was often the same as the detection limit. These chemicals are italicized in Tables 5-1 and 5-2 to note the uncertainty in the HQs. Additionally, for some chemicals, the detection limit was greater than the TRV used to calculate HQs. Since one-half the detection limit was used to calculate HQs for samples where a chemical was not detected, risk may be overestimated in these cases. Chemicals with detection limits greater than their respective TRVs have been flagged in Tables 5-1 and 5-2 to note the uncertainty.

The HQs discussed in the preceding paragraph are based on the exposure concentrations and toxicities of individual chemicals. However, chemical toxicity can be additive, particularly when the modes of toxic action are the same. The additive toxicity of chemicals with narcosis as the mode of action (e.g., PAHs) is a common example (e.g., Di Toro et al. 2000), as is the additive toxicity of certain divalent metals. The toxicities of mixtures with different modes of action may also be additive, or even synergistic (Van der Geest et al. 2000), but additivity across chemical classes was not evaluated given the lack of an acceptable approach for site-specific chemical mixtures. Note that chemical mixtures may also possess synergistic or antagonistic effects, but it is not possible for analyzing the possibility of these effects without conducting testing with the chemical mixture of interest. The most common approach for evaluating the additivity of mixtures is through the use of toxic units (TUs). Toxic units in surface water are defined as the ratio of the concentration in a medium to the effect concentration in the medium (Sprague and Ramsay 1965). The TU in surface water is thus defined as:

$$TU_{wi} = \frac{C_{w,i}}{C_{wQC,i}}$$

In the equation, 'W' refers to water, 'i' denotes the individual chemicals, and the denominator is the water quality criterion (or similar toxicity guideline value). Note that the TU is calculated the same as the HQ used in this SLRA. Accordingly, additivity of appropriate chemicals groups is evaluated through summation of their HQs. Chemical additivity in this SLRA was evaluated for PAHs and divalent metals (i.e., cadmium, cobalt, copper, lead, manganese, mercury, nickel, and zinc). It is important to note that the effects of mixtures of metals cannot always be predicted from the effects of single metals. For example, the relative concentrations of metals in a mixture may influence the bioaccumulation and toxicity of each individual metal (Harrahy and Clements 1997). The toxicity of a metal mixture may also deviate from additivity when low concentrations of one metal are present with high concentrations of another (Harrahy and Clements 1997). The toxicity of mixtures was termed  $\Sigma$ HQ in this analysis.

**Table 5-1. Chemicals with Individual Acute HQs Exceeding 1.0 (HQ is shown in parentheses)**

	River Segment			
	0-3.2	3.2-4.9	4.9-6.5	
<b>Metals</b>				
Aluminum (4.6)	Aluminum (3.2)	Aluminum (1.9)	Aluminum (1.5)	-
Manganese (3.8)	Manganese (5.0)	Manganese (21)	Manganese (6.1)	-
<b>PAHs</b>				
<i>Benzo(a)pyrene</i> (2.1) <sup>b</sup>	<i>Benzo(a)pyrene</i> (2.1) <sup>b</sup>	<i>Benzo(a)pyrene</i> (2.1) <sup>b</sup>	<i>Benzo(a)pyrene</i> (2.1) <sup>b</sup>	-
<i>Benzo(b)fluoranthene</i> (3.0) <sup>b</sup>	<i>Benzo(b)fluoranthene</i> (3.0) <sup>b</sup>	<i>Benzo(b)fluoranthene</i> (3.0) <sup>b</sup>	<i>Benzo(b)fluoranthene</i> (3.0) <sup>b</sup>	-
<i>Benzo(g,h,i)perylene</i> (4.6) <sup>b</sup>	<i>Benzo(g,h,i)perylene</i> (4.6) <sup>b</sup>	<i>Benzo(g,h,i)perylene</i> (4.6) <sup>b</sup>	<i>Benzo(g,h,i)perylene</i> (4.6) <sup>b</sup>	-
<i>Benzo(k)fluoranthene</i> (3.1) <sup>b</sup>	<i>Benzo(k)fluoranthene</i> (3.1) <sup>b</sup>	<i>Benzo(k)fluoranthene</i> (3.1) <sup>b</sup>	<i>Benzo(k)fluoranthene</i> (3.1) <sup>b</sup>	-
<i>Dibenz(a,h)anthracene</i> (3.5) <sup>b</sup>	<i>Dibenz(a,h)anthracene</i> (3.5) <sup>b</sup>	<i>Dibenz(a,h)anthracene</i> (3.5) <sup>b</sup>	<i>Dibenz(a,h)anthracene</i> (3.5) <sup>b</sup>	-
<i>Indeno(1,2,3-cd)pyrene</i> (3.6) <sup>b</sup>	<i>Indeno(1,2,3-cd)pyrene</i> (3.6) <sup>b</sup>	<i>Indeno(1,2,3-cd)pyrene</i> (3.6) <sup>b</sup>	<i>Indeno(1,2,3-cd)pyrene</i> (3.6) <sup>b</sup>	-
<b>Other Organics</b>				
-	-	-	2,4-Dinitrophenol (2.5)	-
-	-	-	4,6-Dinitro-2-methylphenol (2.3)	-
<i>Pentachlorophenol</i> (5.8) <sup>b</sup>	<i>Pentachlorophenol</i> (5.8) <sup>b</sup>	-	<i>Pentachlorophenol</i> (5.8) <sup>b</sup>	-
-	-	-	<i>Phenol</i> (1.3)	-

<sup>a</sup> No surface water chemistry data available for this segment.

<sup>b</sup> Analytical detection limit for this chemical was greater than the acute screening value.

Note: Italicized chemicals were rarely detected and, when detected, only at a concentration equal to the detection limit. Accordingly, the exposure concentration is largely influenced by an uncertain value that is one-half the detection limit.

Table 5-2. Chemicals with Individual Chronic HQs Exceeding 1.0 (HQ is shown in parentheses)

		River Segment			
		0-3.2	3.2-4.9	4.9-6.5	6.5-8.8
<b>Metals</b>					
	Aluminum (30)	Aluminum (25)	Aluminum (14)	Aluminum (13)	-
	Iron (4.3)	Iron (4.2)	Iron (2.8)	Iron (1.9)	-
	Manganese (17)	Manganese (19)	Manganese (68)	Manganese (24)	-
<b>Pesticides</b>					
	Atrazine (1.2) <sup>b</sup>	Atrazine (1.2) <sup>b</sup>	Atrazine (1.2) <sup>b</sup>	Atrazine (1.2) <sup>b</sup>	-
<b>PAHs</b>					
	Benzo(a)anthracene (1.3) <sup>b</sup>	Benzo(a)anthracene (1.3) <sup>b</sup>	Benzo(a)anthracene (1.3) <sup>b</sup>	Benzo(a)anthracene (1.3) <sup>b</sup>	-
	Benzo(a)pyrene (4.1) <sup>b</sup>	Benzo(a)pyrene (4.1) <sup>b</sup>	Benzo(a)pyrene (4.1) <sup>b</sup>	Benzo(a)pyrene (4.1) <sup>b</sup>	-
	Benzo(b)fluoranthene (6.0) <sup>b</sup>	Benzo(b)fluoranthene (6.0) <sup>b</sup>	Benzo(b)fluoranthene (6.0) <sup>b</sup>	Benzo(b)fluoranthene (6.0) <sup>b</sup>	-
	Benzo(g,h,i)perylene (8.9) <sup>b</sup>	Benzo(g,h,i)perylene (8.9) <sup>b</sup>	Benzo(g,h,i)perylene (8.9) <sup>b</sup>	Benzo(g,h,i)perylene (8.9) <sup>b</sup>	-
	Benzo(k)fluoranthene (6.1) <sup>b</sup>	Benzo(k)fluoranthene (6.1) <sup>b</sup>	Benzo(k)fluoranthene (6.1) <sup>b</sup>	Benzo(k)fluoranthene (6.1) <sup>b</sup>	-
	Chrysene (1.4) <sup>b</sup>	Chrysene (1.4) <sup>b</sup>	Chrysene (1.4) <sup>b</sup>	Chrysene (1.4) <sup>b</sup>	-
	Dibenz(a,h)anthracene (14) <sup>b</sup>	Dibenz(a,h)anthracene (14) <sup>b</sup>	Dibenz(a,h)anthracene (14) <sup>b</sup>	Dibenz(a,h)anthracene (14) <sup>b</sup>	-
	Indeno(1,2,3-cd)pyrene (14) <sup>b</sup>	Indeno(1,2,3-cd)pyrene (14) <sup>b</sup>	Indeno(1,2,3-cd)pyrene (14) <sup>b</sup>	Indeno(1,2,3-cd)pyrene (14) <sup>b</sup>	-
	-	-	-	Pyrene (1.4)	-
<b>Other Organics</b>					
	2,4-Dinitrophenol (2.1) <sup>b</sup>	2,4-Dinitrophenol (2.1) <sup>b</sup>	2,4-Dinitrophenol (2.1) <sup>b</sup>	2,4,5-Trichlorophenol (1.3)	-
	4,6-Dinitro-2-methylphenol (1.9) <sup>b</sup>	4,6-Dinitro-2-methylphenol (1.9) <sup>b</sup>	4,6-Dinitro-2-methylphenol (1.9) <sup>b</sup>	2,4,6-Trichlorophenol (1.3)	-
	Pentachlorophenol (1.4) <sup>b</sup>	Pentachlorophenol (1.4) <sup>b</sup>	Pentachlorophenol (1.4) <sup>b</sup>	2,4-Dinitrophenol (2.1) <sup>b</sup>	-
	Phenol (1.1) <sup>b</sup>	Phenol (1.1) <sup>b</sup>	Phenol (1.1) <sup>b</sup>	Phenol (1.1) <sup>b</sup>	-
				4,6-Dinitro-2-methylphenol (1.9) <sup>b</sup>	-
				Pentachlorophenol (1.4) <sup>b</sup>	-
				Phenol (1.1) <sup>b</sup>	-

<sup>a</sup> No surface water chemistry data available for this segment.

<sup>b</sup> Analytical detection limit for this chemical was greater than the chronic screening value.

Note: Italicized chemicals were rarely detected and, when detected, only at a concentration equal to the detection limit. Accordingly, the exposure concentration is largely influenced by an uncertain value that is one-half the detection limit.

The  $\Sigma$ HQs for divalent metals (cadmium, copper, lead, mercury, nickel, and zinc) are shown, by reach, in Table 5-3. Acute  $\Sigma$ HQs were all less than 1.0, while chronic  $\Sigma$ HQs ranged between 1.2 and 1.5 depending on the river segment. However, these chronic  $\Sigma$ HQs were not considered significant because only total recoverable metal concentrations (particulate bound and dissolved fraction) were available. As discussed in previous sections, the toxicity of divalent metals is almost entirely a function of the free ion (dissolved concentration). Accordingly, since 1993, the national AWQC promulgated by the U.S. EPA have been based on dissolved metal (filtered through a 0.45  $\mu$ m filter) (Prothro 1993). However, even dissolved metal can be a conservative estimate of bioavailable metal because other dissolved surface water constituents, such as dissolved organic carbon (DOC), can reduce bioavailability (Taylor et al. 2000). The bioavailable fraction of divalent metals in natural waters may be up to 26 times less than laboratory waters that are typically used to derive aquatic life toxicity values and water quality standards/criteria (Welsh et al. 2000). Accordingly,  $\Sigma$ HQs of 1.2 to 1.5 for divalent metals were considered unlikely to pose risk to aquatic life from direct toxicity.

Overall, the risks posed by chemicals in lower Ottawa River surface water are uncertain. Hazard quotients exceeded 1.0 for a number of chemicals, but uncertainty exists with each of these. For example, HQs greater than 1.0 for aluminum, iron, and manganese are likely overconservative because these metals are often elevated at background concentrations and largely non-bioavailable. For the organic chemicals with HQs greater than 1.0, the risk posed to aquatic life are highly uncertain because they are largely influenced by values one-half the detection limit. Many of these compounds, PAHs in particular, are extremely hydrophobic, so they would not be expected to pose a high risk in surface water. Further analyses can assist in resolving these uncertainties. For example, measurements of dissolved metal or surface water bioassays can both provide information on metal bioavailability. For organics, achievement of detection limits below risk-based toxicity values would allow determination of whether these chemicals are posing unacceptable risks. Lastly, the risk characterization of hydrophobic chemicals in sediment provide a more adequate assessment risk, so the uncertainty in the surface water risk characterization for these compounds may be considered relatively insignificant to the overall conclusions of the ecological SLRA.

### **5.2.1.2 Sediment**

For sediment, a weight-of-evidence approach was also used to screen chemical risks in sediment. As discussed in the Effects Characterization (Section 4.2.2), a variety of different types of sediment guidelines (e.g., ER-L, ER-M) from multiple sources (e.g., Environment Canada 1995; Ingersoll et al. 1996) were used in this SLRA. The ability of these different types of guidelines to predict toxicity (or lack of toxicity) to benthic organisms was reviewed by Long et al. (1998). Long et al. assessed the toxicity of hundreds of field-collected sediment samples using various laboratory bioassays. Based on the data provided in their paper, it is clearly evident that several ER-L values, and even more TEL values, need to be exceeded before sediment toxicity is observed with any consistency.

The following rules were followed in determining whether a chemical was a COPC. First, if the concentration of a chemical exceeded its corresponding PEL or ER-M, it was considered a COPC. For TEL and ER-L exceedances, the analyses of Long et al. (1998) were considered to interpret the sediment HQs. A step-wise ANOVA was conducted to determine how many ER-L values for metals need to be exceeded before excess sediment toxicity is observed (i.e., before the degree of toxicity is significantly different than when no ER-L values are exceeded) and how many TEL values need to be exceeded in a common sample before excess sediment toxicity is observed. The end result is that if four or more metals exceed their ER-L values for a given site, or nine or more metals exceed their TEL, those metals are considered COPCs.

**Table 5-3. ΣHQs for Divalent Metals in Surface Water**

	River Segment				
	0-3.2	3.2-4.9	4.9-6.5	6.5-8.8	>8.8 <sup>a</sup>
Acute	< 1.0	< 1.0	< 1.0	< 1.0	-
Chronic	1.5	1.4	1.2	1.3	-

<sup>a</sup> No surface water chemistry data available for this segment.

For metals, it is important to note that dry-weight sediment concentrations are not predictive of bioavailability, while sediment pore water concentrations have been shown to be correlated with biological effects (Ankley et al. 1996). The primary partitioning phase controlling cationic metal activity and toxicity in the sediment-pore water system is acid volatile sulfide (AVS) (Di Toro et al. 1990, 1992). On a molar basis, AVS binds with cationic metals, resulting in insoluble sulfide complexes with minimum biological bioavailability (Ankley et al. 1996). According to U.S. EPA (2000b), if the simultaneously extracted sum of molar concentrations of cadmium, copper, lead, nickel, silver, and zinc are less than the molar AVS concentrations, toxicity will not be observed. Of the 19 AVS:SEM samples, the molar ratio of SEM (based on the sum of cadmium, copper, lead, nickel, silver, and zinc) to AVS was always greater than 1.0 (mean, minimum, and maximum were 1,664, 1.1, and 8,562, respectively). Accordingly, the AVS:SEM data cannot be used to support an absence of metal toxicity in sediment. It is important to note that exceedance of the molar AVS concentration does not necessarily suggest that the SEM metals are present at toxic concentrations because other factors can reduce metal bioavailability as well, such as dissolved organic carbon (which was not measured).

For sediment guidelines that are normalized to the organic carbon content of the sediment, the 95 percent lower confidence limit on the mean organic carbon concentration was conservatively used for each river segment. When only two organic carbon samples were available for a reach (such as segment four in Inventory 20), the minimum organic carbon concentration was used.

The chemicals with HQs greater than 1.0 based on ER-Ms or PELs from the year 1998 sampling event are shown in Figure 5-6. Lead and PCB (total) HQs consistently exceeded 1.0 for the lower four river segments. More COPCs were identified for RMs 3.2 to 4.9, with cadmium and chromium also being identified, but the highest HQs for PCBs were observed between RMs 4.9 to 6.5. The land adjacent to this segment of the river contains a number of landfills and industrial facilities (see Figure 1-1). Accordingly, based on these data, metals concentrations appeared most elevated from RMs 3.2 to 4.9, and PCBs were most elevated from RMs 4.9 to 6.5. Using the PEL from Ingersoll et al. (1996), HQs from 0 to 24" samples were compared to those from greater than 24" (Figure 5-7). With a few exceptions, HQs decline in the core sediment samples (i.e., greater than 24"). These lower HQs likely reflect lower concentrations as a result of the larger compositing volume, rather than lower toxicity per se.

Based on the year 2000 sediment sampling results, HQs for those chemicals exceeding Ingersoll et al. (1996) ER-M and PEL values or Environment Canada (1995) PEL values are shown graphically in Figure 5-8. Like the 1998 sediment HQs shown in Figure 5-6, lead and PCB HQs consistently exceed 1.0. The highest HQ for lead occurs between RMs 4.9 and 6.5, and is largely affected by the single sample discussed in the wildlife risk characterization. With this exception, HQs for PCBs tend to be the highest, followed closely by organochlorine pesticides.

Figure

**5-6 Sediment HQs > 1.0 Based on ERMs and PELs Using 1998 Surface (< 24")  
Sediment Data**

Figure

**5-7 Comparison of Sediment HQs Based on 1998 Surface (< 24") and Core (> 24") Sediment Data**

Figure

**5-8 Sediment HQs > 1.0 Based on ERMs and PELs Using 2000 Surface Sediment Data**

The sediment HQs, by station, were plotted on maps for three chemicals or chemical classes: (1) lead; (2) total PCBs; and (3) total PAHs (Appendix D). Lead and total PCBs were plotted since they appear to be the driving chemicals for potential risk to both wildlife and benthic aquatic life. Although HQs for total PAHs did not exceed 1.0, the potential risk they pose is uncertain since all PAHs and their key derivatives were not analyzed<sup>10</sup>. The maps were derived as a tool to visually identify potential hot spots. The sediment data, on a sample-by-sample basis, were plotted by color to denote whether the chemicals exceed their respective sediment guideline value. For the 1998 data, samples were also plotted by depth (0 to 24" and greater than 24").

### 5.2.1.3 Tissue Residues

When tissue residue-based effects data were available for chemicals detected in fish tissue, HQs were calculated using the concentrations measured in Ottawa River fish. As discussed in Section 3.2.2, the dietary pathway was more important for certain chemicals (e.g., selenium, PCBs), making it more difficult to predict effects based on water exposure and toxicity data. Chemicals with HQs exceeding 1.0 were lead and PCBs (Figure 5-9). The HQs are provided in Table C-7 of Appendix C. As presented above, these two chemicals were identified as COPCs for aquatic life in sediment. Thus, sediment concentrations may be sufficiently high to transfer to lower levels of the food chain and reach levels in fish tissue that may cause adverse effects.<sup>11</sup>

## 5.2.2 Bioassays

Bioassay results provide a corroborative line of evidence to the HQ screening risk predictions. The bioassays reflect site-specific conditions that can modify toxicity. For example, sediment bioassays provide evidence on the bioavailability of chemicals to the test organism. Moreover, the bioassays assess the toxicity of chemical mixtures, so potential antagonistic, additive, or synergistic impacts are implicitly addressed in the measured effect. Bioassay results, thus, provide information on whether the HQ predictions were conservative or non-conservative. In 1998, the Ohio EPA evaluated the toxicity of Ottawa River sediment samples to the amphipod *Hyaella azteca* and the oligochaete *Lumbriculus variegatus* (OEPA 1998). The sediment samples were collected up to a depth of approximately 10 cm where possible and homogenized. The *H. azteca* and *L. variegatus* bioassays were conducted for 10 and 4 days, respectively. The locations and dates of the bioassays are provided in Table 5-4.

None of the sediment samples was observed to be toxic to *H. azteca* or *L. variegatus*. *H. azteca* survival was 96.3 percent at Site 09. Comparison of each of the nine Ottawa River sampling sites to the University of Toledo reference station using Steel's many one-rank test did not indicate a significant difference on survival of *L. variegatus*. Accordingly, the sediments do not appear to be acutely toxic to either of these test organisms.

---

<sup>10</sup> According to U.S. EPA (2000a), these include the PAHs on the EPA's Priority Pollutant list, as well as alkylated naphthalenes, phenanthrenes, fluoranthenes, fluorenes, and chrysenes.

<sup>11</sup> It should be clarified that lead and PCBs are transferred through the food chain much differently. Lead concentrations tend to decline with increasing trophic level (Vighi 1981), while PCB concentrations tend to increase with increasing trophic level (i.e., biomagnify) (Suedel et al. 1994).

Figure

**5-9 Chronic Hazard Quotients for Fish in the Ottawa River Using Tissue-Based TRVs**

**Table 5-4. Location and Dates of Whole Sediment Toxicity Bioassays**

<b>Sample Site</b>	<b>Species Tested</b>	<b>Date Collected</b>	<b>Dates Tested</b>
Site 09/Downstream Summit St.	<i>H. azteca</i> , <i>L. variegatus</i>	12 Aug 98	17-28 Aug 98
Site 10/@ I-75	<i>L. variegatus</i>	3 Aug 98	8-12 Aug 98
Site 11/Downstream of Stickney Ave.	<i>L. variegatus</i>	3 Aug 98	8-12 Aug 98
Site 12/Adjacent to Stickney Ave.	<i>L. variegatus</i>	3 Aug 98	8-12 Aug 98
Site 13/Upstream of Railroad Trestle	<i>L. variegatus</i>	10 Aug 98	12-16 Aug 98
Site 14/@ Lagrange St.	<i>L. variegatus</i>	10 Aug 98	12-16 Aug 98
Site 15/@ Berdan Ave.	<i>L. variegatus</i>	10 Aug 98	12-16 Aug 98
Site 16/@ Jeep Parkway	<i>L. variegatus</i>	18 Aug 98	24-28 Aug 98
Site 17/@ Auburn Ave.	<i>L. variegatus</i>	18 Aug 98	24-28 Aug 98
Site 18/@ U. of Toledo (Ref/Station)	<i>L. variegatus</i>	19 Aug 98	24-28 Aug 98

Chemistry data were available for Site 09. Some key chemicals detected and their concentrations are listed in Table 5-5. Sediment TEL and PEL guidelines are provided for comparison to the metal, organochlorine, and PCB concentrations, while the sediment quality guidelines from Di Toro and McGrath (2000) are provided for comparison to the PAH data. Most of the metal, pesticide, and PCB concentrations exceeded their respective TELs, and nickel, zinc, DDE, and PCB concentrations also exceeded their respective PELs. Accordingly, toxicity in this sediment sample would have been predicted using sediment guidelines. This may suggest that the bioavailability of the chemicals is reduced. However, the lack of toxicity may also be due to the length of the bioassays. Ingersoll et al. (2001) recently compared the 10- to 14-day *H. azteca* test, which measures survival, and the 28- to 42-day *H. azteca* test, which measured survival and reproduction. The 28- to 42-day test was typically found to be six times more sensitive than the 10- to 14-day test. Accordingly, since the *H. azteca* bioassay used in the Ottawa River sample was a 10-day study, the lack of toxicity is probably more a function of the type of test that was conducted, rather than reduced bioavailability. Accordingly, the lack of toxicity in this sample provides only limited corroborative information for the predicted sediment HQs.

### **5.2.3 Biological Criteria**

Biological monitoring data were used to assess whether there was a relationship between the condition of the biological community and the HQs calculated and described above. The condition of the biological community in the lower Ottawa River was evaluated using biological indices for benthic macroinvertebrates and fish communities. The biological index data used in the analysis were calculated by the Ohio EPA and no changes were made to any indices. However, means of indices were used when more than one sample event occurred in a year and when more than one sample location fell within the river segments evaluated in this SLRA. For example, five different benthic macroinvertebrate sample locations were assessed by Ohio EPA between RMs 4.9 and 6.5.

**Table 5-5. Comparison of Sediment Chemistry from Bioassay Sample Site 09 to Sediment Quality Guidelines**

Chemical	Concentration (mg/kg dw)	Ingersoll et al. (1996)		Di Toro and McGrath (2000)
		TEL	PEL	C <sub>SQG</sub>
<b>Metals</b>				
Arsenic	9.76	11	48	-
Cadmium	1.67	0.58	3.2	-
Chromium	67.8	36	120	-
Copper	55.8	28	100	-
Lead	75.7	37	82	-
Nickel	41.8	20	33	-
Zinc	220	98	540	-
<b>Pesticides/PCBs</b>				
DDD	0.0185	-	-	-
DDE	0.0375	0.00142 <sup>a</sup>	0.00675 <sup>a</sup>	-
Aroclor 1248	2.5			-
Aroclor 1260	0.115			-
Total PCBs <sup>b</sup>	2.6	0.032	0.24	-
<b>PAHs</b>				
Benzo(a)pyrene	1.2	-	-	50.6
Benzo(b)fluoranthene	1.6	-	-	51.3
Benzo(g,h,i)perylene	1.2	-	-	57.4
Benzo(k)fluoranthene	1.2	-	-	51.4
Chrysene	1.5	-	-	44.2
Fluoranthene	1.9	-	-	37.1
Indeno(1,2,3-cd)pyrene	1.3	-	-	58.5
Pyrene	1.6	-	-	36.6

<sup>a</sup> TEL and PEL from Environment Canada (1995).

<sup>b</sup> Total PCBs estimated by summing Aroclor 1248 and Aroclor 1260.

### 5.2.3.1 Biological Indices Evaluated

A select group of the benthic macroinvertebrates indices developed by Ohio EPA were used in this risk assessment. The indices include those that were based on quantitative (Hester Dendy sampler<sup>12</sup>) and qualitative (kicknet) macroinvertebrate collection. The Ohio EPA (appropriately) uses both methods to evaluate streams and rivers in their assessment. These indices include those that are important indicators for metal stress (mayfly richness), as well as those that are important for evaluating stress from organic chemicals (caddisfly richness). An additional summary index being developed by Ohio EPA was also used, the Lacustrary Invertebrate Community Index (LICI). This index was used for stream and river sites that are influenced by Lake Erie.

<sup>12</sup> Hester Dendy samplers are artificial substrate samplers that are placed in the water column by securing the sampler by an anchor and then held in the water column by a float.

Multiple indices for fish were also used. These indices include those that consider general fish community health based on fish richness, relative abundance, and those that consider the health of individual fish health based on external anomalies, or DELT (deformities, fin erosions, lesions/ulcers, and tumors). Two different summary indices used by the Ohio EPA were used in this evaluation: an Index of Well Being (IWB2) and an Index of Biotic Integrity (IBI) for lacustrine conditions.

**5.2.3.2 Results**

In general, the biological indices suggested that benthic macroinvertebrate and fish communities within the lower Ottawa River are not in good condition. For benthic macroinvertebrates, the LICI values were all much lower than the interim goal of 42 for LICI rated streams and rivers (Table 5-6) (OEPA 1998). The IBI scores for fish were also low compared to the goal of 42 for lacustrine sites (OEPA 1998). The IWB2 scores are also slightly below their respective goals of 8.6 for boat sites and 7.3 for wading sites (OEPA 1999). Thus, the biological index results suggest non-attainment of aquatic life use designations in the lower Ottawa River.

**Table 5-6. Summary Indices for Benthic Macroinvertebrates and Fish**

<b>River Mile</b>	<b>0-3.2</b>	<b>3.2-4.9</b>	<b>4.9-6.5</b>	<b>6.5-8.8</b>
<b>Benthic Macroinvertebrates</b>				
LICI	8	14	13	15
<b>Fish</b>				
IWB2	7.1	6.2	6.1	6.6
IBI, Lacustrine	24.0	20.0	21.3	21.3

Surface water HQs from Table 5-2 were compared to benthic invertebrate and fish indices in Table 5-7. There does not seem to be a clear relationship between many of the benthic invertebrate indices and the surface water HQs. In general, the surface water HQs decrease from downstream to upstream, yet many of the macroinvertebrate indices tend to increase. Most of the indices should decrease if the macroinvertebrate community was being stressed. However, macroinvertebrate richness based on kicknet samples increased and caddisfly richness decreased as the surface water HQs increased. Diptera richness is often found to increase when other taxa are stressed because they tend to be more tolerant of stress. In fact, in this evaluation, Diptera richness does increase with increasing surface water HQs. The lack of a clear relationship for some indices may exist because the Hester Dendy sampler is a device that is used in the water column, i.e., the sample is set in the water column. Thus, Hester Dendy results reflect conditions in the water column but not in the sediment.

With the fish there were better relationships between surface water HQs and the indices. Most of the indices decreased as the surface water HQs increased in a downstream manner. Contrary to most indices, the DELT index should increase as the surface water HQs increase, however, there was no clear trend for this index. Although a predictable pattern was not observed with the DELT index, approximately seven percent deformities were observed in multiple river reaches. An example of a carp exhibiting severe deformities is provided in Figure 5-10. It is possible that PAHs are contributing to these deformities since certain PAH compounds can induce teratogenic effects (U.S. EPA 2000). Accordingly, the comparison of biological indices to sediment HQs in the following paragraph may be more relevant because PAHs are hydrophobic. Ultimately, however, further studies would be necessary to determine whether PAH

concentrations are sufficiently elevated to induce the observed deformities. Clearly, fish do seem to be affected and the biotic indices reflect this conclusion.

**Table 5-7. Summary of Chronic HQs for Surface Water and Invertebrate and Fish Biotic Indices**

<b>River Mile</b>	<b>0-3.2</b>	<b>3.2-4.9</b>	<b>4.9-6.5</b>	<b>6.5-8.8</b>
<b>HQs</b>				
Aluminum	30	25	14	13
Manganese	17	19	68	24
Selenium	2.9	2.0	1.5	1.3
Sum of Divalent Metals	1.5	1.4	1.2	1.3
Cyanide	1.0	1.0	2.4	0.7
Sum PAHs	56	43	77	200
Sum SVOs	6.5	6.5	6.4	30
Benzo[b]fluoranthene	6.0	4.5	8.4	21
Dibenz[a,h]anthracene	14	10	19	49
Indeno[1,2,3-cd]pyrene	14	11	20	50
2,4-Dinitrophenol	2.1	2.1	2.1	9.7
Phenol	1.1	1.1	1.1	5.2
<b>Invertebrate Biotic Indices</b>				
Abundance, Hester Dendy	2875	814	1108	213
Taxa Richness, Hester Dendy	11	14	15	19
Taxa Richness, Kicknet	25	27	14	9
Mayfly Richness, Hester Dendy	1	0	0	1
Caddisfly Richness, Hester Dendy	1	1	0	0
Diptera Richness, Hester Dendy	3	5	9	11
Ephemeroptera Plecoptera Trichoptera (EPT), Kicknet	2	2	0	0
EPT, Hester Dendy	2	1	0	1
ICI, Estuary	8	14	13	15
<b>Fish Biotic Indices</b>				
Fish Richness	13	13	11	11
Cumulative Number of Fish Species based on all sample events	19	20	18	17
Fish Abundance	624	495	362.5	400
Biomass, kg	280	133	50	55
Percentage of fish with DELT anomalies	6.8	6.3	7.5	6.4
IWB2	7.1	6.2	6.1	6.6
IBI, Lacustrine	24.0	20.0	21.3	21.3



**Figure 5-10 Deformed carp caught in the Lower Ottawa River**

Sediment HQs were also compared to benthic invertebrate and fish indices (Table 5-8). There appeared to be a better relationship between the sediment HQs and the indices than there was for the surface water HQs. When the sediment HQs increased, many of the biotic indices correspondingly decreased. Like the surface water HQs, macroinvertebrate richness based on kicknet samples and caddisfly richness also decreased as the HQs increased, and Diptera richness increased as the HQs tended to increase. Sediment HQs and fish indices did show a relationship that would be expected, as the biotic indices generally decreased as the HQs increased. Clearly, stressor conditions in the sediment are associated with conditions in the water column for fish. Furthermore, stressor conditions in sediment are associated with benthic macroinvertebrates.

**Table 5-8. Sediment HQs and Invertebrate and Fish Biotic Indices<sup>a</sup>**

<b>River Mile</b>	<b>0-3.2</b>	<b>3.2-4.9</b>	<b>4.9-6.5</b>	<b>6.5-8.8</b>
<b>HQs</b>				
Cadmium	0.7	0.5	1.2	0.7
Chromium	0.5	0.5	1.1	1.0
Copper	0.9	0.6	1.0	1.0
Lead	1.2	3.2	199.1	3.6
Nickel	1.3	1.1	1.2	0.7
Zinc	0.5	0.5	0.6	1.1
PAH (total)	0.7	0.7	0.6	1.3
PCB (total)	2.2	11.2	10.6	10.7
<b>Invertebrate Biotic Indices</b>				
Abundance, Hester Dendy	2875	814	1108	213
Taxa Richness, Hester Dendy	11	14	15	19
Taxa Richness, Kicknet	25	27	14	9
Mayfly Richness, Hester Dendy	1	0	0	1
Caddisfly Richness, Hester Dendy	1	1	0	0
Diptera Richness, Hester Dendy	3	5	8.5	11
Ephemeroptera Plecoptera Trichoptera (EPT), Kicknet	2	2	0	0
EPT, Hester Dendy	2	1	0	1
ICI, Estuary	8	14	13	15
<b>Fish Biotic Indices</b>				
Fish Richness	13	13	11	11
Cumulative Number of Fish Species based on all sample events	19	20	18	17
Fish Abundance	624	495	363	400
Biomass, kg	280	133	50	55
Percentage of fish with DELT anomalies	6.8	6.3	7.5	6.4
IWB2	7.1	6.2	6.1	6.6
IBI, Lacustrine	24.0	20.0	21.3	21.3

<sup>a</sup> Based on data from Inventory 20 and using PELs or C<sub>50q</sub>.

### 5.3 UNCERTAINTIES

A discussion of uncertainties is important in any risk assessment and can be critical in making risk management decisions. A consideration of uncertainties is also imperative in using the lines of evidence approach discussed above. For example, the lines of evidence need to be balanced by considering the amount of uncertainty associated with each (U.S. EPA 1998a).

Wherever possible, conservative assumptions were used in estimating receptor exposures to chemicals and in identifying toxicity thresholds. The largest sources of data to the ecological SLRA were the chemistry data for sediment, fish tissue, and surface water. These data were used to estimate whether individual chemicals, and in some cases classes of chemicals, were present at sufficiently high concentrations to pose a potential risk to ecological receptors. This approach uses site-specific chemistry data, but assumptions are required in estimating the magnitude of exposure by biota. These assumptions include the fraction of time a wildlife receptor feeds in a river segment and the bioavailability of chemicals. In the SLRA, it was assumed that a wildlife receptor may feed in a given river segment 100 percent of the time over a chronic exposure duration and that chemicals were 100 percent bioavailable.

A key uncertainty in this SLRA was the effects of seiches on the screening risk estimates. Seiches likely remobilize chemicals in sediment and increase the likelihood that exposure by ecological receptors will occur, particularly over acute durations. As an analogous example, seiches have been shown to lift nutrients from sediment to the water column (Korgen 2000). As nutrients are released into the water column, organisms are attracted to these nutrients, thereby further increasing the exposure potential to chemicals by ecological receptors. Over shorter durations, seiches may also result in greater exposure of mudflats that certain shorebirds (e.g., spotted sandpiper) may feed upon. To further understand the effects of seiches on chemical mobility and influences on receptors, specific studies over varying flow conditions would be necessary.

Potential risks to the snapping turtle were also a potentially significant uncertainty. Measurement of PCBs in snapping turtle eggs was recommended as a monitoring tool (Pagano et al. 1999), but no data are available linking PCB congeners in eggs to toxic effects. However, it is known that snapping turtles are capable of storing high concentrations of PCBs in their fat without any apparent detrimental effects (Olafsson et al. 1983). Further studies would be necessary to understand whether turtles in the lower Ottawa are at risk from PCBs or other chemicals.

The 1998 sediment data were used in the SLRA for benthic aquatic life because they represented a thorough sampling of the lower Ottawa River. However, the relevance of these data to biological exposures is highly uncertain because they were composited over depths much greater than the biologically active zone (i.e., approximately the top 2 inches). Compositing sediment depths of up to 24" or more may result in sediment exposure concentrations for benthos being under- or overestimated, depending on the magnitude of historical chemical loading to the river sediment. Consequently, the HQ results based on the 1998 and 2000 data should both be considered since the 2000 data are based on sediment from the biologically active zone.

As discussed in the aquatic life SLRA for surface water, concentrations of several organic chemicals (e.g., PAHs) exceeded their toxicity reference values. However, these chemicals were infrequently detected and the exposure concentration was influenced by the magnitude of the detection limit (because half the detection limit was used for non-detect data). Achievement of lower detection limits for these chemicals would confirm whether they are truly posing unacceptable risk.

Lastly, conducting chronic bioassays would strengthen this line of evidence for the estimated aquatic life risks. The most recent bioassays are based on acute exposure durations and no toxicity was observed. These results did not corroborate with the sediment HQs calculated for benthic aquatic life which were greater than 1.0 for multiple chemicals. These HQs were calculated using sediment quality guidelines that were largely influenced by chronic toxicity values. Accordingly, chronic bioassays would be more appropriate for interpreting the significance of the HQs.

## 6. CONCLUSIONS

Using conservative assumptions on chemical exposure and toxicity, the SLRA identified potential hot spots of chemical risk to wildlife and aquatic life. The SLRA focused largely on sediment, surface water, and tissue data collected in 2000, as these data are most relevant to current conditions in the Ottawa River. However, the extensive sediment data from 1998, sediment bioassays, and aquatic community biocriteria were also considered.

The HQ evaluation for wildlife and aquatic life identified chemicals of potential concern for multiple segments of the lower 9 miles of the Ottawa River. Lead and PCB HQs consistently exceeded 1.0 for both wildlife and aquatic life, although HQs for other chemicals also exceeded 1.0 for specific ecological receptors and locations (Table 6-1). For wildlife, lead HQs were influenced by concentrations in sediment, while PCB HQs were influenced by tissue (i.e., food item concentrations). Figure 6-1 graphically shows the sum of HQs greater than 1.0 (i.e., driver chemicals) by river segment and ecological receptor. Segment 3 (RM 4.9 to 6.5) was identified as posing the highest risk to all ecological receptors (Figure 6-1). The high potential risk in this river segment is largely driven by a single sediment sample with a lead concentration of 26,000 mg/kg dry weight (based on the year 2000 data). A lead concentration of this magnitude was not detected in the 1998 sampling event. It is interesting that maximum concentrations of other metals were also detected in the same sample, suggesting that the lead result is not a lead-specific anomaly. Further sampling of this location would be useful to understand the extent of contamination.

PCB concentrations in tissue and sediment were consistently elevated, posing potential risk to wildlife and aquatic life in the following river segments: RMs 3.2 to 4.9, RMs 4.9 to 6.5, and RMs 6.5 to 8.8. The river locations with the highest PCB levels were quite variable and differed between the 1998 and 2000 data. In 1998, for example, the highest PCB concentrations occurred between RMs 1.8 to 3.8 in the top 24" of sediment. In 2000, the highest PCB concentrations occurred between RMs 3.6 to 5.8. These results suggest that identification and potential remediation of hot spots should rely most heavily on the most current data. In addition, it should be noted that PCB and lead hot spots were not co-located, suggesting different sources, and possibly transport, within the river.

Given that many of the lines of evidence used in this evaluation were based on independent studies from multiple years, it is recommended that the COPCs and locations of greatest concern identified here be further evaluated using temporally and spatially co-located chemistry data and chronic bioassays. These studies would support whether the COPCs identified here are truly of concern and would assist in prioritizing further remediation options.

**Table 6-1. Chemicals with Chronic HQs > 1.0 by Ecological Receptor and River Segment**

<b>Receptor</b>	<b>Segment 1 (RM 0-3.2)</b>	<b>Segment 2 (RM 3.2-4.9)</b>	<b>Segment 3 (RM 4.9-6.5)</b>	<b>Segment 4 (RM 6.5-8.8)</b>
Aquatic Life - Pelagic	Aluminum (30) <sup>a</sup> Iron (4.3) Manganese (17)	Aluminum (25) Iron (4.2) Manganese (19)	Aluminum (14) Iron (2.8) Manganese (68)	Aluminum (13) Iron (1.9) Manganese (24)
Aquatic Life - Benthic	Lead (1.2) Nickel (1.3) PCBs (2.2)	Lead (3.2) Nickel (1.1) PCBs (11)	Cadmium (1.2) Chromium (1.2) Lead (199) Nickel (1.2) PCBs (11)	Chromium (1.1) Copper (1.0) Lead (3.6) Zinc (1.1) PCBs (11)
Bald Eagle	PCBs (1.3)	PCBs (4.3)	Lead (2.6) PCBs (3.5)	PCBs (2.9)
Common Tern	Selenium (1.1) PCBs (6.8)	PCBs (22) DDT (1.9)	Lead (13) Selenium (1.2) PCBs (18) DDT (2.4)	PCBs (15) DDT (4.0)
Spotted Sandpiper	Aluminum (1.7) PCBs (2.7)	Aluminum (1.9) Chromium (1.0) Lead (1.0) PCBs (17)	Aluminum (1.6) Chromium (2.3) Lead (71) PCBs (11) DDT (1.6)	Aluminum (1.6) Chromium (2.3) Lead (1.4) Cyanide (1.2) PCBs (11) DDT (1.7)
Mink	Aluminum (16) Lead (1.7) Selenium (1.6) Thallium (1.7)	Aluminum (18) Iron (1.0) Lead (4.1) Thallium (2.1) PCBs (1.9)	Aluminum (15) Lead (254) Selenium (1.6) Thallium (1.5)	Aluminum (15) Lead (6.0) Selenium (1.1) Thallium (2.7)

<sup>a</sup> Value in parentheses is the chronic HQ.

Figure

## 6-1 Ecological Hazard Quotient Comparison by River Segment

## 7. REFERENCES

- Ankley, G.T., D.M. Di Toro, D.J. Hansen, and W.J. Berry. 1996. Technical basis and proposal for deriving sediment quality criteria for metals. *Environ. Toxicol. Chem.* 15(12):2056-2066.
- Bengtsson, B.E. 1980. Long-term effects of PCB (Clophen A50) on growth, reproduction, and swimming performance in the minnow *Phoxinus phoxinus*. *Water Res.* 14:681-687.
- Bryson, W.T., K.A. MacPherson, M.A. Mallin, W.E. Partin, and S.E. Woock. 1985. Hyco Reservoir 1984 bioassay report. Carolina Power and Light Company, New Hill, North Carolina. 51 pp.
- Casas, G.A. and E.A. Crecelius. 1994. Relationships between acid-volatile sulfide and the toxicity of zinc, lead, and copper in marine sediments. *Environ. Toxicol. Chem.* 13:529-536.
- Cook, R.B., G.W. Suter II, and E.R. Sain. 1999. Ecological risk assessment in a large river-reservoir: 1. Introduction and background. *Environ. Toxicol. Chem.* 18(4):581-588.
- Coyle, J.J., D.R. Buckler, C.G. Ingersoll, J.F. Fairchild, and T.W. May. 1993. Effect of dietary selenium on the reproductive success of bluegills (*Lepomis macrochirus*). *Environ. Toxicol. Chem.* 12:551-565.
- DeVault, D. 2000. Personal communication. U.S. Fish and Wildlife Service.
- Di Toro, D.M., S.M. Mahond, D.J. Hansen, K.J. Scott, A.R. Carlson, and G.T. Ankley. 1990. Toxicity of cadmium in sediments: the role of acid-volatile sulfide. *Environ. Toxicol. Chem.* 9:1487-1502.
- Di Toro, D.M., S.M. Mahond, D.J. Hansen, K.J. Scott, A.R. Carlson, and G.T. Ankley. 1992. Acid-volatile sulfide predicts the toxicity of cadmium and nickel in sediments. *Environ. Toxicol. Chem.* 26:96-101.
- Di Toro, D.M. and J.A. McGrath. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ. Toxicol. Chem.* 19:1971-1982.
- Dunning, J.B., Jr. 1993. CRC handbook of avian body masses. CRC Press, Inc., Boca Raton, Florida. 371 pp.
- Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.7.
- Environment Canada. 1995. Interim sediment quality guidelines: Soil and sediment quality section guidelines division. Ecosystem Conservation Directorate Evaluation and Interpretation Branch, Ottawa, Ontario.
- Ferraro, S.P., H. Lee II, R.J. Ozretich, and D.T. Specht. 1990. Predicting bioaccumulation potential: A test of a fugacity-based model. *Arch. Environ. Contam. Toxicol.* 19:386-394.
- Fischer, D.L. and G.A. Hancock. 1997. Interspecies extrapolation of acute toxicity in birds: Body scaling vs. phylogeny. Poster presented at 1997 SETAC meeting, San Francisco, California.

- Gliet, A. 1985. Estimation for small normal data sets with detection limits. *Environmental Science and Technology* 19:1201-1206.
- Grieb, T.M., C.T. Driscoll, S.P. Gloss, C.L. Schofield, G.L. Bowie, and D.B. Porcella. 1990. Factors affecting mercury accumulation in the upper Michigan Peninsula. *Environ. Toxicol. Chem.* 9:919-930.
- Hamilton, S.J., K.J. Buhl, N.L. Faerber, R.H. Wiedmeyer, and F.A. Bullard. 1990. Toxicity of organic selenium in the diet of chinook salmon. *Environ. Toxicol. Chem.* 9:347-358.
- Hare, L., R. Carignan, and M.A. Huerta-Diaz. 1994. A field study of metal toxicity and accumulation by benthic invertebrates; Implications for the acid-volatile sulfide (AVS) model. *Limnol. Oceanogr.* 39(7):1653-1668.
- Harrahy, E.A. and W.H. Clements. 1997. Toxicity and bioaccumulation of a mixture of metals in *Chironomus tentans* (Diptera: Chironomidae) in synthetic sediment. *Environ. Toxicol. Chem.* 16(2):317-327.
- Hopkins, W.A. 2000. Reptile ecotoxicology: Challenges and opportunities on the last frontier in vertebrate ecotoxicology. *Environ. Toxicol. Chem.* 19(10):2391-2393.
- Ingersoll, C.G., P.S. Haverland, E.L. Brunson, T.J. Canfield, F.J. Dwyer, C.E. Henke, N.E. Kemble, D.R. Mount, and R.G. Fox. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod *Hyalella azteca* and the midge *Chironomus riparius*. *J. Great Lakes Res.* 22(3):602-623.
- Ingersoll, C.G., D.D. MacDonald, N. Wang, J.L. Crane, L.J. Field, P.S. Haverland, N.E. Kemble, R.A. Lindskoog, C. Severn, and D.E. Smorong. 2001. Predictions of sediment toxicity using consensus-based freshwater sediment quality guidelines. *Arch. Environ. Contam. Toxicol.* 41:8-21.
- Jarvinen and Ankley. 1999. Linkage of effects to tissue residues: Development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SETAC Technical Publications Series. SETAC Press, Society of Environmental Toxicology and Chemistry, Pensacola, Florida.
- Korgen, B. 2000. Bonanza for Lake Superior: Seiches do more than move water. Minnesota Sea Grant, February 2000.
- Long, E.R., L.J. Field, and D.D. MacDonald. 1998. Predicting toxicity in marine sediments with numerical sediment quality guidelines. *Environ. Toxicol. Chem.* 17(4):714-727.
- LTI (Limno-Tech, Inc.), Intertox, and Parametrix, Inc. 2000. Proposal for the Ottawa River environmental hot spot delineation and risk assessment. 31 pp.
- LTI (Limno-Tech, Inc.). 2001. Database for Ottawa River, Ohio. Database report version 1.2. Prepared by Limno-Tech, Inc., Ann Arbor, Michigan. 44 pp.

- O'Connor, T.P., K.D. Daskalakis, J.L. Hyland, J.F. Paul, and J.K. Summers. 1998. Comparisons of sediment toxicity with predictions based on chemical guidelines. *Environ. Toxicol. Chem.* 17(3):468-471.
- Ohio EPA. 1987. Biological criteria for the protection of aquatic life: Volume II. User's manual for biological field assessment of Ohio's surface waters. Division of Water Quality Monitoring and Assessment, Surface Water Section, Columbus, Ohio.
- Ohio EPA. 1998. Biological, fish tissue, and sediment study of the Ottawa River Dura Avenue Landfill 1996. Lucas County, Ohio. OEPA Technical Report MAS/1997-12-8. January 30, 1998.
- Ohio EPA. 1998. A report on whole sediment toxicity of sediments from sites on the Ottawa River to *Hyalella azteca* and *Lumbriculus variegatus* for the Great Lakes National Program Office. Bioassay Section, Division of Environmental Services, Ohio EPA. 18 pp. + appendices.
- Ohio EPA. 1999. Ohio water quality standards. OAC 3745-1-07.
- Ohio EPA. 2000. Ottawa River geographic initiative (GI) work plan. June 2000.
- Olafsson, P.G., A.M. Bryan, B. Bush, and W. Stone. 1983. Snapping turtles—A biological screen for PCBs. *Chemosphere* 12(11/12):1525-1532.
- Ontario Ministry of the Environment and Energy (OMEE). 1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Prepared by D. Persaud, R. Jaagumagi, and A. Hayton, Water Resources Branch, Ontario Ministry of the Environment and Energy. 24 pp. + figures.
- Pagano, J.J., P.A. Rosenbaum, R.N. Roberts, G.M. Sumnee, and L.V. Williamson. 1999. Assessment of maternal contaminant burden by analysis of snapping turtle eggs. *J. Great Lakes Res.* 25(4):950-961.
- Parkhurst, B.R., W. Warren-Hicks, R.D. Cardwell, J. Volosin, T. Etchison, J.B. Butcher, and S.M. Covington. 1996. Methodology for aquatic ecological risk assessment: A multi-tiered approach. Project 91-AER-1. Prepared for Water Environment Research Foundation, Alexandria, Virginia.
- Porter, P.S., R.C. Ward, and H.F. Bell. 1988. The detection limit. *Environmental Science and Technology* 22(8):856-861.
- Prothro, M.G. 1993. Office of Water policy and technical guidance on interpretation and implementation of aquatic life metals criteria. Memorandum from M.G. Prothro (Acting Assistant Administrator for Water) to Water Management Division Directors, Environmental Services Division Directors, and Regions I-X. October 1, 1993.
- Rabuck, L., M. Sandheinrich, and R. Rada. 1997. Bioaccumulation of methylmercury in fathead minnows fed a naturally contaminated diet. Presented at the SETAC 18<sup>th</sup> Annual Meeting, 16-20 November 1997, San Francisco, California.
- Rand, G.M. 1995. Fundamentals of aquatic toxicology: Effects, environmental fate and risk assessment. Second Edition. Taylor and Francis, Washington D.C. 1125 pp.

- Rodgers, D.W. and F.W.H. Beamish. 1982. Dynamics of dietary methylmercury in rainbow trout, *Salmo gairdneri*. *Aquat. Toxicol.* 2:271-290.
- Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological benchmarks for wildlife: 1996 Revision. U.S. Department of Energy, ES/ER/TM-86/R3.
- Shieldcastle, M. 2000. Personal communication of September 22, 2000. Wildlife officer, Crane Creek Wildlife Research Station.
- Skorupa, J.W., S.P. Morman, and J.S. Sefchick-Edwards. 1996. Guidelines for interpreting selenium exposures of biota associated with non-marine aquatic habitats. Technical Report. U.S. Fish and Wildlife Service, Ecological Services Field Office, Sacramento, California.
- Sprague, J.B. and B.A. Ramsay. 1965. Lethal levels of mixed copper-zinc solutions for juvenile salmon. *J. Fish. Res. Board Can.* 22:425-432.
- Stalmaster, M.V. 1987. *The bald eagle*. Universe Books, New York, New York.
- Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. U.S. EPA, Washington, D.C. NTIS No. PB85-227049. 98 pp.
- Suedel, B.C., J.A. Boraczek, R.K. Peddicord, P.A. Clifftort, and T.M. Dillon. 1994. Trophic transfer and biomagnification potential of contaminants in aquatic ecosystems. *Rev. Environ. Contam. Toxicol.* 136:21-89.
- Suter, G.W., L.W. Bamthouse, R.A. Efrogmson, and H. Jager. 1999. Ecological risk assessment in a large river-reservoir: 2. Fish Community. *Environ. Toxicol. Chem.* 18(4):589-598.
- Suter, II, G.W. 1993. *Ecological risk assessment*. Lewis Publishers, Boca Raton, Florida. 538 pp.
- Szebedinszky, C., J.C. McGeer, D.G. McDonald, and C.M. Wood. 2001. Effects of chronic Cd exposure via the diet or water on internal organ-specific distribution and subsequent gill Cd uptake kinetics in juvenile rainbow trout (*Oncorhynchus mykiss*). *Environ. Toxicol. Chem.* 20(3):597-607.
- Tracey, G.A., and D.J. Hansen. 1996. Use of biota-sediment accumulation factors to assess similarity of nonionic organic chemical exposure to benthically-coupled organisms of differing trophic mode. *Arch. Environ. Contam. Toxicol.* 30:467-475.
- Travis, C.C., R.K. White, and R.C. Ward. 1990. Interspecies extrapolation of pharmacokinetics. *J. Theoret. Biol.* 142:285-304.
- Travis, C.C. and R.K. White. 1988. Interspecific scaling of toxicity data. *Risk Anal.* 8:119-125.
- U.S. EPA. 1991a. Technical support document for water quality-based toxics control. Office of Water, Technical Document, EN-336. EPA/505/2-90-001.
- U.S. EPA. 1991b. U.S. EPA Region III guidance on handling chemical concentration data near the detection limit in risk assessments. Roy L. Smith, Interim Final, November 4, 1991.

- U.S. EPA. 1992. Draft report: A cross-species scaling factor for carcinogen risk assessment based on equivalent of  $\text{mg/kg}^{3/4}/\text{day}$ ; Notice. Federal Register. 57: 24152-24173.
- U.S. EPA. 1993a. Wildlife exposure factors handbook. Volume 1 of 2. Office of Research and Development, U.S. Environmental Protection Agency, Washington, D.C. EPA/600/R-93/187a.
- U.S. EPA. 1993b. Sediment quality criteria for the protection of benthic organisms: Fluoranthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-012.
- U.S. EPA. 1993c. Sediment quality criteria for the protection of benthic organisms: Acenaphthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-013.
- U.S. EPA. 1993d. Sediment quality criteria for the protection of benthic organisms: Phenanthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-014.
- U.S. EPA. 1993e. Sediment quality criteria for the protection of benthic organisms: Dieldrin. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-015.
- U.S. EPA. 1993f. Sediment quality criteria for the protection of benthic organisms: Endrin. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-016.
- U.S. EPA. 1994. Equilibrium partitioning approach to predicting metal bioavailability in sediments and the derivation of sediment quality criteria for metals. U.S. EPA, Office of Water and Office of Research and Development. EPA/822-D-94-002.
- U.S. EPA. 1997. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. U.S. Environmental Protection Agency, Washington, D.C. EPA 540-R-97-006.
- U.S. EPA. 1998a. Guidelines for ecological risk assessment. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, D.C. EPA/630/R-95/002F.
- U.S. EPA. 1998b. 1998 Update of ambient water quality criteria for ammonia. Office of Water, U.S. EPA, Washington, D.C. EPA 822-R-98-008.
- U.S. EPA. 2000a. Equilibrium sediment partitioning sediment guidelines (ESGs) for the protection of benthic organisms: PAH mixtures. Draft report, Washington, D.C.
- U.S. EPA. 2000b. Draft implementation framework for the use of equilibrium partitioning sediment guidelines: Guidance for using equilibrium partitioning sediment guidelines (ESGs) in water quality programs. U.S. EPA, Office of Science and Technology. December 2000.
- Van der Geest, H.G., G.D. Greve, M.-E. Boivin, M.H.S. Kraak, and C.A.M. van Gestel. 2000. Mixture toxicity of copper and diazinon to larvae of the mayfly (*Ephoron virgo*) judging additivity at different effect levels. Environ. Toxicol. Chem. 19(12):2900-2905.

- Vighi, M. 1981. Lead uptake and release in an experimental trophic chain. *Ecotoxicol. Environ. Safety*. 5:177-193.
- Welsh, P.G., J. Lipton, and G.A. Chapman. 2000. Evaluation of water-effect ratio methodology for establishing site-specific water quality criteria. *Environ. Toxicol. Chem.* 19(6):1616-1623.
- Williams, P. 2000. Personal communication of June 6, 2000. Ohio Environmental Protection Agency.
- Wobeser, G. 1975. Prolonged oral administration of methyl mercury chloride to rainbow trout (*Salmo gairdneri*) fingerlings. *J. Fish. Res. Board Can.* 32:2015-2023.
- Woock, S. E., W.R. Garrett, W.E. Partin, and W.T. Bryson. 1987. Decreased survival and teratogenesis during laboratory selenium exposures to bluegill, *Lepomis macrochirus*. *Bull. Environ. Contam. Toxicol.* 39:998-1005.

**APPENDIX A**

---

**Exposure Data**

## APPENDIX A - EXPOSURE DATA

As discussed in Section 3.1 of the main report, wildlife and aquatic life exposures to chemicals were evaluated using measured chemical concentrations, where available. For some chemicals and environmental media, however, it was necessary to estimate chemical concentrations. Measured and estimated chemistry data are summarized in Sections A.1 and A.2 below.

### A.1 MEASURED CHEMICAL CONCENTRATIONS

The summary statistics for chemicals with a detection frequency greater than 5 percent are provided in Tables A-1, A-2, and A-3 for fish tissue, sediment, and surface water, respectively. The 95 percent UCL on the mean was used to estimate chronic exposure (Equation A-1) and the 95<sup>th</sup> percentile of the data was used to estimate acute exposures (Equation A-2).

$$95\% \text{ UCL (Population)} = \text{Mean} + t_{0.05, n-1} \times \text{SD} \quad (\text{A-1})$$

$$95\% \text{ UCL (Mean)} = \text{Mean} + t_{0.05, n-1} \times \frac{\text{SD}}{\sqrt{n}} \quad (\text{A-2})$$

Where:	95% UCL (Mean)	=	Upper 95% confidence limit of the mean concentration
	$t_{0.05, n-1}$	=	Critical value of the Student's <i>t</i> distribution at n-1 degrees of freedom, one-tailed
	SD	=	Standard deviation
	n	=	Sample size

### A.2 ESTIMATION OF TISSUE CONCENTRATIONS

As summarized in Tables A-1, A-2, and A-3, concentration data were available for a variety of chemical classes and environmental media. However, to thoroughly evaluate exposure of all receptors to the chemicals likely to be present in the Ottawa River, it was necessary to estimate the concentrations of some chemicals in tissue.

No chemistry data are currently available for macroinvertebrates in the lower Ottawa River; however, these data are necessary for estimating potential risks to the spotted sandpiper. Because benthos are primarily exposed to chemicals associated with sediment (e.g., pore water, detritus), chemical concentrations in invertebrate tissue were estimated using biota-sediment accumulation factors (BSAFs). This is applicable to lipophilic organic chemicals. The BSAFs are expressed as the ratio of a chemical's concentration in biological tissue normalized for the fraction lipid to the chemical's concentration in sediment normalized for organic carbon<sup>13</sup>. Equation A-3 shows how benthic tissue concentrations were estimated from sediment concentrations using BSAFs:

---

<sup>13</sup> Given the multitude of site-specific factors that influence the bioaccumulation of metals from sediment, methods do not exist for adequately estimating metal concentrations in tissue from sediment concentrations.

$$C_{\text{tissue}} = \frac{\text{BSAF} \times C_{\text{sed}} \times F_1}{F_{\text{oc}}} \quad (\text{A-3})$$

Where:

- $C_{\text{tissue}}$  = Chemical concentration in tissue (mg/kg wet weight)
- $\text{BSAF}$  = Biota-sediment accumulation factor (kg organic carbon/kg lipid)
- $C_{\text{sed}}$  = Chemical concentration in sediment (mg/kg wet weight)
- $F_1$  = Fraction lipid in tissue
- $F_{\text{oc}}$  = Fraction organic carbon in sediment

The BSAFs used in this ecological SLRA were estimated using modeled data from Di Toro and McGrath (2000) or measured data from other sources (e.g., Tracey and Hansen 1996, Ferraro et al. 1990). The empirically derived BSAFs met the guidelines outlined by Tracey and Hansen (1996)<sup>14</sup>. The BSAFs and the estimated macroinvertebrate tissue concentrations using this approach are provided in Table A-4.

---

<sup>14</sup> These guidelines include: (1) BSAF studies must report the sediment total organic carbon, organism lipid, lipid method, and paired tissue and sediment chemical concentrations; (2) exposures must occur from “naturally-contaminated” (i.e., not laboratory-spiked) sediments; (3) laboratory studies must be a minimum of 28 days; and (4) aggregated chemical data are to be excluded.

## REFERENCES – APPENDIX A

- AQUIRE. 2001. AQUatic toxicity Information REtrieval database. Environmental Research Laboratory. United States Environmental Protection Agency, Duluth, Minnesota. Available at: [http://www.epa.gov/ecotox/ecotox\\_search\\_driver.htm](http://www.epa.gov/ecotox/ecotox_search_driver.htm)
- Call, D.J., L.T. Brooke, N. Ahmad and J.E. Richter. 1983. Toxicity and metabolism studies with EPA priority pollutants and related chemicals in freshwater organisms. PB83-263665. National Technical Information Service, Springfield, Virginia.
- Carr, K.H., G.T. Coyle, and R.A. Kimerle. 1997. Bioconcentration of [<sup>14</sup>C]butyl benzyl phthalate in bluegill sunfish (*Lepomis macrochirus*). Environ. Toxicol. Chem. 16(10):2200-2203.
- Di Toro, D.M. and J.A. McGrath. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. Environ. Toxicol. Chem. 19:1971-1982.
- Ferraro, S.P., H. Lee II, R.J. Ozretich, and D.T. Specht. 1990. Predicting bioaccumulation potential: A test of a fugacity-based model. Arch. Environ. Contam. Toxicol. 19:386-394.
- Lang, P.-Z., Y. Wang, D.-B. Chen, N. Wang, X.-M. Zhao, and Y.-Z. Ding. 1997. Bioconcentration, elimination and metabolism of 2,4-dinitrotoluene in carps (*Cyprinus carpio*L.). Chemosphere 35(8):1799-1815.
- McCarty, L.S. 1986. The relationship between aquatic toxicity QSARs and bioconcentration for some organic chemicals. Environ. Toxicol. Chem. 5:1071-1080.
- Tracey, G.A., and D.J. Hansen. 1996. Use of biota-sediment accumulation factors to assess similarity of nonionic organic chemical exposure to benthically-coupled organisms of differing trophic mode. Arch. Environ. Contam. Toxicol. 30:467-475.
- U.S. EPA. 1993. Ambient aquatic life water quality criteria for 2,4-dimethylphenol (draft). Office of Water and Office of Research and Development, U.S. EPA, Washington, D.C., Duluth, Minnesota, and Narragansett, Rhode Island. 822/R93025.

---

**APPENDIX B**

**Toxicity Data**

## APPENDIX B – TOXICITY DATA

The toxicity data used for wildlife receptors and aquatic life are presented in this appendix. Issues associated with the wildlife and aquatic life toxicity data are discussed separately below.

### B.1 WILDLIFE

The acute and chronic toxicity data for wildlife receptors (birds and mammals) are provided in Tables B-1 and B-2, respectively. As mentioned in Section 4.1, the toxicity data for mink were scaled for the relative weight of mink to the laboratory test organism. The equation for body weight scaling, from Sample et al. (1996), is as follows:

$$\text{NOAEL}_w = \text{NOAEL}_t \left( \frac{\text{BW}_t}{\text{BW}_w} \right)^{1/4} \quad (\text{B-1})$$

Where:

NOAEL <sub>w</sub>	=	No Observed Adverse Effects Level for mammalian wildlife receptor
NOAEL <sub>t</sub>	=	No Observed Adverse Effects Level for mammalian test species
BW <sub>t</sub>	=	Body weight of mammalian test species
BW <sub>w</sub>	=	Body weight of mammalian wildlife receptor

### B.2 AQUATIC LIFE

The acute and chronic surface water toxicity data for aquatic life are presented in Table B-3; the sediment guidelines used are provided in Table B-4.

The toxicity of PAH mixtures in surface water and sediment were also evaluated using the target lipid narcosis model derived by Di Toro et al. (2000) and Di Toro and McGrath (2000). Di Toro et al. (2000) provided a method for deriving PAH criteria for surface water and Di Toro and McGrath (2000) provided a method for deriving PAH criteria for sediments and mixtures. The methods for these approaches, summarized from their respective papers, are provided below.

PAHs are type I narcotic chemicals. Accordingly, the toxicity of a mixture of PAHs should be additive. The target lipid model was developed to describe the toxicity of all type I narcotics. This model relates narcotic lethality to the target tissue of an organism, in this case, the lipid. The partitioning of the narcotics into the lipid is assumed to be species independent, but the threshold at which the narcotic concentration in the lipid results in mortality is species specific and dependent on chemical differences. However, the slope of the relationship between toxicity and the chemical's octanol-water partition coefficient (K<sub>ow</sub>) is essentially constant between species. Using this relationship, species-specific body burdens can be used to calculate water quality criteria using an approach analogous to the U.S. EPA's current guidelines. Acute and chronic toxicity values using this approach are provided in Table B-2 of Appendix B.

The target lipid model can also be applied to sediments and PAH mixtures. Using the chronic water-based toxicity values described in the preceding paragraph, equilibrium partitioning (EqP) can be used to calculate sediment guidelines. EqP theory holds that nonionic chemicals in sediment partition between sediment organic carbon, interstitial water, and benthic organisms (Di Toro et al. 1991). At equilibrium, if the concentration of one phase is known, the concentrations in the others can be predicted. Di Toro et

al. (1991) reported that the biological responses of benthic organisms to nonionic chemicals in sediments are different across sediments when the sediment concentrations are reported on a dry-weight basis, but similar when the concentrations are normalized for the organic carbon content of the sediment. Accordingly, the use of narcosis theory and EqP allows sediment guidelines to be readily developed for nonionic chemicals, such as PAHs, with only data on the chemicals' Kow.

## REFERENCES – APPENDIX B

- Ahdaya, S.M., P.V. Shah, and F.E. Guthrie. 1976. Thermoregulation in mice treated with Parathion, Carbaryl, or DDT. *Toxicol. Appl. Pharmacol.* 35:575-580.
- Ambrose, A.M., P.S. Larson, J.F. Borzelleca, and G.R. Hennigar, Jr. 1976. Long-term toxicologic assessment of nickel in rats and dogs. *J. Food Sci.Tech.* 13:181-187.
- AQUIRE. 2000. AQUatic toxicity Information REtrieval database. Environmental Research Laboratory. United States Environmental Protection Agency, Duluth, Minnesota. Available at: [http://www.epa.gov/ecotox/ecotox\\_search\\_driver.htm](http://www.epa.gov/ecotox/ecotox_search_driver.htm)
- ATSDR. 1989. Toxicological profile for pentachlorophenol. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1989. Toxicological profile for phenol. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1989. Toxicological profile for di-n-butyl phthalate. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1990. Toxicological profile for 2-methylphenol. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1991. Toxicological profile for cyanide. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1991a. Toxicological profile for selected PCBs (Aroclor-1260, -1254, -1248, -1242, -1232, -1221, and -1016). U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1991b. Toxicological profile for aldrin/dieldrin. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1991c. Toxicological profile for heptachlor/heptachlor epoxide. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1992a. Toxicological profile for 4,4'-DDT, 4,4'-DDE, and 4,4'-DDD. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1992b. Toxicological profile for alpha, beta, gamma, and delta hexachlorocyclohexane. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1992c. Toxicological profile for methoxychlor. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1992d. Toxicological profile for vanadium. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.

- ATSDR. 1993. Toxicological profile for endosulfan. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1994. Toxicological profile for mercury. U.S. Department of Health and Human Services, ATSDR, Atlanta, Georgia.
- ATSDR. 1997a. Toxicological profile for chlorophenols. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.
- ATSDR. 1997b. Toxicological profile for manganese. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.
- ATSDR. 1997c. Toxicological profile for 2,4-dinitrotoluene and 2,6-dinitrotoluene. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.
- Aulerich, R.J., R.K. Ringer, M.R. Bleavins, et al. 1982. Effects of supplemental dietary copper on growth, reproductive performance and kit survival of standard dark mink and the acute toxicity of copper to mink. *J. Animal Sci.* 55:337-343.
- Battelle. 1980. Subchronic toxicity study: Naphthalene (C52904), B6C3F1 mice. Report to U.S. Department of Health and Human Services, National Toxicology Program, Research Triangle Park, North Carolina, by Battelle's Columbus Laboratories, Columbus, Ohio.
- Bedford, C.T., D.H. Hutson, and I.L. Natoff. 1975. The acute toxicity of Endrin and its metabolites to rats. *Toxicol. Appl. Pharmacol.* 33:115-121.
- Begearmi, M.M., H.E. Ganther, and M.L. Sunde. 1980. Toxicity of mercuric chloride in Japanese quail as affected by methods of incorporation into the diet. *Poult. Sci.* 59(10):2216-2220.
- Cain, B.W., and E.A. Pafford. 1981. Effects of dietary nickel on survival and growth of Mallard ducklings. *Arch. Environm. Contam. Toxicol.* 10:737-745.
- Carriere, D., K.L. Fischer, D.B. Peakall, and P. Anghern. 1986. Effects of dietary aluminum sulfate on reproductive success and growth of ring doves. *Can. J. Zool.* 64:1500-5.
- Cecil, H.C., J. Bitman, R.J. Lillie, G.F. Fries, and J. Verrett. 1974. Embryotoxic and teratogenic effects on unhatched fertile eggs from hens fed PCBs. *Bull. Environ. Contam. Toxicol.* 11:489-495.
- Di Toro, D.M. et al. 1991. Technical basis for the equilibrium partitioning method for establishing sediment quality criteria. *Environ. Toxicol. Chem.* 11:1541-1583.
- Di Toro, D.M., J.A. McGrath, and D.J. Hansen. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water and tissue. *Environ. Toxicol. Chem.* 19:1951-1970.
- Di Toro, D.M. and J.A. McGrath. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ. Toxicol. Chem.* 19:1971-1982.

- Dieter, M.P., M.I. Luster, G.A. Boorman, C.W. Jameson, J.H. Dean, and J.W. Cox. 1983. Immunological and biochemical responses in mice treated with mercuric chloride. *Toxicol. Appl. Pharmacol* 68: 218-228.
- DOTN (Department of the Navy). 1997. Development of toxicity reference values as part of a regional approach for conducting ecological risk assessments at naval facilities in California. DOTN, San Bruno, California. 134p. Eco-SSL (Ecological Soil Screening Level). 2000a. Ecological soil screening level guidance-wildlife TRVs. Appendix 4-6. Available at: <http://www.epa.gov/oerrpage/superfund/programs/risk/>. 79 pp.
- Eco-SSL (Ecological Soil Screening Level). 2000. Ecological soil screening level guidance. Available at: <http://www.epa.gov/oerrpage/superfund/programs/risk/>. 113 pp.
- Edens, F., W.E. Benton, S.J. Bursian, and G.W. Morgan. 1976. Effect of Dietary Lead on Reproductive Performance in Japanese Quail, *Coturnix coturnix japonica*. *Toxicol. Appl. Pharmacol.* 38:307-314.
- Eisler, R. 1985. Toxaphene hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.4.
- Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.7.
- Eisler, R. 1988. Arsenic hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.12.
- Eisler, R. 1989. Molybdenum hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.19.
- Eisler, R. 1990. Chlordane hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Laurel, Maryland. Biological Report No. 85:1.21.
- Environment Canada. 1995. Interim sediment quality guidelines: Soil and sediment quality section guidelines division. Ecosystem Conservation Directorate Evaluation and Interpretation Branch, Ottawa, Ontario.
- Gersich, F.M., E.A. Bartlett, P.G. Murphy, and D.P. Milazzo. 1989. Chronic toxicity of biphenyl to *Daphnia magna* Straus. *Bull. Environ. Contam. Toxicol.* 43:355-362.
- HEAST (Health Effects Assessment Summary Tables). 1995. Office of Solid Waste and Emergency Response, U.S. EPA. EPA/540/R-95/036.
- Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, and D.M.G. Weller. 1987. Reproduction in mallards fed selenium. *Environ. Toxicol. Chem.* 6:423-433.
- Hill, E. F. 1981. Inorganic and organic mercury chloride toxicity to coturnix: Sensitivity related to age and quantal assessment of physiological responses. Ph.D. Thesis, Univ. Maryland, College Park. 221 pp.

- Hill, E. F. and M. B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to *Coturnix*. U.S. Fish and Wildlife Service. Tech. Report No. 2.
- Hudson, R.H., M.A. Haegele, and R.K. Tucker. 1979. Acute oral and percutaneous toxicity of pesticides to mallards: correlations with mammalian toxicity data. *Toxicol. Appl. Pharmacol.* 47:451-460.
- Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. U.S. Fish Wildl. Serv. Resour. Publ. 153. 90 pp.
- Ingersoll, C.G., P.S. Haverland, E.L. Brunson, T.J. Canfield, F.J. Dwyer, C.E. Henke, N.E. Kemble, D.R. Mount, and R.G. Fox. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod (*Hyalella azteca*) and the midge (*Chironomus riparius*). *J. Great Lakes Res.* 22(3):602-623.
- Johnson, D., Jr., A.L. Mehring, J.R., and H.W. Titus. 1960. Tolerance of chickens for barium. *Proc. Soc. Exp. Biol. Med.* 104:436-438.
- Keplinger, M.L., O.E. Fancher, and J.C. Calandra. 1971. Toxicologic studies with polychlorinated biphenyls. *Toxicol. Appl. Pharmacol.* 19:402-403.
- Kostial, K. M. Blanus, and T. Maljkovic. 1989. Effect of a metal mixture in diet on the toxicokinetics and toxicity of cadmium, mercury and manganese in rats. *Toxicol. Ind. Health* 5(5):685-698.
- Laskey, J.W., G.L. Rehnberg, J.F. Hein, and S.D. Carter. 1982. Effects of chronic manganese (Mn<sub>3</sub>O<sub>4</sub>) exposure on selected reproductive parameters in rat. *J. Toxicol. Environ. Health* 9:677-687.
- Lillie, R.J., H.C. Cecil, J. Bitman, G.F. Fries, and J. Verret. 1975. Toxicity of certain polychlorinated and polybrominated efficiency of caged chickens. *Poult. Sci.* 54:1550-1555.
- Linder, R.E., T.B. Gaines, and R.D. Kimbrough. 1974. The effect of polychlorinated biphenyls on rat reproduction. *Food Cosmet. Toxicol.* 12:63-77.
- Mackenzie, K.M. and D.M. Angevine. 1981. Infertility in mice exposed *in utero* to benzo(a)pyrene. *Biol. Reprod.* 24:183-191.
- Mackenzie, R.D., R.U. Byerrum, D.F. Decker, C.A. Hoppert, and R.F. Langham. 1958. Chronic toxicity studies, II. Hexavalent and trivalent chromium administered in drinking water to rats. *Am. Med. Assoc. Arch. Ind. Health.* 18:232-234.
- Matuk, Y., M. Ghosh, and C. McCulloch. 1981. Distribution of silver in the eyes and plasma proteins of the albino rat. *Can J. Ophthalmol.* 16:145-150.
- Mehring, A.L., J.H. Brumbaugh, A.J. Sutherland, and H.W. Titus. 1960. The tolerance of growing chickens for dietary copper. *Poult. Sci.* 39:713-719.
- National Research Council (NRC). 1980. Mineral tolerance in domestic animals. National Academy of Sciences Press, Washington, D.C.
- Oak Ridge National Research Laboratory (ORNL). 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. ES/ER/TM-86/R3. Health Sciences Research Division, Oak Ridge, Tennessee.

- Ondreicka, R., E. Ginter, and J. Kortus. 1966. Chronic toxicity of aluminum in rats and mice and its effects on phosphorus metabolism. *Brit. J. Ind. Med.* 23:305-313.
- Ontario Ministry of the Environment and Energy. 1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Prepared by D. Persaud, R. Jaagumagi, and A. Hayton, Water Resources Branch, Ontario Ministry of the Environment and Energy. 24 pp. + figures.
- U.S. EPA. 1999b. 1999 update of ambient water quality criteria for ammonia. *Federal Register*: December 22, 1999 (Volume 64, Number 245), page 71974.
- Patton, J.F. and M.P. Dieter. 1980. Effects of petroleum hydrocarbons on hepatic function in the duck. *Comp. Biochem. Physiol.* 65C:33-36.
- Perry, H.M., E.F. Perry, M.N. Erlanger, and S.J. Kopp. 1983. Cardiovascular effects of chronic barium ingestion. In: *Proc. 17<sup>th</sup> Ann. Conf. Trace Substances in Environ. Health*, Vol. 17. University of Missouri Press, Columbia, Missouri.
- Podowski, A.A., B.C. Banerjee, and M. Feroz. 1979. Photolysis of heptachlor and cis-chlordane and toxicity of their photoisomers to animals. *Arch. Environ. Contam. Toxicol.* 8:509-518.
- Ringer, R.K. 1983. Toxicology of PCBs in mink and ferrets. Pages 227-240 in F.M. D'Itri and M.A. Kamrin, editors. *PCBs: human and environmental hazards*. Butterworth Publishing, Woburn, Massachusetts.
- RTECS (Registry of Toxic Effects of Chemical Substances). 1995. On-line computer database. NIOSH: U.S. Dept of Health and Human Services, Center for Disease Control.
- RTECS (Registry of Toxic Effects of Chemical Substances). 1997. On-line computer database. NIOSH: U.S. Dept of Health and Human Services, Center for Disease Control.
- Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological benchmarks for wildlife: 1996 Revision. U.S. Department of Energy, ES/ER/TM-86/R3.
- Schafer, Jr., E.W., W.A. Bowles, Jr., and J. Hurblet. 1983. The acute and oral toxicity repellency and hazard potential of 998 chemicals to one or more species of wild and domestic birds. *Arch. Environ. Contam. Toxicol.* 12(3):355-382.
- Schafer, Jr., E.W., and W.A. Bowles, Jr. 1985. Acute oral toxicity and repellency of 933 chemicals to house and deer mice. *Arch. Environ. Contam. Toxicol.* 14(1):111-129.
- Schlicker, S.A. and D.H. Cox. 1968. Maternal dietary zinc, and development and zinc, iron, and copper content of the rat fetus. *J. Nutr.* 95:287-294.
- Schroeder, H.A. and M. Mitchener. 1971. Toxic effects of trace elements on the reproduction of mice and rats. *Arch. Environ. Health* 23:102-106.
- Stahl, J.L., J.L. Greger, and M.E. Cook. 1990. Breeding-hen and progeny performance when hens are fed excessive dietary zinc. *Poult. Sci.* 69:259-263.

- Szebedinszky, C., J.C. McGeer, D.G. McDonald, and C.M. Wood. 2001. Effects of chronic Cd exposure via the diet or water on internal organ-specific distribution and subsequent gill Cd uptake kinetics in juvenile rainbow trout (*Oncorhynchus mykiss*). *Environ. Toxicol. Chem.* 20(3):597-607.
- Tewe, O.O., and J.H. Maner. 1981. Long-term and carry-over effect of dietary inorganic cyanide (KCN) in the life cycle performance and metabolism of rats. *Toxicol. Appl. Pharmacol.* 58:1-7
- Tucker, R.K., and M.A. Haegele. 1971. Comparative acute oral toxicity of pesticides to six species of birds. *Toxicol. Appl. Pharmacol.* 20(1):57-65.
- U.S. EPA. 1980a. Ambient aquatic life water quality criteria for thallium. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-074.
- U.S. EPA. 1980b. Ambient aquatic life water quality criteria for polychlorinated biphenyls. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-068.
- U.S. EPA. 1980c. Ambient aquatic life water quality criteria for aldrin/dieldrin. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-019.
- U.S. EPA. 1980d. Ambient aquatic life water quality criteria for DDT. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-038.
- U.S. EPA. 1980e. Ambient aquatic life water quality criteria for heptachlor. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-052.
- U.S. EPA. 1980f. Ambient aquatic life water quality criteria for hexachlorocyclohexane. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-80-054.
- U.S. EPA. 1984. Health effects assessment for iron (and compounds). Office of Research and Development. United States Environmental Protection Agency, Cincinnati, Ohio. EPA/540/1-86-054.
- U.S. EPA. 1986a. Quality criteria for water 1986. Office of Water, Regulations and Standards. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-86-001.
- U.S. EPA. 1986b. Ambient aquatic life water quality criteria for toxaphene. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-86-006.
- U.S. EPA. 1988. Ambient aquatic life water quality criteria for aluminum. Office of Water, Regulations and Standards, Criteria and Standards Division. United States Environmental Protection Agency, Washington, D.C. EPA 440/5-88-008.

- U.S. EPA. 1991. Water quality criteria summary chart. Office of Science and Technology, Health and Ecological Criteria Division. Washington, D.C.
- U.S. EPA. 1993a. Sediment quality criteria for the protection of benthic organisms: Fluoranthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-012.
- U.S. EPA. 1993b. Sediment quality criteria for the protection of benthic organisms: Acenaphthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-013.
- U.S. EPA. 1993c. Sediment quality criteria for the protection of benthic organisms: Phenanthene. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-014.
- U.S. EPA. 1993d. Sediment quality criteria for the protection of benthic organisms: Dieldrin. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-015.
- U.S. EPA. 1993e. Sediment quality criteria for the protection of benthic organisms: Endrin. United States Environmental Protection Agency, Health and Ecological Criteria Division, Washington, D.C. EPA-822-R-93-016.
- U.S. EPA. 1995a. Integrated risk information system (IRIS) on-line computer database. Information system updated regularly by United States Environmental Protection Agency, Washington, D.C.
- U.S. EPA. 1995b. Final water quality guidance for the Great Lakes system; final rule. Federal Register, Vol. 60, No.56. March 23, 1995. pp. 15366-15425.
- U.S. EPA 1995c. Office of Pesticide Programs. Environmental Effects Database (EEDB).
- U.S. EPA. 1996. 1995 updates: Water quality criteria documents for the protection of aquatic life in ambient water. Office of Water, U.S. Environmental Protection Agency, Washington, D.C. EPA-820-B-96-001.
- U.S. EPA. 1999. National recommended water quality criteria – correction. Office of Water, U.S. EPA. EPA/822-Z-99-001.
- U.S. FWS. 1964. Pesticide-wildlife studies. 1963: A review of Fish and Wildlife Service investigations during the calendar year. FWS Circular 199.
- Walsh, G.M., and G.B. Fink. 1972. Comparative toxicity and distribution of Endrin and Dieldrin after intravenous administration in mice. Toxicol. Appl. Pharmacol. 23:408-416.
- White, D.H., and M.T. Finley. 1978. Uptake and retention of dietary cadmium in mallard ducks. Environ. Res. 17:53-59.

**APPENDIX C**

---

**Hazard Quotients**

**APPENDIX D**

---

**Sediment HQ Maps**