FINAL REPORT
LOWER LEY CREEK SITE
STREAMLINED BASELINE ECOLOGICAL RISK ASSESSMENT
SYRACUSE, NEW YORK

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LIST OF ACRONYMS AND ABBREVIATIONS

AE	Assessment Endpoint
AFDW	Ash-free dry weight
Al	Aluminum
As	Arsenic
ATSDR	Agency for Toxic Substances and Disease Registry
AUF	Area Use Factor
AWQC	Ambient Water Quality Criteria
BAF	Bioaccumulation factor
BERA	Baseline Ecological Risk Assessment
BHC	Benzene hexachloride
BW	Body Weight
Ca	Calcium
CCC	Criteria continuous concentration
Cd	Cadmium
C_F	Concentration in Food
cm	Centimeter
CMC	Criteria maximum concentration
COPC	Contaminant of Potential Concern
Cr	Chromium
C_{Sed}	Concentration in sediment
CTC	Critical Tissue Concentration
Cu	Copper
C_W	Concentration in water
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethylene
DDT	Dichlorodiphenyltrichlorethane
DL	Detection limit
EC	Exposure concentration
EcoSSLs	Ecological Soil Screening Levels
EPA	United States Environmental Protection Agency
EPC	Exposure point concentration
ERA	Ecological risk assessment
ERAGS	Ecological Risk Assessment Guidance for Superfund
Fe	Iron
FIR	Food ingestion rate
FS	Feasibility Study
Hg	Mercury
HHRA	Human health risk assessment
HMWPAH	High molecular weight polycyclic aromatic hydrocarbon
HQ	Hazard quotient
K	Potassium
Kg/kgBW/day	Kilograms per kilogram body weight per day
LD_{50}	Dose that is lethal to 50% of exposed organisms
L/kgBW/day	Liters per kilogram body weight per day
LMWPAH	Low molecular weight polycyclic aromatic hydrocarbon
LOAEL	Lowest observed adverse effect level
LOEC	Lowest observed effect concentration
LLC	Lower Ley Creek

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MeHg	Methylmercury
Mg	Magnesium
mg/kg	milligrams per kilogram
mg/kgBW/day	milligrams per kilogram body weight per day
µg/kg	micrograms per kilogram
µg/L	Micrograms per Liter
Mn	Manganese
Na	Sodium
ND	Not detected
ng/L	Nanograms per Liter
Ni	Nickel
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No observed adverse effect level
NOEC	No observed effect concentration
NYSDEC	New York State Department of Environmental Conservation
OME	Ontario Ministry of the Environment
ORNL	Oak Ridge National Laboratories
PAHs	Polycyclic aromatic hydrocarbons
Pb	Lead
PCBs	Polychlorinated biphenyls
PRG	Preliminary remediation goal
RI	Remedial Investigation
Sb	Antimony
SIR	Sediment ingestion rate
Se	Selenium
SERAS	Screening Ecological Risk Assessment System
SLERA	Screening Level Ecological Risk Assessment
SMDP	Scientific Management Decision Point
SVOC	Semi-volatile organic compound
TCDD	2,3,7,8-Tetrachlorodibenzodioxin
Tl	Thallium
TOC	total organic carbon
TRV	Toxicity reference value
U.S.	United States
V	Vanadium
VOC	Volatile organic compound
WIR	Water ingestion rate
w.w.	wet weight
Zn	zinc
%	percent
≥	Greater than or equal to
<	Less than

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EXECUTIVE SUMMARY

An ecological risk assessment (ERA) was conducted for the Lower Ley Creek (LLC) Superfund Site, in Syracuse NY. Lower Ley Creek is the downstream portion of Ley Creek that drains into Onondaga Lake, which is the subject of a separate environmental investigation. Lower Ley Creek is an impaired water body which has been impacted physically through re-routing of the streambed and other changes to the creek. There is concern that Lower Ley Creek has been impacted by industrial contaminants and has released and continues to release contaminants to Onondaga Lake. The present study presents a screening level ecological risk assessment (SLERA) and a streamlined baseline ecological risk assessment (BERA) developed to evaluate the potential impact of contaminants within the stream channel on ecological receptors in and adjacent to Lower Ley Creek.

For the purposes of this BERA, Lower Ley Creek is defined as approximately two miles of Ley Creek between the Brewerton Road (U.S. Route 1) bridge downstream to its confluence with Onondaga Lake. The site consists of channel sediments, surface water, and sediment/soil that may have migrated into the creek from adjacent lands. Due to concerns regarding the potential impacts to Lower Ley Creek from historic and ongoing releases of contamination, a field sampling program was conducted in November 2009 to more fully characterize the nature and extent of chemical contamination at the site.

Five assessment endpoints (AEs) were selected to evaluate risk to ecological receptors at the LLC site: survival, growth and reproduction of aquatic plants, benthic invertebrates, fish, piscivorous birds, and piscivorous mammals.

A SLERA was conducted in which measured concentrations in abiotic media were compared with conservative screening benchmarks. The measured (maximum detected) concentration of several inorganics in surface water, and numerous contaminants of potential concern (COPCs) measured in surface sediment samples, exceeded their screening benchmarks, indicating the potential for adverse effects to the aquatic community in Lower Ley Creek.

For the streamlined BERA, measured concentrations of selected COPCs in fish tissue were compared with concentrations reported in the literature that are associated with adverse effects in fish. Dietary exposure of piscivorous birds and mammals feeding on prey captured from Lower Ley Creek was also evaluated. Solid-phase toxicity tests were conducted using two invertebrate species. Risk to the aquatic plant community in Lower Ley Creek was assessed by comparing measured concentrations of COPCs in surface water with selected surface water quality benchmarks and by comparing measured concentrations of COPCs in sediment with soil benchmarks for plants.

Exceedances of surface water quality benchmarks and sediment benchmarks suggest potential risk to aquatic plants, benthic invertebrates, and fish. In sediment, inorganics (particulary cadmium [Cd], chromium [Cr] and nickel [Ni]), polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and some pesticides resulted in exceedances of screening values, indicating potential risk to aquatic plants and benthic invertebrates. The sediment sampling locations at which the most exceedances of sediment screening benchmarks were calculated include R3-3 (PAHs); R2-2 and R2-12 (inorganics); R2-6, R3-9, and R3-11 (pesticides); R2-12 (dioxins/furans); and R2-6 (volatile organic compounds; VOCs).

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Reduced growth was observed in invertebrates exposed to sediment samples collected from several locations in Lower Ley Creek; significant mortality was observed in one sample. No significant correlations with measured COPC concentrations in sediment samples was observed within the test results.

Total equivalent concentrations (TEC) of dioxin in fish tissue collected from Lower Ley Creek exceeded concentrations reported to be associated with adverse effects in fish.

Piscivorous mammals are at risk from dietary exposure to measured total PCB concentrations in fish from Lower Ley Creek. It may also be concluded that piscivorous birds are at risk from dietary exposure to PAHs and potentially Cr.

Because no dose-response relationship was observed in the toxicity tests, no-effect concentrations for direct toxicity of inorganics and PAHs to *Hyalella azteca* and *Chironomus dilutus* were identified and compared with maximum concentrations measured during the 2009 sampling event, screening benchmarks, and effect concentrations for growth and mortality in *H. azteca*.

The maximum no effect concentration for exposure of *H. azteca* to PAHs (45.19 mg/kg) was lower than the measured concentrations at which significant effects on growth and survival were observed. A LOAEL concentration for adverse effects on growth and survival of 54.78 mg PAHs/kg (growth) and 156.56 mg PAHs/kg (survival) were identified.

Because concentrations measured in samples collected in 2009 were higher than screening benchmarks and the observed no-effect concentrations in the toxicity tests, the following inorganics were retained as COPCs potentially resulting in direct toxicity to benthic invertebrates: arsenic (As), Cd, Cr, copper (Cu), lead (Pb), mercury (Hg), Ni, silver (Ag), and zinc (Zn). The maximum no-effect concentration observed in the toxicity tests was identified as the preliminary remediation goal (PRG) (As, 5.6 mg/kg; Cd, 6.4 mg/kg; Cr, 94.2 mg/kg; Cu, 102 mg/kg; Pb, 87.8 mg/kg; Hg, 0.29 mg/kg; Ni, 34.4 mg/kg; Ag, 2.1 mg/kg; and Zn, 342 mg/kg). A remedial action objective of 0.8 mg Hg/kg was identified for Onondaga Lake (U.S. EPA 2005). The concentration of 0.29 mg Hg/kg identified in this risk assessment would meet the objective of eliminating Ley Creek as a source of Hg to Onondaga Lake.

Site-specific bioaccumulation factors for PCBs were calculated for forage fish in the upper, middle and lower sections of Lower Ley Creek. Lowest observed adverse effect level-based and NOAEL-based sediment concentrations were calculated to identify a range of sediment PCB concentrations below which adverse effects on wildlife receptors would not be expected Sediment concentrations that would result in calculated HQs less than 1.0 for mink (the most sensitive receptor at this site based on the food chain models) were calculated. The LOAEL-based sediment PCB concentrations protective of ecological receptors ranged from 0.08 to 2.28 mg/kg. The NOAEL-based sediment PCB concentrations protective of ecological receptors ranged from 0.01 to 0.23 mg/kg.

Based upon the results, risk characterization, and interpretation, ecological risks exist at the Site from contaminants in sediments, specifically PAHs and several inorganics including Cd which may pose a risk via exposure to surface water in addition to exposure to sediment. Ecological risk exists from concentrations of dioxin-like COPCs in fish tissue, and PCB concentrations in sediment and forage fish pose a risk to piscivorous mammals.

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1.0 INTRODUCTION

This report presents a screening level ecological risk assessment (SLERA) and a streamlined baseline ecological risk assessment (BERA) for the Lower Ley Creek (LLC) Site in Syracuse, NY. The LLC Site is defined as approximately two miles of stream channel between the Route 11 /Brewerton Road bridge and the confluence of Onondaga Lake (Figure 1). The site consists of channel sediments and surface water, along with floodplain soil/sediment that may have migrated from adjacent and upgradient properties. The site is thought to have been impacted by a wide range of contaminants, particularly PCBs and heavy metals, discharged or released from industrial operations in the local vicinity. Ley Creek is considered to be a potential source of PCBs, heavy metals, and other contaminants to Onondaga Lake.

The SLERA and streamlined BERA follow the eight-step ERA process developed by the United States (U.S.) Environmental Protection Agency (EPA) (U.S. EPA 1997). The results of this BERA will be used to support risk management decision-making for the LLC Site.

In November 2009, surface sediment (0 to 6 inch sample depth), surface water, and whole body fish samples were collected from Lower Ley Creek (Figures 1 and 2). Sediment samples were analyzed for metals, volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), pesticides, polychlorinated biphenyls (PCBs), and a subset were submitted for analysis of dioxins/furans. Surface water samples were analyzed for VOCs, SVOCs, and metals. Fish tissue samples were analyzed for metals, pesticides/PCBs, and a subset were submitted for analysis of dioxins/furans.

Additional samples were collected in May of 2010 to address data gaps identified in the original SLERA. Ten of the original sediment sample locations were re-sampled (Figure 3). Sediment samples were submitted for analysis of metals, semivolatiles, TOC, grain site, pH, 10-day *Chironomus* toxicity tests, and 28-day *Hyalella* toxicity tests (five of the above locations). Sediment samples were also collected at seven locations within a swale area connected to the original creek channel. Swale samples were collected at two depth intervals: 0 to 6 inches and 6 to 12 inches, and were submitted for analysis of metals and PCBs.

The objective of this effort was to generate site-specific ecological and contaminant data, and to define ecological risks associated with surface water and surficial sediment contamination present in Lower Ley Creek. As a streamlined BERA, details of some of the risk assessment steps are referenced to the LLC Remedial Investigation (RI) and are presented in summary form in the BERA and its appendices.

2.0 SITE DESCRIPTION AND SITE CHARACTERIZATION ACTIVITIES

This section describes the general characteristics of the site and the sampling performed during the RI field investigation in 2009. The LLC Site is defined as approximately two miles of stream channel of Ley Creek between the Route 11 /Brewerton Road bridge and its confluence with Onondaga Lake (Figure 1). The site consists of surface water and sediment within the LLC channel. Lower Ley Creek is approximately 35 feet in width with an average depth of 2.5 feet, and under base flow conditions is a slow moving stream. As a result of stream modifications including the re-routing of Ley Creek by the Army Corps of Engineers (ACOE) in the 1970s, Ley Creek is generally straight, without any major bends or contours. In addition, the habitat diversity

and quality are reduced as a result of the stream modifications (e.g., the routing of the creek through a portion of the Town of Salina Landfill and the presence of waste materials (e.g., solvay waste). The site is accessible from the shoreline, and also by small boat.

New York State Department of Environmental Conservation (NYSDEC) has classified Ley Creek as a Class C water from its confluence with Onondaga Lake to the Ley Creek Sewage Treatment Plant outfall sewer, and as a Class B water from the outfall sewer to South Branch (6 NYCRR § 895) (NYSDEC 2010). The portion of Lower Ley Creek sampled for this investigation from the confluence with Onondaga Lake to above the 7th Street Bridge is a Class C water. The upper portion of the upper sampling reach is a Class B water (Figure 4). The class and standard designation assigned to state waters is based on the existing or expected best usage of each waterway segment. Class B waters are best used for swimming and other contact recreation, but not for drinking water. Class C waters support fisheries and are suitable for non-contact activities.

2.1 Site Investigation

Lower Ley Creek, a tributary of Onondaga Lake, is thought to have been impacted by environment contaminants released from a variety of upstream sources. Prior to the current RI study, a detailed assessment of contamination in Ley Creek has not been conducted. Collection of environmental chemical data in and around Ley Creek (e.g., from Onondaga Lake Lake) has indicated that Ley Creek may be contaminated and thus serve as an ongoing source of contamination to Onondaga Lake. These studies have detected cadmium (Cd), chromium (Cr), (Cu), lead (Pb), mercury (Hg), nickel (Ni), PCBs, and dioxins/furans at elevated concentrations in sediments at the confluence of Ley Creek and Onondaga Lake.

Potential sources of contamination for Lower Ley Creek include various current and historical industrial operations, as well as runoff and other urban sources of contamination. While groundwater may discharge contaminants to Lower Ley Creek, the sources of that contamination are not considered in this BERA. Identification and evaluation of the potential sources of contamination to Lower Ley Creek is outside the scope of this BERA and the RI projects.

In addition to toxic chemical contaminants, Lower Ley Creek may have been impacted by ionic wastes (e.g., Solvay waste) associated with industrial processes in the region. These ionic wastes may have been released directly into Ley Creek or may have migrated upstream from Onondaga Lake. These wastes may build up on the creek bottom and have indirect (i.e., not direct toxicity) effects on the ecosystem due to alterations in water and sediment quality. The potential impacts are being considered as part of the RI/Feasibility Study (FS) for Onondaga Lake and are not addressed in the LLC BERA.

Due to concerns regarding the potential impacts to Lower Ley Creek from historic and ongoing releases of contamination, a field investigation study was conducted in Lower Ley Creek in November 2009 to define the nature and extent of chemical contamination at the site. Fish samples were collected on November 9 and 10, 2009; sediment and surface water samples were collected during the week of November 16, 2009. The distribution of chemicals within each medium (surface water, surface sediment, and fish) to which ecological receptors may be exposed was examined and exposure point concentrations (EPCs) were calculated. The 0 to 6 inch zone of surficial sediment is considered to be the primary zone of exposure to aquatic receptors in Lower Ley Creek. Although deeper sediments may be exposed by bioturbation or mechanical disturbance, the potential exposure to and risk from the surface sediment was considered to be the of concern for this ERA.

The primary contaminants of interest were VOCs, SVOCs, pesticides/PCBs, polycyclic aromatic hydrocarbons (PAHs), metals, and dioxins/furans. The surface water and sediment and sediment sampling locations are shown in Figure 1. Maximum and mean sediment and surface water sampling COPC concentrations are presented in Tables 1 and 2, respectively. Sediment sample locations for the May 2010 sampling event are shown in Figure 3.

Fish samples were collected in three discrete reaches of Lower Ley Creek, defined as upstream (between the Brewerton Road bridge and 7th N. St.), midstream (directly below 7th N. St.) and downstream (from the midstream sampling location to near the confluence with Onondaga Lake). The locations of the three discrete fish sampling reaches are shown in Figure 2. Maximum and mean fish tissue COPC concentrations used in this ERA are presented in Table 3.

The complete analytical results for the samples collected are presented in the LLC RI report. A more comprehensive discussion of the physical and chemical properties of Lower Ley Creek is also presented in the LLC RI report.

3.0 SCREENING-LEVEL PROBLEM FORMULATION

The initial ecological screening assessment includes the development of the problem formulation for the site and an ecological effects evaluation (Step 1of the ERA process in the Ecological Risk Assessment Guidance for Superfund [ERAGS], U.S. EPA 1997). During this step, a preliminary conceptual site model is developed, COPCs and ecological effects of COPCs are identified, complete exposure pathways and potential ecological receptors are identified, and assessment endpoints (AEs) and measures of exposure and effects are selected.

3.1 Preliminary Conceptual Site Model

The preliminary conceptual site model for Lower Ley Creek is presented in Figure 5 and identifies the following:

- Potential contaminant sources
- Potential exposure routes
- Potential ecological receptors
- Potential ecological effects

The primary contaminant sources in Lower Ley Creek are considered to be the sediments and surface water within the stream channel. Potential contaminant transport pathways include surface water and sediment migration from upstream locations, groundwater transport and subsequent migration into Lower Ley Creek, and surface runoff from adjacent properties.

Plants and animals are directly exposed to chemicals primarily from contaminated sediments, and surface water of the LLC site, while animals may be exposed through ingestion of food (e.g., prey) or water containing contaminants.

3.2 Identification of Chemicals of Potential Concern

The preliminary selection of COPCs is based on the results of the field investigation conducted in November 2009. The analytes evaluated in this sampling event were based on known or

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suspected historical chemical releases and the results of environmental investigations conducted in the vicinity of Ley Creek (e.g., Onondaga Lake).

The following groups of chemicals were identified as COPCs for this SLERA:

- metals
- VOCs
- SVOCs
- PCBs
- Pesticides
- PAHs
- Dioxins/furans.

3.3 Preliminary Identification of Ecological Receptors and Exposure Pathways

Potential ecological receptors are those organisms that may be exposed to COPCs through direct contact with chemicals or through trophic (food chain) transfer via ingestion of contaminated prey. Based on field observations, review of the scientific literature, and a general understanding of the site ecological setting, the following groups of ecological receptors are found in and around Lower Ley Creek and are considered in this SLERA:

- Aquatic plants
- Benthic invertebrates
- Fish
- Piscivorous birds and mammals

Groups that are not included in this list, such as reptiles, amphibians, and herbivorous bird and mammals, are considered to be at lower risk than some of the receptors considered, based on their feeding habits and potential for exposure to site contaminants.

The aquatic plant community is exposed to contaminants through direct contact with the sediment and surface water. Vegetation may also be indirectly affected by a reduction in ecosystem functions (e.g., nutrient cycling or energy transfer) that are important for growth and reproduction.

Benthic organisms inhabit the sediment and directly absorb contaminants through dermal contact with sediment particles and interstitial water, as well as through ingestion of contaminated food items and incidental ingestion of sediment. For benthic organisms, the interstitial water phase may represent a substantial portion of the respired fluids.

The water column represents the primary source of exposure for fish. Fish absorb contaminants through dermal exposure and through respiration. Fish may also be exposed to contaminants through the ingestion of contaminated food items and incidental ingestion of sediment.

Birds and mammals that utilize Lower Ley Creek as a foraging area may be exposed to contaminants through dermal contact with soil and water, through ingestion of contaminated food items or incidental ingestion of soil or sediment, and through direct contact with surface water.

3.4 Identification of Assessment Endpoints and Measures of Exposures and Effects

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Assessment endpoints are explicit expressions of the ecological resources that are to be protected. Valuable ecological resources include those without which ecosystem function would be significantly impaired or those providing critical resources (e.g., habitat). Assessment endpoints help to focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants at the site. Because it is not practical or possible to directly evaluate risks to all of the individual components of the ecosystem on-site, appropriate selection and definition of AEs are critical to the identification of key sensitive components of the ecosystem that could be adversely affected by the contaminants associated with the site.

Measures of exposures and effects are defined as measureable ecological characteristics that are related to the valued characteristic selected as the assessment endpoint. A measure of effect is a change in an attribute of an AE or its surrogate in response to a stressor to which it is exposed. Measures of exposure and effects typically include measured or modeled concentrations of chemicals in various media, and may also consider the results of laboratory toxicity studies and field observations. For the LLC BERA, all measures of effects and exposure were based on measured concentrations of COPCs in sediment, surface water and fish tissue.

The following AEs and measures of exposure and effects were selected for this SLERA and are directed towards the survival, growth and reproduction of aquatic organisms (Table 4):

3.4.1 Assessment Endpoint #1: Survival, growth and reproduction of aquatic plants.

Aquatic plants play a key role in ecosystem functions such as carbon fixation and nutrient cycling. They are an important source of food for herbivorous and omnivorous species, provide shelter and nursery habitat for fish, and reduce erosion. Aquatic plants may be directly exposed to contaminants present in sediment, surface water, and discharging groundwater. The sedentary nature of plants suggests a high potential for exposure to COPCs in sediment and surface water.

Aquatic plants were determined to be of concern due to their role in energy low and materials cycling, their role as a food source for higher trophic level organisms, and their potential exposure to contaminants. The survival, growth and reproduction of aquatic plants were therefore selected as an AE for this risk assessment.

Overall Risk Question: Are concentrations of contaminants in sediment and surface water high enough to adversely affect the survival, growth, or reproduction of aquatic plants?

Measure of Effects: Determine whether maximum measured concentrations of COPCs in sediment and surface water exceed screening benchmark values.

Measure of Effects: Determine whether maximum concentrations of COPCs in sediment exceed screening benchmark values for plants.

3.4.2 Assessment Endpoint #2: Survival, growth and reproduction of benthic macroinvertebrates

The benthic macroinvertebrate community of small streams is typically diverse taxonomically, morphologically, and physiologically, and often consists of numerically abundant populations. The benthic macroinvertebrate community plays a key role in ecosystem functions such as nutrient cycling and organic matter processing, and is a food

resource for the pelagic community, including fish, as well as semiaquatic organisms such as birds and mammals.

Many benthic invertebrate species are detritus feeders. The role of invertebrates in recycling organic material contributes to the productivity of aquatic ecosystems by mineralizing nutrients, making them available for plant production. They also make sediment and detrital carbon available to higher trophic levels, as many invertebrates serve as an important food source for fish and other higher trophic level organisms.

Benthic macroinvertebrates were determined to be of concern due to their role in energy flow and materials cycling, their role as a food source for higher trophic level organisms, and their potential exposure to contaminants. The survival, growth, and reproduction of benthic macroinvertebrates was selected as an AE for this risk assessment.

Overall Risk Question: Are concentrations of contaminants in sediment and surface water high enough to adversely affect the survival, growth, or reproduction of benthic macroinvertebrates?

Measure of Effects: Determine whether maximum measured concentrations of COPCs in sediment and surface water exceed screening benchmark values.

3.4.3 Assessment Endpoint #3: Survival, growth and reproduction of fish

Fish serve a vital role in nutrient and energy transfer within an aquatic system. Specifically, fish act as a link between aquatic and terrestrial ecosystems and between the benthic and pelagic environments. Fish typically comprise a large proportion of the biomass in an aquatic ecosystem and fill a wide range of trophic positions (e.g., predatory, planktivores, bottom feeders). Fish serve as predators of zooplankton, periphyton, benthic invertebrates, and other fish. Some fish also serve as food items for avian and mammalian predators that forage in aquatic systems. These predator-prey interactions represent a transfer of energy from and within the aquatic ecosystem.

Since fish can be mid- to upper trophic level predators, they are susceptible to exposure to contaminants which bioaccumulate or biomagnify in the organisms upon which they feed. Aquatic plants, invertebrates and fish have been shown to accumulate certain contaminants that are present in aquatic ecosystems. In addition, fish can uptake dissolved contaminants directly from the water and bioconcentrate them in their tissues.

The fish community was determined to be of concern due to its role in energy flow, their role as a food source for higher trophic level organisms, and their potential for exposure to contaminants. Based on the above discussion, it was determined that viability and function of the fish community was a valid assessment endpoint for this risk assessment.

Overall Risk Question: Are concentrations of contaminants in surface water and sediment high enough to adversely affect the survival, growth, or reproduction of fish?

Measure of Effects: Determine whether maximum concentrations of COPCs in sediment and surface water exceed screening benchmark values.

4.0 ANALYSIS

4.1 Characterization of Exposure

Analytical results for surface water, sediment, and fish tissue collected from Lower Ley were summarized in the RI report. For this SLERA, the maximum concentration of each analyte measured in surface water or surface sediment samples collected on-site was identified.

In cases where COPCs in sediment or surface water were not detected, conservative assumptions were made to account for the "non-detect" (ND) results when evaluating risk. All data qualified as "U" or "UJ" were considered non-detect values. When an inorganic COPC was not detected in a particular sample, it was assumed that the actual concentration of that COPC in that sample was one-half the reported detection limit (DL). If an organic COPC was not detected in a sample, it was assumed that the actual concentration of that COPC in that sample was one-tenth the DL. The 10% DL for organics was chosen because values much lower than the DL can be detected. For this reason, maximum concentrations identified in this BERA may differ from maximum concentrations identified in the RI.

All data qualified as "U" or "UJ" were considered non-detect values. Some of the inorganic fish tissue data was qualified "UJ" due to contamination in the blank, therefore the data, the data validation reports, and guidance for utilizing qualified data were reviewed. For several inorganics, the substituted value in the laboratory data table was much higher than the method detection limit (MDL), and there was no consistency in how much higher than the MDL the substituted value was. When a particular COPC was detected, the measured concentration was orders of magnitude lower than substituted value. For example, antimony (Sb) was detected in one fish tissue sample at an estimated ("J" qualified) concentration of 0.004 mg/kg. All of the other antimony data was qualified "UJ", with a substituted result value ranging from 23 to 29 mg/kg. The "UJ" fish tissue data was utilized in this ERA in the following manner: All "UJ" qualified data were considered to be non-detect data. A value of five times the MDL was calculated to be the sample quantitation limit (SQL), which was the value that was substituted into the "Result" column (U.S. EPA 1989). To be consistent with how all non-detect data was handled for this risk assessment, it was assumed that the actual concentration of a non-detected inorganic in a fish tissue sample was one-half the reported DL.

4.2 Characterization of Effects

The screening-level exposure estimate and risk calculation is the second step of the ERAGS risk assessment process. Risk to aquatic receptors is estimated by comparing the maximum detected chemical concentration with ecotoxicity screening benchmark values. The screening-level assessment identifies the exposure pathways and chemicals that pose the greatest potential site risk to be further evaluated in the BERA. The results of the screening-level assessment can also be used to identify data gaps or areas of uncertainty in the ecological risk analysis.

4.2.1 Screening-Level Exposure Estimates

A screening evaluation was conducted for sediment and surface water by comparing the maximum detected concentration of each contaminant with conservative benchmark screening values. Conservative benchmark values derived from several sources were used to ensure that potential ecological threats were not overlooked. The benchmarks are

chronic No Adverse Effect Concentrations (NOECs), or the highest exposure concentration at which ecological effects are not expected.

Surface water screening benchmarks were obtained from EPA sources (US EPA 1996a, US EPA 2002), NYSDEC (NYSDEC 1998), and Oak Ridge National Laboratories (ORNL; Suter and Tsao 1996). The surface water screening benchmarks used in this SLERA are presented in Table 5. Sediment screening benchmarks were obtained from EPA (US EPA 1996a, US EPA 1996b), NYSDEC (NYSDEC 1999), ORNL (Jones *et al.* 1997), Ontario Ministry of the Environment (OME; Persaud *et al.* 1993), and the National Oceanic and Atmospheric Administration (NOAA; Buchman 2008). The sediment benchmarks used in this SLERA are presented in Tables 6 and 7. Screening benchmarks for plants were obtained from EPA (Ecological Soil Screening Levels; EcoSSLs) and ORNL (Efroymson *et al.* 1997).

The Screening Ecological Risk Assessment System (SERAS) program developed by Signal Corporation (Signal Corporation 2004) was used to calculate risk to aquatic organisms from exposure to chemical compounds in sediment and surface water. Ecological risk was estimated by comparing maximum and mean sediment and surface water exposure concentrations (ECs) with sediment and surface water screening benchmark values to calculate hazard quotients (HQs). This comparison is expressed as a ratio of exposure concentration to effect concentration, as follows:

$$\text{Hazard Quotient} = \frac{\text{Exposure Concentration (Maximum Detected)}}{\text{Screening Benchmark Value}}$$

5.0 RISK CHARACTERIZATION

The comparison of the maximum detected concentrations of COPCs in surface water and sediment and their associated HQs are presented in Tables 5-7 and the findings are summarized below.

5.1 Screening Assessment for Surface Water

A comparison of surface water COPC concentrations versus screening benchmarks is presented in Table 5. Several chemicals had detection limits that exceeded their respective screening benchmarks; these chemicals are identified in the list below as non-detect (ND).

The following chemicals were found to have an HQ $\geq$ 1.0 for at least one of its surface water screening benchmark concentrations:

- Barium
- Benzo(a)anthracene (ND)
- Benzo(a)pyrene (ND)
- Cadmium (ND)
- Dissolved copper
- Hexachloropentadiene (ND)
- Lead
- Manganese

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- Silver

The surface water sampling locations with exceedances of screening benchmarks include R1-4, R2-1, and R3-2, shown in Figure 1. It should be noted that the three organic compounds which this SLERA would retain based upon the detection limit being above the screening level are not retained as COPCs from the sediment SLERA (Section 5.2). It is concluded that these organic compounds should be eliminated from the list of COPCs.

5.2 Screening Assessment for Sediment

A comparison of the maximum detected sediment contaminant concentrations versus screening benchmarks is presented in Table 6 for organic COPCs and in Table 7 for inorganic COPCs. All sediment COPCs had detection limits lower than their respective screening benchmarks.

The following chemicals were found to have an HQ $\geq$ 1 for at least one of its sediment screening benchmark concentrations:

VOCs:

- 1,1,1-trichloroethane
- 1,1-dichloroethane
- 1,1-dichloroethylene
- 2-hexanone
- 4-methyl-2-pentanone
- acetone
- carbon disulfide
- chloroform
- xylene
- xylenes, total

SVOCs:

- 1,1'-biphenyl
- 2-methylnaphthalene
- acenaphthene
- anthracene
- benzo(a)anthracene
- benzo(a)pyrene
- benzo(g,h,i)perylene
- benzo(k)flouranthene
- biphenyl
- bis(2-ethylhexyl) phthalate
- dibenzo(a,h)anthracene
- dibenzofuran
- flouranthene
- flourene
- hexachlorobenzene
- hexachlorobutadiene
- hexachlorocyclopentadiene
- hexachloroethane
- high molecular weight PAHs (HMWPAH)

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- indeno(1,2,3-c,d)pyrene
- low molecular weight PAHs (LMWPAH)
- naphthalene
- PAH's, total
- pentachlorophenol
- phenanthrene
- phenol
- pyrene

Pesticides/PCBs

- aldrin
- Aroclor 1016
- Aroclor 1221
- Aroclor 1242
- Aroclor 1248
- Aroclor 1254
- Aroclor 1260
- Benzene hexachloride (BHC), alpha
- BHC, beta
- BHC, gamma (Lindane)
- Chlordane
- Dichlorodiphenyldichloroethane (DDD)
- Dichlorodiphenyldichloroethylene (DDE)
- Dichlorodiphenyltrichlorethane (DDT)
- DDT, total
- Dieldrin
- Endosulfan
- Endosulfan (alpha and beta)
- Endrin
- Heptachlor
- Heptachlor epoxide
- hexachlorocyclohexane
- Methoxychlor
- PCBs, total
- Toxaphene

Dioxins/Furans

- 2,3,7,8 –tetrachlorodibenzo-*p*-dioxin (TCDD)

Inorganics (metals)

- antimony
- arsenic
- arsenic III
- cadmium
- chromium
- chromium III
- copper
- iron

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- lead
- manganese
- mercury
- nickel
- silver
- zinc

The sediment sampling locations with the most exceedances of sediment screening benchmarks include R2-6 (VOCs), R3-3 (PAHs), R2-2 and R2-12 (inorganics), R2-12 (dioxins/furans). The sediment sampling locations are shown in Figure 1.

5.3 Screening Assessment for Plants

A comparison of the maximum detected sediment contaminant concentrations versus plant screening benchmarks is presented in Table 6 for organic COPCs and in Table 7 for inorganic COPCs. All sediment COPCs had detection limits lower than their respective screening benchmarks.

The following chemicals were found to have an HQ $\geq$ 1 for at least one of its plant screening benchmark concentrations:

- arsenic
- cadmium
- chromium
- copper
- lead
- manganese
- mercury
- nickel
- silver
- zinc
- pentachlorophenol

5.4 Freshwater Screening Benchmarks Not Available

There were a number of COPCs detected in surface water and sediment for which screening benchmarks are not available. Due to the lack of eco-toxicity information for these COPCs, it is not possible to estimate risk from them. The COPCs lacking screening benchmarks are included in Tables 8 (surface water) and 9 (sediment).

6.0 UNCERTAINTY AND ASSUMPTIONS

This SLERA evaluated exposure to COPCs in sediment and surface water. The SLERA process requires the use of conservative benchmarks and assumptions. This must be considered when interpreting results; for example:

- Conservative benchmark values were used in this screen to ensure that potential ecological risk was not overlooked. The benchmarks are chronic NOECs, or the highest

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exposure concentrations at which ecological effects are not observed. It is assumed that the most sensitive receptor organism and life stages are exposed.

- An HQ less than 1.0 does not indicate a lack of risk, but there is a high degree of confidence that minimal risk exists for that particular COPC, given the use of conservative screening benchmarks.
- Several COPCs were not detected in surface water samples, however calculated HQs were greater than 1.0 because a value one-tenth of the DL exceeded the conservative benchmark values. It is possible that these compounds may not actually be present, and that the risk is overestimated.
- Screening benchmarks are only available for exposure of plants to soil. It was assumed that soil concentrations toxic to plants are equivalent to sediment concentrations toxic to plants.
- All COPCs were assumed to be 100% bioavailable. No assumptions were made regarding the ionic species of metal present, or partitioning of the inorganic or organic COPCs.
- This SLERA was conducted using data from 32 sediment and ten surface water samples. It was assumed that the sampling effort was adequate to sufficiently characterize site conditions.

7.0 SCIENTIFIC MANAGMENT DECISION POINT (SMDP)

At the conclusion of a SLERA, there are only three possible decisions:

- There is adequate information to conclude that ecological risks are negligible and therefore there is no need for remediation on the basis of ecological risk. Ecological risks are negligible for the analytes on Tables 4 and 5 that do not result in HQs $\geq$ 1.0 when compared with any of the screening benchmarks.

- The information is not adequate to make a decision at this point, and the ERA process should continue to Step 3. Because reported DLs for benzo(a)anthracene, benzo(a)pyrene, and hexachloropentadiene exceed screening benchmarks for water, information regarding these analytes is not adequate to make a decision at this point based upon water data; however, these compounds were eliminated in the sediment screening. Based upon these compounds not being detected in water and being eliminated from sediment exposure risk, these compounds are not carried forward as COPCs. Due to the lack of screening benchmarks, no conclusion can be made regarding potential ecological risk from the analytes on Tables 8 and 9.

- The information indicates a potential for adverse ecological effects, and a more thorough assessment is warranted (continue to Step 3 of the ERA process). The results of this SLERA indicate that five inorganic chemicals exceed surface water screening benchmarks (Table 5), and numerous organic and inorganic chemicals exceed sediment benchmark values (HQ $\geq$ 1.0; Tables 6 and7) and may pose a potential risk to aquatic plants, benthic invertebrates, and fish.

8.0 BASELINE ECOLOGICAL RISK ASSESSMENT

This section (ERAGS Step 3) outlines the approaches needed, if any, to refine the problem formulation approach developed in ERAGS Step 1 (Section 3). The major components of the BERA problem formulation include:

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- Refinement of COPCs
- Further characterization of the ecological effects of the COPCs
- Review of information on COPC fate and transport, complete exposure pathways, and ecosystems potentially at risk
- Refinement of AEs and measures of exposure and effects, and
- Refinement of the conceptual site model

The above components are further discussed in this section.

8.1 Refinement of Contaminants of Potential Concern

The SLERA identified the Contaminants of Potential Concern (COPCs) for each environmental medium for which ecological risk exists based on those contaminants that exceeded benchmark values, if the screening benchmark was less than the DL, or for which a screening benchmark was not available. Within Step 3 of the ERAGS process, the COPCs retained from the SLERA are re-evaluated, to determine whether any of the COPCs may be considered as posing negligible risk when other pertinent information related to the site and the contaminants are examined.

Criteria that were used to further identify whether COPCs will or will not be retained for this BERA include:

- COPCs that were undetected at the reported DLs were eliminated from further evaluation in this BERA. The COPCs that were not analytically detected in surface water are shown on Table 5. The uncertainty of assuming that these COPCs at the not detected at the MDL pose negligible risk will be addressed in the uncertainty section (Section 12).

- Any COPC for which toxicity information is lacking was not eliminated from further consideration, but will be discussed as an uncertainty (Section 12). Tables 8 and 9 list COPCs that were analytically detected in surface water or sediment for which no screening benchmark is available.

- Essential nutrients

The screening-level assessment identified a number of chemicals that had an HQ $\geq$ 1.0 in both surface water and sediment. This list of chemicals included all the major classes of chemicals analyzed, including VOCs, SVOCs, PCBs, pesticides, metals, and dioxins/furans. Comparison with conservative screening benchmarks indicates potential ecological risk from the analytes listed in Sections 5.1 and 5.2. Based on the number and magnitude of the exceedances, metals and SVOCs will be evaluated further.

Based on their bioaccumulation potential and/or identification as COPCs retained for the Onondaga Lake BERA, a select list of analytes in addition to metals and SVOCs will be carried forward into this BERA for further evaluation based on measured tissue concentrations and estimated food chain exposure concentrations. These analytes include dioxins/furans, chlordane, total DDT, dieldrin, endrin, hexachlorocyclohexanes, dichlorobenzenes, trichlorobenzenes, and xylenes.

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8.1.1 Essential Elements

The following analytes will not be considered further quantitatively in this risk assessment: calcium (Ca), magnesium (Mg), potassium (K), sodium (Na), iron (Fe), manganese (Mn). These compounds are naturally abundant in many environments. They are all essential nutrients or essential trace elements, and as such their concentrations in tissues are regulated by living organisms; even at relatively high levels of exposure, internal concentrations of these chemicals generally do not become sufficiently high to cause toxic effects, and they are unlikely to be risk drivers. Therefore, a brief summary of the characteristics, fate, and transport of these analytes is provided below, but they will be assumed to be relatively unimportant sources of risk compared to other metals under consideration in this assessment, and they will not be evaluated further.

8.1.1.1 Calcium

Calcium is the fifth most abundant element in the earth's crust and is found in numerous mineral compounds. It occurs in the earth's crust at a concentration of 3.65 %, and in sea water at approximately 400 grams per ton. The average Ca concentration in soils is reported to be approximately 1.4%; this number varies due to differences in the parent materials and the level of rainfall (Bodek *et al.* 1988). No benchmark values are available for Ca. Most Ca compounds have high water solubility and are mobile in soils. Once in the soil solution, Ca will bind to cation exchange sites and movement through the soil profile is limited. In aquatic systems, Ca ions form weak soluble complexes with carbonates. Under acidic conditions, solubility and mobility is increased. Basic conditions decrease solubility and cause Ca to precipitate out of solution.

Calcium is an essential macronutrient that is internally regulated by most organisms. It is an important constituent of bones, teeth, and shells of living organisms, and is essential for muscular, nervous system, and renal function, as well as for blood coagulation and respiration. The maximum measured concentration of Ca in surface sediment in Lower Ley Creek is 30 percent (Table 9); the mean Ca concentration is 7.5%. Given that Ca is regulated within the body, risk to aquatic assessment endpoints from Ca will not be evaluated further in this ERA.

8.1.1.2 Magnesium

Magnesium is the eighth most abundant element in the earth's crust, and is widely distributed in a variety of rocks and minerals. Magnesium does not exist in a pure state in nature but is generally found in one of the following forms: dolomite, magnesite, brucite, periclase, carnallite, and kiersite. It is present in the earth's crust at about 2.1% by weight. Rocks and minerals contain a higher percentage of Mg than do soils; weathering results in leaching of Mg compounds to water. Once released to water, Mg compounds are precipitated to the sediment (Micromedex 1999). The maximum measured concentration of Mg in Lower Ley Creek sediment is 1.5% (Table 9).

Magnesium is an essential element for animals and plants and is internally regulated by most organisms. It is a required cofactor for more than 300 enzyme systems and is required for enzyme production in the body. Magnesium helps maintain appropriate intracellular concentrations of Ca, Na, and K. Given that concentrations of Mg are not elevated relative to naturally occurring concentrations, and that Mg is regulated within the body, the risk of Mg to aquatic assessment endpoints will not be evaluated further in this ERA.

8.1.1.3 Potassium

Potassium occurs in the earth's crust at a concentration of 2.59% by weight and in seawater at a concentration of 3.8×10^{-5} micrograms per liter (μg/L). It is never found free in nature (Micromedex 1999). The maximum measured K concentration in Lower Ley Creek sediment was 0.186% (Table 9).

Potassium is an essential macromineral and is internally regulated by most organisms. It is the primary regulator of fluids inside cells. It is critical to the transmission of nerve impulses and muscle contractions, helps regulate the acid-base balance, and participates in respiration via the chloride shift (Underwood and Suttle 1999). Information regarding toxic effects of high levels of K in aquatic organisms could not be found.

Given that K is not found free in nature, that concentrations of K are not elevated relative to naturally occurring concentrations, and that K is regulated within the body, the risk to aquatic assessment endpoints from K will not be evaluated further in this ERA.

8.1.1.4 Sodium

Sodium is found in the earth's crust at approximately 2.83% by weight and is the principal cation in the hydrosphere. It does not exist in its free form in nature, but rather in its halide, silicate, or carbonate forms (Micromedex 1999). The maximum Na concentration measured in Lower Ley Creek sediment was 0.629%.

Sodium is an essential macromineral and is internally regulated. Sodium helps maintain osmotic pressure, regulate the acid-base equilibrium, and control water metabolism in the body (Underwood and Suttle 1999).

Given that Na does not exist in its free form in nature, that measured concentrations at the Site are not elevated relative to naturally occurring concentrations, and that Na is an essential nutrient and is internally regulated, the risk of Na to aquatic assessment endpoints will not be evaluated further in this ERA.

8.1.1.5 Iron

Iron is the fourth most common element in the earth's crust. Iron concentrations in soil can range from 0.2% to 55% (Bodek *et al.* 1988), and concentrations in soil, surface water and sediment can vary significantly, even within localized

areas. The maximum Fe concentration measured in sediment at this site was 2.66% (Table 1).

Iron is an essential micro-nutrient to most forms of life from plants to man and is internally regulated by most organisms. Iron is a component of hemoglobin, which carries oxygen to the cells. It is also a component of other proteins and enzymes.

Environmental threats posed by the release of Fe generally relate to the precipitation of Fe oxides in aquatic systems, resulting in the smothering and embedding of the bottom substrate of the water body. This embedding of the aquatic bed material eliminates available habitat for benthic organisms and destroys spawning areas for fish. Essentially, the adverse effect of Fe oxides is the imposing of a physical impact to the aquatic system, which can usually be visibly observed. This precipitation has not been observed at this Site. Because Fe is regulated within the body, and because Fe oxide precipitation has not been observed within the streambed, Fe will not be evaluated further as a COPC in this assessment.

8.1.1.6 Manganese

Mangenese is the tenth most abundant metal in the earth's crust (Venugopal ad Luckey 1978). Background concentrations in soil range from 40 to 900 mg/kg (Barceloux 1999). The maximum Mn concentration measured at this site is 886 mg/kg.

Manganese is an essential trace element that plays a role in bone mineralization, protein and energy metabolism, metabolic regulation, and cellular protection from damaging free radical species. Manganese acts as both a constituent of metalloenzymes and an enzyme activator (ATSDR 2008). In addition to variable absorption of Mn from the gastrointestinal tract, Mn homeostasis is controlled by efficient excretion (Finley and Davis 1999). Manganese is the least toxic of the essential metals.

Because Mn is regulated within the body, and that measured concentrations at the Site are not elevated relative to naturally occurring concentrations, Mn will not be evaluated further in this risk assessment.

8.1.2 Aluminum

Aluminum (Al) is a major constituent of clays and other complex minerals. Al is ubiquitous and highly variable in the environment. It is a major component of most common inorganic soil particles, with concentrations varying widely and reaching to 30% (EPA 2003). Due to the ubiquitous nature of Al, the natural variability of Al in soil, and the use of conservative soil screening benchmarks, Al is often identified as a COPC for ERAs. The commonly used soil screening benchmark values are based on laboratory toxicity testing using an Al solution that is added to test soils, which the EPA has deemed inappropriate for assessing potential toxicity in soils. The following conclusions were derived from the

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available data on the environmental chemistry and toxicity of Al in soils to plants, soil invertebrates, mammals, and birds (EPA 2003):

- Total Al in soil is not correlated with toxicity to tested plants and soil invertebrates.
- Aluminum toxicity is associated with soluble Al.
- Soluble Al and not total Al is associated with the uptake and bioaccumulation of Al from soils to plants.
- The oral toxicity of Al compounds in soil is dependent upon the chemical form (Storer and Nelson 1968). Insoluble Al compounds such as the mineral forms associated with clays or soil particles are considerably less toxic than soluble forms.

Because total Al values are not reliable for predicting toxicity or bioaccumulation, EPA recommends Al be identified as a COPC only when soil pH is lower than 5.5. At higher pHs, Al is generally not bioavailable (EPA 2003). The minimum measured pH in Lower Ley Creek sediment was 7.4 (Table 15).

Ecological risk associated with Al is associated with low pH. Under low pH conditions, Al exerts toxicologic effects primarily by binding chemically with phosphorus, thereby interfering with phosphorous availability and/or absorption, resulting in a phosphorous deficiency in an organism. The digestive tract of many animals, particularly mammals and birds, have low pH conditions; toxicological evidence of adverse effects by Al appears to be limited to instances where Al is administered in a form that can immediately react with the phosphorous in an animal's system, unlike the insoluble mineral forms of Al typically associated with sediment or soil particles. Thus, while Al in soils can potentially be a source of soluble Al as a result of mobility from exposure to low pH water, under most conditions, Al ingested via soil would not be expected to impose adverse effects.

Given that Al is generally not bioavailable when pH is > 5.5, and the minimum pH concentration measured in sediment collected from Lower Ley Creek is 7.4, the risk of Al to aquatic assessment endpoints will not be evaluated further in this ERA.

8.2 Characterization of Ecological Effects

A literature search was conducted to obtain information on the ecological effects of the COPCs identified for this site. Toxicological profiles for the COPCs are presented in Appendix A.

8.3 Contaminant Transport and Fate and Ecosystems Potentially at Risk, and Complete Exposure Pathways

As previously discussed, the primary media of concern in Lower Ley Creek are sediment and surface water present within the Ley Creek channel. Contaminants from various offsite and/or upgradient industrial properties may have been discharged (directly or indirectly) into Lower Ley Creek. Evaluating the transport and fate of contaminants from specific source areas that have or

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may be contributing contamination to the ecosystem are not within the scope of this streamlined BERA.

8.4 Complete Exposure Pathways

The ecosystem potentially at risk at this site is the aquatic community in Lower Ley Creek. The aquatic plant community is exposed to contaminants through direct contact with the sediment and surface water, and may also be indirectly affected by a reduction in ecosystem functions (e.g., nutrient cycling or energy transfer) due to contamination. Benthic organisms inhabit the sediment and directly absorb contaminants through dermal contact with sediment particles and interstitial water, as well as through ingestion of contaminated food items and incidental ingestion of sediment. The water column represents the primary source of exposure for fish. Fish absorb contaminants through dermal exposure and through respiration. Fish may also be exposed to contaminants through the ingestion of contaminated food items and incidental ingestion of sediment.

Terrestrial receptors which obtain prey items from Lower Ley Creek are also of concern. Birds and mammals that utilize Lower Ley Creek as a foraging area may be exposed to contaminants through dermal contact with soil and water, through ingestion of contaminated food items or incidental ingestion of soil or sediment, and through direct contact with surface water.

8.5 Selection of Assessment Endpoints and Measures of Exposure and Effects

Three AEs evaluating the survival, growth, and reproduction of aquatic plants, benthic invertebrates, and fish, were identified in the SLERA. Additional risk questions for benthic macroinvertebrates and fish were identified for the BERA. Two additional AEs were identified related to food chain impacts and exposure to COPCs by higher trophic level organisms.

8.5.1 Assessment Endpoint #2: Survival, growth and reproduction of benthic macroinvertebrates

Survival, growth and reproduction of the benthic invertebrate community was identified as a valid assessment endpoint for the SLERA. Measured concentrations of numerous COPCs in surface water and sediment exceeded screening benchmarks for aquatic organisms. Risk to the benthic macroinvertebrate community in Lower Ley Creek will be further evaluated in this BERA.

<u>Overall Risk Question</u>: Does exposure to COPCs in sediment adversely affect survival, growth, or reproduction of benthic macroinvertebrates?

<u>Measure of Effects:</u> A 10-day solid phase bioassay will be conducted with the chironomid *Chironomus dilutus.* Survival and growth in site sediments and in a control sediment will be monitored.

<u>Measure of Effects</u>: A 28-day solid phase bioassay will be conducted with the amphipod *Hyalella azteca.* Survival and growth in site sediments and in a control sediment will be monitored.

8.5.2 Assessment Endpoint #3: Survival, growth and reproduction of fish.

Survival, growth and reproduction of the fish community was identified as a valid assessment endpoint for the SLERA. Measured concentrations of five metals in surface water exceeded screening benchmarks for aquatic organisms. Fish can bioaccumulate COPCs they are exposed to via direct contact with surface water or sediment, and can also be exposed to COPCs via ingestion of contaminated prey items. Risk to the fish community in Lower Ley Creek will be further evaluated in this BERA.

Overall Risk Question: Are concentrations of contaminants in water high enough to adversely affect survival, growth, or reproduction of fish?

Measure of Effects: Compare maximum detected concentrations of COPCs in water to Ambient Water Quality Criteria (AWQC) for protection of aquatic life.

Overall Risk Question: Are concentrations of contaminants in fish high enough to adversely affect their survival, growth, or reproduction?

Measure of Effects: Determine whether measured PCB and dioxin concentrations in fish tissue exceed concentrations reported in the literature as associated with adverse effects.

8.5.3 Assessment Endpoint #4: Survival, growth and reproduction of piscivorous birds

Foraging of piscivorous birds represents a pathway by which nutrients and energy are transferred from aquatic to terrestrial ecosystems. There is a close relationship between terrestrial and aquatic systems due to the nutrient and energy flow between them. Nutrients enter aquatic ecosystems via surface water runoff, stream input, and water infiltration through the soil. Energy enters aquatic ecosystems via sunlight and biological inputs such as detritus and leaves. Nutrients and energy are used to fix carbon in the production of plant and animal biomass, and are transferred from aquatic to terrestrial ecosystems through the food chain. Nutrient and energy cycles between aquatic and terrestrial systems are delicately balanced. Since nutrients and energy are limiting factors in the production of an ecosystem, the transfer of energy from an aquatic to a terrestrial system and back is essential.

Because piscivorous birds are upper trophic level predators, they are susceptible to exposure to contaminants which bioaccumulate or biomagnify in the organisms upon which they feed. Fish have been shown to accumulate certain contaminants that are present in aquatic ecosystems. Therefore, birds that consume fish have the potential to accumulate large concentrations of contaminants in their tissues. Survival, growth and reproduction of piscivorous birds was identified as an AE for this risk assessment.

Overall Risk Question: Are dietary exposure concentrations of bioaccumulative COPCs high enough to cause adverse effects to piscivorous birds?

Measure of Effects: The mean and maximum concentration of bioaccumulative COPCs measured in forage fish tissue collected from Lower Ley Creek will be used in food chain models to calculate dietary exposure concentrations for piscivorous birds. Receptor species selected as surrogates representative of piscivorous birds are the belted kingfisher (*Ceryle alcyon*) and great blue heron (*Ardea herodias*). The calculated dietary exposure concentrations will be compared with TRVs obtained from the literature for birds.

8.5.4 Assessment Endpoint #5: Survival, growth and reproduction of piscivorous mammals.

Foraging of piscivorous mammals also represents a pathway by which nutrients and energy are transferred from aquatic to terrestrial ecosystems. Piscivorous mammals are upper trophic level predators and are susceptible to exposure to contaminants which bioaccumulate or biomagnify in the organisms upon which they feed. Fish have been shown to accumulate certain contaminants that are present in aquatic ecosystems. Therefore, mammals that consume fish have the potential to accumulate large concentrations of contaminants in their tissues. Survival, growth and reproduction of piscivorous mammals was identified as an AE for this risk assessment.

Overall Risk Question: Are dietary exposure concentrations of bioaccumulative COPCs high enough to cause adverse effects to piscivorous mammals?

Measure of Effects: The mean and maximum concentration of bioaccumulative COPCs measured in fish tissue collected from Lower Ley Creek will be used in food chain models to calculate dietary exposure concentrations for piscivorous mammals. Receptor species selected as surrogates representative of piscivorous mammals are the mink (*Mustela vison*) and river otter (*Lutra canadensis*). The calculated dietary exposure concentrations will be compared with TRVs obtained from the literature for mammals.

9.0 STUDY DESIGN

Prior to the November 2009 field sampling and data collection study, an evaluation of the presence and distribution of contaminants in Lower Ley Creek had not been conducted. The November 2009 field sampling study, which collected sediment, surface water, and fish tissue samples, served as the basis for this risk assessment. The LLC RI describes in detail the study objectives, design, and findings.

The November 2009 field investigation study and results are considered to be valid and appropriate for the BERA and the objectives of ERAGS Steps 4 (study design and data quality objectives) and 5 (field verification of sampling design) have been met.

9.1 Additional Sampling, May 2010

Based on initial BERA results, additional sediment samples were collected in May 2010 to fill data gaps identified in the initial BERA. Ten of the original sediment sample locations were re-sampled (Figure 3). Sediment samples were submitted for analysis of metals, semivolatiles, total organic carbon (TOC), grain site, pH, 10-day *Chironomus* toxicity tests, and 28-day *Hyalella* toxicity tests (five of the above locations).

Sediment samples were also collected at seven locations within a swale area connected to the original creek channel, between Sample Locations R3-10 and R3-11 (Figure 3). Swale samples were collected at two depth intervals: 0 to 6 inches and 6 to 12 inches, and were submitted for analysis of metals and PCBs.

10.0 ANALYSIS PHASE

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The analysis phase is Step 6 of the eight-step Superfund process (EPA 1997), and is the technical evaluation of existing and potential exposure and ecological effects at the site. The analysis phase examines the two primary components of risk, exposure and effects. The objective is to determine or predict ecological responses under site-specific exposure conditions.

10.1 Characterization of Exposure

The maximum measured concentration of a COPC in surface water or sediment was the exposure concentration for plants and benthic invertebrates. Individual PAHs were screened against their screening benchmarks. Concentrations of LWMPAHs, HMWPAHs, and total PAHs were calculated for each sample location, and screened against the appropriate screening benchmark.

For fish and terrestrial receptors (i.e., piscivorous birds and mammals), two separate exposure point calculations were performed. For these receptors, Lower Ley Creek was divided into three distinct reaches. These reaches were defined as upstream (between the Brewerton Road bridge and 7th N. St.), midstream (directly below 7th N. St.) and downstream (from the midstream location to near the confluence with Onondaga Lake). Mean and maximum COPC concentrations measured in surface water and surface sediment samples collected within each reach were calculated (Tables 1 and 2).

Several species of fish were collected for whole body and fillet tissue analysis from three reaches (upstream, midstream, and downstream reach) of Lower Ley Creek (Figure 2) as part of the November 2009 field investigation. Fish species used for whole-body analysis include creek chub (*Semolitus atromaculatus)*, white sucker (*Catastomus commersoni)*, sunfish (*Lepomis* sp.), and minnows (*Cyprinidae sp*). Fish species used for fillet analysis include carp (*Cyprinus carpio)*, white sucker and northern pike (*Esox lucius)*. Details of fish weight, length, and capture location are presented in the LLC RI. The fish tissue COPC concentrations used for exposure calculations were the maximum measured concentration and the mean measured concentration (wet weight; w.w.) of contaminants in fish from each of the three reaches of Lower Ley Creek (Table 3).

Maximum and mean PCB and dioxin concentrations measured in forage fish collected from the upstream, midstream, and downstream reaches of Lower Ley Creek were compared with tissue concentrations of PCBs and dioxin reported in the literature that were associated with an adverse biological response. Fillets from larger fish captured from the upper and lower reach were collected primarily for use in the human health risk assessment (HHRA); fillets were not collected from the middle reach. Although the measured concentration in the fillet is an underestimate of the total body CTC, these data were also compared to CTCs derived from the literature, as higher tissue COPC concentrations would be expected in larger fish.

Whole-body tissue samples of forage fish were used for the food chain models in this BERA. Because there is little data available to derive dietary TRVs for individual PAH compounds, toxicity from dietary exposure to total PAHs was evaluated.

Because it was assumed that the actual concentration of a non-detected inorganic in a sample was one-half the reported DL, and the actual concentrations of a non-detected organic COPC was one-tenth the DL, maximum and mean COPC concentrations used for this BERA may differ from maximum and mean fish tissue concentrations reported in the RI or used in the HHRA.

10.2 Characterization of Effects

The HQ method (Barnthouse *et al.* 1986; U.S. EPA 1997) was employed in this BERA. Briefly, the HQ method compares exposure concentrations with screening benchmarks or toxicity reference values (TRVs) based on ecologically relevant endpoints such as mortality, reproductive failure, or reduced growth. The comparison is expressed as a ratio of exposure concentration to effect concentration. Hazard quotients less than 1.0 are assumed to indicate that the COPC is unlikely to cause adverse ecological effects; HQs of 1.0 or greater are assumed to indicate that the COPC may present ecological risk.

The following analyses were conducted to characterize potential effects from exposure to sediment, surface water, or fish within Lower Ley Creek to ecological receptors.

10.2.1 Solid-Phase Toxicity Tests

Survival, growth and reproduction of two invertebrate species (*H. azteca* and *C. dilutus*) exposed to site sediment was compared with survival, growth and reproduction of *H. azteca* and *C. dilutus* exposed to a control sediment. This information contributed to the risk characterization for the following AE:

- Survival, Growth and Reproduction of the Benthic Invertebrate Community

10.2.2 Comparison of Measured COPC Concentrations in Surface Water With Ambient Water Quality Criteria

Mean and maximum concentrations of COPCs in surface water were compared with acute and chronic AWQC. This information contributed to the risk characterization for the following AE:

- Survival, Growth and Reproduction of the Fish Community

10.2.3 Comparison of Measured Fish Tissue COPC Concentrations with Critical Tissue Concentrations Reported in the Literature

Mean and maximum PCB and dioxin concentrations measured in fish collected from Lower Ley Creek were compared with tissue concentrations of PCBs and dioxin reported in the literature that were associated with an adverse biological response (Table 10).

This information contributed to the risk characterization for the following assessment endpoint:

- Survival, Growth and Reproduction of the Fish Community

10.2.4 Food Chain Models

Site-specific COPC concentrations in sediment, surface water, and forage fish were entered into food chain models for selected receptor species to estimate the daily exposure dose for each COPC for species representative of each assessment endpoint. The estimated daily dose was compared to adverse effect concentrations derived from the literature. If the resulting calculated HQ is greater than 1.0, it will be concluded that there is risk to the assessment endpoint. This information will be used to characterize risk for the following assessment endpoints:

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- Survival, Growth and Reproduction of Piscivorous Birds
- Survival, Growth and Reproduction of Piscivorous Mammals

The selected receptor species were assumed to be exposed to COPCs via ingestion of food, ingestion of surface water, and incidental ingestion of sediment. The sediment, surface water, and tissue residue concentrations measured in this investigation were used to model the total dose to which the receptor species were exposed, per the following formula:

$$\text{Total dose} = [(FIR * C_F) + (WIR * C_w) + (SIR * C_S)]$$

where:

FIR = food ingestion rate (kilograms per kilogram body weight per day; kg/kgBW/day)
C_F = contaminant concentration in the food item (mg/kg)
WIR = water ingestion rate (liters per kilogram body weight per day; L/kgBW/day)
C_w = contaminant concentration in water (mg/L)
SIR = sediment ingestion rate (kg/kgBW/day)
C_S = contaminant concentration in the sediment (mg/kg)

Organisms likely to be exposed to contaminants because of specific behaviors, patterns of habitat use, or feeding habits were selected as receptor species representative of an assessment endpoint. The availability of appropriate toxicity information on which risk calculations was based was also an important consideration. The surrogate receptor species selected for this ERA included the belted kingfisher, great blue heron, mink, and river otter.

Toxicity reference values for inorganics, selected organic COPCS, PCBs, and dioxin were obtained from studies published in the literature (Table 11). Life history parameters from published literature sources were used in the exposure models (Table 12). A summary of the studies used to derive the TRVs used in this BERA is presented in Appendix A. Life history information and exposure profiles for the selected receptor species can be found in Appendix B. Two exposure scenarios were evaluated for each receptor species: one scenario using the maximum measured concentration of COPCs in fish; and one scenario, using mean measured COPC concentrations.

This BERA utilized simplifying assumptions in the food chain models, since it is difficult to mimic a complete diet. Site-specific data were only available for one prey species for each of the selected receptor species (fish). Measured COPC concentrations in forage fish were considered representative of concentrations that would be present in all prey species found on-site.

Fish tissue contaminant concentrations were entered into the models as wet weights to be compared to the TRVs derived from the literature, since most TRVs are reported in the literature as wet weight concentrations. In addition, the water contaminant concentrations entered into the models were for unfiltered water samples because this represented a more realistic exposure via ingestion of water than the use of data from filtered water samples.

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The results of these models were used to determine the contamination values that bound the threshold for adverse effects to each assessment endpoint (EPA 1997) and can be used as one line of evidence for evaluating site management alternatives.

Based on the results of the food chain models, the following conclusions were drawn:

- If the no observed adverse effect level (NOAEL)-based HQ was greater than or equal to 1.0 but the lowest observed adverse effect level (LOAEL)-based HQ was not, it was concluded that model-calculated risk could not be determined.

- A contaminant concentration was considered to demonstrate model- calculated risk to the given receptor if both the NOAEL-based HQ and LOAEL-based HQ were greater than or equal to 1.0.

- If neither the NOAEL- or LOAEL-based HQs were greater than or equal to 1.0, it was concluded that there is no model-calculated risk to the given receptor.

11.0 RISK CHARACTERIZATION

This section presents the risk characterization and analysis of ecological effects for the receptors selected for the LLC BERA (ERAGS Step 7). The risk questions developed for the assessment endpoints are also addressed.

11.1 Solid-Phase Toxicity Tests

Toxicity tests (chronic exposure) were conducted with two invertebrate species, *Hyalella azteca* and *Chironomus dilutus* using EPA test methods 100.4 and 100.5 (USEPA 2000). Endpoints measured were survival, growth, and reproduction. Results for organisms exposed to site sediment were compared to those for organisms exposed to a control sediment to determine whether any significant toxic effects are attributable to the site. Five sediment samples were submitted for the 28-day *H. azteca* test, and ten sediment samples were submitted for the ten-day *C. dilutus* bioassay.

Tables 13 and 14 present the toxicity test results. The survival, growth and reproductive effects were reported by the testing laboratory in accordance with standard test protocols protocols and are summarized in the tables.

Survival of *H. azteca* in the sample collected at location R3-11 was significantly lower than survival of controls (Table 13). Growth of amphipods in sample R3-11 was not evaluated due to the observed mortality. Growth of *H. azteca* in the sample collected at location R3-5 was significantly lower than growth of controls.

Surivival of *C. dilutus* in all site samples was similar to control survival. Growth of *C. dilutus* in samples collected at locations R1-1, R2-13, R2-2, R2-12, R2-8, and R3-11 was significantly lower than growth of controls (Table 14). Due to a laboratory error, dry weight was reported for chironomids instead of ash-free dry weight (AFDW).

Table 15 presents the analytical results from the sediment samples used for the bioassays. Correlation analyses were run on log-transformed data to determine whether there was any relationship between the measured COPC concentrations and growth or survival.

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None of the measured analyte concentrations were significantly correlated with the observed effects on growth or survival at a significance level of 0.05.

The locations to be re-sampled during the May 2010 sampling event were selected to represent a range of inorganic and PAH concentrations, and included locations where maximum concentrations were measured in November of 2009. The objective of the toxicity testing effort was to test a range of concentrations present on-site, and to identify a NOEC and LOEC concentration to support risk-management decisions. Although locations with maximum measured concentrations of inorganics and PAHs in November 2009 were re-sampled in May of 2010, the range of concentrations tested in the bioassays was much lower than concentrations of COPCs measured on-site in November 2009.

11.2 Comparison of Measured COPC Concentrations in Surface Water with AWQC

The maximum measured cadmium concentration exceeded the AWQC freshwater criteria maximum concentration (CMC; Table 5). Maximum measured concentrations of cadmium and lead exceeded the AWQC freshwater criteria continuous concentration (CCC).

11.3 Comparison of Measured Fish Tissue COPC Concentrations with Critical Tissue Concentrations Reported in the Literature

Maximum and mean PCB and dioxin concentrations measured in forage fish collected from the upstream, midstream, and downstream reaches of Lower Ley Creek were compared with tissue concentrations of PCBs and dioxin reported in the literature that were associated with an adverse biological response (Table 16). Measured PCBs in forage fish did not exceed CTCs in any stream reach. In forage fish tissue, NOAEL-based HQs exceeded 1.0 for dioxins/furans in all three stream reaches.

Fillets from larger fish captured within each reach were collected primarily for use in the human health risk assessment (HHRA). Pike, white sucker and carp were collected in the upper reach, and carp and white suckers were collected in the lower reach. The highest COPC concentrations were measured in carp in both reaches. Although the measured concentration in the fillet is an underestimate of the total body CTC, these data were also compared to CTCs derived from the literature, as higher tissue COPC concentrations would be expected in larger fish (Table 16). In fillet fish tissue, LOAEL-based HQs exceeded 1.0 for dioxins/furans in the upstream and downstream reaches. Fillets were not collected in the middle reach.

11.4 Food Chain Models, Piscivorous Birds

Food chain models for piscivorous receptors were run using COPC concentrations measured in sediment, surface water, and fish collected from the upstream, midstream, and downstream reaches of Lower Ley Creek, assuming the predators obtained all their prey from that particular reach of stream. The complete food chain modeling and risk calculations for all piscivorous receptors, exposure scenarios and COPCs are presented in Tables 17-40. A summary of these findings is presented below.

11.4.1 Belted Kingfisher

Using maximum measured COPC concentrations in sediment, surface water and fish, LOAEL-based HQs exceeded 1.0 for Cr and PAHs in the upstream reach of Lower Ley

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Creek, and for PAHs in the middle reach. Calculated NOAEL-based HQs exceeded 1.0 for dioxins/furans, methylmercury (MeHg) and zinc (Zn) in all three reaches; Pb in the upstream and middle reach; total PCBs and Aroclor 1254 in the upper reach; and Cr and in the middle reach (Tables 17-19).

Using mean measured COPC concentrations, NOAEL-based HQs exceeded 1.0 for dioxins/furans and MeHg in all three reaches; PAHs in the upper and middle reach; and Zn in the lower reach (Tables 20-22).

11.4.2 Great Blue Heron

Using maximum measured COPC concentrations in sediment, surface water and fish, NOAEL-based HQs exceed 1.0 for MeHg in all three reaches; Cr in the upstream reach, and total PAHs in the upstream and middle reach (Tables 23-25).

Using mean measured COPC concentrations, the calculated NOAEL-based HQ exceeded 1.0 for MeHg in the upstream and downstream reach; and PAHs in the upper reach (Tables 26-28).

11.5 Food Chain Models, Piscivorous Mammals

11.5.1 River Otter

Using maximum measured COPC concentrations in sediment, surface water and fish, LOAEL-based HQs exceeded 1.0 for total PCBs in the upstream and middle reaches of Lower Ley Creek. Calculated NOAEL-based based exceeded 1.0 for MeHg, dioxins/furans, and Aroclors 1254 and 1260 in all three stream reaches; and for total PCBs in the downstream reach (Tables 29-31).

The LOAEL-based HQ exceeded 1.0 for mean concentrations of total PCBs in the upstream reach. Using mean measured COPC concentrations, the calculated NOAEL-based HQ exceeded 1.0 for MeHg, dioxins/furans, Aroclor 1254, and Aroclor 1260 in all three stream reaches, and for total PCBs in the middle and downstream reach (Tables 32-34).

11.5.2 Mink

Using maximum measured COPC concentrations in sediment, surface water and fish, LOAEL-based HQs exceeded 1.0 for total PCBs in all three stream reaches; and for dioxins/furans and Aroclor 1260 in the middle reach. Calculated NOAEL-based HQs exceeded 1.0 for MeHg and Aroclor 1254 in all three reaches; and for dioxins/furans and Aroclor 1260 in the upstream and downstream reach (Tables 35-37).

The LOAEL-based HQ exceeded 1.0 for mean concentrations of total PCBs in the upstream reach of Lower Ley Creek. Using mean measured COPC concentrations, the calculated NOAEL-based HQ exceeded 1.0 for MeHg, dioxins/furans, and Arcolors 1254 and 1260 in all three stream reaches; and for total PCBs in the middle and downstream reaches (Tables 38-40).

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11.6 Food Chain Models, Swale Samples

Aroclor 1248 was the only Aroclor detected in surface sediment samples collected from the swale area (Table 41). Maximum and mean total PCB concentrations in swale sediment samples and forage fish from the upper reach (Table 3) were used in food chain models for piscivorous receptors to evaluate potential exposure to this area in wet seasons/years.

Using maximum or mean measured PCB concentrations in the swale area, NOAEL-based HQs exceeded 1.0 for piscivorous birds (Table 42). Using maximum or mean measured PCB concentrations in the swale area, LOAEL-based HQs exceeded 1.0 for piscivorous mammals.

11.7 Summary of Risk Conclusions

Based upon the results, risk characterization, and interpretation, ecological risks exist at the Site from exposure to contaminants in sediment, specifically PAHs and several metals including Cd which may pose a risk through water exposure in addition to sediment exposures. Ecological risk exists from dioxin-like risk in fish, and PCB concentrations in sediment and fish pose a risk to piscivorous mammals.

12.0. UNCERTAINTY ANALYSIS/ASSUMPTIONS

Uncertainties and limitations are inherent in all risk assessments and need to be considered when interpreting results. Knowledge of the sources of uncertainty, how the ERA dealt with the sources of uncertainty, and an understanding of the magnitude of the effects resulting from the sources of uncertainty allows for informed management decisions. The nature and magnitude of uncertainties depend on the amount and quality of data available, the degree of knowledge concerning the site conditions, and the assumptions made to perform the risk assessment. Within this BERA, decisions regarding the direction of uncertainty were made to err towards the conservative side; however, there are some instances for which insufficient information was available to estimate the direction of the uncertainty. The uncertainties related to problem formulation, exposure characterization, effects characterization, and risk characterization are provided in the following sections.

12.1 Problem Formulation

There are several sources of uncertainty within the problem formulation phase of the ERA. These include: the selection of assessment endpoints; and assumptions within the site conceptual model.

The selection of appropriate assessment endpoints and the receptors that will serve to characterize risk of the assessment endpoints is a critical step within the problem formulation of an ERA. If a particular assessment endpoint or receptor that may potentially be exposed to site contaminants is overlooked or not identified, there will be an underestimation of risk.

The site conceptual model presents the pathways by which contaminants are released from source areas and receptors are exposed. However, some exposure pathways are

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difficult to evaluate or information does not exist to allow for a quantitative evaluation of exposure from particular exposure pathways. Within this ERA, dermal and inhalation exposure pathways are not addressed quantitatively. It is believed that these exposure pathways are not substantive relative to other exposure pathways (e.g., ingestion). In addition, exposure to COPCs potentially present in groundwater that may discharge to Ley Creek was not addressed in this risk assessment.

In May 2010, soil samples were collected from a swale area adjacent to the original creek channel, between Sample Locations R3-10 and R3-11 (Figure 3). Although standing water was not present within the swale at the time the samples were collected, water and fish may be present at this location during years or time periods with heavy precipitation. Exposure of receptors to COPCs present within the swale area was not incorporated into the original site conceptual model. However, during wet periods, aquatic-foraging birds and mammals may be exposed prey or sediment within this area.

12.2 Characterization of Exposure

The uncertainties associated with exposure characterization include: quality of the data available; the exposure parameters used in the food chain models; exposure pathways not retained for quantitative evaluation; identification of ecological receptors; selection of representative species; exposure route assumptions; speciation of inorganic COPCs, and partitioning of organic COPCs.

Within this BERA, receptor model species were selected with the intent of targeting highly exposed, sensitive species. The uncertainty associated with the selection of species models is often in the direction of over-estimation of risk for most species included within the assessment endpoint; however, it is possible that a species not selected as a receptor or individual organisms could have higher exposures that those calculated within this ERA.

Certain assumptions were made relating to several exposure parameters that deviated from the information developed within the life history profiles. For the area use factor (AUF), which is the foraging area utilized by the receptor for the exposure model, a factor of 1.0 was applied. The AUF of 1.0 being applied to each reach of Lower Ley Creek may overestimate the actual risk to the receptors.

Another assumption is that the contaminants in food items were assumed to exhibit 100 percent (%) absorption efficiency and were assumed not to be excreted during the life of the receptor. That is, the risk estimated from dietary exposure is based on administered dose, not the absorbed dose. This conservative assumption should overestimate actual risk to the receptors.

All water and food ingestion rates used in this risk assessment were calculated using an allometric equation. It was assumed that these estimated ingestion rates were representative of the true ingestion rates for the receptor species in question.

Modeling the dietary component of the avian or mammalian receptors utilized maximum and mean contaminant levels in fish. For this BERA, sampling of forage fish and fish fillets was performed to estimate the bioaccumulation of contaminants. It was assumed that this data is representative of the bioaccumulation within each reach and therefore appropriate for use in the exposure assessment. Samples sizes were small for forage fish

samples within each reach. Data limitations related to variations in tissue concentrations due to seasonal changes, bioaccumulation in species not captured in this sampling effort, and specific exposure/bioaccumulation relationships could not be evaluated. The direction of the uncertainty due to these data limitations cannot be conclusively stated.

There is very little information available in the literature regarding the rates of incidental sediment ingestion by wildlife species. A sediment ingestion rate of 1% of food intake was estimated for all of the receptor species evaluated in this risk assessment. The uncertainty related to incidental sediment ingestion could either underestimate or overestimate the exposure depending upon the species.

The food chain models used simplified diets of one item with a static ingestion rate; in reality, each receptor organism's diet is varied, and the ingestion rate varies with food availability and metabolic needs (such as during growth of young and periods of metabolic stress). While reliance on a single forage item is not realistic over long time periods or even a growing season, it may not be implausible within the time frame relevant to the toxic mechanism of the contaminants. Organisms do not use the environment uniformly, but rather forage where food is most readily available to them. For this reason, the use of a single food item may not dramatically over-estimate exposure but also should not under-estimate actual exposures within relevant time frames.

Information concerning speciation of inorganic COPCs (metals) and organic COPCs was lacking. It is widely recognized that bioavailability and toxicity can vary dramatically as a function of the speciation and/or partitioning of COPCs. As a consequence, exposure and risks may be either underestimated or overestimated. However, given that the toxicological studies used to generate the TRVs for this BERA generally used bioavailable/toxic forms of the contaminants, it is unlikely that risk is substantively underestimated.

12.3 Characterization of Effects

Numerous COPCs that were analytically detected but for which toxicity benchmarks were not available were not evaluated any further in this ERA (Tables 7 and 8 list the COPCs with no benchmarks). Because ecotoxicity information for these COPCs is not available, risk to ecological receptors from exposure to these COPCs cannot be evaluated quantitatively.

Not all TRVs for birds and mammals represent the same degree of certainty. Toxicity reference values were mostly derived from laboratory animal studies. Toxicity reference values were selected through a systematic process to minimize the potential for under-estimating the toxicity of contaminants to the assessment endpoints. A literature search was conducted to determine the chronic toxicity of the contaminants of concern when ingested by the indicator species. If no toxicity values could be located for the receptor species, values reported for a closely related species were used. All studies were critically reviewed to determine whether study design and methods were appropriate. For the purposes of this ERA, a factor of 10 was used to convert the reported LOAEL to a NOAEL if adverse effects were observed at the lowest tested dose. When several toxicity values were reported for a receptor species, the most conservative value that resulted in an ecologically significant adverse effect was used in the risk calculations, regardless of toxic mechanism. Toxicity values obtained from long-term feeding studies were used in

preference to those obtained from single dose oral studies. No other safety factors were incorporated into this BERA.

Uncertainty related to the toxicity estimates of effects including NOAELs, LOAELs, LD_{50}s, and other mathematical calculations derived from the literature have inherent variability. These values are statistically determined and are reflective of the experimental design. For example, within a particular toxicity study the reported LOAEL and/or NOAEL is dependent upon the exposure levels selected within the study design. It is not known within these studies how much lower the LOAEL may be or how much higher the NOAEL may be. However, within the risk calculations this error is believed to be relatively minor as compared to other sources of error within the risk calculations of the ERA.

Error can be introduced by use of invalid assumptions in the conceptual model. Conservative assumptions were made in light of the uncertainty associated with the risk assessment process. This was done to minimize the possibility of concluding that no risk is present when a threat actually does exist (i.e., to eliminate false negatives). Whenever possible, risk calculations were based on conservative values. For example, LOAELs used to calculate HQs were the lowest values found in the literature, regardless of toxic mechanism.

12.4 Risk Characterization

This ERA evaluates exposure to contaminants via direct contact of ingestion of food, water, or sediment. Major sources of uncertainty include natural variability, error, and insufficient knowledge. Natural variability is an inherent characteristic of ecological receptors, their stressors, and their combined behavior in the environment. Biotic and abiotic parameters in these systems may vary to such a degree that the exposure of similar ecological receptors in the same system may differ temporally and spatially. Factors that contribute to temporal and spatial variability include differences in individual organism behavior (within a species), changes in the weather or ambient temperature, unanticipated interference from other stressors, interactions with other species in the community, differences between microenvironments, and numerous other factors.

Dioxins, furans, and many PCB congeners act through the same receptor-mediated toxic mechanism. These COPCs typically have additive effects, and their toxicity is best described using a toxic equivalence approach. Individual PCB congeners were not analyzed in sediment or fish tissue collected at this site, therefore the analytical data are not sufficient to conduct this type of analysis. This BERA identified risk to fish (based on CTCs) and piscivorous ecological receptors from dietary exposure to PCBs and dioxins/furans. Effects were characterized by looking at individual COPCs. Because dioxin-like COPCs act via the same toxic mechanism, risk from exposure to dioxin-like COPCs is additive. Risk from exposure to dioxin-like COPCs may be underestimated in this BERA.

13.0 CONCLUSIONS

Several lines of evidence were used to assess the risk to ecological receptors from COPCs present within the LLC ecosystem. All of these lines of evidence suggest that contaminants in the sediment and surface water may pose a risk to ecological receptors within Ley Creek. However,

the risk assessment indicates that for most COPCs the risks do not exceed the range bounded by the NOAEL and LOAEL.

In addition to chemical contaminants present in Lower Ley Creek, it is physically impaired and has received road/urban runoff. Lower Ley Creek has been impacted physically through re-routing of the streambed and other changes to the creek. Waste material historically discharged into Onondaga Lake may have migrated into Lower Ley Creek and affected the overall water and sediment quality. The potential impacts from physical changes to the creek and import of waste materials from Onondaga Lake on ecological receptors in Lower Ley Creek is unknown, however they may have indirect effects on aquatic organisms due to adverse impacts on habitat quality.

Exceedances of surface water quality benchmarks and sediment benchmarks suggest potential risk to aquatic plants, benthic invertebrates, and fish. In sediment, inorganics (particulary Cd, Cr, Cu and Ni), PAHs, and PCBs pose potential risk to aquatic plants and benthic invertebrates, and Zn, which may pose potential risk to aquatic plants. The sediment sampling locations at which the most exceedances of sediment screening benchmarks were calculated include R3-3 (PAHs); R2-2, R3-9 and R2-12 (inorganics); R2-15 (PCBs); R2-6, R3-9, and R3-11 (pesticides); R2-12 (dioxins/furans); and R2-6 (VOCs).

Reduced growth was observed in invertebrates exposed to sediment samples collected from several locations in Lower Ley Creek; significant mortality was observed in one sample. While significant adverse effects were observed in the sediment toxicity tests, no significant correlations with measured COPC concentrations in sediment samples were observed. The range of COPC concentrations in the samples selected for toxicity testing was much lower than many of the concentrations measured in samples collected in November of 2009 (Table 43).

Concentrations of dioxin in fish tissue collected from Lower Ley Creek exceeded concentrations reported to be associated with adverse effects in fish.

Piscivorous mammals are at risk from dietary exposure to measured total PCB concentrations in fish from Lower Ley Creek. Piscivorous mammals are also at risk from exposure to total PCB concentrations measured in surface soil samples collected in the swale area.

14.0 DEVELOPMENT OF THRESHOLDS FOR ADVERSE EFFECTS

Because no dose-response relationship was observed in the toxicity tests, no-effect concentrations for direct toxicity of inorganics and PAHs to *H. azteca* and *C.dilutus* were identified and compared with maximum concentrations measured during the 2009 sampling event, screening benchmarks, and effect concentrations for growth and mortality in *H. azteca* (Table 43).

The maximum no effect concentration for exposure of *H. azteca* to PAHs (45.19 mg/kg) was lower than the measured concentrations at which significant effects on growth and survival were observed. A LOAEL concentration for adverse effects on growth and survival of 54.78 mg PAHs/kg (growth) and 156.56 mg PAHs/kg (survival) were identified.

A maximum no-effect concentration of 14.2 mg/kg was identified for Sb. This concentration was greater than the maximum concentration measured in samples collected in 2009, therefore Sb was eliminated from further consideration as a COPC resulting in direct toxicity to benthic invertebrates.

Because concentrations measured in samples collected in 2009 were higher than screening benchmarks and the observed no-effect concentrations in the toxicity tests, the following inorganics were retained as COPCs potentially resulting in direct toxicity to benthic invertebrates: As, Cd, Cr, Cu, Pb, Hg, Ni, Ag, and Zn. The maximum no-effect concentration observed in the toxicity tests was identified as the PRG (Table 43).

A remedial action objective of 0.8 mg Hg/kg was identified for Onondaga Lake (U.S. EPA 2005). The concentration of 0.29 mg Hg/kg identified in this risk assessment will meet the objective of eliminating Ley Creek as a source of Hg to Onondaga Lake.

Site-specific bioaccumulation factors for PCBs were calculated for forage fish in the upper, middle and lower sections of Lower Ley Creek (Table 44). Lowest observed adverse effect level-based and NOAEL-based sediment concentrations were calculated to identify a range of sediment PCB concentrations below which adverse effects on wildlife receptors would not be expected (Table 45). The equation used for calculating the risk-based sediment concentrations is:

$$C_{sed} = (LOAEL * BW) / [SIR + (FIR * BAF)]$$ where:

C_{sed}	=	the sediment cleanup goal in mg/kg
LOAEL	=	the lowest observed adverse effect level (mg/kgBW/day)
BW	=	the body weight of the wildlife receptor (mink)
SIR	=	Sediment ingestion rate (kg/day)
FIR	=	Food ingestion rate (kg/day)
BAF	=	Bioaccumulation factor (ratio of concentration of PCBs in fish tissue to concentration of PCBs in sediment

Sediment concentrations that would result in calculated HQs less than 1.0 for mink (the most sensitive receptor at this site based on the food chain models) were calculated. The LOAEL-based sediment concentrations that would be protective of ecological receptors ranged from 0.08 to 2.28 mg/kg. The NOAEL-based sediment concentration protective of ecological receptors ranged from 0.01 to 0.23 mg/kg.

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Tables

Lower Ley Creek Site
Streamlined Baseline Ecological Risk Assessment
Final Report
January 2011

TABLE 1. Maximum and Mean Sediment COPC Concentrations used in Ecological Risk Assessment
Lower Ley Creek Site, Syracuse, New York

Analyte	Upstream (mg/kg)		Middle (mg/kg)		Downstream (mg/kg)	
	mean	maximum	mean	maximum	mean	maximum
Inorganic Analytes						
Antimony	0.7300	1.8000	1.5515	4.0000	0.8750	1.4000
Arsenic	5.6182	10.5000	8.4647	19.0000	1.4375	2.3000
Barium	56.3364	120.0000	105.0676	210.0000	98.4750	118.0000
Cadmium	1.7773	7.0000	14.8313	107.0000	0.8900	1.3000
Chromium	171.3727	1090.0000	63.6382	247.0000	8.2000	19.8000
Copper	106.2455	405.0000	97.4706	433.0000	11.1250	27.0000
Lead	101.9273	273.0000	82.1706	284.0000	21.2000	63.7000
Manganese	206.8571	261.0000	348.7059	886.0000	229.5000	258.0000
Mercury	0.1022	0.2700	0.3481	1.3000	0.0340	0.0610
Nickel	69.6000	447.0000	23.8029	52.4000	7.6750	8.7000
Selenium	0.4618	0.7800	1.5471	4.5000	0.4025	0.4700
Thallium	0.3191	0.4200	0.6822	2.0000	0.4413	0.7000
Vanadium	10.2143	15.3000	17.3500	31.8000	3.0000	4.1000
Zinc	181.0545	436.0000	352.6941	1640.0000	25.7500	56.6000
Organic Compounds						
Bis(2-ethylhexyl)phthalate	0.2970	1.1000	0.4840	4.9000	0.0260	0.0300
Chlordane	0.0065	0.0274	0.0049	0.0370	0.0005	0.0006
DDD/DDE/DDT	0.0332	0.1230	0.0124	0.0490	0.0025	0.0054
Dichlorobenzenes	0.0042	0.0140	0.0199	0.0990	0.0032	0.0033
Dieldrin	0.0005	0.0007	0.0094	0.1100	0.0005	0.0006
Endrin	0.0084	0.0220	0.0045	0.0280	0.0005	0.0006
Hexachlorobenzene	0.2580	1.1000	0.1910	0.8400	0.0260	0.0300
Hexachlorocyclohexanes	0.0431	0.2943	0.0051	0.0260	0.0010	0.0012
Methylmercury	0.1022	0.2700	0.3481	1.3000	0.0340	0.0610
Total PAHs	164.3140	671.2000	39.9290	331.6400	3.6780	9.8430
Trichlorobenzenes	0.0018	0.0032	0.0133	0.0660	0.0021	0.0022
Xylenes	0.0045	0.0303	0.0140	0.0660	0.0021	0.0022
Dioxins/furans	0.0002	0.0004	0.0006	0.0017	0.0000	0.0000
Total PCBs	4.6450	19.0460	1.7440	18.0330	0.1020	0.2680
Aroclor-1016	0.0051	0.0069	0.0240	0.1200	0.0050	0.0055
Aroclor-1221	0.0051	0.0069	0.0240	0.1200	0.0050	0.0055
Aroclor-1232	0.0051	0.0069	0.0240	0.1200	0.0050	0.0055
Aroclor-1242	4.6000	19.0000	0.4520	2.7000	0.0610	0.2300
Aroclor-1248	0.0051	0.0069	0.0240	0.1200	0.0050	0.0055
Aroclor-1254	0.0051	0.0069	0.0240	0.1200	0.0050	0.0055
Aroclor-1260	0.0051	0.0069	1.0930	18.0000	0.0160	0.0480

mg/kg = milligrams per kilogram
DDD = 1,1'-(2,2 dichloroethylidene)bis[4-chlorobenzene]
DDE = 1,1'-(2,2 dichloroethenylidene)bis[4-chlorobenzene]
DDT = dichlorodiphenyltrichloroethane
PCB = Polychlorinated biphenyl
PAHs = Polycyclic aromatic hydrocarbons

Only total mercury analysis was conducted; total mercury was used for both the inorganic and methylmercury exposure

TABLE 2. Maximum and Mean COPC Concentrations in Surface Water used in Ecological Risk Assessment
Lower Ley Creek Site, Syracuse, New York

Analyte	Upstream (mg/L)		Middle (mg/L)		Downstream (mg/L)	
	mean	max	mean	max	mean	max
Antimony	0.0050	0.0050	0.0050	0.0050	0.0050	0.0050
Arsenic	0.0016	0.0018	0.0012	0.0013	0.0014	0.0014
Barium	0.1015	0.1050	0.1044	0.1080	0.1130	0.1130
Cadmium	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025
Chromium	0.0044	0.0084	0.0010	0.0010	0.0010	0.0010
Copper	0.0056	0.0066	0.0029	0.0038	0.0028	0.0028
Lead	0.0041	0.0067	0.0024	0.0099	0.0005	0.0005
Manganese	0.1100	0.1200	0.0953	0.1020	0.1080	0.1080
Mercury	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Nickel	0.0049	0.0062	0.0030	0.0033	0.0030	0.0030
Selenium	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025
Thallium	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025
Vanadium	0.0014	0.0015	0.0028	0.0050	0.0013	0.0013
Zinc	0.0134	0.0181	0.0068	0.0093	0.0060	0.0060
Organic Compounds						
Bis(2-ethylhexyl)phthalate	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005
Chlordane	NA	NA	NA	NA	NA	NA
DDT and metabolites	NA	NA	NA	NA	NA	NA
Dichlorobenzenes	0.0015	0.0015	0.0015	0.0015	0.0015	0.0015
Dieldrin	NA	NA	NA	NA	NA	NA
Endrin	NA	NA	NA	NA	NA	NA
Hexachlorobenzene	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005
Hexachlorocyclohexans	NA	NA	NA	NA	NA	NA
Methylmercury	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Total PAHs	0.0084	0.0084	0.0127	0.0130	0.0123	0.0130
Trichlorobenzenes	0.0010	0.0010	0.0010	0.0010	0.0010	0.0010
Xylenes	0.0010	0.0010	0.0010	0.0010	0.0010	0.0010
Dioxins/furans	NA	NA	NA	NA	NA	NA
Total PCBs	NA	NA	NA	NA	NA	NA
Aroclor 1016	NA	NA	NA	NA	NA	NA
Aroclor 1221	NA	NA	NA	NA	NA	NA
Aroclor 1232	NA	NA	NA	NA	NA	NA
Aroclor 1242	NA	NA	NA	NA	NA	NA
Aroclor 1248	NA	NA	NA	NA	NA	NA
Aroclor 1254	NA	NA	NA	NA	NA	NA
Aroclor 1260	NA	NA	NA	NA	NA	NA

mg/L = Milligrams per Liter
DDD = 1,1'-(2,2 dichloroethylidene)bis[4-chlorobenzene]
DDE = 1,1'-(2,2 dichloroethenylidene)bis[4-chlorobenzene]
DDT = dichlorodiphenyltrichloroethane
NA = Not Analyzed
PCB = Polychlorinated biphenyl
PAHs = Polycyclic aromatic hydrocarbons

Only total mercury analysis was conducted; total mercury was used for both the inorganic and methylmercury exposure

TABLE 3. Maximum and Mean Fish Tissue Concentrations used in Ecological Risk Assessment, mg/kg Wet Weight
Lower Ley Creek Site, Syracuse, New York

	Upstream (n = 3)		Middle (n = 1)	Downstream (n = 2)	
Analyte	Mean	Maximum	Mean	Mean	Maximum
Antimony	0.09	0.09	0.09	0.09	0.09
Arsenic	0.21	0.30	0.20	0.21	0.23
Barium	0.69	0.93	1.18	1.07	1.29
Cadmium	0.04	0.04	0.04	0.04	0.04
Chromium	0.36	0.60	0.05	0.22	0.39
Copper	0.66	1.35	0.04	0.17	0.30
Lead	0.18	0.35	0.65	0.18	0.25
Manganese	3.61	4.33	6.88	3.63	3.65
Mercury	0.04	0.06	0.03	0.04	0.05
Nickel	0.06	0.06	0.06	0.06	0.06
Selenium	0.73	0.80	0.70	0.64	0.70
Thallium	0.19	0.19	0.19	0.19	0.19
Vanadium	0.10	0.10	0.17	0.10	0.10
Zinc	28.7	29.9	24.3	32.3	39.4
Organic Compounds					
Bis(2-ethylhexyl)phthalate	NA	NA	NA	NA	NA
Chlordane	0.00164	0.00177	0.00054	0.00167	0.00225
DDD/DDE/DDT	0.00126	0.00130	R	0.00203	0.00203
Dichlorobenzenes	NA	NA	NA	NA	NA
Dieldrin	0.00363	0.00363	R	0.00195	0.00195
Endrin	0.00301	0.00350	0.00169	0.00158	0.00225
Hexachlorobenzene	NA	NA	NA	NA	NA
Hexachlorocyclohexanes	0.00030	0.00047	0.00038	0.00039	0.00043
Mercury	0.04	0.06	0.03	0.04	0.05
PAHs	NA	NA	NA	NA	NA
Trichlorobenzenes	NA	NA	NA	NA	NA
Xylenes	NA	NA	NA	NA	NA
Dioxins/furans	0.000033		0.000043	0.000048	
Total Aroclor	0.3125	0.3500	0.1125	0.1475	0.2150
Aroclor-1016	0.00083	0.00083	0.00083	0.00083	0.00083
Aroclor-1221	0.00083	0.00083	0.00083	0.00083	0.00083
Aroclor-1232	0.00083	0.00083	0.00083	0.00083	0.00083
Aroclor-1242	0.00083	0.00083	0.00083	0.00083	0.00083
Aroclor-1248	0.00083	0.00083	0.00083	0.00083	0.00083
Aroclor-1254	0.2446	0.3000	0.0850	0.1156	0.1800
Aroclor-1260	0.1013	0.1125	0.0364	0.0506	0.0725

mg/kg = milligrams per kilogram
DDD = 1,1'-(2,2 dichloroethylidene)bis[4-chlorobenzene]
DDE = 1,1'-(2,2 dichloroethenylidene)bis[4-chlorobenzene]
DDT = dichlorodiphenyltrichloroethane
Only total mercury analysis was conducted; total mercury was used for both the inorganic and methylmercury exposure concentration
Dioxin/furan analysis only conducted on one forage fish sample per reach
NA indicates not analyzed

TABLE 4. Assessment Endpoints and Measures of Exposure/Effects for the Lower Ley Creek Ecological Risk Assessment
Lower Ley Creek Site
Syracuse, New York

Assessment Endpoint	Measures of Exposure/Effects	Receptor Species
Survival, growth and reproduction of aquatic plants	Compare mean and maximum measured concentrations of COPCs in sediment and surface water to screening benchmark values	
Survival, growth and reproduction of benthic invertebrates	Compare mean and maximum measured concentrations of COPCs in sediment and surface water to screening benchmark values	
Survival, growth and reproduction of fish	Compare mean and maximum measured concentrations of COPCs in sediment and surface water to screening benchmark values Determine whether measured COPC concentrations in fish tissue exceed critical tissue concentrations	
Survival, growth and reproduction of piscivorous birds	Mean and maximum concentrations of COPCs measured in fish tissue will be used in food chain models to calculate dietary exposure of selected receptor species. Calculated dietary exposure concentrations will be compared with TRVs for COPCs obtained from the literature for birds.	Belted Kingfisher Great Blue Heron
Survival, growth and reproduction of piscivorous mammals	Mean and maximum concentrations of COPCs measured in fish tissue will be used in food chain models to calculate dietary exposure of selected receptor species. Calculated dietary exposure concentrations will be compared with TRVs for COPCs obtained from the literature for mammals.	Mink River Otter

COPC = Contaminant of Potential Concern
TRV = Toxicity Reference Value

TABLE 5. Surface Water Concentrations Versus Screening Benchmarks
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum Measured Concentration (ug/L)	Location	EPA AWQC-FCV (ug/L)	Hazard Quotient	EPA CMC (2002) (ug/L)	Hazard Quotient	EPA CCC (2002) (ug/L)	Hazard Quotient	NYSDEC Acute (ug/L)	Hazard Quotient	NYSDEC Chronic (ug/L)	Hazard Quotient	ORNL FW Tier II acute (ug/L)	Hazard Quotient	ORNL FW Tier II chronic (ug/L)	Hazard Quotient
1,1,1-Trichloroethane	0.5	ND (5)		NB		NB		NB		NB		NB	200	0.0	11	0.0
1,1,2,2-Tetrachloroethane	0.5	ND (5)	420	0.0		NB		NB		NB		NB	2100	0.0	610	0.0
1,1,2-Trichloroethane	0.5	ND (5)		NB		NB		NB		NB		NB	5200	0.0	1200	0.0
1,1'-Biphenyl	0.5	ND (5)	14	0.0		NB		NB		NB		NB		NB	14	0.0
1,1-Dichloroethane	0.4	R3-2	47	0.0		NB		NB		NB		NB	830	0.0	47	0.0
1,1-Dichloroethylene (1,1-Dichloroethene)	0.5	ND (5)		NB		NB		NB		NB		NB	450	0.0	25	0.0
1,2,4-Trichlorobenzene	0.5	ND (5)	110	0.0		NB		NB		NB		NB	700	0.0	110	0.0
1,2-Dichlorobenzene	0.5	ND (5)	14	0.0		NB		NB		NB		NB	260	0.0	14	0.0
1,2-Dichloroethane	0.5	ND (5)		NB		NB		NB		NB		NB	8800	0.0	910	0.0
1,3-Dichlorobenzene	0.5	ND (5)	71	0.0		NB		NB		NB		NB	630	0.0	71	0.0
1,4-Dichlorobenzene	0.5	ND (5)	15	0.0		NB		NB		NB		NB	180	0.0	15	0.0
2-Butanone	1	ND (10)		NB		NB		NB		NB		NB	240000	0.0	14000	0.0
2-Hexanone	1	ND (10)		NB		NB		NB		NB		NB	1800	0.0	99	0.0
2-Methylnaphthalene	0.5	ND (5)		NB		NB		NB	42	0.0	4.7	0.1		NB		NB
4-Bromophenyl phenyl ether	0.5	ND (5)	1.5	0.3		NB		NB		NB		NB		NB	1.5	0.3
4-Methyl-2-Pentanone	1	ND (10)		NB		NB		NB		NB		NB	2200	0.0	170	0.0
4-Nitrophenol	1	ND (10)		NB		NB		NB		NB		NB	1200	0.0	300	0.0
Acenaphthene	0.16	R2-11	23	0.0		NB		NB	48	0.0	5.3	0.0		NB		NB
Acetone	1	ND (10)		NB		NB		NB		NB		NB	28000	0.0	1500	0.0
Anthracene	0.5	ND (5)		NB		NB		NB	35	0.0	3.8	0.1	13	0.0	0.73	0.7
Antimony	5	ND (10)		NB		NB		NB		NB		NB	180	0.0	30	0.2
Arsenic	1.8	R3-2		NB	340	0.0	150	0.0	340	0.0	150	0.0		NB		NB
Arsenic III	1.8	R3-2	190	0.0		NB		NB		NB		NB		NB		NB
Arsenic V	1.8	R3-2	8.1	0.2		NB		NB		NB		NB	66	0.0	3.1	0.6
Barium	113	R1-4	3.9	29.0		NB		NB		NB		NB	110	1.0	4	28.3
Benzene	0.5	ND (5)	46	0.0		NB		NB	760	0.0	210	0.0	2300	0.0	130	0.0
Benzo(a)anthracene	0.5	ND (5)		NB		NB		NB	0.23	2.2	0.03	16.7	0.49	1.0	0.027	18.5
Benzo(a)pyrene	0.5	ND (5)	0.014	35.7		NB		NB		NB		NB	0.24	2.1	0.014	35.7
Beryllium	0.5	ND (5)		NB		NB		NB		NB		NB	35	0.0	0.66	0.8
Beryllium (hardness <=75)	0.5	ND (5)		NB		NB		NB		NB	11	0.0		NB		NB
Beryllium (hardness > 75)	0.5	ND (5)		NB		NB		NB		NB	1100	0.0		NB		NB
Bis(2-ethylhexyl) phthalate	0.5	ND (5)	32	0.0		NB		NB		NB	0.6	0.8	27	0.0	3	0.2
Bromoform	0.5	ND (5)	320	0.0		NB		NB		NB		NB	2300	0.0	320	0.0
Butyl benzyl phthalate	0.5	ND (5)	19	0.0		NB		NB		NB		NB		NB	19	0.0
Cadmium	2.5	ND (5)	1	2.5	2	1.3	0.25	10.0		NB		NB		NB		NB
Cadmium (hardness=100)	2.5	ND (5)		NB		NB		NB	3.8397	0.7	2.0927	1.2		NB		NB
Carbon Disulfide	0.5	ND (5)		NB		NB		NB		NB		NB	17	0.0	0.92	0.5
Carbon Tetrachloride	0.5	ND (5)	240	0.0		NB		NB		NB		NB	180	0.0	9.8	0.1
Chlorobenzene	0.5	ND (5)	130	0.0		NB		NB		NB	5	0.1	1100	0.0	64	0.0
Chloroform	0.5	ND (5)		NB		NB		NB		NB		NB	490	0.0	28	0.0
Chromium (hardness = 100)	8.4	R3-7		NB		NB		NB	569.76	0.0	74.115	0.1		NB		NB
Copper	6.6	R3-2		NB	13	0.5		NB		NB		NB		NB		NB
Copper (hardness=100)	6.6	R3-2		NB		NB		NB	13.439	0.5	8.956	0.7		NB		NB
Copper (dissolved)	2.6	R1-4	1.45	4.6												
Dibenzofuran	0.3	R2-11	20	0.0		NB		NB		NB		NB	66	0.0	3.7	0.1
Diethylphthalate	0.5	ND (5)	220	0.0		NB		NB		NB		NB	1800	0.0	210	0.0
Di-n-butyl phthalate	0.5	ND (5)	33	0.0		NB		NB		NB		NB	190	0.0	35	0.0
Ethylbenzene	0.5	ND (5)		NB		NB		NB	150	0.0	17	0.0	130	0.0	7.3	0.1
Fluoranthene	0.24	R3-2	8.1	0.0		NB		NB		NB		NB		NB		NB
Fluorene	0.28	R2-11	3.9	0.1		NB		NB	4.8	0.1	0.54	0.5	70	0.0	3.9	0.1
Hexachlorobutadiene	0.5	ND (5)		NB		NB		NB	10	0.1	1	0.5		NB		NB
Hexachlorocyclopentadiene	0.5	ND (5)		NB		NB		NB	4.5	0.1	0.45	1.1		NB		NB

TABLE 5. Surface Water Concentrations Versus Screening Benchmarks
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum Measured Concentration (ug/L)	Location	EPA AWQC-FCV (ug/L)	Hazard Quotient	EPA CMC (2002) (ug/L)	Hazard Quotient	EPA CCC (2002) (ug/L)	Hazard Quotient	NYSDEC Acute (ug/L)	Hazard Quotient	NYSDEC Chronic (ug/L)	Hazard Quotient	ORNL FW Tier II acute (ug/L)	Hazard Quotient	ORNL FW Tier II chronic (ug/L)	Hazard Quotient
Hexachloroethane	0.5	ND (5)	12	0.0		NB		NB		NB		NB	210	0.0	12	0.0
Isopropylbenzene	0.5	ND (5)		NB		NB		NB	23	0.0	2.6	0.2		NB		NB
Lead	9.9	R2-1	2.5	4.0	65	0.2	2.5	4.0		NB		NB		NB		NB
Manganese	120	R3-2	80	1.5		NB		NB		NB		NB	2300	0.1	120	1.0
Mercury	0.1	ND (0.2)	1.3	0.1	1.4	0.1	0.77	0.1	1.4	0.1	0.77	0.1		NB		NB
Methylene chloride	0.5	ND (5)		NB		NB		NB		NB		NB	26000	0.0	2200	0.0
Naphthalene	0.41	R1-4	24	0.0		NB		NB	110	0.0	13	0.0	190	0.0	12	0.0
Nickel	6.2	R3-7		NB	470	0.0	52	0.1		NB		NB		NB		NB
N-Nitrosodiphenylamine	0.5	ND (5)		NB		NB		NB		NB		NB	3800	0.0	210	0.0
Pentachlorophenol	0.67	R1-4		NB	19	0.0	15	0.0		NB		NB		NB		NB
Pentachlorophenol (pH = 7.8)	0.67	R1-4		NB	19	0.0	15	0.0	19.492	0.0	14.953	0.0		NB		NB
Phenanthrene	0.17	R3-7	6.3	0.0		NB		NB	45	0.0	5	0.0		NB		NB
Pyrene	0.25	R3-2		NB		NB		NB	42	0.0	4.6	0.1		NB		NB
Selenium	2.5	ND (5)	5	0.5		NB	5	0.5		NB		NB		NB		NB
Silver	1	R1-4		NB	3.2	0.3		NB		NB		NB		NB	0.36	2.8
Silver (hardness = 100)	1	R1-4		NB		NB		NB	4.0588	0.2	0.1	10.0		NB		NB
Tetrachloroethylene (Tetrachloroethene)	0.5	R3-7	120	0.0		NB		NB		NB		NB	830	0.0	98	0.0
Thallium	2.5	ND (5)		NB		NB		NB	20	0.1	8	0.3	110	0.0	12	0.2
Toluene	0.94	R3-7	130	0.0		NB		NB	480	0.0	100	0.0	120	0.0	9.8	0.1
Trichloroethylene (Trichloroethene)	0.69	R3-5		NB		NB		NB		NB		NB	440	0.0	47	0.0
Vanadium	1.5	R3-2, R3-5	19	0.1		NB		NB	190	0.0	14	0.1	280	0.0	20	0.1
Xylene	0.5	ND (5)		NB		NB		NB		NB		NB	230	0.0		NB
Xylenes, total	0.5	ND (5)		NB		NB		NB		NB		NB		NB	13	0.0
Zinc	18.1	R3-2	100	0.2	120	0.2	120	0.2		NB		NB		NB		NB
Zinc (hardness=100)	18.1	R3-2		NB		NB		NB	117.18	0.2		NB		NB		NB

NB indicates no benchmark available
ND indicates analyte not detected in any surface water sample, number in parentheses is the detection limit.
Pesticide/PCB analyses were not conducted on surface water samples
Shading indicates Hazard Quotient greater than 1.0

EPA AWQC-FCV: U.S. EPA. OSWER (Office of Solid Waste and Emergency Response). 1996. Eco Update: Ecotox thresholds. Washington, D.C. EPA 540/F-95/038.
EPA CMC (1999): U.S. EPA. 1999. National Recommended Water Quality Criteria - Correction. EPA 822-Z-99-001. Freshwater Criteria Maximum Concentrations (CMC).
EPA CCC (1999): U.S. EPA. 1999. National Recommended Water Quality Criteria - Correction. EPA 822-Z-99-001. Freshwater Criteria Continuous Concentrations (CCC).
EPA CMC (2002): U.S. EPA. 2002. National Recommended Water Quality Criteria. EPA 822-R-02-047.
EPA CCC (2002): U.S. EPA. 2002. National Recommended Water Quality Criteria. EPA 822-R-02-047.
NYSDEC acute: NY DEC (New York State Department of Environmental Conservation), Division of Water. 1998. Technical and Operational Guidance Series (1.1.1). Ambient Water Quality Standards and Guidance Values and Groundwater Effluent Limitations.
NYSDEC chronic: NY DEC (New York State Department of Environmental Conservation), Division of Water. 1998. Technical and Operational Guidance Series (1.1.1). Ambient Water Quality Standards and Guidance Values and Groundwater Effluent Limitations.
ORNL FW Tier II acute: Suter, G.W. II, and Tsao, C.L. 1996. Toxicological benchmarks for screening potential contaminants of concern for effects on aquatic biota: 1996 Revision. ES/ER/TM-96/R2.
ORNL FW Tier II chronic: Suter, G.W. II, and Tsao, C.L. 1996. Toxicological benchmarks for screening potential contaminants of concern for effects on aquatic biota: 1996 Revision. ES/ER/TM-96/R2.

Although the 1999 EPA CMC and CCC values were utilized in the SLERA for Onondaga Lake and Geddes Brook/Nine Mile Creek, the 2002 values are current and were utilized in this risk assessment.

TABLE 6. Sediment Concentrations Versus Screening Benchmarks, Organic COPCs
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum measured concentration (mg/kg)	Location	NYSDEC chronic (mg/kg)	Hazard Quotient	NY SDEC acute (mg/kg)	Hazard Quotient	NYSDEC bioaccum (mg/kg)	Hazard Quotient	EPA SQB (mg/kg)	Hazard Quotient	OME sed LELs (mg/kg	Hazard Quotient	OME sed SELs (mg/kg)	Hazard Quotient	EPA TEC (mg/kg)	Hazard Quotient	ORNL Secondary Chronic (mg/kg)	Hazard Quotient	ORNL Plant (mg/kg)	Hazard Quotient	EcoSSL for Plants (mg/kg)	Hazard Quotient
Volatile Organic Compounds																						
1,1,1-Trichloroethane	0.035	R3-1		NB		NB		NB	0.17	0.2		NB		NB		NB	0.03	1.2		NB		NB
1,1,2,2-Tetrachloroethane	0.033	R2-6		NB		NB		NB	0.94	0.0		NB		NB		NB	1.4	0.0		NB		NB
1,1,2-Trichloroethane	0.033	R2-6		NB		NB		NB		NB		NB		NB		NB	1.2	0.0		NB		NB
1,1-Dichloroethane	0.033	R2-6		NB		NB		NB		NB		NB		NB		NB	0.027	1.2		NB		NB
1,1-Dichloroethylene (1,1-Dichloroethene)	0.033	R2-6		NB		NB		NB		NB		NB		NB		NB	0.031	1.1		NB		NB
1,2,4-Trichlorobenzene	0.033	R2-6		NB		NB		NB	9.2	0.0		NB		NB		NB	9.6	0.0		NB		NB
1,2-Dichlorobenzene	0.033	R2-6		NB		NB		NB	0.34	0.1		NB		NB		NB	0.33	0.1		NB		NB
1,2-Dichloroethane	0.033	R2-6		NB		NB		NB		NB		NB		NB		NB	0.25	0.1		NB		NB
1,3-Dichlorobenzene	0.033	R2-6		NB		NB		NB	1.7	0.0		NB		NB		NB	1.7	0.0		NB		NB
1,4-Dichlorobenzene	0.033	R2-6		NB		NB		NB	0.35	0.1		NB		NB		NB	0.34	0.1		NB		NB
2-Hexanone	0.065	R2-6		NB		NB		NB		NB		NB		NB		NB	0.022	3.0		NB		NB
4-Methyl-2-Pentanone	0.065	R2-6		NB		NB		NB		NB		NB		NB		NB	0.033	2.0		NB		NB
Acetone	0.39	R2-6		NB		NB		NB		NB		NB		NB		NB	0.0087	44.8		NB		NB
Benzene	0.05	R3-7	0.28	0.2	1.03	0.0		NB	0.057	0.9		NB		NB		NB	0.16	0.3		NB		NB
Carbon Disulfide	0.0092	R2-7		NB		NB		NB		NB		NB		NB		NB	0.00085	10.8		NB		NB
Carbon Tetrachloride	0.033	R2-6		NB		NB		NB	1.2	0.0		NB		NB		NB	0.047	0.7		NB		NB
Chlorobenzene	0.033	R2-6	0.035	0.9	0.346	0.1		NB	0.82	0.0		NB		NB		NB	0.41	0.1		NB		NB
Chloroform	0.033	R2-6		NB		NB		NB		NB		NB		NB		NB	0.022	1.5		NB		NB
Ethylbenzene	0.033	R2-6	0.24	0.1	2.12	0.0		NB	3.6	0.0		NB		NB		NB	0.089	0.4		NB		NB
Methylene chloride	0.0088	R2-6		NB		NB		NB		NB		NB		NB		NB	0.37	0.0		NB		NB
Tetrachloroethylene (Tetrachloroethene)	0.033	R2-6		NB		NB		NB	0.53	0.1		NB		NB		NB	0.41	0.1		NB		NB
Toluene	0.033	R2-6	0.49	0.1	2.35	0.0		NB	0.67	0.0		NB		NB		NB	0.05	0.7	200	0.00		NB
Trichloroethylene (Trichloroethene)	0.033	R2-6		NB		NB		NB	1.6	0.0		NB		NB		NB	0.22	0.2		NB		NB
Xylene	0.066	R2-6	0.92	0.1	8.33	0.0		NB	0.025	2.6		NB		NB		NB	0.16	0.4		NB		NB
Xylenes, total	0.066	R2-6		NB		NB		NB	0.025	2.6		NB		NB		NB	0.16	0.4		NB		NB
Semi-Volatile Organic Compounds																						
1,1'-Biphenyl	1.4	R3-3		NB		NB		NB	1.1	1.3		NB		NB		NB	1.1	1.3	60	0.02		NB
2-Methylnaphthalene	4.6	R3-3	0.34	13.5	3.04	1.5		NB		NB		NB		NB		NB		NB		NB		NB
Acenaphthene	3.7	R2-13	1.4	2.6		NB		NB	0.62	6.0		NB		NB		NB	1.3	2.8	20	0.19		NB
Acenaphthylene	0.59	R2-6		NB		NB		NB		NB		NB		NB		NB		NB		NB		NB
Anthracene	30	R3-3	1.07	28.0	9.86	3.0		NB		NB	0.22	136.4	3.7	8.1	0.0316	949.4	0.22	136.4		NB		NB
Benzo(a)anthracene	47	R3-3	0.12	391.7	0.94	50.0		NB		NB	0.32	146.9	14.8	3.2	0.26	180.8	0.11	427.3		NB		NB
Benzo(a)pyrene	31	R3-3		NB		NB		NB	0.43	72.1	0.37	83.8	14.4	2.2	0.35	88.6	0.14	221.4		NB		NB
benzo(b)fluoranthene	42	R3-3		NB		NB		NB		NB		NB		NB		NB		NB		NB		NB
Benzo(g,h,i)perylene	17	R3-3		NB		NB		NB		NB	0.17	100.0	3.2	5.3	0.29	58.6		NB		NB		NB
Benzo(k)fluoranthene	17	R3-3		NB		NB		NB		NB	0.24	70.8	13.4	1.3		NB		NB		NB		NB
Biphenyl	1.4	R3-3		NB		NB		NB	1.1	1.3		NB		NB		NB	1.1	1.3	60	0.02		NB
Bis(2-ethylhexyl) phthalate	4.9	R2-12	1.995	2.5		NB		NB		NB		NB		NB		NB	890	0.0		NB		NB
Butyl benzyl phthalate	0.59	R2-6		NB		NB		NB	11	0.1		NB		NB		NB	11	0.1		NB		NB
Dibenzo(a,h)anthracene	4.9	R3-3		NB		NB		NB		NB	0.06	81.7	1.3	3.8		NB		NB		NB		NB
Dibenzofuran	13	R3-3		NB		NB		NB	2	6.5		NB		NB		NB	0.42	31.0		NB		NB
Diethylphthalate	0.51	R2-7		NB		NB		NB	0.63	0.8		NB		NB		NB	0.6	0.9	100	0.01		NB
Fluoranthene	130	R3-3	10.2	12.7		NB		NB	2.9	44.8	0.75	173.3	10.2	12.7	0.0642	2025	6.2	21.0		NB		NB
Fluorene	19	R3-3	0.08	237.5	0.73	26.0		NB	0.54	35.2	0.19	100.0	1.6	11.9	0.0346	549	0.54	35.2		NB		NB
Hexachlorobenzene	0.59	R2-6	55.7	0.0	90.81	0.0	0.12	4.9		NB	0.02	29.5	0.24	2.5		NB		NB		NB		NB
Hexachlorobutadiene	0.59	R2-6	0.055	10.7	0.55	1.1	0.04	14.8		NB		NB		NB		NB		NB		NB		NB
Hexachlorocyclopentadiene	0.59	R2-6	0.044	13.4	0.44	1.3		NB		NB		NB		NB		NB		NB	10	0.06		NB
Hexachloroethane	0.59	R2-6		NB		NB		NB	1	0.6		NB		NB		NB	1	0.6		NB		NB
High Molecular Weight PAHs	451.9	R3-3		NB		NB		NB		NB		NB		NB	0.19295	2342		NB		NB		NB
Indeno(1,2,3-c,d)pyrene	20	R3-3		NB		NB		NB		NB	0.2	100.0	3.2	6.3	0.078	256		NB		NB		NB
Low Molecular Weight PAHs	249.2	R3-3		NB		NB		NB		NB		NB		NB	0.07642	3261		NB		NB		NB
Naphthalene	8.6	R3-3	0.3	28.7	2.58	3.3		NB	0.48	17.9		NB		NB	0.0328	262	0.24	35.8		NB		NB

TABLE 6. Sediment Concentrations Versus Screening Benchmarks, Organic COPCs
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum measured concentration (mg/kg)	Location	NYSDEC chronic (mg/kg)	Hazard Quotient	NY SDEC acute (mg/kg)	Hazard Quotient	NYSDEC bioaccum (mg/kg)	Hazard Quotient	EPA SQB (mg/kg)	Hazard Quotient	OME sed LELs (mg/kg	Hazard Quotient	OME sed SELs (mg/kg)	Hazard Quotient	EPA TEC (mg/kg)	Hazard Quotient	ORNL Secondary Chronic (mg/kg)	Hazard Quotient	ORNL Plant (mg/kg)	Hazard Quotient	EcoSSL for Plants (mg/kg)	Hazard Quotient
PAH's, total	671.2	R3-3		NB		NB		NB	4	167.8	4	167.8	100	6.7	0.26405	2542		NB		NB		NB
Pentachlorophenol	21	R3-3	0.4	52.5	1	21.0		NB		NB		NB		NB		NB		NB	3	7.00	5	4.20
Phenanthrene	160	R3-3	1.2	133.3		NB		NB	0.85	188.2	0.56	285.7	9.5	16.8		NB	1.8	88.9		NB		NB
Phenol	0.28	R2-12	0.005	56.0		NB		NB		NB		NB		NB		NB	0.031	9.0	70	0.00		NB
Pyrene	100	R3-3	9.61	10.4	87.75	1.1		NB	0.66	151.5	0.49	204.1	8.5	11.8	0.57	175.4		NB		NB		NB
Pesticides/Polychlorinated Biphenyls																						
Aldrin	0.0064	R2-6		NB		NB	0.0077	0.8		NB	0.002	3.2	0.08	0.1		NB		NB		NB		NB
Aroclor 1016	0.12	R2-6		NB		NB		NB		NB	0.007	17.1	5.3	0.0		NB		NB		NB		NB
Aroclor 1221	0.12	R2-6		NB		NB		NB		NB		NB		NB		NB	0.12	1.0		NB		NB
Aroclor 1232	0.12	R3-9		NB		NB		NB		NB		NB		NB		NB	0.6	0.2		NB		NB
Aroclor 1242	19	R2-6		NB		NB		NB		NB		NB		NB		NB	0.17	111.8		NB		NB
Aroclor 1248	0.12	R2-6		NB		NB		NB		NB	0.03	4.0	1.5	0.1		NB	1	0.1		NB		NB
Aroclor 1254	0.12	R2-15		NB		NB		NB		NB	0.06	2.0	0.34	0.4		NB	0.81	0.1		NB		NB
Aroclor 1260	18	R2-6		NB		NB		NB		NB	0.005	3600	0.24	75.0		NB	4500	0.0		NB		NB
BHC (non Lindane)	0.0064	R2-6		NB		NB		NB		NB		NB		NB		NB	0.12	0.1		NB		NB
BHC, alpha	0.0064	R2-6		NB		NB		NB		NB	0.006	1.1	0.1	0.1		NB		NB		NB		NB
BHC, beta	0.0064	R2-6		NB		NB		NB		NB	0.005	1.3	0.21	0.0		NB		NB		NB		NB
BHC, delta	0.0064	R2-6		NB		NB		NB		NB		NB		NB		NB		NB		NB		NB
BHC, gamma (Lindane)	0.29	R3-9		NB		NB		NB	0.0037	78.4	0.003	96.7	0.01	29.0		NB	0.0037	78.4		NB		NB
Chlordane	0.031	R2-6	0.0003	103.3	0.014	2.2	0.00006	516.7		NB	0.007	4.4	0.06	0.5		NB	2.8	0.0		NB		NB
Chlordane, alpha	0.0064	R2-12		NB		NB		NB		NB		NB		NB		NB		NB		NB		NB
Chlordane, gamma	0.031	R2-12		NB		NB		NB		NB		NB		NB		NB		NB		NB		NB
DDD	0.012	R2-6		NB		NB	0.01	1.2		NB	0.008	1.5	0.06	0.2		NB	0.11	0.1		NB		NB
DDE	0.028	R3-11		NB		NB	0.01	2.8		NB	0.005	5.6	0.19	0.1		NB		NB		NB		NB
DDT	0.084	R3-11	0.01	8.4	11	0.0	0.01	8.4	0.0016	52.5	0.008	10.5	0.71	0.1		NB	0.34	0.2		NB		NB
DDT/DDE/DDD (total)	0.124	R3-11, R2-6		NB		NB	0.01	12.4		NB		NB		NB		NB		NB		NB		NB
Dieldrin	0.11	R2-15	0.09	1.2		NB	0.0077	14.3	0.052	2.1	0.002	55.0	0.91	0.1		NB	0.11	1.0		NB		NB
Endosulfan	0.031	R3-11, R2-6	0.0003	103.3	0.0078	4.0		NB		NB		NB		NB		NB	0.0055	5.6		NB		NB
Endosulfan (alpha and beta)	0.031	R3-11, R2-6		NB		NB		NB	0.0054	5.7		NB		NB		NB		NB		NB		NB
Endosulfan I (a-endosulfan)	0.019	R3-11		NB		NB		NB	0.0029	6.6		NB		NB		NB		NB		NB		NB
Endosulfan II (b-endosulfan)	0.012	R2-6		NB		NB		NB	0.014	0.9		NB		NB		NB		NB		NB		NB
Endrin	0.028	R2-15	0.04	0.7		NB	0.008	3.5	0.02	1.4	0.003	9.3	1.3	0.0		NB	0.042	0.7		NB		NB
HCH, a-	0.0013	R2-6		NB		NB		NB		NB	0.006	0.2	0.1	0.0		NB		NB		NB		NB
HCH, b-	0.0032	R2-6		NB		NB		NB		NB	0.005	0.6	0.21	0.0		NB		NB		NB		NB
HCH, g- (Lindane)	0.29	R3-9		NB		NB		NB	0.0037	78.4	0.003	96.7	0.01	29.0		NB	0.0037	78.4		NB		NB
Heptachlor	0.15	R3-9		NB	0.131	1.1		NB		NB		NB		NB		NB	0.068	2.2		NB		NB
Heptachlor & Heptachlor epoxide	0.159	R3-9, R2-10	0.001	159.0		NB	0.0003	530.0		NB		NB		NB		NB		NB		NB		NB
Heptachlor epoxide	0.0086	R2-10		NB	0.131	0.1		NB		NB	0.005	1.7	0.05	0.2		NB		NB		NB		NB
Hexachlorocyclohexane (HCH, BHC)	0.29	R3-9	0.0006	483.3	0.126	2.3	0.015	19.3		NB	0.003	96.7	0.12	2.4		NB		NB		NB		NB
Lindane (g-BHC)	0.29	R3-9		NB		NB		NB	0.0037	78.4	0.003	96.7	0.01	29.0		NB	0.0037	78.4		NB		NB
Methoxychlor	0.064	R2-6	0.006	10.7		NB		NB	0.019	3.4		NB		NB		NB	0.019	3.4		NB		NB
PCB's, total	19.046	R2-15	0.193	98.7	27.608	0.7	0.014	1360	0.023	828.1	0.07	272.1	5.3	3.6	0.0316	602.7		NB	40	0.48		NB
Toxaphene	0.64	R2-6	0.0001	6400	0.032	20.0		NB	0.028	22.9		NB		NB		NB		NB		NB		NB
Dioxins/Furans																						
2,3,7,8-TCDD-Dioxin	0.0002	R2-12		NB		NB	0.000002	100.0		NB		NB		NB		NB		NB		NB		NB

Shading indicates Hazard Quotient greater than 1.0

EPA SQB: U.S. EPA. OSWER (Office of Solid Waste and Emergency Response). 1996. Eco Update: Ecotox thresholds. Washington, D.C. EPA 540/F-95/038.
EPA TEC: U.S. EPA. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod Hyalella azteca and the midge Chironomus riparius. EPA 905-R96-008, Great Lakes National Program Office, Chicago, IL. 28-day H. azteca TELs.
NYSDEC acute: NY DEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments. converted to mg/kg assuming 1%TOC.
NYSDEC chronic: NY DEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments. Converted to mg/kg assuming 1% TOC.

TABLE 6. Sediment Concentrations Versus Screening Benchmarks, Organic COPCs
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum measured concentration (mg/kg)	Location	NYSDEC chronic (mg/kg)	Hazard Quotient	NY SDEC acute (mg/kg)	Hazard Quotient	NYSDEC bioaccum (mg/kg)	Hazard Quotient	EPA SQB (mg/kg)	Hazard Quotient	OME sed LELs (mg/kg	Hazard Quotient	OME sed SELs (mg/kg)	Hazard Quotient	EPA TEC (mg/kg)	Hazard Quotient	ORNL Secondary Chronic (mg/kg)	Hazard Quotient	ORNL Plant (mg/kg)	Hazard Quotient	EcoSSL for Plants (mg/kg)	Hazard Quotient

NYSDEC bioaccum: NY DEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments. Converted to mg/kg assuming 1% TOC.
OME sed LELs: Persaud, D., Jaagumagi, R. and Hayton, A. Aug.,1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Ontario Ministry of Environment, Queen's Printer for Ontario. Lowest Effet Levels (LELs).
OME sed SELs: Persaud, D., Jaagumagi, R. and Hayton, A. Aug.,1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Ontario Ministry of Environment. Severe Effect Levels (SELs). Converted to mg/kg assuming 1% TOC.
ORNL secondary chronic: Jones, D.S., Suter, G.W. II, and Hull, R.N. 1997. Toxicological benchmarks for screening contaminants of potential concern for effects on sediment-associated biota: 1997 Revision. ES/ER/TM-95/R4. Oak Ridge National Laboratory, Oak Ridge, TN.
ORNL Plant: Efroymson, R.A., M.E. Will, G.W. Suter II, A.C. Wooten. 1997. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision. ES/ER/TM-85/R3. ORNL, Oak Ridge, TN.

TABLE 7. Sediment Concentrations Versus Screening Benchmarks, Inorganic COPCs
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Analyte	Maximum measured concentration (mg/kg)	Location	NYSDEC LEL (mg/kg)	Hazard Quotient	NYSDEC SEL (mg/kg)	Hazard Quotient	NOAA ER-L (mg/kg)	Hazard Quotient	OME sed LELs (mg/kg)	Hazard Quotient	OME sed SELs (mg/kg)	Hazard Quotient	EPA TEC (mg/kg)	Hazard Quotient	ORNL Plant (mg/kg)	Hazard Quotient	EcoSSL for Plants (mg/kg)	Hazard Quotient
Antimony	3	R2-2	2	1.5	25	0.1		NB		NB		NB		NB	5	0.6		NB
Arsenic	19	R2-2	6	3.2	33	0.6		NB	6	3.2	33	0.6	12.1	1.6	10	1.9	18	0.33
Arsenic III	19	R2-2		NB		NB	8.2	2.3		NB		NB		NB	NB	NB		NB
Cadmium	107	R2-12	0.6	178.3	9	11.9	1.2	89.2	0.6	178.3	10	10.7	0.592	180.7	4	26.75	32	0.02
Chromium	1090	R3-9	26	41.9	110	9.9		NB	26	41.9	110	9.9	56	19.5	1	1090		NB
Chromium III	1090	R3-9		NB		NB	81	13.5		NB		NB		NB	NB	NB		NB
Copper	433	R2-2	16	27.1	110	3.9	34	12.7	16	27.1	110	3.9	28	15.5	100	4.33	70	0.23
Iron	26600	R2-3	20000	1.3	40000	0.7		NB	20000	1.3	40000	0.7		NB	NB	NB		NB
Lead	284	R2-12	31	9.2	110	2.6	47	6.0	31	9.2	250	1.1	34.2	8.3	50	5.68	120	0.26
Manganese	886	R2-12	460	1.9	1100	0.8		NB	460	1.9	1100	0.8	1673	0.5	500	1.77	220	2.09
Mercury	1.3	R2-4	0.15	8.7	1.3	1.0		NB	0.2	6.5	2	0.7		NB	0.3	4.33		NB
Nickel	447	R3-9	16	27.9	50	8.9	21	21.3	16	27.9	75	6.0	39.6	11.3	30	14.9	38	0.42
Silver	18	R2-2	1	18.0	2.2	8.2		NB		NB		NB		NB	2	9	560	0.00
Zinc	1640	R2-2	120	13.7	270	6.1	150	10.9	120	13.7	820	2.0	159	10.3	50	32.8	160	0.75

NOAA ER-Ls: U.S. EPA. OSWER (Office of Solid Waste and Emergency Response). 1996. Eco Update: Ecotox thresholds. Washington, D.C. EPA 540/F-95/038.
EPA TEC: U.S. EPA. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod Hyalella azteca and the midge Chironomus riparius. EPA 905-R96-008, Great Lakes National Program Office, Chicago, IL.
NYSDEC LELs: NY DEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments. Metals - LEL's.
NYSDEC SELs: NY DEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments. Metals - SEL's.
OME sed LELs: Persaud, D., Jaagumagi, R. and Hayton, A. Aug.,1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Ontario Ministry of Environment, Queen's Printer for Ontario. Lowest Effet Levels (LELs).
OME sed SELs: Persaud, D., Jaagumagi, R. and Hayton, A. Aug.,1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Ontario Ministry of Environment. Severe Effect Levels (SELs). Converted to mg/kg assuming 1% TOC.
ORNL Plant: Efroymson, R.A., M.E. Will, G.W. Suter II, A.C. Wooten. 1997. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision. ES/ER/TM-85/R3. ORNL, Oak Ridge, TN.

Arsenic III and Chromium III not measured in sediment samples. The screening benchmark is for As III and Cr III; analytical results for total As and total Cr were used for this screen.

Location is the sampling location where the maximum concentration was measured.

Shading indicates Hazard Quotient greater than 1.0

TABLE 8 (cont). Maximum Concentration of Analytes Measured in Surface Water, No Screening Benchmark Available
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured (ug/L)
	1,1,2-Trichloro-1,2,2-trifluoroethane	ND (5)
	1,2,3-Trichlorobenzene	ND (5)
	1,2,4,5-Tetrachlorobenzene	ND (5)
	1,2-Dibromo-3-chloropropane	ND (5)
	1,2-Dibromoethane	ND (5)
	1,2-Dichloropropane	ND (5)
	2,2'-Oxybis(1-chloropropane)	ND (5)
R3-7	2,3,3-Trimethyl-1-hexene	17
R1-4	2,3,4,6-Tetrachlorophenol	0.18
R2-11	2,4,5-Trichlorophenol	0.23
	2,4,6-Trichlorophenol	ND (5)
	2,4-Dichlorophenol	ND (5)
R1-4	2,4-Dimethylphenol	0.35
	2,4-Dinitrophenol	ND (10)
	2,4-Dinitrotoluene	ND (5)
	2,6-Dinitrotoluene	ND (5)
	2-Chloronaphthalene	ND (5)
	2-Chlorophenol	ND (5)
	2-Methylphenol	ND (5)
	2-Nitroaniline	ND (10)
	2-Nitrophenol	ND (5)
	3,3'-Dichlorobenzidine	ND (5)
	3-Nitroaniline	ND (10)
	4,6-Dinitro-2-methylphenol	ND (10)
R2-11	4-Chloro-3-methylphenol	0.19
	4-Chloroaniline	ND (5)
R2-11	4-Chlorophenyl-phenylether	0.28
R1-4	4-Methylphenol	0.16
	4-Nitroaniline	ND (10)
	Acenaphthylene	ND (5)
	Acetophenone	ND (5)
	Atrazine	ND (5)
	Benzaldehyde	ND (5)
R3-2	Benzo(b)fluoranthene	0.16
R1-4	Benzo(g,h,i)perylene	5
	Benzo(k)fluoranthene	ND (5)
	Bis(2-chloroethoxy)methane	ND (5)
	Bis(2-chloroethyl)ether	ND (5)
	Bromochloromethane	ND (5)
	Bromodichloromethane	ND (5)
	Bromomethane	ND (5)
	Caprolactam	ND (5)
	Carbazole	ND (5)

TABLE 8 (cont). Maximum Concentration of Analytes Measured in Surface Water, No Screening Benchmark Available
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured (ug/L)
R3-2	Carbonic acid, dipentyl ester	3.1
	Chloroethane	ND (5)
	Chloromethane	ND (5)
	Chrysene	ND (5)
R3-2	cis-1,2-Dichloroethene	4.2
	cis-1,3-Dichloropropene	ND (5)
	Cyclohexane	ND (5)
	Dibenzo(a,h)anthracene	ND (5)
	Dibromochloromethane	ND (5)
	Dichlorodifluoromethane	ND (5)
	Dimethylphthalate	ND (5)
	Di-n-octylphthalate	ND (5)
R3-5	Ethanone, 1-(2,2-dimethylcyclopentyl)-	2.6
	Hexachlorobenzene	ND (5)
	Indeno(1,2,3-cd)pyrene	ND (5)
R1-4	Indole	3.2
	Isophorone	ND (5)
	Methyl acetate	ND (5)
	Methyl tert-butyl ether	ND (5)
	Methylcyclohexane	ND (5)
	Nitrobenzene	ND (5)
	N-Nitroso-di-n-propylamine	ND (5)
	Phenol	ND (5)
	Styrene	ND (5)
R3-7	Total Alkanes	96
	trans-1,2-Dichloroethene	ND (5)
	trans-1,3-Dichloropropene	ND (5)
	Trichlorofluoromethane	ND (5)
R3-2	Vinyl chloride	1.1

ND indicates analyte not detected in any surface water sample, number in parentheses is the detection limit.

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
Inorganic Analytes (mg/kg)		
R2-4A	Aluminum	15100
R2-2A	Barium	210
R2-5A	Beryllium	0.96
R1-1A	Calcium	300000
R2-2A	Cobalt	11.2
R3-11A	Magnesium	15500
R2-2A	Potassium	1860
R2-5A	Selenium	4.5
R1-1A	Sodium	6290
R2-6A	Thallium	2
R2-5A	Vanadium	31.8
Organic Analytes (ug/kg)		
R1-2A	(4,6-Dichloro[1,3,5]triazin-2-yl)-phenyl-amine	140
R1-2A	.alpha.-Ketostearic acid	170
R2-7A	.beta.-Amyrin	840
R2-7A	.delta. Nonalactone	270
R2-10A	[1,1'-Bicyclohexyl]-4-carboxylic acid, 4'-propyl	270
R3-3A	[1,1'-Biphenyl]-2-carbonitrile	5500
R2-10A	[1,2'-Binaphthalene]-5,5',8,8'-tetrone, 1',4-dih	240
R1-2A	1-(1-Trimethylsiloxyethenyl)-4-trimethylsiloxy-b	130
R2-12A	1(4H)-Naphthalenone, 4a,5,8,8a-tetrahydro-4-hydr	6000
R2-6A	1,1,2-Trichloro-1,2,2-trifluoroethane	33
R3-5A	1,1'-Binaphthalene	1400
R3-11A	1,1'-Biphenyl, 2,2',3,4-tetrachloro-	1200
R3-9A	1,1'-Biphenyl, 2,3,3'-trichloro-	4000
R3-11A	1,1'-Biphenyl, 2,3',4,5,5'-pentachloro-	1100
R3-11A	1,1'-Biphenyl, 2,3',4',5-tetrachloro-	1000
R3-9A	1,1'-Biphenyl, 2,3,4',6-tetrachloro-	1500
R2-8A	1,1'-Biphenyl, 2',3,4-trichloro-	190
R3-11A	1,1'-Biphenyl, 2,3',5,5'-tetrachloro-	2800
R3-11A	1,1'-Biphenyl, 2,4,4'-trichloro-	2500
R3-9A	1,1'-Biphenyl, 2,4',5-trichloro-	5000
R3-11A	1,1'-Biphenyl, 3,3',4,4'-tetrachloro-	1700
R3-9A	1,1'-Biphenyl, 4,4'-dichloro-	14000
R3-9A	1,1'-Biphenyl, 4-chloro-	3700
R3-11A	1,1'-Biphenyl, trichloro-	5000
R2-6A	1,2,3-Trichlorobenzene	33
R3-3A	1,2,4,5-Tetrachlorobenzene	ND (11000)

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-3A	1,2-Benzenedicarboxylic acid, diheptyl ester	270
R2-6A	1,2-Dibromo-3-chloropropane	33
R2-6A	1,2-Dibromoethane	33
R2-6A	1,2-Dichloropropane	33
SP-4A	1,3-Benzenediol, 4-(6-methoxy-2-benzofuranyl)-	230
R3-9A	1,4-Benzenediol, 2-[(1,4,4a,5,6,7,8,8a-octahydro	2800
R3-7A	1,4-Dioxane	1200
R2-5A	1,4-Methanoazulen-7-ol, decahydro-4,8,8,9-tetram	360
R1-2A	10-Bromo-1,2,3,4-tetrahydro-phenanthren-9-ol	140
R2-5A	10-Nonadecanone	220
R2-1A	11H-Benzo[a]carbazole	640
R3-4A	11H-Benzo[a]fluorene	2100
R3-3A	11H-Benzo[b]fluorene	5800
R3-2A	11H-Indeno[2,1-a]phenanthrene	730
R3-5A	13H-Dibenzo[a,h]fluorene	1600
R3-6A	13H-Dibenzo[a,i]carbazole	1400
R2-2A	1-Cyclohexanol-2-(3-methyl-1,3-butadienyl)-1,3,3	240
R3-9A	1-Cyclohexene, 1,3,3-trimethyl-2-(1-methylbut-1-	1800
R3-11A	1-Cyclohexene-1-glyoxylamide, N-1-naphthyl-	990
R2-7A	1-Docosene	1500
R2-7A	1-Dotriacontanol	2300
R2-7A	1-Eicosanol	2900
R2-12A	1H-Cyclopropa[l]phenanthrene,1a,9b-dihydro-	12000
R2-12A	1-Hexadecanol, 3,7,11,15-tetramethyl-	6100
R2-4A	1-Hexanol, 2-ethyl-	1600
R2-12A	1H-Indene, 2,3-dihydro-1,2-dimethyl-	11
R3-9A	1H-Indene, 2,3-dihydro-5,6-dimethyl-	18
R2-9A	1H-Indene, 2-butyl-5-hexyloctahydro-	240
R2-14A	1H-Indene, octahydro-2,2,4,4,7,7-hexamethyl-, tr	14
R2-8A	1H-Phenanthro[9,10-c]pyrazole	170
R2-10A	1-Hydroxy-p-menth-3-one	430
R2-4A	1-Mercapto-2-heptadecanone	490
R3-11A	1-Methyldecahydronaphthalene	89
R2-2A	1-Naphthalenol, decahydro-4a-methyl-	270
R2-5A	1-Octadecanol	1500
R2-2A	1-Octadecene	340
R1-1A	1-Octanol	300
R2-10A	1-Pentacosanol	390
R2-11A	1-Pentadecene	170

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R3-2A	1-Phosphacyclopent-2-ene, 1,5-diphenyl-2,3-dimet	720
R2-14A	1R-.alpha.-Pinene	38
R2-12A	2(1H)-Naphthalenone, octahydro-1-methyl-, (1.alp	12
R2-12A	2(1H)-Naphthalenone, octahydro-4a,5-dimethyl-, (	11
R2-12A	2(1H)-Naphthalenone, octahydro-4a,7,7-trimethyl-	11
R3-7A	2-(7-Methoxymethylphenanthren-3-yl)propan-2-ol	1700
R3-3A	2,2'-Oxybis(1-chloropropane)	ND (11000)
R3-3A	2,3,4,6-Tetrachlorophenol	ND (11000)
R3-11A	2,4,5,5,8a-Pentamethyl-6,7,8,8a-tetrahydro-5H-ch	1500
R3-3A	2,4,5-Trichlorophenol	ND (11000)
R2-9A	2,4,6,8-Tetrathiatricyclo[3.3.1.13,7]decane, 1,3	210
R3-3A	2,4,6-Trichlorophenol	ND (11000)
R3-3A	2,4-Dichlorophenol	ND (11000)
R2-12A	2,4-Dimethylphenol	150
R3-3A	2,4-Dinitrophenol	ND (21000)
R3-3A	2,4-Dinitrotoluene	ND (11000)
R2-9A	2,6,10,14,18-Pentamethyl-2,6,10,14,18-eicosapent	350
R3-3A	2,6-Dinitrotoluene	ND (11000)
R2-10A	2-[p-Cyanophenyl]-5-chlorobenzimidazole	200
R2-8A	2-Benzylidenehydrazono-3-methyl-2,3-dihydrobenzo	170
R2-6A	2-Butanone	130
R3-7A	2-Butyne	26
R3-3A	2-Chloronaphthalene	ND (11000)
R3-3A	2-Chlorophenol	ND (11000)
R2-10A	2-Cyclohexen-1-one, 4-(3-hydroxy-1-butenyl)-3,5,	480
R2-12A	2-Dodecen-1-yl(-)succinic anhydride	6700
R2-8A	2-Heptanone, 4-methyl-	290
R2-16A	2-Methyl-3-phenylpyridine	1400
R3-3A	2-Methylphenol	ND (11000)
R2-12A	2-Naphthalenol, 6,7-epoxy-5,6,7,8-tetrahydro-5-i	6000
R3-3A	2-Nitroaniline	ND (21000)
R3-3A	2-Nitrophenol	ND (11000)
R2-14A	2-Oxo-6-phenyl-4-(2-hydroxyphenyl)-1,2-dihydropy	750
R2-12A	2-Phenyl-3-amino-4,7-dimethyl-4,5(3H)dihydroimid	3900
R2-8A	2-Phenylnaphthalene	350
R2-16A	2-Phenylnaphthalene	1500
R2-9A	2-Undecanone, 6,10-dimethyl-	170
R3-9A	3-(3,4-Dichlorophenyl)-1-(5-methyl-2-thiazolin-2	1600
R3-3A	3,3'-Dichlorobenzidine	ND (11000)

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R3-1A	3,4-Biphenyldicarbonitrile	1100
R3-3A	3,4-Dihydrocyclopenta(cd)pyrene (acepyrene)	6800
R3-2A	3,4-Epoxy-4a-ethyl-2,3,4,4a,5,6-hexahydro-1H-pyr	700
R3-7A	3-Buten-2-one, 3-methyl-4-(1,3,3-trimethyl-7-oxa	1100
R3-7A	3-Chloro-11H-pyrido[3',2'-4,5]pyrrolo[3,2-c]quin	990
R3-3A	3-Methylcholanthrene	5200
R3-3A	3-Nitroaniline	ND (21000)
R2-14A	3-Undecene, 9-methyl-, (Z)-	1300
R2-11A	4,4,6a,6b,8a,11,11,14b-Octamethyl-1,4,4a,5,6,6a,	330
R2-2A	4,4'-Dichlorodiphenylsulphide	600
R3-2A	4,6'-Biazulenyl	1300
R3-3A	4,6-Dinitro-2-methylphenol	ND (21000)
R2-1A	4,8a-Dimethyl-6-(2-methyl-oxiran-2-yl)-4a,5,6,7,	590
R3-11A	4,8-Dimethyl-4Z,8E-tetracosadienal	1300
R2-16A	4-Amino-2-chloro-6,7-dimethoxyquinazoline	530
R3-3A	4-Bromophenyl-phenylether	ND (11000)
R2-7A	4-Chloro-3-methylphenol	30
R3-3A	4-Chloroaniline	ND (11000)
R3-3A	4-Chlorophenyl-phenylether	ND (11000)
R3-3A	4H-Cyclopenta[def]phenanthrene	24000
R3-7A	4H-Dibenzo[de,g]quinoline, 5,6,6a,7-tetrahydro-1	1100
R1-4A	4-Methyl-dodec-3-en-1-ol	130
R2-2A	4-Methylphenol	520
R2-1A	4-Methylpyrrolo[1,2-a]pyrazine	580
R3-3A	4-Nitroaniline	ND (21000)
R3-3A	4-Nitrophenol	ND (21000)
R3-1A	4-Nonylphenol	2500
R2-7A	4-Pyridinamine, N,N,2,6-tetramethyl-	460
R2-7A	5-(1-Naphthyl)tricyclo[4.1.0.0]hept-3-ene	340
R3-11A	5,6-Dihydrochrysene	1300
R3-11A	5,6-Dimethyl-4-phenyl-3-cyanopyridine-2(1H)-thio	1200
R2-7A	5-Chloroquinoxaline	1400
R2-11A	5H-3,5a-Epoxynaphth[2,1-c]oxepin, dodecahydro-3,	300
R2-12A	5-Methyl-2,6-diphenyl-5,6-dihydro-4H-1,3-oxazine	6800
R2-10A	6H-Dibenzo[b,d]pyran-6-one, 3,7,9-trihydroxy-1-m	200
R3-11A	6-Tridecene, 7-methyl-	64
R3-9A	7H-Benz[de]anthracen-7-one	1800
R3-9A	7H-Benzo[c]carbazole	1200
R3-4A	7H-Benzo[c]fluorene	2500

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-12A	7-Ketooctanoic acid oxime, bis(trimethylsilyl)	4500
R3-3A	9,10-Anthracenedione	24000
R2-10A	9,10-Anthracenedione, 1-hydroxy-2-(hydroxymethyl	160
R2-12A	9,10-di(Chloromethyl)anthracene	11000
R2-11A	9,10-Dimethylanthracene	170
R3-7A	9-Chloro-9-phenyl-9-silafluorene	790
R3-3A	9H-Fluoren-9-one	9200
R2-11A	9H-Fluorene, 1-methyl-	170
R3-3A	9H-Fluorene, 2-methyl-	6300
R1-2A	9H-Fluorene, 4-methyl-	140
R2-1A	9H-Fluorene-2-carbonitrile	580
R2-9A	9-Octadecenoic acid (Z)-, octadecyl ester	320
R3-3A	Acetophenone	ND (11000)
R2-16A	Androstan-17-one, 3-hydroxy-, (3.beta.,5.beta.)-	500
R2-11A	Anthracene, 1,4-dimethyl-	520
R2-13A	Anthracene, 1-methyl-	7800
R3-3A	Anthracene, 2-methyl-	13000
R2-10A	Anthracene, 9-dodecyltetradecahydro-	170
R2-16A	Anthracene, 9-phenyl-	570
R2-6A	Aroclor-1262	120
R2-6A	Aroclor-1268	120
R2-12A	Aromatic	15
R3-3A	Atrazine	ND (11000)
R2-12A	Azulene, 7-ethyl-1,4-dimethyl-	4800
R2-8A	Azuleno[4,5-b]furan-2(3H)-one, decahydro-8,9-dih	170
R2-2A	Baccharane	270
R3-11A	Benz[a]anthracene, 8-methyl-	920
R3-5A	Benz[j]aceanthrylene, 3-methyl-	1300
R3-3A	Benzaldehyde	ND (11000)
R2-2A	Benzaldehyde, 3,5-dichloro-2-hydroxy-	330
R2-2A	Benzenamine, 2-bromo-4,6-dinitro-	460
R2-12A	Benzenamine, 3-chloro-N-(2-pyridinylmethylene)-	5900
R3-7A	Benzene	50
R3-5A	Benzene, [1-(2,4-cyclopentadien-1-ylidene)ethyl]	660
R3-1A	Benzene, 1-(1,1-dimethylethyl)-4-methoxy-	1100
R3-7A	Benzene, 1,1'-(1,2-dimethyl-1,2-ethenediyl)bis[4	1800
R2-12A	Benzene, 1,2,3,4-tetramethyl-	84
R2-12A	Benzene, 1,2,4,5-tetramethyl-	26
R3-9A	Benzene, 1-ethyl-4-(1-methylethyl)-	11

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-2A	Benzene, 1-fluoro-4-(2-phenylethenyl)-	250
R3-9A	Benzene, 1-methyl-2-(1-methylethyl)-	17
R2-12A	Benzene, 1-methyl-3-(1-methylethyl)-	55
R2-12A	Benzene, 1-methyl-4-(1-methylethyl)-	100
R2-12A	Benzene, 2,4-dimethyl-1-(1-methylethyl)-	14
R2-8A	Benzeneacetonitrile, 4-cyano-	140
R3-2A	Benzo[1,2-b:4,3-b']dithiophene, 1-phenyl-	800
R3-7A	Benzo[b]chrysene	1700
R3-3A	Benzo[b]naphtho[2,1-d]thiophene	4400
R2-12A	Benzo[b]naphtho[2,3-d]furan	4200
R3-2A	Benzo[b]naphtho[2,3-d]thiophene	790
R3-11A	Benzo[b]naphtho[2,3-d]thiophene, 6-methyl-	960
R3-3A	Benzo[b]triphenylene	4200
R3-2A	Benzo[c]phenanthrene	1500
R3-3A	Benzo[e]pyrene	19000
R3-11A	Benzo[j]fluoranthene	1700
R2-11A	Benzoic acid, 2,4-dimethoxy-6-methyl-, methyl es	170
R2-4A	Benzoic acid, 2,4-dimethyl-	170
R2-3A	Benzoic acid, 2-amino-4-nitro-	4200
R3-9A	Bicyclo Hydrocarbon	21
R3-3A	Bis(2-chloroethoxy)methane	ND (11000)
R3-3A	Bis(2-chloroethyl)ether	ND (11000)
R2-6A	Bromochloromethane	33
R2-6A	Bromodichloromethane	33
R2-6A	Bromoform	33
R2-6A	Bromomethane	33
R2-12A	C3 Benzene	120
R2-12A	C4 Benzene	120
R2-12A	C5 Benzene	45
R2-7A	Campesterol	1000
R2-14A	Camphene	32
R2-7A	Caprolactam	110
R3-3A	Carbazole	21000
R2-14A	Chlorinated aromatic	1000
R3-7A	Chloroethane	420
R2-9A	Chloromethane	8.8
R2-6A	Chloroxylenol	560
R3-3A	Chrysene	43000
R3-1A	Chrysene, 1-methyl-	1100

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-7A	cis-1,2-Dichloroethene	18
R2-6A	cis-1,3-Dichloropropene	33
R2-16A	Clorophene	470
R2-13A	Copaene	5800
R3-9A	Cyclohexane	1.1
R3-9A	Cyclohexanone, 5-methyl-2-(1-methylethylidene)-	30
R3-3A	Cyclopenta(def)phenanthrenone	5600
R3-6A	Cyclopenta[cd]pyrene	2100
R1-2A	Cyclopropanemethanol, 2-methyl-2-(4-methyl-3-pen	100
R3-7A	D:A-Friedooleanan-7-ol, (7.alpha.)-	910
R2-13A	D:C-Friedooleanan-3-one	17000
R3-11A	Decahydro-4,4,8,9,10-pentamethylnaphthalene	80
R2-9A	D-Homoestra-1,3,5(10),14-tetraen-17-one, 3-metho	220
R2-12A	Delta BHC	ND (6.4)
R3-6A	Dibenz(a,h)acridine	1200
R3-6A	Dibenzanthracene-5,6-quinone	2200
R3-4A	Dibenzo[def,mno]chrysene	4500
R3-3A	Dibenzofuran, 4-methyl-	6800
R3-3A	Dibenzothiophene	11000
R2-11A	Dibenzothiophene, 3-methyl-	320
R3-7A	Diboron(.mu.-selenium)diethylbis[.mu.-(1H-pyrazo	920
R2-6A	Dibromochloromethane	33
R2-6A	Dichlorodifluoromethane	33
R3-7A	Diethyl sulfide	22
R3-3A	Dimethylphthalate	ND (11000)
R2-8A	Dinaphtho[2,1-b:1',2'-d]furan	120
R3-3A	Di-n-butylphthalate	ND (11000)
R3-3A	Di-n-octylphthalate	ND (11000)
R2-2A	Endosulfan sulfate	0.74
R2-15A	Endrin aldehyde	250
R2-16A	Endrin ketone	5.9
R2-9A	Equilenin	150
R3-6A	Estra-1,3,5,7,9-pentaen-17-one, 3-methoxy-	1500
R2-6A	Ethanol, 2-(tetradecyloxy)-	1200
R2-12A	Famophos	4300
R1-2A	Farnesol isomer a	180
R2-9A	Fenthion	220
R3-1A	Fluoranthene, 2-methyl-	1800
R2-2A	Fluorenone oxime	230

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-6A	Friedelin	4200
R2-5A	Heptafluorobutyric acid, n-octadecyl ester	1200
R2-4A	Heptane, 5-ethyl-2-methyl-	630
R2-2A	Hexadecanal, 2-methyl-	470
R2-11A	Hexadecanoic acid, 2-hydroxy-, methyl ester	170
R2-9A	Hexadecanoic acid, 4-nitrophenyl ester	640
R2-7A	Hexadecenoic acid, Z-11-	430
R2-5A	Hexahydroindole	210
R3-6A	Indeno[1,2,3-cd]fluoranthene	3700
R3-3A	Isophorone	ND (11000)
R2-12A	Isopropylbenzene	3.1
R2-9A	Isoquinoline, 1,2,3,4-tetrahydro-7-methoxy-2-met	260
R2-6A	Ledol	630
R2-6A	Methyl acetate	33
R3-7A	Methyl tert-butyl ether	0.2
R2-12A	Methylcyclohexane	12
R3-9A	Naphthalene, 1,6,7-trimethyl-	1400
R3-5A	Naphthalene, 1,6-dimethyl-	730
R2-12A	Naphthalene, 1,6-dimethyl-4-(1-methylethyl)-	5400
R3-5A	Naphthalene, 1-methyl-	570
R2-16A	Naphthalene, 1-phenyl-	600
R3-11A	Naphthalene, 2,3,6-trimethyl-	1500
R3-5A	Naphthalene, 2,3-dimethyl-	570
R3-9A	Naphthalene, 2,7-dimethyl-	26
R3-7A	Naphthalene, 2-phenyl-	1700
R2-12A	Naphthalene, decahydro-, trans-	68
R3-9A	Naphthalene, decahydro-2,6-dimethyl-	26
R2-14A dup	Naphthalene, decahydro-2-methyl-	17
R2-7A	n-Hexadecanoic acid	530
R3-3A	Nitrobenzene	ND (11000)
R3-3A	N-Nitroso-di-n-propylamine	ND (11000)
R3-3A	N-Nitrosodiphenylamine	ND (11000)
SP-5A	Nonanoic acid	200
R2-7A	Octadecanal	1000
R2-11A	Octadecanoic acid, ethenyl ester	360
R2-5A	Octyl thioglycolate	1200
R2-7A	Olean-12-ene, 3-methoxy-, (3.beta.)-	1600
R3-1A	o-Terphenyl	1200
R3-11A	Oxalic acid, cyclohexylmethyl propyl ester	38

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R2-7A	Oxirane, heptadecyl-	420
R2-13A	Perylene	4900
R3-6A	Perylene, 3-methyl-	2500
R2-12A	Phenanthrene, 1-methyl-	6200
R3-3A	Phenanthrene, 2,3-dimethyl-	7000
R3-11A	Phenanthrene, 2,5-dimethyl-	1300
R3-3A	Phenanthrene, 2-methyl-	19000
R3-11A	Phenanthrene, 3,6-dimethyl-	1000
R2-13A	Phenanthrene, 4-methyl-	3500
R1-4A	Phenol, 2,2'-methylenebis[6-(1,1-dimethylethyl)-	320
R2-11A	Phenol, 2-methyl-4-(1,1,3,3-tetramethylbutyl)-	190
R3-1A	Phenol, 4-(1,1,3,3-tetramethylbutyl)-	2500
R3-1A	Phenol, nonyl-	1800
R2-7A	Phytol	450
R2-4A	Piperonal	200
R3-7A	PNA, MW = 278	860
R3-9A	Podocarp-12-en-14a-ol	1500
R2-12A	Propyphenazone	6100
R2-1A	p-Terphenyl	710
R3-5A	Pyrene, 1,3-dimethyl-	860
R2-13A	Pyrene, 1-methyl-	6300
R3-5A	Pyrene, 1-phenyl-	1000
R3-5A	Pyrene, 2-methyl-	930
R2-3A	Pyrene, 4,5,9,10-tetrahydro-	170
R2-11A dup	Pyrene, 4,5-dihydro-	480
R2-5A	Sesquirosefuran	7900
R2-10A	Silane, 1,2-ethanediylbis[triethenyl-	190
R2-12A	Silane, dimethyldi(2,4-cyclopentadien-1-yl)-	4100
R3-7A	Spiro[4.5]decan-7-one, 1,8-dimethyl-8,9-epoxy-4-	1300
R3-7A	Spiro[cyclopropane-1,4'(6'H)-[6,10]ethenonaphtho	1600
R2-7A	Squalene	850
R3-7A	Spiro[4.5]decan-7-one, 1,8-dimethyl-8,9-epoxy-4-	1300
R3-7A	Spiro[cyclopropane-1,4'(6'H)-[6,10]ethenonaphtho	1600
R2-7A	Squalene	850
R2-6A	Styrene	33
R2-11A	Sulfur	200
R2-6A	Tetrachloroethene	33
R3-7A	Thiophanate methyl	1000
R3-11A	threo-7,8-Bromochlorodisparlure	1400

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured
R3-9A	Total Alkanes	56000
R2-14A	trans,trans-1,8-Dimethylspiro[4.5]decane	15
R3-7A	trans-1,2-Dichloroethene	1.2
R2-6A	trans-1,3-Dichloropropene	33
R2-14A dup	trans-Decalin, 2-methyl-	24
R2-6A	Trichlorofluoromethane	1.6
R3-1A	Triphenylene	1600
R2-14A dup	Triphenylene, 2-methyl-	830
R1-1A	Valeric acid, 3,5-dihydroxy-2,4-dimethyl-, .delt	200
R1-2A	Vanadium, (.eta.5-2,4-cyclopentadien-1-yl)[(1,2,	120
R2-12A	Vinyl chloride	2.1
R2-12A	Z-8-Methyl-9-tetradecen-1-ol formate	3800
Dioxins/Furans (ng/kg)		
R2-12A	1,2,3,4,6,7,8-heptachlorodibenzo-*p*- dioxin	3900
R2-12A	1,2,3,4,6,7,8-heptachlorodibenzofuran	470
R2-12A	1,2,3,4,7,8,9-heptachlorodibenzofuran	36
R2-12A	1,2,3,4,7,8-hexachlorodibenzo-p-dioxin	98
R2-12A	1,2,3,4,7,8-hexachlorodibenzofuran	36
R2-12A	1,2,3,6,7,8-hexachlorodibenzo-p-dioxin	480
R2-14A dup	1,2,3,6,7,8-hexachlorodibenzofuran	17
R2-12A	1,2,3,7,8,9-hexachlorodibenzo-p-dioxin	600
R2-14A dup	1,2,3,7,8,9-hexachlorodibenzofuran	8.4
R2-12A	1,2,3,7,8-pentachlorodibenzo-p-dioxin	330
R2-12A	1,2,3,7,8-pentachlorodibenzofuran	23
R2-12A	2,3,4,6,7,8-hexachlorodibenzofuran	35
R2-12A	2,3,4,7,8-pentachlorodibenzofuran	35
R2-12A	2,3,7,8-tetrachlorodibenzo-p-dioxin	200
R2-12A	2,3,7,8-tetrachlorodibenzofuran	27
R2-12A	Octachlorinated dibenzo-*p*- dioxin	34000
R2-12A	Octachlorinated dibenzofuran	700
R2-12A	Total Heptachlorodibenzo-p-dioxi	18000
R2-12A	Total Heptachlorodibenzofuran	1600
R2-12A	Total Hexachlorodibenzo-p-dioxin	10000
R2-12A	Total Hexachlorodibenzofuran	1300
R2-12A	Total Pentachlorodibenzo-p-dioxin	2400
R2-12A	Total Pentachlorodibenzofuran	700
R2-12A	Total Tetrachlorodibenzo-p-dioxin	970
R2-12A	Total Tetrachlorodibenzofuran	740

TABLE 9 (cont.). Maximum Concentration of Analytes Measured in Sediment, No Screening Benchmark
Lower Ley Creek Site
Screening-Level Ecological Risk Assessment

Sampling Location where Measured	Analyte	Maximum Concentration Measured

ND indicates analyte not detected in any sediment sample, number in parentheses is the detection limit.

TABLE 10. Critical Tissue Concentrations
Fish Tissue
Lower Ley Creek Site, Syracuse, New York

Analyte	Fish (mg/kg wet weight tissue)	
	LOAEL	NOAEL
PCBs	1.44	0.91
Dioxins/furans	0.00005	0.000023

LOAEL = Lowest observed adverse effect level
NOAEL = No observed adverse effect level
PCB = Polychlorinated biphenyl

TABLE 11. Toxicity Reference Values
Avian and Mammalian Receptors
Lower Ley Creek Site, Syracuse, New York

Analyte	Mammals (mg/kgBW/day)		Birds (mg/kgBW/day	
	LOAEL	NOAEL	LOAEL	NOAEL
Inorganic Analytes				
Antimony	1.25	0.125	NA	NA
Arsenic	1.26	0.126	7.38	2.46
Barium	75	45	41.7	20.8
Cadmium	10	1	20	1.45
Chromium	13.14	3.28	5	1
Copper	15.14	11.7	61.7	47
Lead	80	8	11.8	1.18
Manganese	284	88	NA	977
Mercury (inorganic)	10	1	0.9	0.45
Nickel	80	40	107	77.4
Selenium	0.33	0.2	0.8	0.4
Thallium	0.74	0.074	NA	NA
Vanadium	2.1	0.21	114	11.4
Zinc	320	160	131	14.5
Organic Compounds				
Bis(2-ethylhexyl)phthalate	NA	NA	11	1.1
Chlordane	0.75	0.15	12.4	2.4
DDT and metabolites	4	0.8	0.028	0.0028
Dichlorobenzenes	NA	NA	60	6
Dieldrin	0.018	0.009	0.77	0.077
Endrin	0.92	0.092	0.1	0.01
Hexachlorobenzene	0.14	0.014	2.25	0.225
Hexachlorocyclohexanes	1	0.1	0.34	0.11
Methylmercury	0.025	0.0025	0.064	0.0064
Polycyclic aromatic hydrocarbons	10	1	1.43	0.143
Trichlorobenzenes	53.6	14.8	NA	NA
Xylenes	2.6	2.1	NA	NA
Dioxins/furans	0.00001	0.000001	0.00014	0.000014
Total PCBs	0.034	0.0034	1.8	0.18
Aroclor 1016	NA	0.44	NA	1.24
Aroclor 1221	NA	0.44	NA	1.3
Aroclor 1232	0.034	0.0034	0.62	0.31
Aroclor 1242	NA	0.44	12.2	1.22
Aroclor 1248	0.034	0.0034	1.8	0.18
Aroclor 1254	0.1	0.01	1.12	0.112
Aroclor 1260	0.034	0.0034	1.8	0.18

NA = Not available
mg/kgBW/day = milligrams per kilogram body weight per day
LOAEL = Lowest observed adverse effect level
NOAEL = No observed adverse effect level
PCB = Polychlorinated biphenyl
If information for an individual Aroclor was not available, the TRV for total PCBs was used for that Aroclor.

TABLE 12. Life History Exposure Parameters for Food Chain Model Receptor Species
Lower Ley Creek Site
Syracuse, New York

Receptor	Body Weight (kg)	Water Ingestion Rate (L/kgBW/day)	Food Ingestion Rate (kg/kgBW/day w.w.)	Sediment Ingestion Rate (kg/kBW/day w.w.)	Dietary Composition		
					Fish	Plant	Invertebrate
Belted Kingfisher	0.136	0.114	0.448	0.0045	100	0	0
Great Blue Heron	2.2	0.045	0.189	0.0019	100	0	0
River Otter	5.45	0.084	0.128	0.0013	100	0	0
Mink	0.6	0.104	0.198	0.0020	100	0	0

kg = kilogram
kg/kgBW/day w.w. = kilograms per kilogram body weight per day wet weight
L/kgBW/day = Liters per kilogram body weight per day

Table 13. Toxicity Test Results, *Hyalella azteca*
Lower Ley Creek Site
Syracuse, New York

Sample	Sample Location	Percent Survival	Growth (mg)
		Day 28	Day 28
Control		86	0.1537
TOX-01	R1-1	84	0.2325
TOX-02	R2-16	86	0.1165
TOX-06	R2-8	82	0.1654
TOX-08	R3-11	48 a	
TOX-09	R3-5	88	0.0821 a

a = Indicates a statistically significant reduction ($P < 0.05$) in the response relative to the corresponding response in the laboratory control.

mg = milligrams

Correlation analyses were run on log-transformed data to determine whether there was any relationship between the measured COPC concentrations and growth or survival. None of the analytes were significantly correlated with growth or survival at a significance level of 0.05.

Table 14. Toxicity Test Results, *Chironomus dilutus*
Lower Ley Creek Site
Syracuse, New York

Sample	Sample Location	Percent Survival	Growth (mg)
		Day 10	Day 10
Control		94	1.610
TOX-01	R1-1	92	1.117 a
TOX-02	R2-16	76	1.529
TOX-03	R2-13	92	0.951 a
TOX-04	R2-2	72	0.862 a
TOX-05	R2-12	70	1.128 a
TOX-06	R2-8	76	0.894 a
TOX-07	R3-9	94	1.546
TOX-08	R3-11	76	0.669 a
TOX-09	R3-5	88	1.499
TOX-10	R3-3	84	1.659

a = Indicates a statistically significant reduction ($P < 0.05$) in the response relative to the corresponding response in the laboratory control.

mg = milligrams

Correlation analyses were run on log-transformed data to determine whether there was any relationship between the measured COPC concentrations and growth or survival. None of the analytes were significantly correlated with growth or survival at a significance level of 0.05.

Table 15. Analytical Results, Sediment Samples Submitted for Toxicity Testing
Lower Ley Creek Site
Syracuse, New York

Analyte	TOX-01	TOX-02	TOX-03	TOX-04	TOX-05	TOX-06	TOX-07	TOX-08	TOX-09	TOX-10	Max Concentration November 2009	Max Concentration May 2010
1,1'-Biphenyl	4500 (U)	76	3000 (U)	620	620	2100 (U)	250	2100 (U)	440	440	3700	620
2,4-Dimethylphenol	4500 (U)	110	3000 (U)	4000 (U)	11000 (U)	4100 (U)	2100 (U)	2200 (U)	2100 (U)	7500 (U)	350	110
3,3'-Dichlorobenzidine	4500 (U)	2100 (U)	3000 (U)	4000 (U)	1000	4100 (U)	2100 (U)	2200 (U)	2100 (U)	7500 (U)	ND	1000
4-Methylphenol	4500 (U)	100	3000 (U)	4000 (U)	11000 (U)	4100 (U)	2100 (U)	120	2100 (U)	250	160	250
Acetophenone	4500 (U)	77	3000 (U)	4000	11000 (U)	4100 (U)	2100 (U)	2200 (U)	2100 (U)	7500 (U)	ND	4000
Benzaldehyde	230	74	150	4000 (U)	11000 (U)	150	83	140	79	7500 (U)	ND	230
Bis(2-ethylhexyl)phthalate	1000	220	600	380	1100	840	210	1200	600	790	4.9	1200
Carbazole	370	820	1200	150	9400	170	440	1800	580	6000	21000	9400
Dibenzofuran	140	650	570	4000 (U)	6000	4100 (U)	190	1500	370	4000	13	6000
Di-n-butylphthalate	4500 (U)	160	3000 (U)	4000 (U)	11000 (U)	4100 (U)	2100 (U)	2200 (U)	98	7500 (U)	ND	160
Di-n-octylphthalate	4500 (U)	2100 (U)	3000 (U)	4000 (U)	780	4100 (U)	2100 (U)	2200 (U)	2100 (U)	7500 (U)	ND	780
Total Alkanes	5900	6300	5000	3400	(U)	4300	(U)	4300	(U)	(U)	56000	6300
PAHs (µg/kg)												
2-Methylnaphthalene	4500 (U)	250	270	4000 (U)	2200	4100 (U)	92	720	180	1600	4600	2200
Acenaphthene	200	650	780	4000 (U)	6300	4100 (U)	270	2100	630	5000	3700	6300
Acenaphthylene	4500 (U)	2100 (U)	170	4000 (U)	11000 (U)	4100 (U)	110	1100	120	230	590	1100
Anthracene	470	890	1800	160	9300	200	740	4700	1100	7600	30000	9300
Benzo(a)anthracene	2300	2400	6600	1000	23000	1400	2700	11000	4200	18000	47000	23000
Benzo(a)pyrene	2700	2200	6200	1300	20000	1600	2700	8900	4100	17000	31000	20000
Benzo(b)fluoranthene	3300	2100	6700	1400	20000	1900	2300	7900	4100	15000	42000	20000
Benzo(g,h,i)perylene	2300	1300	4000	1100	13000	1400	1600	4500	2700	9800	17000	13000
Benzo(k)fluoranthene	2300	1900	6000	1000	19000	1400	2100	8700	3500	14000	17000	19000
Chrysene	3100	2500	7200	1400	26000	1900	2700	11000	4200	19000	43000	26000
Dibenzo(a,h)anthracene	540	440	1200	300	5400	390	570	1600	870	3000	4900	5400
Fluoranthene	6200	6800	18000	2700	75000	3700	6200	32000	10000	54000	130000	75000
Fluorene	210	720	760	4000 (U)	6800	4100 (U)	290	3400	530	4600	19000	6800
Indeno(1,2,3-cd)pyrene	1800	1200	3600	910	12000	1100	1400	4100	2300	8600	20000	12000
Naphthalene	170	290	440	4000 (U)	4600	4100 (U)	190	640	370	2800	8600	4600
Phenanthrene	2700	6800	11000	1100	75000	1300	3600	26000	6600	49000	160000	75000
Pyrene	5000	5500	14000	2200	54000	3100	5000	25000	8300	42000	100000	54000
HMWPAH	32440	28440	70400	13310	267400	19490	28970	117900	45250	199700	451900	267400
LMWPAH	12750	11700	15220	21260	115200	22000	5292	38660	9530	70830	249200	115200
Total PAH	45190	40140	92220	34570	382600	41490	34262	156560	54780	288530	671200	382600
Inorganic Compounds (mg/kg)												
Aluminum	6070	14000	4620	7870	5260	7520	2540	2890	2860	3690	15100	14000
Antimony	14.2	7.4	10.4 (U)	13.6	12.1	13.1	7.2	7.7	7.5 (U)	7.9 (U)	3	14.2
Arsenic	5.4	3.1	4.3	6.1	6.7	5.6	3	2.4	2.3	3	19	6.7
Barium	112	255	81.8	101	119	112	22.2	33.3	24.9 (U)	62.6	210	255
Cadmium	5.9	1.7	5.7	5.8	5.3	6.4	1.3	0.76	0.76	1	107	6.4
Calcium	75100	39500	32400	87600	46600	65500	70800	49800	85800	59000	300000	87600
Chromium	67.4	16.6	51.6	70.5	51.2	94.2	14.8	31.9	20.4	41.5	1090	94.2
Cobalt	4.9	3	4.8	5.6	5.1	5.8	3.1	2.7	3.4	4.6	11.2	5.8
Copper	83.8	R	57.1	94.1	66.1	105	R	R	102	77.8	433	105
Cyanide	3.5	0.22	0.79	0.65	1.1	0.52	3 (U)	0.72	3.2 (U)	0.49		3.5
Iron	17200	9950	10300	16800	15100	17000	8550	7560	7570	8980	26600	17200
Lead	82.7	15.5	45.2	64	86.6	87.8	15.9	30.8	32.9	84.3	284	87.8
Magnesium	11900	6040	5640	10400	9000	13000	12800	7810	15700	11400	15500	15700
Manganese	389	516	191	307	283	354	255	191	206	185	886	516
Mercury	0.21	0.065	0.088	0.19	0.17	0.29	0.1	0.071	0.12 (U)	0.14 (U)	1.3	0.29
Nickel	25.2	15.7	20.4	26.6	22.4	34.4	8.7	14.2	12.6	22.3	447	34.4
Potassium	1310	550	786	1440	1050	1460	563	621	645	788	1860	1460

Table 15. Analytical Results, Sediment Samples Submitted for Toxicity Testing
Lower Ley Creek Site
Syracuse, New York

Analyte	TOX-01	TOX-02	TOX-03	TOX-04	TOX-05	TOX-06	TOX-07	TOX-08	TOX-09	TOX-10	Max Concentration November 2009	Max Concentration May 2010
Selenium	8.3	4.3 (U)	6.1 (U)	1.1	7.1	7.6	4.2 (U)	4.5 (U)	4.4 (U)	4.6 (U)	4.5	8.3
Silver	0.94	1.2 (U)	0.45	1.3	0.7	2.1	0.14	0.33	0.075	0.24	18	2.1
Sodium	1180	8400	868 (U)	2230	1900	1090	598 (U)	641 (U)	622 (U)	661 (U)	6290	8400
Thallium	5.9	3.1 (U)	4.3 (U)	5.6	5.1	5.5	3 (U)	3.2 (U)	3.1 (U)	3.3 (U)	2	5.9
Vanadium	18	4.8	11.1	19.7	14.6	19.8	6 (U)	7.7	6.8	8.4	31.8	19.8
Zinc	296	R	198	253	310	342	R	R	81.6	133	1640	342
Total Organic Carbon (mg/kg)	100,000	24,000	71,000	110,000	78,000	100,000	35000	43000	30000	42000		
pH	7.4	7.8	7.7	7.4	7.5	7.7	8.2	7.8	8.1	7.9		
Percent > 2 mm	0	19.6	0	25.4	0	0	13.8	1.2	5.8	1.9		
Percent Sand	46.1	74.7	79.3	27.4	47.3	29.9	83.5	88	90.3	84.9		
Percent Silt	56.1	2.6	20.6	42.2	53.5	73	0.5	8.7	3.2	13.2		
Percent Clay	0	3.2	0.1	5	0	0	2.1	2	0.7	0.1		
Significant difference from control in bioassay, *H. azteca*	No	No	NT	NT	NT	No	NT	Yes	Yes	NT		
Significant difference from control in bioassay, *C. dilutus*	Yes	No	Yes	Yes	Yes	Yes	No	Yes	No	No		

Only chemicals with detected concentrations in at least one sample are reported.
U = not detected at the indicated detection limit
NT = Not tested
R = Rejected
µg/kg = micrograms per kilogram
mg/kg = milligrams per kilogram

TABLE 16. Comparison of Fish Tissue CTCs with Maximum and Mean PCB and Dioxin Concentrations in Fish Tissue (mg/kg wet weight)
Concentrations in Forage Fish Collected for the Ecological Risk Assessment
Lower Ley Creek Site, Syracuse, New York

	Fish CTC (mg/kg w.w.)		Upstream Reach			Middle Reach			Downstream Reach		
	LOAEL	NOAEL	Concentration (mg/kg w.w.)	LOAEL HQ	NOAEL HQ	Concentration (mg/kg w.w.)	LOAEL HQ	NOAEL HQ	Concentration (mg/kg w.w.)	LOAEL HQ	NOAEL HQ
Whole Body Forage Fish											
Dioxins/furans	0.00005	0.000023	0.000033	0.66	1.43	0.000043	0.86	1.87	0.000048	0.96	2.09
Total PCBs, Maximum	1.44	0.91	0.4	0.28	0.44	0.1525	0.11	0.17	0.215	0.15	0.24
Total PCBs, Mean	1.44	0.91	0.3063	0.21	0.34	0.1125	0.08	0.12	0.125	0.09	0.14
Fillet Tissue from Larger Fish											
Dioxins/furans	0.00005	0.000023	0.0000708	1.42	3.08	NA	NA	NA	0.0000735	1.47	3.20
Total PCBs, Maximum	1.44	0.91	0.35	0.24	0.38	NA	NA	NA	0.7	0.49	0.77
Total PCBs, Mean	1.44	0.91	0.143	0.10	0.16	NA	NA	NA	0.245	0.17	0.27

LOAEL = Lowest Observed Adverse Effect Level
NOAEL = No Observed Adverse Effect Level
PCB = Polychlorinated biphenyl
CTC = Critical Tissue Concentration
NA = Not Available

mg/kg w.w. = milligrams per kilogram wet weight
HQ = Hazard Quotient
COPC = Contaminant of Potential Concern
Shading indicates calculated hazard quotient greater than 1.0

TABLE 17. Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Maximum Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.8	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	10.5	0	0.0000	0.002	0.000	0.0000	0.30	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	120	0	0.0000	0.105	0.000	0.0000	0.93	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	7	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	1090	0	0.0000	0.008	0.000	0.0000	0.60	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	405	0	0.0000	0.007	0.000	0.0000	1.35	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	273	0	0.0000	0.007	0.000	0.0000	0.35	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	261	0	0.0000	0.120	0.000	0.0000	4.33	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.27	0	0.0000	0.000	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	447	0	0.0000	0.006	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.78	0	0.0000	0.003	0.000	0.0000	0.80	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.42	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	15.3	0	0.0000	0.002	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	436	0	0.0000	0.018	0.000	0.0000	29.88	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	1.1	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.0274	0	0.0000	NA	0.000	NA	0.0018	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.123	0	0.0000	NA	0.000	NA	0.0013	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.014	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.00068	0	0.0000	NA	0.000	NA	0.0036	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.022	0	0.0000	NA	0.000	NA	0.0035	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	1.1	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.2943	0	0.0000	NA	0.000	NA	0.0005	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.27	0	0.0000	0.000	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	671.2	0	0.0000	0.008	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0032	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0303	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.000357	0	0.0000	NA	0.000	NA	0.000033	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	19.046	0	0.0000	NA	0.000	NA	0.3500	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	19	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.0069	0	0.0000	NA	0.000	NA	0.3000	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.0069	0	0.0000	NA	0.000	NA	0.1125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 18. Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Maximum Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	4	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	19	0	0.0000	0.001	0.000	0.0000	0.20	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	210	0	0.0000	0.108	0.000	0.0000	1.18	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	107	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	247	0	0.0000	0.003	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	433	0	0.0000	0.004	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	284	0	0.0000	0.010	0.000	0.0000	0.65	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	886	0	0.0000	0.102	0.000	0.0000	6.88	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	1.3	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	52.4	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	4.5	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	2	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	31.8	0	0.0000	0.005	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	1640	0	0.0000	0.009	0.000	0.0000	24.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	4.9	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.037	0	0.0000	NA	0.000	NA	0.0005	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.049	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.099	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.11	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.028	0	0.0000	NA	0.000	NA	0.0017	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.84	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.026	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	1.3	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	331.64	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.066	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.066	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.00171	0	0.0000	NA	0.000	NA	0.000043	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	18.033	0	0.0000	NA	0.000	NA	0.1125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	2.7	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.12	0	0.0000	NA	0.000	NA	0.0850	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	18	0	0.0000	NA	0.000	NA	0.0364	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0
Only one fish sample (two field duplicates; results from the two duplicate samples were averaged to obtain a fish tissue concentration) was collected in the middle reach. Maximum sediment concentrations and the only available fish tissue concentration are used in this model.

TABLE 19. Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Maximum Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.4	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	2.3	0	0.0000	0.001	0.000	0.0000	0.23	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	118	0	0.0000	0.113	0.000	0.0000	1.29	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	1.3	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	19.8	0	0.0000	0.001	0.000	0.0000	0.39	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	27	0	0.0000	0.003	0.000	0.0000	0.30	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	63.7	0	0.0000	0.001	0.000	0.0000	0.25	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	258	0	0.0000	0.108	0.000	0.0000	3.65	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.061	0	0.0000	0.000	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	8.7	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.47	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.7	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	4.1	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	56.6	0	0.0000	0.006	0.000	0.0000	39.38	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.03	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.00058	0	0.0000	NA	0.000	NA	0.0023	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.0054	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.0033	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.0006	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.0006	0	0.0000	NA	0.000	NA	0.0023	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.03	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.0012	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.061	0	0.0000	0.000	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	9.843	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0022	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0022	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.0000072	0	0.0000	NA	0.000	NA	0.000048	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	0.268	0	0.0000	NA	0.000	NA	0.2150	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.23	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.0055	0	0.0000	NA	0.000	NA	0.1800	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.048	0	0.0000	NA	0.000	NA	0.0725	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 20. Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Mean Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.730	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	5.618	0	0.0000	0.002	0.000	0.0000	0.21	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	56.336	0	0.0000	0.102	0.000	0.0000	0.69	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	1.777	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	171.373	0	0.0000	0.004	0.000	0.0000	0.36	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	106.245	0	0.0000	0.006	0.000	0.0000	0.66	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	101.927	0	0.0000	0.004	0.000	0.0000	0.18	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	206.857	0	0.0000	0.110	0.000	0.0000	3.61	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.102	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	69.600	0	0.0000	0.005	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.462	0	0.0000	0.003	0.000	0.0000	0.73	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.319	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	10.214	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	181.055	0	0.0000	0.013	0.000	0.0000	28.71	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.297	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.007	0	0.0000	NA	0.000	NA	0.0016	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.033	0	0.0000	NA	0.000	NA	0.0013	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.004	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.001	0	0.0000	NA	0.000	NA	0.0036	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.008	0	0.0000	NA	0.000	NA	0.0030	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.258	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.043	0	0.0000	NA	0.000	NA	0.0003	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.102	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	164.314	0	0.0000	0.008	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.002	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.005	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.000206	0	0.000000	NA	0.000	NA	0.000033	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	4.645	0	0.0000	NA	0.000	NA	0.3125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	4.600	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.005	0	0.0000	NA	0.000	NA	0.2446	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.005	0	0.0000	NA	0.000	NA	0.1013	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 21 . Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Mean Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.55	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	8.46	0	0.0000	0.001	0.000	0.0000	0.20	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	105.07	0	0.0000	0.104	0.000	0.0000	1.18	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	14.83	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	63.64	0	0.0000	0.001	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	97.47	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	82.17	0	0.0000	0.002	0.000	0.0000	0.65	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	348.71	0	0.0000	0.095	0.000	0.0000	6.88	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.35	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	23.80	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	1.55	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.68	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	17.35	0	0.0000	0.003	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	352.69	0	0.0000	0.007	0.000	0.0000	24.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.48	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.00	0	0.0000	NA	0.000	NA	0.001	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.01	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.02	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.01	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.00	0	0.0000	NA	0.000	NA	0.002	0	0	R	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.19	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.01	0	0.0000	NA	0.000	NA	0.000	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.35	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	39.93	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.01	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.01	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.000561	0	0.0000	NA	0.000	NA	0.000043	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	1.74	0	0.0000	NA	0.000	NA	0.113	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.02	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.02	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.02	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.45	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.02	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.02	0	0.0000	NA	0.000	NA	0.085	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	1.09	0	0.0000	NA	0.000	NA	0.036	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 22. Hazard Quotient Calculations for a Piscivorous Bird (Belted Kingfisher)
Mean Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.875	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	1.4375	0	0.0000	0.001	0.000	0.0000	0.21	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	98.475	0	0.0000	0.113	0.000	0.0000	1.07	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	0.89	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	8.2	0	0.0000	0.001	0.000	0.0000	0.22	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	11.125	0	0.0000	0.003	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	21.2	0	0.0000	0.001	0.000	0.0000	0.18	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	229.5	0	0.0000	0.108	0.000	0.0000	3.63	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.034	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	7.675	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.4025	0	0.0000	0.003	0.000	0.0000	0.64	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.44125	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	3	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	25.75	0	0.0000	0.006	0.000	0.0000	32.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.026	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.00052	0	0.0000	NA	0.000	NA	0.0017	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.0025	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.0032	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.0005	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.0005	0	0.0000	NA	0.000	NA	0.0016	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.026	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.001	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.034	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	3.678	0	0.0000	0.012	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0021	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0021	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.0000072	0	0.0000	NA	0.000	NA	0.000048	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	0.102	0	0.0000	NA	0.000	NA	0.1475	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.061	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.005	0	0.0000	NA	0.000	NA	0.1156	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.016	0	0.0000	NA	0.000	NA	0.0506	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 23. Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Maximum Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.8	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	10.5	0	0.0000	0.002	0.000	0.0000	0.30	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	120	0	0.0000	0.105	0.000	0.0000	0.93	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	7	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	1090	0	0.0000	0.008	0.000	0.0000	0.60	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	405	0	0.0000	0.007	0.000	0.0000	1.35	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	273	0	0.0000	0.007	0.000	0.0000	0.35	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	261	0	0.0000	0.120	0.000	0.0000	4.33	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.27	0	0.0000	0.000	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	447	0	0.0000	0.006	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.78	0	0.0000	0.003	0.000	0.0000	0.80	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.42	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	15.3	0	0.0000	0.002	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	436	0	0.0000	0.018	0.000	0.0000	29.88	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	1.1	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.0274	0	0.0000	NA	0.000	NA	0.0018	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.123	0	0.0000	NA	0.000	NA	0.0013	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.014	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.00068	0	0.0000	NA	0.000	NA	0.0036	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.022	0	0.0000	NA	0.000	NA	0.0035	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	1.1	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.2943	0	0.0000	NA	0.000	NA	0.0005	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.27	0	0.0000	0.000	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	671.2	0	0.0000	0.008	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0032	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0303	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.000357	0	0.0000	NA	0.000	NA	0.000033	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	19.046	0	0.0000	NA	0.000	NA	0.3500	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	19	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.0069	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.0069	0	0.0000	NA	0.000	NA	0.3000	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.0069	0	0.0000	NA	0.000	NA	0.1125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 24 . Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Maximum Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	4	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	19	0	0.0000	0.001	0.000	0.0000	0.20	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	210	0	0.0000	0.108	0.000	0.0000	1.18	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	107	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	247	0	0.0000	0.003	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	433	0	0.0000	0.004	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	284	0	0.0000	0.010	0.000	0.0000	0.65	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	886	0	0.0000	0.102	0.000	0.0000	6.88	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	1.3	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	52.4	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	4.5	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	2	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	31.8	0	0.0000	0.005	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	1640	0	0.0000	0.009	0.000	0.0000	24.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	4.9	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.037	0	0.0000	NA	0.000	NA	0.0005	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.049	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.099	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.11	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.028	0	0.0000	NA	0.000	NA	0.0017	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.84	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.026	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	1.3	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	331.64	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.066	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.066	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.00171	0	0.0000	NA	0.000	NA	0.000043	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	18.033	0	0.0000	NA	0.000	NA	0.1125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	2.7	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.12	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.12	0	0.0000	NA	0.000	NA	0.0850	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	18	0	0.0000	NA	0.000	NA	0.0364	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 25 . Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Maximum Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.4	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	2.3	0	0.0000	0.001	0.000	0.0000	0.23	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	118	0	0.0000	0.113	0.000	0.0000	1.29	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	1.3	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	19.8	0	0.0000	0.001	0.000	0.0000	0.39	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	27	0	0.0000	0.003	0.000	0.0000	0.30	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	63.7	0	0.0000	0.001	0.000	0.0000	0.25	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	258	0	0.0000	0.108	0.000	0.0000	3.65	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.061	0	0.0000	0.000	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	8.7	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.47	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.7	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	4.1	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	56.6	0	0.0000	0.006	0.000	0.0000	39.38	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.03	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.00058	0	0.0000	NA	0.000	NA	0.0023	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.0054	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.0033	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.0006	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.0006	0	0.0000	NA	0.000	NA	0.0023	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.03	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.0012	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.061	0	0.0000	0.000	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	9.843	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0022	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0022	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.0000072	0	0.0000	NA	0.000	NA	0.000048	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	0.268	0	0.0000	NA	0.000	NA	0.2150	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.23	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.0055	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.0055	0	0.0000	NA	0.000	NA	0.1800	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.048	0	0.0000	NA	0.000	NA	0.0725	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 26. Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Mean Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.730	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	5.618	0	0.0000	0.002	0.000	0.0000	0.21	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	56.336	0	0.0000	0.102	0.000	0.0000	0.69	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	1.777	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	171.373	0	0.0000	0.004	0.000	0.0000	0.36	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	106.245	0	0.0000	0.006	0.000	0.0000	0.66	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	101.927	0	0.0000	0.004	0.000	0.0000	0.18	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	206.857	0	0.0000	0.110	0.000	0.0000	3.61	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.102	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	69.600	0	0.0000	0.005	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.462	0	0.0000	0.003	0.000	0.0000	0.73	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.319	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	10.214	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	181.055	0	0.0000	0.013	0.000	0.0000	28.71	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.297	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.007	0	0.0000	NA	0.000	NA	0.0016	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.033	0	0.0000	NA	0.000	NA	0.0013	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.004	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.001	0	0.0000	NA	0.000	NA	0.0036	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.008	0	0.0000	NA	0.000	NA	0.0030	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.258	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.043	0	0.0000	NA	0.000	NA	0.0003	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.102	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	164.314	0	0.0000	0.008	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.002	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.005	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.000	0	0.0000	NA	0.000	NA	0.000033	0	0	0.0000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	4.645	0	0.0000	NA	0.000	NA	0.3125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	4.600	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.005	0	0.0000	NA	0.000	NA	0.2446	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.005	0	0.0000	NA	0.000	NA	0.1013	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 27 . Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Mean Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.551	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NA	NA	NA	NA
Arsenic	8.465	0	0.0000	0.001	0.000	0.0000	0.20	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	105.068	0	0.0000	0.104	0.000	0.0000	1.18	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	14.831	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	63.638	0	0.0000	0.001	0.000	0.0000	0.05	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	97.471	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	82.171	0	0.0000	0.002	0.000	0.0000	0.65	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	348.706	0	0.0000	0.095	0.000	0.0000	6.88	0	0	0.0000	1	0.0000	NA	NA	977	0.00
Mercury (inorganic)	0.348	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	23.803	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	1.547	0	0.0000	0.003	0.000	0.0000	0.70	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.682	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NA	NA	NA	NA
Vanadium	17.350	0	0.0000	0.003	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	352.694	0	0.0000	0.007	0.000	0.0000	24.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.484	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.005	0	0.0000	NA	0.000	NA	0.0005	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.012	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.020	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.009	0	0.0000	NA	0.000	NA	R	0	0	R	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.005	0	0.0000	NA	0.000	NA	0.0017	0	0	R	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.191	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.005	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.348	0	0.0000	0.000	0.000	0.0000	0.03	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	39.929	0	0.0000	0.013	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.013	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NA	NA	NA	NA
Xylenes	0.014	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NA	NA	NA	NA
Dioxins/furans	0.000561	0	0.0000	NA	0.000	NA	0.000043	0	0	0.000000	1	0.0000	0.00014	0.00	0.000014	0.00
Total PCBs	1.744	0	0.0000	NA	0.000	NA	0.1125	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.024	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NA	NA	1.24	0.00
Aroclor 1221	0.024	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NA	NA	1.3	0.00
Aroclor 1232	0.024	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.452	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.024	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.024	0	0.0000	NA	0.000	NA	0.0850	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	1.093	0	0.0000	NA	0.000	NA	0.0364	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 28. Hazard Quotient Calculations for a Piscivorous Bird (Great Blue Heron)
Mean Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0			0.000			0	0		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.875	0	0.0000	0.005	0.000	0.0000	0.09	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Arsenic	1.4375	0	0.0000	0.001	0.000	0.0000	0.21	0	0	0.0000	1	0.0000	7.38	0.00	2.46	0.00
Barium	98.475	0	0.0000	0.113	0.000	0.0000	1.07	0	0	0.0000	1	0.0000	41.7	0.00	20.8	0.00
Cadmium	0.89	0	0.0000	0.003	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	20	0.00	1.45	0.00
Chromium	8.2	0	0.0000	0.001	0.000	0.0000	0.22	0	0	0.0000	1	0.0000	5	0.00	1	0.00
Copper	11.125	0	0.0000	0.003	0.000	0.0000	0.17	0	0	0.0000	1	0.0000	61.7	0.00	47	0.00
Lead	21.2	0	0.0000	0.001	0.000	0.0000	0.18	0	0	0.0000	1	0.0000	11.8	0.00	1.18	0.00
Manganese	229.5	0	0.0000	0.108	0.000	0.0000	3.63	0	0	0.0000	1	0.0000	NB	NB	977	0.00
Mercury (inorganic)	0.034	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.9	0.00	0.45	0.00
Nickel	7.675	0	0.0000	0.003	0.000	0.0000	0.06	0	0	0.0000	1	0.0000	107	0.00	77.4	0.00
Selenium	0.4025	0	0.0000	0.003	0.000	0.0000	0.64	0	0	0.0000	1	0.0000	0.8	0.00	0.4	0.00
Thallium	0.44125	0	0.0000	0.003	0.000	0.0000	0.19	0	0	0.0000	1	0.0000	NB	NB	NB	NB
Vanadium	3	0	0.0000	0.001	0.000	0.0000	0.10	0	0	0.0000	1	0.0000	114	0.00	11.4	0.00
Zinc	25.75	0	0.0000	0.006	0.000	0.0000	32.31	0	0	0.0000	1	0.0000	131	0.00	14.5	0.00
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.026	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	11	0.00	1.1	0.00
Chlordane	0.00052	0	0.0000	NA	0.000	NA	0.0017	0	0	0.0000	1	0.0000	12.4	0.00	2.4	0.00
DDT and metabolites	0.0025	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.028	0.00	0.0028	0.00
Dichlorobenzenes	0.0032	0	0.0000	0.002	0.000	0.0000	NA	0	0	NA	1	0.0000	60	0.00	6	0.00
Dieldrin	0.0005	0	0.0000	NA	0.000	NA	0.0020	0	0	0.0000	1	0.0000	0.77	0.00	0.077	0.00
Endrin	0.0005	0	0.0000	NA	0.000	NA	0.0016	0	0	0.0000	1	0.0000	0.1	0.00	0.01	0.00
Hexachlorobenzene	0.026	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	2.25	0.00	0.225	0.00
Hexachlorocyclohexanes	0.001	0	0.0000	NA	0.000	NA	0.0004	0	0	0.0000	1	0.0000	0.34	0.00	0.11	0.00
Methylmercury	0.034	0	0.0000	0.000	0.000	0.0000	0.04	0	0	0.0000	1	0.0000	0.064	0.00	0.0064	0.00
Polycyclic aromatic hydrocarb	3.678	0	0.0000	0.012	0.000	0.0000	NA	0	0	NA	1	0.0000	1.43	0.00	0.143	0.00
Trichlorobenzenes	0.0021	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Xylenes	0.0021	0	0.0000	0.001	0.000	0.0000	NA	0	0	NA	1	0.0000	NB	NB	NB	NB
Dioxins/furans	0.0000072	0	0.0000	NA	0.000	NA	0.000048	0	0	0.000000	1	0.000000	0.00014	0.00	0.000014	0.00
Total PCBs	0.102	0	0.0000	NA	0.000	NA	0.1475	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1016	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.24	0.00
Aroclor 1221	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	NB	NB	1.3	0.00
Aroclor 1232	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	0.62	0.00	0.31	0.00
Aroclor 1242	0.061	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	12.2	0.00	1.22	0.00
Aroclor 1248	0.005	0	0.0000	NA	0.000	NA	0.00083	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00
Aroclor 1254	0.005	0	0.0000	NA	0.000	NA	0.1156	0	0	0.0000	1	0.0000	1.12	0.00	0.112	0.00
Aroclor 1260	0.016	0	0.0000	NA	0.000	NA	0.0506	0	0	0.0000	1	0.0000	1.8	0.00	0.18	0.00

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 29 . Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Maximum Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.8	0.0013	0.0023	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0148	1.25	0.01	0.125	0.12
Arsenic	10.5	0.0013	0.0137	0.002	0.084	0.0002	0.30	100	0.128	0.0384	1	0.0522	1.26	0.04	0.126	0.41
Barium	120	0.0013	0.1560	0.105	0.084	0.0088	0.93	100	0.128	0.1184	1	0.2832	75	0.00	45	0.01
Cadmium	7	0.0013	0.0091	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.0145	10	0.00	1	0.01
Chromium	1090	0.0013	1.4170	0.008	0.084	0.0007	0.60	100	0.128	0.0768	1	1.4945	13.14	0.11	3.28	0.46
Copper	405	0.0013	0.5265	0.007	0.084	0.0006	1.35	100	0.128	0.1728	1	0.6999	15.14	0.05	11.7	0.06
Lead	273	0.0013	0.3549	0.007	0.084	0.0006	0.35	100	0.128	0.0448	1	0.4003	80	0.01	8	0.05
Manganese	261	0.0013	0.3393	0.120	0.084	0.0101	4.33	100	0.128	0.5536	1	0.9030	284	0.00	88	0.01
Mercury (inorganic)	0.27	0.0013	0.0004	0.000	0.084	0.0000	0.06	100	0.128	0.0077	1	0.0080	10	0.00	1	0.01
Nickel	447	0.0013	0.5811	0.006	0.084	0.0005	0.06	100	0.128	0.0078	1	0.5895	80	0.01	40	0.01
Selenium	0.78	0.0013	0.0010	0.003	0.084	0.0002	0.80	100	0.128	0.1024	1	0.1036	0.33	0.31	0.2	0.52
Thallium	0.42	0.0013	0.0005	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0248	0.74	0.03	0.074	0.33
Vanadium	15.3	0.0013	0.0199	0.002	0.084	0.0001	0.10	100	0.128	0.0128	1	0.0328	2.1	0.02	0.21	0.16
Zinc	436	0.0013	0.5668	0.018	0.084	0.0015	29.88	100	0.128	3.8240	1	4.3923	320	0.01	160	0.03
													0		0	
Organic Compounds													0		0	
Bis(2-ethylhexyl)phthalate	1.1	0.0013	0.0014	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0015	NB	NB	NB	NB
Chlordane	0.0274	0.0013	0.0000	NA	0.084	NA	0.0018	100	0.128	0.0002	1	0.0003	0.75	0.00	0.15	0.00
DDT and metabolites	0.123	0.0013	0.0002	NA	0.084	NA	0.0013	100	0.128	0.0002	1	0.0003	4	0.00	0.8	0.00
Dichlorobenzenes	0.014	0.0013	0.0000	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.00068	0.0013	0.0000	NA	0.084	NA	0.0036	100	0.128	0.0005	1	0.0005	0.018	0.03	0.009	0.05
Endrin	0.022	0.0013	0.0000	NA	0.084	NA	0.0035	100	0.128	0.0004	1	0.0005	0.92	0.00	0.092	0.01
Hexachlorobenzene	1.1	0.0013	0.0014	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0015	0.14	0.01	0.014	0.11
Hexachlorocyclohexanes	0.2943	0.0013	0.0004	NA	0.084	NA	0.0005	100	0.128	0.0001	1	0.0004	1	0.00	0.1	0.00
Methylmercury	0.27	0.0013	0.0004	0.000	0.084	0.0000	0.06	100	0.128	0.0077	1	0.0080	0.025	0.32	0.0025	3.22
Polycyclic aromatic hydrocarb	671.2	0.0013	0.8726	0.008	0.084	0.0007	NA	100	0.128	NA	1	0.8733	10	0.09	1	0.87
Trichlorobenzenes	0.0032	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0303	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.000357	0.0013	0.0000	NA	0.084	NA	0.000033	100	0.128	0.0000	1	0.0000	0.00001	0.47	0.000001	4.69
Total PCBs	19.046	0.0013	0.0248	NA	0.084	NA	0.3500	100	0.128	0.0448	1	0.0696	0.034	2.05	0.0034	20.46
Aroclor 1016	0.0069	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1221	0.0069	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1232	0.0069	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1242	19	0.0013	0.0247	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0248	NB	NB	0.44	0.06
Aroclor 1248	0.0069	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1254	0.0069	0.0013	0.0000	NA	0.084	NA	0.3000	100	0.128	0.0384	1	0.0384	0.1	0.38	0.01	3.84
Aroclor 1260	0.0069	0.0013	0.0000	NA	0.084	NA	0.1125	100	0.128	0.0144	1	0.0144	0.034	0.42	0.0034	4.24

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 30. Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Maximum Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	4	0.0013	0.0052	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0176	1.25	0.01	0.125	0.14
Arsenic	19	0.0013	0.0247	0.001	0.084	0.0001	0.20	100	0.128	0.0252	1	0.0500	1.26	0.04	0.126	0.40
Barium	210	0.0013	0.2730	0.108	0.084	0.0091	1.18	100	0.128	0.1504	1	0.4325	75	0.01	45	0.01
Cadmium	107	0.0013	0.1391	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.1445	10	0.01	1	0.14
Chromium	247	0.0013	0.3211	0.003	0.084	0.0002	0.05	100	0.128	0.0060	1	0.3273	13.14	0.02	3.28	0.10
Copper	433	0.0013	0.5629	0.004	0.084	0.0003	0.04	100	0.128	0.0055	1	0.5688	15.14	0.04	11.7	0.05
Lead	284	0.0013	0.3692	0.010	0.084	0.0008	0.65	100	0.128	0.0832	1	0.4532	80	0.01	8	0.06
Manganese	886	0.0013	1.1518	0.102	0.084	0.0086	6.88	100	0.128	0.8800	1	2.0404	284	0.01	88	0.02
Mercury (inorganic)	1.3	0.0013	0.0017	0.000	0.084	0.0000	0.03	100	0.128	0.0036	1	0.0053	10	0.00	1	0.01
Nickel	52.4	0.0013	0.0681	0.003	0.084	0.0003	0.06	100	0.128	0.0078	1	0.0762	80	0.00	40	0.00
Selenium	4.5	0.0013	0.0059	0.003	0.084	0.0002	0.70	100	0.128	0.0896	1	0.0957	0.33	0.29	0.2	0.48
Thallium	2	0.0013	0.0026	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0268	0.74	0.04	0.074	0.36
Vanadium	31.8	0.0013	0.0413	0.005	0.084	0.0004	0.17	100	0.128	0.0219	1	0.0637	2.1	0.03	0.21	0.30
Zinc	1640	0.0013	2.1320	0.009	0.084	0.0008	24.31	100	0.128	3.1120	1	5.2448	320	0.02	160	0.03
Organic Compounds																
Bis(2-ethylhexyl)phthalate	4.9	0.0013	0.0064	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0064	NB	NB	NB	NB
Chlordane	0.037	0.0013	0.0000	NA	0.084	NA	0.0005	100	0.128	0.0001	1	0.0001	0.75	0.00	0.15	0.00
DDT and metabolites	0.049	0.0013	0.0001	NA	0.084	NA	R	100	0.128	R	1	0.0001	4	0.00	0.8	0.00
Dichlorobenzenes	0.099	0.0013	0.0001	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0001	NB	NB	NB	NB
Dieldrin	0.11	0.0013	0.0001	NA	0.084	NA	R	100	0.128	R	1	0.0001	0.018	0.01	0.009	0.02
Endrin	0.028	0.0013	0.0000	NA	0.084	NA	0.0017	100	0.128	0.0002	1	0.0003	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.84	0.0013	0.0011	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0011	0.14	0.01	0.014	0.08
Hexachlorocyclohexanes	0.026	0.0013	0.0000	NA	0.084	NA	0.0004	100	0.128	0.0000	1	0.0001	1	0.00	0.1	0.00
Methylmercury	1.3	0.0013	0.0017	0.000	0.084	0.0000	0.03	100	0.128	0.0036	1	0.0053	0.025	0.21	0.0025	2.13
Polycyclic aromatic hydrocarb	331.64	0.0013	0.4311	0.013	0.084	0.0011	NA	100	0.128	NA	1	0.4322	10	0.04	1	0.43
Trichlorobenzenes	0.066	0.0013	0.0001	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0002	53.6	0.00	14.8	0.00
Xylenes	0.066	0.0013	0.0001	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0002	2.6	0.00	2.1	0.00
Dioxins/furans	0.00171	0.0013	0.0000	NA	0.084	NA	0.000043	100	0.128	0.0000	1	0.0000	0.00001	0.77	0.000001	7.73
Total PCBs	18.033	0.0013	0.0234	NA	0.084	NA	0.1125	100	0.128	0.0144	1	0.0378	0.034	1.11	0.0034	11.13
Aroclor 1016	0.12	0.0013	0.0002	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0003	NB	NB	0.44	0.00
Aroclor 1221	0.12	0.0013	0.0002	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0003	NB	NB	0.44	0.00
Aroclor 1232	0.12	0.0013	0.0002	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0003	0.034	0.01	0.0034	0.08
Aroclor 1242	2.7	0.0013	0.0035	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0036	NB	NB	0.44	0.01
Aroclor 1248	0.12	0.0013	0.0002	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0003	0.034	0.01	0.0034	0.08
Aroclor 1254	0.12	0.0013	0.0002	NA	0.084	NA	0.0850	100	0.128	0.0109	1	0.0110	0.1	0.11	0.01	1.10
Aroclor 1260	18	0.0013	0.0234	NA	0.084	NA	0.0364	100	0.128	0.0047	1	0.0281	0.034	0.83	0.0034	8.25

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 31. Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Maximum Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.4	0.0013	0.0018	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0142	1.25	0.01	0.125	0.11
Arsenic	2.3	0.0013	0.0030	0.001	0.084	0.0001	0.23	100	0.128	0.0296	1	0.0327	1.26	0.03	0.126	0.26
Barium	118	0.0013	0.1534	0.113	0.084	0.0095	1.29	100	0.128	0.1648	1	0.3277	75	0.00	45	0.01
Cadmium	1.3	0.0013	0.0017	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.0071	10	0.00	1	0.01
Chromium	19.8	0.0013	0.0257	0.001	0.084	0.0001	0.39	100	0.128	0.0494	1	0.0752	13.14	0.01	3.28	0.02
Copper	27	0.0013	0.0351	0.003	0.084	0.0002	0.30	100	0.128	0.0380	1	0.0733	15.14	0.00	11.7	0.01
Lead	63.7	0.0013	0.0828	0.001	0.084	0.0000	0.25	100	0.128	0.0317	1	0.1145	80	0.00	8	0.01
Manganese	258	0.0013	0.3354	0.108	0.084	0.0091	3.65	100	0.128	0.4672	1	0.8117	284	0.00	88	0.01
Mercury (inorganic)	0.061	0.0013	0.0001	0.000	0.084	0.0000	0.05	100	0.128	0.0068	1	0.0068	10	0.00	1	0.01
Nickel	8.7	0.0013	0.0113	0.003	0.084	0.0003	0.06	100	0.128	0.0078	1	0.0194	80	0.00	40	0.00
Selenium	0.47	0.0013	0.0006	0.003	0.084	0.0002	0.70	100	0.128	0.0896	1	0.0904	0.33	0.27	0.2	0.45
Thallium	0.7	0.0013	0.0009	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0251	0.74	0.03	0.074	0.34
Vanadium	4.1	0.0013	0.0053	0.001	0.084	0.0001	0.10	100	0.128	0.0128	1	0.0182	2.1	0.01	0.21	0.09
Zinc	56.6	0.0013	0.0736	0.006	0.084	0.0005	39.38	100	0.128	5.0400	1	5.1141	320	0.02	160	0.03
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.03	0.0013	0.0000	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0001	NB	NB	NB	NB
Chlordane	0.00058	0.0013	0.0000	NA	0.084	NA	0.0023	100	0.128	0.0003	1	0.0003	0.75	0.00	0.15	0.00
DDT and metabolites	0.0054	0.0013	0.0000	NA	0.084	NA	0.0020	100	0.128	0.0003	1	0.0003	4	0.00	0.8	0.00
Dichlorobenzenes	0.0033	0.0013	0.0000	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.0006	0.0013	0.0000	NA	0.084	NA	0.0020	100	0.128	0.0002	1	0.0003	0.018	0.01	0.009	0.03
Endrin	0.0006	0.0013	0.0000	NA	0.084	NA	0.0023	100	0.128	0.0003	1	0.0003	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.03	0.0013	0.0000	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0001	0.14	0.00	0.014	0.01
Hexachlorocyclohexanes	0.0012	0.0013	0.0000	NA	0.084	NA	0.0004	100	0.128	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.061	0.0013	0.0001	0.000	0.084	0.0000	0.05	100	0.128	0.0068	1	0.0068	0.025	0.27	0.0025	2.74
Polycyclic aromatic hydrocarb	9.843	0.0013	0.0128	0.013	0.084	0.0011	NA	100	0.128	NA	1	0.0139	10	0.00	1	0.01
Trichlorobenzenes	0.0022	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0022	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.0000072	0.0013	0.0000	NA	0.084	NA	0.000048	100	0.128	0.0000	1	0.0000	0.00001	0.62	0.000001	6.15
Total PCBs	0.268	0.0013	0.0003	NA	0.084	NA	0.2150	100	0.128	0.0275	1	0.0279	0.034	0.82	0.0034	8.20
Aroclor 1016	0.0055	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1221	0.0055	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1232	0.0055	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1242	0.23	0.0013	0.0003	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0004	NB	NB	0.44	0.00
Aroclor 1248	0.0055	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1254	0.0055	0.0013	0.0000	NA	0.084	NA	0.1800	100	0.128	0.0230	1	0.0230	0.1	0.23	0.01	2.30
Aroclor 1260	0.048	0.0013	0.0001	NA	0.084	NA	0.0725	100	0.128	0.0093	1	0.0093	0.034	0.27	0.0034	2.75

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 32. Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Mean Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.730	0.0013	0.0009	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0134	1.25	0.01	0.125	0.11
Arsenic	5.618	0.0013	0.0073	0.002	0.084	0.0001	0.21	100	0.128	0.0264	1	0.0338	1.26	0.03	0.126	0.27
Barium	56.336	0.0013	0.0732	0.102	0.084	0.0085	0.69	100	0.128	0.0878	1	0.1695	75	0.00	45	0.00
Cadmium	1.777	0.0013	0.0023	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.0077	10	0.00	1	0.01
Chromium	171.373	0.0013	0.2228	0.004	0.084	0.0004	0.36	100	0.128	0.0463	1	0.2694	13.14	0.02	3.28	0.08
Copper	106.245	0.0013	0.1381	0.006	0.084	0.0005	0.66	100	0.128	0.0840	1	0.2226	15.14	0.01	11.7	0.02
Lead	101.927	0.0013	0.1325	0.004	0.084	0.0003	0.18	100	0.128	0.0235	1	0.1563	80	0.00	8	0.02
Manganese	206.857	0.0013	0.2689	0.110	0.084	0.0092	3.61	100	0.128	0.4624	1	0.7406	284	0.00	88	0.01
Mercury (inorganic)	0.102	0.0013	0.0001	0.000	0.084	0.0000	0.04	100	0.128	0.0050	1	0.0051	10	0.00	1	0.01
Nickel	69.600	0.0013	0.0905	0.005	0.084	0.0004	0.06	100	0.128	0.0078	1	0.0987	80	0.00	40	0.00
Selenium	0.462	0.0013	0.0006	0.003	0.084	0.0002	0.73	100	0.128	0.0939	1	0.0947	0.33	0.29	0.2	0.47
Thallium	0.319	0.0013	0.0004	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0246	0.74	0.03	0.074	0.33
Vanadium	10.214	0.0013	0.0133	0.001	0.084	0.0001	0.10	100	0.128	0.0128	1	0.0262	2.1	0.01	0.21	0.12
Zinc	181.055	0.0013	0.2354	0.013	0.084	0.0011	28.71	100	0.128	3.6747	1	3.9112	320	0.01	160	0.02
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.297	0.0013	0.0004	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0004	NB	NB	NB	NB
Chlordane	0.007	0.0013	0.0000	NA	0.084	NA	0.0016	100	0.128	0.0002	1	0.0002	0.75	0.00	0.15	0.00
DDT and metabolites	0.033	0.0013	0.0000	NA	0.084	NA	0.0013	100	0.128	0.0002	1	0.0002	4	0.00	0.8	0.00
Dichlorobenzenes	0.004	0.0013	0.0000	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.001	0.0013	0.0000	NA	0.084	NA	0.0036	100	0.128	0.0005	1	0.0005	0.018	0.03	0.009	0.05
Endrin	0.008	0.0013	0.0000	NA	0.084	NA	0.0030	100	0.128	0.0004	1	0.0004	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.258	0.0013	0.0003	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0004	0.14	0.00	0.014	0.03
Hexachlorocyclohexanes	0.043	0.0013	0.0001	NA	0.084	NA	0.0003	100	0.128	0.0000	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.102	0.0013	0.0001	0.000	0.084	0.0000	0.04	100	0.128	0.0050	1	0.0051	0.025	0.20	0.0025	2.04
Polycyclic aromatic hydrocarb	164.314	0.0013	0.2136	0.008	0.084	0.0007	NA	100	0.128	NA	1	0.2143	10	0.02	1	0.21
Trichlorobenzenes	0.002	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.005	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.000206	0.0013	0.0000	NA	0.084	NA	0.000033	100	0.128	0.0000	1	0.000004	0.00001	0.45	0.000001	4.49
Total PCBs	4.645	0.0013	0.0060	NA	0.084	NA	0.3125	100	0.128	0.0400	1	0.0460	0.034	1.35	0.0034	13.54
Aroclor 1016	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1221	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1232	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1242	4.600	0.0013	0.0060	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0061	NB	NB	0.44	0.01
Aroclor 1248	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1254	0.005	0.0013	0.0000	NA	0.084	NA	0.2446	100	0.128	0.0313	1	0.0313	0.1	0.31	0.01	3.13
Aroclor 1260	0.005	0.0013	0.0000	NA	0.084	NA	0.1013	100	0.128	0.0130	1	0.0130	0.034	0.38	0.0034	3.81

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 33 . Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Mean Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.55	0.0013	0.0020	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0144	1.25	0.01	0.125	0.12
Arsenic	8.46	0.0013	0.0110	0.001	0.084	0.0001	0.20	100	0.128	0.0252	1	0.0363	1.26	0.03	0.126	0.29
Barium	105.07	0.0013	0.1366	0.104	0.084	0.0088	1.18	100	0.128	0.1504	1	0.2958	75	0.00	45	0.01
Cadmium	14.83	0.0013	0.0193	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.0247	10	0.00	1	0.02
Chromium	63.64	0.0013	0.0827	0.001	0.084	0.0001	0.05	100	0.128	0.0060	1	0.0888	13.14	0.01	3.28	0.03
Copper	97.47	0.0013	0.1267	0.003	0.084	0.0002	0.04	100	0.128	0.0055	1	0.1325	15.14	0.01	11.7	0.01
Lead	82.17	0.0013	0.1068	0.002	0.084	0.0002	0.65	100	0.128	0.0832	1	0.1902	80	0.00	8	0.02
Manganese	348.71	0.0013	0.4533	0.095	0.084	0.0080	6.88	100	0.128	0.8800	1	1.3413	284	0.00	88	0.02
Mercury (inorganic)	0.35	0.0013	0.0005	0.000	0.084	0.0000	0.03	100	0.128	0.0036	1	0.0041	10	0.00	1	0.00
Nickel	23.80	0.0013	0.0309	0.003	0.084	0.0002	0.06	100	0.128	0.0078	1	0.0390	80	0.00	40	0.00
Selenium	1.55	0.0013	0.0020	0.003	0.084	0.0002	0.70	100	0.128	0.0896	1	0.0918	0.33	0.28	0.2	0.46
Thallium	0.68	0.0013	0.0009	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0251	0.74	0.03	0.074	0.34
Vanadium	17.35	0.0013	0.0226	0.003	0.084	0.0002	0.17	100	0.128	0.0219	1	0.0447	2.1	0.02	0.21	0.21
Zinc	352.69	0.0013	0.4585	0.007	0.084	0.0006	24.31	100	0.128	3.1120	1	3.5711	320	0.01	160	0.02
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.48	0.0013	0.0006	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0007	NB	NB	NB	NB
Chlordane	0.00	0.0013	0.0000	NA	0.084	NA	0.0005	100	0.128	0.0001	1	0.0001	0.75	0.00	0.15	0.00
DDT and metabolites	0.01	0.0013	0.0000	NA	0.084	NA	R	100	0.128	R	1	0.0000	4	0.00	0.8	0.00
Dichlorobenzenes	0.02	0.0013	0.0000	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.01	0.0013	0.0000	NA	0.084	NA	R	100	0.128	R	1	0.0000	0.018	0.00	0.009	0.00
Endrin	0.00	0.0013	0.0000	NA	0.084	NA	0.0017	100	0.128	0.0002	1	0.0002	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.19	0.0013	0.0002	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0003	0.14	0.00	0.014	0.02
Hexachlorocyclohexanes	0.01	0.0013	0.0000	NA	0.084	NA	0.0004	100	0.128	0.0000	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.35	0.0013	0.0005	0.000	0.084	0.0000	0.03	100	0.128	0.0036	1	0.0041	0.025	0.16	0.0025	1.63
Polycyclic aromatic hydrocarb	39.93	0.0013	0.0519	0.013	0.084	0.0011	NA	100	0.128	NA	1	0.0530	10	0.01	1	0.05
Trichlorobenzenes	0.01	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.01	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.000561	0.0013	0.0000	NA	0.084	NA	0.000043	100	0.128	0.0000	1	0.0000	0.00001	0.62	0.000001	6.23
Total PCBs	1.74	0.0013	0.0023	NA	0.084	NA	0.1125	100	0.128	0.0144	1	0.0167	0.034	0.49	0.0034	4.90
Aroclor 1016	0.02	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1221	0.02	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1232	0.02	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.04
Aroclor 1242	0.45	0.0013	0.0006	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0007	NB	NB	0.44	0.00
Aroclor 1248	0.02	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.04
Aroclor 1254	0.02	0.0013	0.0000	NA	0.084	NA	0.0854	100	0.128	0.0109	1	0.0110	0.1	0.11	0.01	1.10
Aroclor 1260	1.09	0.0013	0.0014	NA	0.084	NA	0.0364	100	0.128	0.0047	1	0.0061	0.034	0.18	0.0034	1.79

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 34. Hazard Quotient Calculations for a Piscivorous Mammal (River Otter)
Mean Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.0013			0.084			100	0.128		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.875	0.0013	0.0011	0.005	0.084	0.0004	0.09	100	0.128	0.0120	1	0.0136	1.25	0.01	0.125	0.11
Arsenic	1.4375	0.0013	0.0019	0.001	0.084	0.0001	0.21	100	0.128	0.0274	1	0.0294	1.26	0.02	0.126	0.23
Barium	98.475	0.0013	0.1280	0.113	0.084	0.0095	1.07	100	0.128	0.1368	1	0.2743	75	0.00	45	0.01
Cadmium	0.89	0.0013	0.0012	0.003	0.084	0.0002	0.04	100	0.128	0.0052	1	0.0066	10	0.00	1	0.01
Chromium	8.2	0.0013	0.0107	0.001	0.084	0.0001	0.22	100	0.128	0.0277	1	0.0385	13.14	0.00	3.28	0.01
Copper	11.125	0.0013	0.0145	0.003	0.084	0.0002	0.17	100	0.128	0.0217	1	0.0364	15.14	0.00	11.7	0.00
Lead	21.2	0.0013	0.0276	0.001	0.084	0.0000	0.18	100	0.128	0.0234	1	0.0510	80	0.00	8	0.01
Manganese	229.5	0.0013	0.2984	0.108	0.084	0.0091	3.63	100	0.128	0.4640	1	0.7714	284	0.00	88	0.01
Mercury (inorganic)	0.034	0.0013	0.0000	0.000	0.084	0.0000	0.04	100	0.128	0.0052	1	0.0052	10	0.00	1	0.01
Nickel	7.675	0.0013	0.0100	0.003	0.084	0.0003	0.06	100	0.128	0.0078	1	0.0181	80	0.00	40	0.00
Selenium	0.4025	0.0013	0.0005	0.003	0.084	0.0002	0.64	100	0.128	0.0816	1	0.0823	0.33	0.25	0.2	0.41
Thallium	0.44125	0.0013	0.0006	0.003	0.084	0.0002	0.19	100	0.128	0.0240	1	0.0248	0.74	0.03	0.074	0.33
Vanadium	3	0.0013	0.0039	0.001	0.084	0.0001	0.10	100	0.128	0.0128	1	0.0168	2.1	0.01	0.21	0.08
Zinc	25.75	0.0013	0.0335	0.006	0.084	0.0005	32.31	100	0.128	4.1360	1	4.1700	320	0.01	160	0.03
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.026	0.0013	0.0000	0.0005	0.084	0.0000	NA	100	0.128	NA	1	0.0001	NB	NB	NB	NB
Chlordane	0.00052	0.0013	0.0000	NA	0.084	NA	0.0017	100	0.128	0.0002	1	0.0002	0.75	0.00	0.15	0.00
DDT and metabolites	0.0025	0.0013	0.0000	NA	0.084	NA	0.0020	100	0.128	0.0003	1	0.0003	4	0.00	0.8	0.00
Dichlorobenzenes	0.0032	0.0013	0.0000	0.002	0.084	0.0001	NA	100	0.128	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.0005	0.0013	0.0000	NA	0.084	NA	0.0020	100	0.128	0.0002	1	0.0003	0.018	0.01	0.009	0.03
Endrin	0.0005	0.0013	0.0000	NA	0.084	NA	0.0016	100	0.128	0.0002	1	0.0002	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.026	0.0013	0.0000	0.001	0.084	0.0000	NA	100	0.128	NA	1	0.0001	0.14	0.00	0.014	0.01
Hexachlorocyclohexanes	0.001	0.0013	0.0000	NA	0.084	NA	0.0004	100	0.128	0.0000	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.034	0.0013	0.0000	0.000	0.084	0.0000	0.04	100	0.128	0.0052	1	0.0052	0.025	0.21	0.0025	2.09
Polycyclic aromatic hydrocarb	3.678	0.0013	0.0048	0.012	0.084	0.0010	NA	100	0.128	NA	1	0.0058	10	0.00	1	0.01
Trichlorobenzenes	0.0021	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0021	0.0013	0.0000	0.001	0.084	0.0001	NA	100	0.128	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.0000072	0.0013	0.0000	NA	0.084	NA	0.000048	100	0.128	0.0000	1	0.0000	0.00001	0.62	0.000001	6.15
Total PCBs	0.102	0.0013	0.0001	NA	0.084	NA	0.1475	100	0.128	0.0189	1	0.0190	0.034	0.56	0.0034	5.59
Aroclor 1016	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1221	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	NB	NB	0.44	0.00
Aroclor 1232	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1242	0.061	0.0013	0.0001	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0002	NB	NB	0.44	0.00
Aroclor 1248	0.005	0.0013	0.0000	NA	0.084	NA	0.00083	100	0.128	0.0001	1	0.0001	0.034	0.00	0.0034	0.03
Aroclor 1254	0.005	0.0013	0.0000	NA	0.084	NA	0.1156	100	0.128	0.0148	1	0.0148	0.1	0.15	0.01	1.48
Aroclor 1260	0.016	0.0013	0.0000	NA	0.084	NA	0.0506	100	0.128	0.0065	1	0.0065	0.034	0.19	0.0034	1.91

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 35. Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Maximum Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.8	0.002	0.0036	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0227	1.25	0.02	0.125	0.18
Arsenic	10.5	0.002	0.0210	0.002	0.104	0.0002	0.30	100	0.198	0.0594	1	0.0806	1.26	0.06	0.126	0.64
Barium	120	0.002	0.2400	0.105	0.104	0.0109	0.93	100	0.198	0.1832	1	0.4341	75	0.01	45	0.01
Cadmium	7	0.002	0.0140	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.0223	10	0.00	1	0.02
Chromium	1090	0.002	2.1800	0.008	0.104	0.0009	0.60	100	0.198	0.1188	1	2.2997	13.14	0.18	3.28	0.70
Copper	405	0.002	0.8100	0.007	0.104	0.0007	1.35	100	0.198	0.2673	1	1.0780	15.14	0.07	11.7	0.09
Lead	273	0.002	0.5460	0.007	0.104	0.0007	0.35	100	0.198	0.0693	1	0.6160	80	0.01	8	0.08
Manganese	261	0.002	0.5220	0.120	0.104	0.0125	4.33	100	0.198	0.8564	1	1.3908	284	0.00	88	0.02
Mercury (inorganic)	0.27	0.002	0.0005	0.000	0.104	0.0000	0.06	100	0.198	0.0119	1	0.0124	10	0.00	1	0.01
Nickel	447	0.002	0.8940	0.006	0.104	0.0006	0.06	100	0.198	0.0121	1	0.9068	80	0.01	40	0.02
Selenium	0.78	0.002	0.0016	0.003	0.104	0.0003	0.80	100	0.198	0.1584	1	0.1602	0.33	0.49	0.2	0.80
Thallium	0.42	0.002	0.0008	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0382	0.74	0.05	0.074	0.52
Vanadium	15.3	0.002	0.0306	0.002	0.104	0.0002	0.10	100	0.198	0.0198	1	0.0506	2.1	0.02	0.21	0.24
Zinc	436	0.002	0.8720	0.018	0.104	0.0019	29.88	100	0.198	5.9153	1	6.7891	320	0.02	160	0.04
Organic Compounds																
Bis(2-ethylhexyl)phthalate	1.1	0.002	0.0022	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0023	NB	NB	NB	NB
Chlordane	0.0274	0.002	0.0001	NA	0.104	NA	0.0018	100	0.198	0.0004	1	0.0004	0.75	0.00	0.15	0.00
DDT and metabolites	0.123	0.002	0.0002	NA	0.104	NA	0.0013	100	0.198	0.0003	1	0.0005	4	0.00	0.8	0.00
Dichlorobenzenes	0.014	0.002	0.0000	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.00068	0.002	0.0000	NA	0.104	NA	0.0036	100	0.198	0.0007	1	0.0007	0.018	0.04	0.009	0.08
Endrin	0.022	0.002	0.0000	NA	0.104	NA	0.0035	100	0.198	0.0007	1	0.0007	0.92	0.00	0.092	0.01
Hexachlorobenzene	1.1	0.002	0.0022	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0023	0.14	0.02	0.014	0.16
Hexachlorocyclohexanes	0.2943	0.002	0.0006	NA	0.104	NA	0.0005	100	0.198	0.0001	1	0.0007	1	0.00	0.1	0.01
Methylmercury	0.27	0.002	0.0005	0.000	0.104	0.0000	0.06	100	0.198	0.0119	1	0.0124	0.025	0.50	0.0025	4.97
Polycyclic aromatic hydrocarb	671.2	0.002	1.3424	0.008	0.104	0.0009	NA	100	0.198	NA	1	1.3433	10	0.13	1	1.34
Trichlorobenzenes	0.0032	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0303	0.002	0.0001	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0002	2.6	0.00	2.1	0.00
Dioxins/furans	0.000357	0.002	0.0000	NA	0.104	NA	0.000033	100	0.198	0.0000	1	0.000007	0.00001	0.72	0.000001	7.25
Total PCBs	19.046	0.002	0.0381	NA	0.104	NA	0.3500	100	0.198	0.0693	1	0.1074	0.034	3.16	0.0034	31.59
Aroclor 1016	0.0069	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1221	0.0069	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1232	0.0069	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1242	19	0.002	0.0380	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0382	NB	NB	0.44	0.09
Aroclor 1248	0.0069	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1254	0.0069	0.002	0.0000	NA	0.104	NA	0.3000	100	0.198	0.0594	1	0.0594	0.1	0.59	0.01	5.94
Aroclor 1260	0.0069	0.002	0.0000	NA	0.104	NA	0.1125	100	0.198	0.0223	1	0.0223	0.034	0.66	0.0034	6.56

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 36. Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Maximum Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	4	0.002	0.0080	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0271	1.25	0.02	0.125	0.22
Arsenic	19	0.002	0.0380	0.001	0.104	0.0001	0.20	100	0.198	0.0390	1	0.0771	1.26	0.06	0.126	0.61
Barium	210	0.002	0.4200	0.108	0.104	0.0112	1.18	100	0.198	0.2327	1	0.6639	75	0.01	45	0.01
Cadmium	107	0.002	0.2140	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.2223	10	0.02	1	0.22
Chromium	247	0.002	0.4940	0.003	0.104	0.0003	0.05	100	0.198	0.0093	1	0.5036	13.14	0.04	3.28	0.15
Copper	433	0.002	0.8660	0.004	0.104	0.0004	0.04	100	0.198	0.0086	1	0.8750	15.14	0.06	11.7	0.07
Lead	284	0.002	0.5680	0.010	0.104	0.0010	0.65	100	0.198	0.1287	1	0.6977	80	0.01	8	0.09
Manganese	886	0.002	1.7720	0.102	0.104	0.0106	6.88	100	0.198	1.3613	1	3.1439	284	0.01	88	0.04
Mercury (inorganic)	1.3	0.002	0.0026	0.000	0.104	0.0000	0.03	100	0.198	0.0056	1	0.0082	10	0.00	1	0.01
Nickel	52.4	0.002	0.1048	0.003	0.104	0.0003	0.06	100	0.198	0.0121	1	0.1173	80	0.00	40	0.00
Selenium	4.5	0.002	0.0090	0.003	0.104	0.0003	0.70	100	0.198	0.1386	1	0.1479	0.33	0.45	0.2	0.74
Thallium	2	0.002	0.0040	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0414	0.74	0.06	0.074	0.56
Vanadium	31.8	0.002	0.0636	0.005	0.104	0.0005	0.17	100	0.198	0.0339	1	0.0980	2.1	0.05	0.21	0.47
Zinc	1640	0.002	3.2800	0.009	0.104	0.0010	24.31	100	0.198	4.8139	1	8.0948	320	0.03	160	0.05
Organic Compounds																
Bis(2-ethylhexyl)phthalate	4.9	0.002	0.0098	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0099	NB	NB	NB	NB
Chlordane	0.037	0.002	0.0001	NA	0.104	NA	0.0005	100	0.198	0.0001	1	0.0002	0.75	0.00	0.15	0.00
DDT and metabolites	0.049	0.002	0.0001	NA	0.104	NA	R	100	0.198	R	1	0.0001	4	0.00	0.8	0.00
Dichlorobenzenes	0.099	0.002	0.0002	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0002	NB	NB	NB	NB
Dieldrin	0.11	0.002	0.0002	NA	0.104	NA	R	100	0.198	R	1	0.0002	0.018	0.01	0.009	0.02
Endrin	0.028	0.002	0.0001	NA	0.104	NA	0.0017	100	0.198	0.0003	1	0.0004	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.84	0.002	0.0017	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0017	0.14	0.01	0.014	0.12
Hexachlorocyclohexanes	0.026	0.002	0.0001	NA	0.104	NA	0.0004	100	0.198	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	1.3	0.002	0.0026	0.000	0.104	0.0000	0.03	100	0.198	0.0056	1	0.0082	0.025	0.33	0.0025	3.28
Polycyclic aromatic hydrocarb	331.64	0.002	0.6633	0.013	0.104	0.0014	NA	100	0.198	NA	1	0.6646	10	0.07	1	0.66
Trichlorobenzenes	0.066	0.002	0.0001	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0002	53.6	0.00	14.8	0.00
Xylenes	0.066	0.002	0.0001	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0002	2.6	0.00	2.1	0.00
Dioxins/furans	0.00171	0.002	0.0000	NA	0.104	NA	0.000043	100	0.198	0.0000	1	0.0000	0.00001	1.19	0.000001	11.93
Total PCBs	18.033	0.002	0.0361	NA	0.104	NA	0.1125	100	0.198	0.0223	1	0.0583	0.034	1.72	0.0034	17.16
Aroclor 1016	0.12	0.002	0.0002	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0004	NB	NB	0.44	0.00
Aroclor 1221	0.12	0.002	0.0002	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0004	NB	NB	0.44	0.00
Aroclor 1232	0.12	0.002	0.0002	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0004	0.034	0.01	0.0034	0.12
Aroclor 1242	2.7	0.002	0.0054	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0056	NB	NB	0.44	0.01
Aroclor 1248	0.12	0.002	0.0002	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0004	0.034	0.01	0.0034	0.12
Aroclor 1254	0.12	0.002	0.0002	NA	0.104	NA	0.0850	100	0.198	0.0168	1	0.0171	0.1	0.17	0.01	1.71
Aroclor 1260	18	0.002	0.0360	NA	0.104	NA	0.0364	100	0.198	0.0072	1	0.0432	0.034	1.27	0.0034	12.71

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 37. Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Maximum Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.4	0.002	0.0028	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0219	1.25	0.02	0.125	0.18
Arsenic	2.3	0.002	0.0046	0.001	0.104	0.0001	0.23	100	0.198	0.0458	1	0.0505	1.26	0.04	0.126	0.40
Barium	118	0.002	0.2360	0.113	0.104	0.0118	1.29	100	0.198	0.2549	1	0.5027	75	0.01	45	0.01
Cadmium	1.3	0.002	0.0026	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.0109	10	0.00	1	0.01
Chromium	19.8	0.002	0.0396	0.001	0.104	0.0001	0.39	100	0.198	0.0764	1	0.1161	13.14	0.01	3.28	0.04
Copper	27	0.002	0.0540	0.003	0.104	0.0003	0.30	100	0.198	0.0587	1	0.1130	15.14	0.01	11.7	0.01
Lead	63.7	0.002	0.1274	0.001	0.104	0.0001	0.25	100	0.198	0.0490	1	0.1765	80	0.00	8	0.02
Manganese	258	0.002	0.5160	0.108	0.104	0.0112	3.65	100	0.198	0.7227	1	1.2499	284	0.00	88	0.01
Mercury (inorganic)	0.061	0.002	0.0001	0.000	0.104	0.0000	0.05	100	0.198	0.0104	1	0.0106	10	0.00	1	0.01
Nickel	8.7	0.002	0.0174	0.003	0.104	0.0003	0.06	100	0.198	0.0121	1	0.0298	80	0.00	40	0.00
Selenium	0.47	0.002	0.0009	0.003	0.104	0.0003	0.70	100	0.198	0.1386	1	0.1398	0.33	0.42	0.2	0.70
Thallium	0.7	0.002	0.0014	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0388	0.74	0.05	0.074	0.52
Vanadium	4.1	0.002	0.0082	0.001	0.104	0.0001	0.10	100	0.198	0.0198	1	0.0281	2.1	0.01	0.21	0.13
Zinc	56.6	0.002	0.1132	0.006	0.104	0.0006	39.38	100	0.198	7.7963	1	7.9101	320	0.02	160	0.05
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.03	0.002	0.0001	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0001	NB	NB	NB	NB
Chlordane	0.00058	0.002	0.0000	NA	0.104	NA	0.0023	100	0.198	0.0004	1	0.0004	0.75	0.00	0.15	0.00
DDT and metabolites	0.0054	0.002	0.0000	NA	0.104	NA	0.0020	100	0.198	0.0004	1	0.0004	4	0.00	0.8	0.00
Dichlorobenzenes	0.0033	0.002	0.0000	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.0006	0.002	0.0000	NA	0.104	NA	0.0020	100	0.198	0.0004	1	0.0004	0.018	0.02	0.009	0.04
Endrin	0.0006	0.002	0.0000	NA	0.104	NA	0.0023	100	0.198	0.0004	1	0.0004	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.03	0.002	0.0001	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	0.14	0.00	0.014	0.01
Hexachlorocyclohexanes	0.0012	0.002	0.0000	NA	0.104	NA	0.0004	100	0.198	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.061	0.002	0.0001	0.000	0.104	0.0000	0.05	100	0.198	0.0104	1	0.0106	0.025	0.42	0.0025	4.23
Polycyclic aromatic hydrocarb	9.843	0.002	0.0197	0.013	0.104	0.0014	NA	100	0.198	NA	1	0.0210	10	0.00	1	0.02
Trichlorobenzenes	0.0022	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0022	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.0000072	0.002	0.0000	NA	0.104	NA	0.000048	100	0.198	0.0000	1	0.000010	0.00001	0.95	0.000001	9.52
Total PCBs	0.268	0.002	0.0005	NA	0.104	NA	0.2150	100	0.198	0.0426	1	0.0431	0.034	1.27	0.0034	12.68
Aroclor 1016	0.0055	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1221	0.0055	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1232	0.0055	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1242	0.23	0.002	0.0005	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0006	NB	NB	0.44	0.00
Aroclor 1248	0.0055	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1254	0.0055	0.002	0.0000	NA	0.104	NA	0.1800	100	0.198	0.0356	1	0.0357	0.1	0.36	0.01	3.57
Aroclor 1260	0.048	0.002	0.0001	NA	0.104	NA	0.0725	100	0.198	0.0144	1	0.0145	0.034	0.43	0.0034	4.25

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 38. Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Mean Measured COPC Concentrations, Upstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.730	0.002	0.0015	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0205	1.25	0.02	0.125	0.16
Arsenic	5.618	0.002	0.0112	0.002	0.104	0.0002	0.21	100	0.198	0.0408	1	0.0522	1.26	0.04	0.126	0.41
Barium	56.336	0.002	0.1127	0.102	0.104	0.0106	0.69	100	0.198	0.1358	1	0.2590	75	0.00	45	0.01
Cadmium	1.777	0.002	0.0036	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.0119	10	0.00	1	0.01
Chromium	171.373	0.002	0.3427	0.004	0.104	0.0005	0.36	100	0.198	0.0716	1	0.4148	13.14	0.03	3.28	0.13
Copper	106.245	0.002	0.2125	0.006	0.104	0.0006	0.66	100	0.198	0.1299	1	0.3430	15.14	0.02	11.7	0.03
Lead	101.927	0.002	0.2039	0.004	0.104	0.0004	0.18	100	0.198	0.0363	1	0.2406	80	0.00	8	0.03
Manganese	206.857	0.002	0.4137	0.110	0.104	0.0114	3.61	100	0.198	0.7153	1	1.1404	284	0.00	88	0.01
Mercury (inorganic)	0.102	0.002	0.0002	0.000	0.104	0.0000	0.04	100	0.198	0.0077	1	0.0079	10	0.00	1	0.01
Nickel	69.600	0.002	0.1392	0.005	0.104	0.0005	0.06	100	0.198	0.0121	1	0.1518	80	0.00	40	0.00
Selenium	0.462	0.002	0.0009	0.003	0.104	0.0003	0.73	100	0.198	0.1452	1	0.1464	0.33	0.44	0.2	0.73
Thallium	0.319	0.002	0.0006	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0380	0.74	0.05	0.074	0.51
Vanadium	10.214	0.002	0.0204	0.001	0.104	0.0001	0.10	100	0.198	0.0198	1	0.0404	2.1	0.02	0.21	0.19
Zinc	181.055	0.002	0.3621	0.013	0.104	0.0014	28.71	100	0.198	5.6843	1	6.0478	320	0.02	160	0.04
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.297	0.002	0.0006	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0006	NB	NB	NB	NB
Chlordane	0.007	0.002	0.0000	NA	0.104	NA	0.0016	100	0.198	0.0003	1	0.0003	0.75	0.00	0.15	0.00
DDT and metabolites	0.033	0.002	0.0001	NA	0.104	NA	0.0013	100	0.198	0.0002	1	0.0003	4	0.00	0.8	0.00
Dichlorobenzenes	0.004	0.002	0.0000	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.001	0.002	0.0000	NA	0.104	NA	0.0036	100	0.198	0.0007	1	0.0007	0.018	0.04	0.009	0.08
Endrin	0.008	0.002	0.0000	NA	0.104	NA	0.0030	100	0.198	0.0006	1	0.0006	0.92	0.00	0.092	0.01
Hexachlorobenzene	0.258	0.002	0.0005	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0006	0.14	0.00	0.014	0.04
Hexachlorocyclohexanes	0.043	0.002	0.0001	NA	0.104	NA	0.0003	100	0.198	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.102	0.002	0.0002	0.000	0.104	0.0000	0.04	100	0.198	0.0077	1	0.0079	0.025	0.32	0.0025	3.16
Polycyclic aromatic hydrocarb	164.314	0.002	0.3286	0.008	0.104	0.0009	NA	100	0.198	NA	1	0.3295	10	0.03	1	0.33
Trichlorobenzenes	0.002	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.005	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.000	0.002	0.0000	NA	0.104	NA	0.000033	100	0.198	0.0000	1	0.0000	0.00001	0.69	0.000001	6.95
Total PCBs	4.645	0.002	0.0093	NA	0.104	NA	0.3125	100	0.198	0.0619	1	0.0712	0.034	2.09	0.0034	20.93
Aroclor 1016	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1221	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1232	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1242	4.600	0.002	0.0092	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0094	NB	NB	0.44	0.02
Aroclor 1248	0.005	0.002	0.0000	NA	0.104	NA	0.0008	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1254	0.005	0.002	0.0000	NA	0.104	NA	0.2446	100	0.198	0.0484	1	0.0484	0.1	0.48	0.01	4.84
Aroclor 1260	0.005	0.002	0.0000	NA	0.104	NA	0.1013	100	0.198	0.0200	1	0.0201	0.034	0.59	0.0034	5.90

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 39 . Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Mean Measured COPC Concentrations, Middle reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	1.55	0.002	0.0031	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0222	1.25	0.02	0.125	0.18
Arsenic	8.46	0.002	0.0169	0.001	0.104	0.0001	0.20	100	0.198	0.0390	1	0.0561	1.26	0.04	0.126	0.44
Barium	105.07	0.002	0.2101	0.104	0.104	0.0109	1.18	100	0.198	0.2327	1	0.4536	75	0.01	45	0.01
Cadmium	14.83	0.002	0.0297	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.0380	10	0.00	1	0.04
Chromium	63.64	0.002	0.1273	0.001	0.104	0.0001	0.05	100	0.198	0.0093	1	0.1367	13.14	0.01	3.28	0.04
Copper	97.47	0.002	0.1949	0.003	0.104	0.0003	0.04	100	0.198	0.0086	1	0.2038	15.14	0.01	11.7	0.02
Lead	82.17	0.002	0.1643	0.002	0.104	0.0003	0.65	100	0.198	0.1287	1	0.2933	80	0.00	8	0.04
Manganese	348.71	0.002	0.6974	0.095	0.104	0.0099	6.88	100	0.198	1.3613	1	2.0686	284	0.01	88	0.02
Mercury (inorganic)	0.35	0.002	0.0007	0.000	0.104	0.0000	0.03	100	0.198	0.0056	1	0.0063	10	0.00	1	0.01
Nickel	23.80	0.002	0.0476	0.003	0.104	0.0003	0.06	100	0.198	0.0121	1	0.0600	80	0.00	40	0.00
Selenium	1.55	0.002	0.0031	0.003	0.104	0.0003	0.70	100	0.198	0.1386	1	0.1420	0.33	0.43	0.2	0.71
Thallium	0.68	0.002	0.0014	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0387	0.74	0.05	0.074	0.52
Vanadium	17.35	0.002	0.0347	0.003	0.104	0.0003	0.17	100	0.198	0.0339	1	0.0689	2.1	0.03	0.21	0.33
Zinc	352.69	0.002	0.7054	0.007	0.104	0.0007	24.31	100	0.198	4.8139	1	5.5200	320	0.02	160	0.03
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.48	0.002	0.0010	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0010	NB	NB	NB	NB
Chlordane	0.00	0.002	0.0000	NA	0.104	NA	0.0005	100	0.198	0.0001	1	0.0001	0.75	0.00	0.15	0.00
DDT and metabolites	0.01	0.002	0.0000	NA	0.104	NA	R	100	0.198	R	1	0.0000	4	0.00	0.8	0.00
Dichlorobenzenes	0.02	0.002	0.0000	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.01	0.002	0.0000	NA	0.104	NA	R	100	0.198	R	1	0.0000	0.018	0.00	0.009	0.00
Endrin	0.00	0.002	0.0000	NA	0.104	NA	0.0017	100	0.198	0.0003	1	0.0003	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.19	0.002	0.0004	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0004	0.14	0.00	0.014	0.03
Hexachlorocyclohexanes	0.01	0.002	0.0000	NA	0.104	NA	0.0004	100	0.198	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.35	0.002	0.0007	0.000	0.104	0.0000	0.03	100	0.198	0.0056	1	0.0063	0.025	0.25	0.0025	2.52
Polycyclic aromatic hydrocarb	39.93	0.002	0.0799	0.013	0.104	0.0013	NA	100	0.198	NA	1	0.0812	10	0.01	1	0.08
Trichlorobenzenes	0.01	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.01	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.000561	0.002	0.0000	NA	0.104	NA	0.000034	100	0.198	0.0000	1	0.0000	0.00001	0.79	0.000001	7.85
Total PCBs	1.74	0.002	0.0035	NA	0.104	NA	0.1125	100	0.198	0.0223	1	0.0258	0.034	0.76	0.0034	7.58
Aroclor 1016	0.02	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1221	0.02	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1232	0.02	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.06
Aroclor 1242	0.45	0.002	0.0009	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0011	NB	NB	0.44	0.00
Aroclor 1248	0.02	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.06
Aroclor 1254	0.02	0.002	0.0000	NA	0.104	NA	0.0854	100	0.198	0.0169	1	0.0170	0.1	0.17	0.01	1.70
Aroclor 1260	1.09	0.002	0.0022	NA	0.104	NA	0.0364	100	0.198	0.0072	1	0.0094	0.034	0.28	0.0034	2.76

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 40. Hazard Quotient Calculations for a Piscivorous Mammal (Mink)
Mean Measured COPC Concentrations, Downstream reach
Lower Ley Creek Site
Syracuse, New York

INITIAL SETTINGS		0.002			0.104			100	0.198		1					
COPC	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Inorganic Analytes																
Antimony	0.875	0.002	0.0018	0.005	0.104	0.0005	0.09	100	0.198	0.0186	1	0.0208	1.25	0.02	0.125	0.17
Arsenic	1.4375	0.002	0.0029	0.001	0.104	0.0001	0.23	100	0.198	0.0458	1	0.0488	1.26	0.04	0.126	0.39
Barium	98.475	0.002	0.1970	0.113	0.104	0.0118	1.29	100	0.198	0.2549	1	0.4636	75	0.01	45	0.01
Cadmium	0.89	0.002	0.0018	0.003	0.104	0.0003	0.04	100	0.198	0.0081	1	0.0101	10	0.00	1	0.01
Chromium	8.2	0.002	0.0164	0.001	0.104	0.0001	0.39	100	0.198	0.0764	1	0.0929	13.14	0.01	3.28	0.03
Copper	11.125	0.002	0.0223	0.003	0.104	0.0003	0.30	100	0.198	0.0587	1	0.0812	15.14	0.01	11.7	0.01
Lead	21.2	0.002	0.0424	0.001	0.104	0.0001	0.25	100	0.198	0.0490	1	0.0915	80	0.00	8	0.01
Manganese	229.5	0.002	0.4590	0.108	0.104	0.0112	3.65	100	0.198	0.7227	1	1.1929	284	0.00	88	0.01
Mercury (inorganic)	0.034	0.002	0.0001	0.000	0.104	0.0000	0.05	100	0.198	0.0104	1	0.0105	10	0.00	1	0.01
Nickel	7.675	0.002	0.0154	0.003	0.104	0.0003	0.06	100	0.198	0.0121	1	0.0278	80	0.00	40	0.00
Selenium	0.4025	0.002	0.0008	0.003	0.104	0.0003	0.70	100	0.198	0.1386	1	0.1397	0.33	0.42	0.2	0.70
Thallium	0.44125	0.002	0.0009	0.003	0.104	0.0003	0.19	100	0.198	0.0371	1	0.0383	0.74	0.05	0.074	0.52
Vanadium	3	0.002	0.0060	0.001	0.104	0.0001	0.10	100	0.198	0.0198	1	0.0259	2.1	0.01	0.21	0.12
Zinc	25.75	0.002	0.0515	0.006	0.104	0.0006	39.38	100	0.198	7.7963	1	7.8484	320	0.02	160	0.05
Organic Compounds																
Bis(2-ethylhexyl)phthalate	0.026	0.002	0.0001	0.0005	0.104	0.0001	NA	100	0.198	NA	1	0.0001	NB	NB	NB	NB
Chlordane	0.00052	0.002	0.0000	NA	0.104	NA	0.0023	100	0.198	0.0004	1	0.0004	0.75	0.00	0.15	0.00
DDT and metabolites	0.0025	0.002	0.0000	NA	0.104	NA	0.0020	100	0.198	0.0004	1	0.0004	4	0.00	0.8	0.00
Dichlorobenzenes	0.0032	0.002	0.0000	0.002	0.104	0.0002	NA	100	0.198	NA	1	0.0000	NB	NB	NB	NB
Dieldrin	0.0005	0.002	0.0000	NA	0.104	NA	0.0020	100	0.198	0.0004	1	0.0004	0.018	0.02	0.009	0.04
Endrin	0.0005	0.002	0.0000	NA	0.104	NA	0.0023	100	0.198	0.0004	1	0.0004	0.92	0.00	0.092	0.00
Hexachlorobenzene	0.026	0.002	0.0001	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	0.14	0.00	0.014	0.01
Hexachlorocyclohexanes	0.001	0.002	0.0000	NA	0.104	NA	0.0004	100	0.198	0.0001	1	0.0001	1	0.00	0.1	0.00
Methylmercury	0.034	0.002	0.0001	0.000	0.104	0.0000	0.05	100	0.198	0.0104	1	0.0105	0.025	0.42	0.0025	4.21
Polycyclic aromatic hydrocarb	3.678	0.002	0.0074	0.012	0.104	0.0013	NA	100	0.198	NA	1	0.0086	10	0.00	1	0.01
Trichlorobenzenes	0.0021	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	53.6	0.00	14.8	0.00
Xylenes	0.0021	0.002	0.0000	0.001	0.104	0.0001	NA	100	0.198	NA	1	0.0001	2.6	0.00	2.1	0.00
Dioxins/furans	0.0000072	0.002	0.0000	NA	0.104	NA	0.000048	100	0.198	0.0000	1	0.0000	0.00001	0.95	0.000001	9.52
Total PCBs	0.102	0.002	0.0002	NA	0.104	NA	0.2150	100	0.198	0.0426	1	0.0428	0.034	1.26	0.0034	12.58
Aroclor 1016	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1221	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	NB	NB	0.44	0.00
Aroclor 1232	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1242	0.061	0.002	0.0001	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0003	NB	NB	0.44	0.00
Aroclor 1248	0.005	0.002	0.0000	NA	0.104	NA	0.00083	100	0.198	0.0002	1	0.0002	0.034	0.01	0.0034	0.05
Aroclor 1254	0.005	0.002	0.0000	NA	0.104	NA	0.1800	100	0.198	0.0356	1	0.0357	0.1	0.36	0.01	3.57
Aroclor 1260	0.016	0.002	0.0000	NA	0.104	NA	0.0725	100	0.198	0.0144	1	0.0144	0.034	0.42	0.0034	4.23

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level
COPC = Contaminant of Potential Concern

PCB = Polychlorinated
DDT = Dichlorodiphenyltrichloroethane
NA = Not Analyzed
R = Data rejected for use
NB = No TRV available for contaminant, receptor

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Only total mercury was measured in samples. Total mercury value was used for both the inorganic and methylmercury calculation.
Shading indicates Hazard Quotient greater than 1.0

TABLE 41. Polychlorinated Biphenyls in Surface Soil (0-6 inches), Swale Samples
May 2010
Lower Ley Creek Site, Syracuse, New York

Location	Concentration (µg/kg)						
	SW-1A	SW-2A	SW-3A	SW-4A	SW-5A	SW-6A	SW-7A
Aroclor-1016	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1221	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1232	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1242	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1248	220	300	2500	1000	210000	230000	390
Aroclor-1254	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1260	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1262	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Aroclor-1268	5.2 (U)	7 (U)	5.7 (U)	9.2 (U)	580 (U)	520 (U)	4.6 (U)
Total PCBs	261.6	356	2545.6	1073.6	214640	234160	426.8
Mean Total PCBs	64780						

TABLE 42. Hazard Quotient Calculations for Exposure of Piscivorous Receptors to Swale Area Sediment
Maximum and Mean Total PCB Concentrations, Upstream Reach and Swale Area
Lower Ley Creek Site
Syracuse, New York

Receptor	Conc. In Sediment	Sediment Ingestion Rate	Total Intake through Sediment	Conc. In Surface Water	Water Ingestion Rate	Total Intake through Water	Conc. In Fish	Percent of Diet Fish	Food Ingestion Rate	Total Intake through Food	AUF	Dose	LOAEL	HQ LOAEL	NOAEL	HQ NOAEL
	mg/kg	kg/kgBW/day	mg/kg/day	mg/L	L/kgBW/day	mg/kgBW/day	mg/kg w.w.		kg/kgBW/day w.w.	mg/kgBW/day		mg/kgBW/day	mg/kgBW/day		mg/kgBW/day	
Maximum																
Belted Kingfisher	234.16	0.0045	1.0537	NA	0.045	NA	0.35	100	0.448	0.1568	1	1.2105	1.8	0.67	0.18	6.73
Great Blue Heron	234.16	0.0019	0.4449	NA	0.045	NA	0.35	100	0.189	0.0662	1	0.5111	1.8	0.28	0.18	2.84
Mink	234.16	0.002	0.4683	NA	0.045	NA	0.35	100	0.198	0.0693	1	0.5376	0.034	15.81	0.0034	158.12
River Otter	234.16	0.0013	0.3044	NA	0.045	NA	0.35	100	0.128	0.0448	1	0.3492	0.034	10.27	0.0034	102.71
Mean																
Belted Kingfisher	64.78	0.0045	0.2915	NA	0.045	NA	0.3125	100	0.448	0.1400	1	0.4315	1.8	0.24	0.18	2.40
Great Blue Heron	64.78	0.0019	0.1231	NA	0.045	NA	0.3125	100	0.189	0.0591	1	0.1821	1.8	0.10	0.18	1.01
Mink	64.78	0.002	0.1296	NA	0.045	NA	0.3125	100	0.198	0.0619	1	0.1914	0.034	5.63	0.0034	56.30
River Otter	64.78	0.0013	0.0842	NA	0.045	NA	0.3125	100	0.128	0.0400	1	0.1242	0.034	3.65	0.0034	36.53

Conc. = Concentration
mg = Milligram
kg = Kilogram
w.w. = wet weight
L = Liter
Shading indicates Hazard Quotient greater than 1.0

AUF = Area Use Factor
LOAEL = Lowest observable adverse effect level
HQ = Hazard quotient
NOAEL = No observable adverse effect level

PCB = Polychlorinated biphenyl
NA = Not Analyzed

mg/kg = milligrams per kilogram
kg/kgBW/day = kilograms per kilogram body weight per day
L/kgBW//day = Liters per kilogram body weight per day
mg/kgBW/day = milligrams per kilogram body weight per day

Table 43. Development of PRGs for Inorganic Contaminants of Concern Based on Direct Toxicity
Lower Ley Creek Site
Syracuse, New York

Analyte	Antimony	Arsenic	Cadmium	Chromium	Copper	Iron	Lead	Manganese	Mercury	Nickel	Silver	Zinc	Total PAH
Effect Level (mg/kg), Hyalella Toxicity Tests. TOX-08, significant mortality. TOX-09, significant decrease in growth.													
TOX-08	7.7	2.4	0.76	31.9	R	7560	30.8	191	0.071	14.2	0.33	R	156.56
TOX-09	3.75 (U)	2.3	0.76	20.4	102	7570	32.9	206	0.06 (U)	12.6	0.075	81.6	54.78
Maximum No Effect Level (mg/kg), Toxicity Tests													
H. azteca	14.2	5.6	6.4	94.2	5.8	17200	87.8	516	0.29	34.4	2.1	342	45.19
C. tentans	7.4	3.1	1.7	41.5	102	9950	84.3	516	0.1	22.3	0.24	133	288.53
Max Conc Nov 2009	3	19	107	1090	433	26600	284	886	1.3	447	18	1640	671.2
Screening Benchmarks													
TEC	NB	9.79	0.99	43.4	31.6	NB	35.8	NB	0.18	22.7	NB	121	1.61
NYSDEC FW sed	2	6	0.6	26	16	20000	31	460	0.15	16	1	120	NB
Is Maximum no effect concentration, H. azteca, greater than measured concentration in TOX-08 and TOX-09?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No
DECISION POINT: No effect concentration for PAHs, 45.19 mg/kg; LOAEL effect concentration for growth = 54.78 mg/kg; LOAEL effect concentration for mortality = 156.56 mg/kg													
Eliminated as COPC due to no effect concentration greater than maximum concentration measured November 2009	Yes	No	No	No	No	No	No	No	No	No	No	No	—
Eliminated as COPC because are essential trace elements; discussed in uncertainty section	—	No	No	No	No	Yes	No	Yes	No	No	No	No	—
Retained as a COPC; PRG is maximum no effect concentration	—	Yes	Yes	Yes	Yes	—	Yes	—	Yes	Yes	Yes	Yes	—
PRG	NA	5.6 mg/kg	6.4 mg/kg	94.2 mg/kg	102 mg/kg	NA	87.8 mg/kg	NA	0.29 mg/kg	34.4 mg/kg	2.1 mg/kg	342 mg/kg	Growth-based, 54.78 mg/kg; survival-based, 156.56 mg/kg

TEC = Threshold Effect Concentration, MacDonald et al. 2000
U = indicates not detected in that sample; value in table is 1/2 the reported detection limit
NB = No benchmark available
NA = Not applicable
MacDonald, D.D., Ingersoll, C.G., and Berger, T.A. 2000. Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. Arch. Environ. Contam. Toxicol. 39:20-31.
NYSDEC (New York State Department of Environmental Conservation), Division of Fish, Wildlife, and Marine Resources. 1999. Technical Guidance for Screening Contaminated Sediments.

Table 44. Site-Specific Bioaccumulation Factor Calculations for Forage Fish
Lower Ley Creek
Syracuse, New York

	Mean Forage Fish Total PCB Concentration (mg/kg ww)	Sediment PCB Concentration		Maximum Forage Fish Total PCB Concentration (mg/kg ww)	Sediment PCB Concentration	
		Mean Concentration (mg/kg)	BAF		Mean Concentration (mg/kg)	BAF
Upstream	0.3063	4.645	0.066	0.4	4.645	0.086
Middle	0.1125	1.744	0.065	0.1525	1.744	0.087
Downstream	0.125	0.102	1.225	0.215	0.102	2.108

PCB = Polychlorinated biphenyl
mg/kg ww = milligrams per kilogram wet weight
mg/kg = milligrams per kilogram
BAF = Site-specific bioaccumulation factor

Table 45. Ecological Risk-Based PRGs Based on Site-Specific Bioaccumulation Factors in Forage Fish
Lower Ley Creek Superfund Site
Syracuse, New York

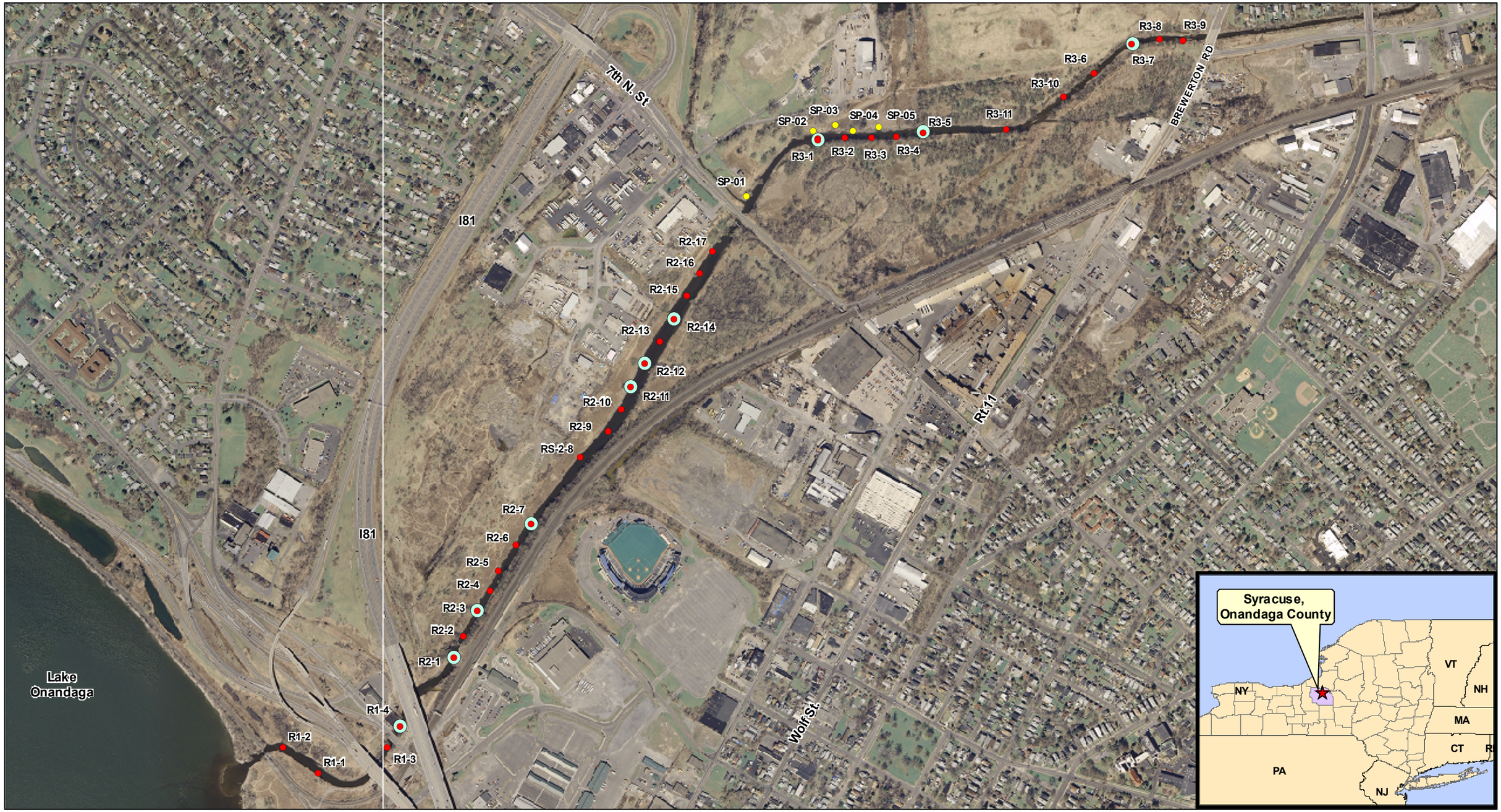
	Sediment PRGs based on consumption of fish by mink							Sediment PRGs based on consumption of fish by mink						
	Mean Forage Fish Concentration							Maximum Forage Fish Concentration						
	LOAEL (mg/kgBW/day	BW (kg)	SIR (kg/day)	FIR (kg/day)	BAF	FIR X BAF	Csed (mg/kg)	LOAEL (mg/kgBW/day	BW (kg)	SIR (kg/day)	FIR (kg/day)	BAF	FIR X BAF	Csed (mg/kg)
Upstream	0.034	0.6	0.0012	0.119	0.066	0.007854	2.25	0.034	0.6	0.0012	0.119	0.086	0.010234	1.78
Middle	0.034	0.6	0.0012	0.119	0.065	0.007735	2.28	0.034	0.6	0.0012	0.119	0.087	0.010353	1.77
Downstream	0.034	0.6	0.0012	0.119	1.225	0.145775	0.14	0.034	0.6	0.0012	0.119	2.108	0.250852	0.08
	NOAEL (mg/kgBW/day	BW (kg)	SIR (kg/day)	FIR (kg/day)	BAF	FIR X BAF	Csed	NOAEL (mg/kgBW/day	BW (kg)	SIR (kg/day)	FIR (kg/day)	BAF	FIR X BAF	Csed
Upstream	0.0034	0.6	0.0012	0.119	0.066	0.007854	0.23	0.0034	0.6	0.0012	0.119	0.086	0.010234	0.18
Middle	0.0034	0.6	0.0012	0.119	0.065	0.007735	0.23	0.0034	0.6	0.0012	0.119	0.087	0.010353	0.18
Downstream	0.0034	0.6	0.0012	0.119	1.225	0.145775	0.01	0.0034	0.6	0.0012	0.119	2.108	0.250852	0.01

LOAEL = Lowest observed adverse effect level
BW = Body weight
SIR = Sediment ingestion rate
FIR = Food ingestion rate
BAF = Site-specific bioaccumulation factor
Csed = Calculated sediment cleanup goal

mg/kgBW/day = milligrams per kilogram body weight per day
kg = kilograms
kg/day = kilograms per day
NOAEL = No observed adverse effect level
mg/kg = milligrams per kilogram

Figures

Lower Ley Creek Site
Streamlined Baseline Ecological Risk Assessment
Final Report
January 2011



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Legend

- Sediment Sampling Location;
- Dredge Area Sampling Location
- Water Sample at this Location

500 0 500 1,000 Feet

Map Creation Date: February 2010

Coordinate system: New York State Plane, Central
FIPS 3102
Datum :NAD83
Units: Feet

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U.S EPA Environmental Response Team
Scientific Engineering Response and Analytical Services
EP-W-09-031
W.A.# 0-007

Figure 1
Sediment and Surface Water Sampling Locations, November 2009
Lower Ley Creek-BERA
Syracuse, New York

Map Creation Date: February 2011

Coordinate system: New York State Plane, Central
FIPS 3102
Datum :NAD83
Units: Feet

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Legend

- Toxicity Testing (10-28)
- Toxicity Testing (10)

500 0 500 1,000
Feet

N
W E
S

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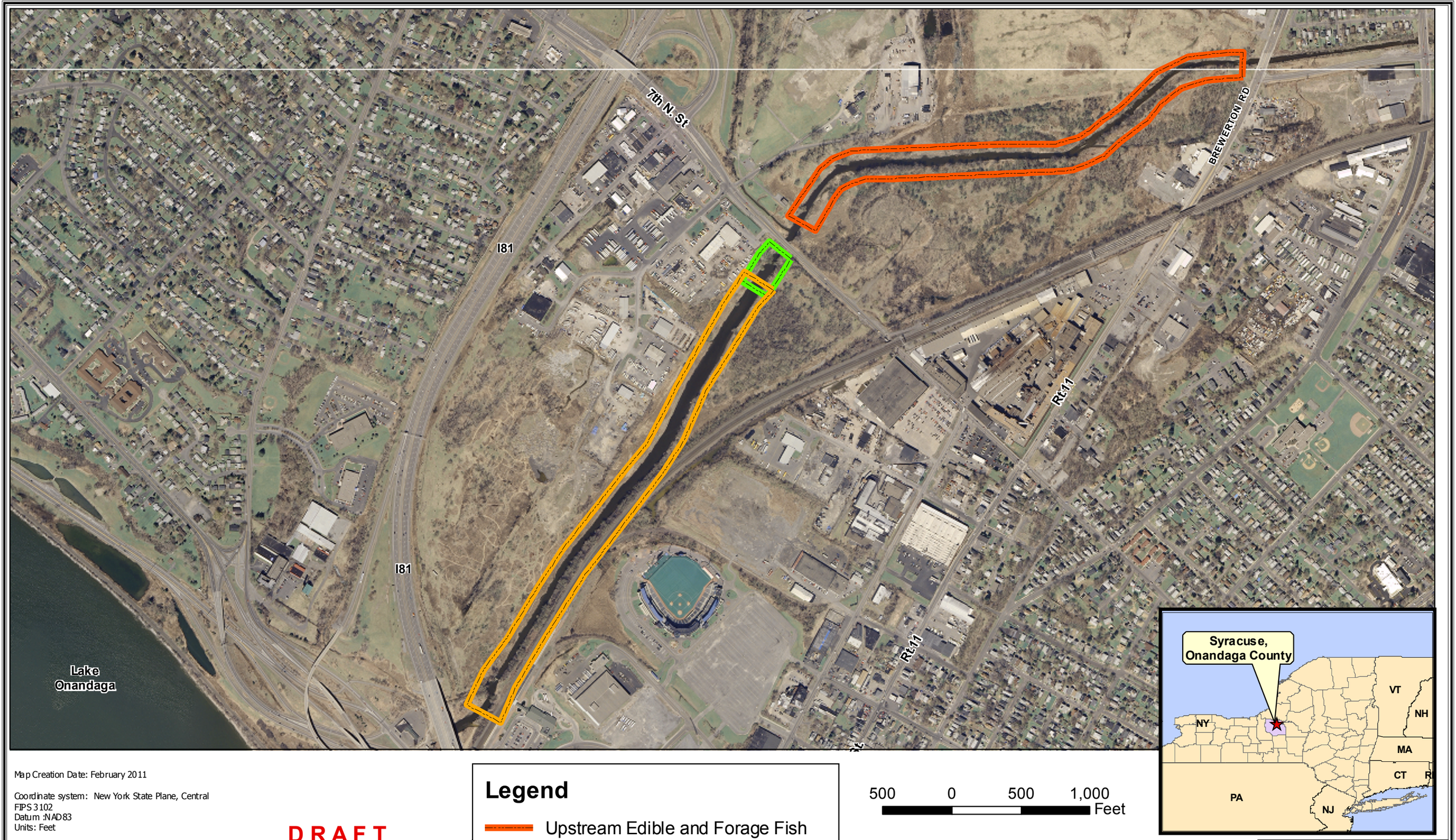
EP-W-09-031
W.A.# 0-007

Figure 2
Sediment Sampling Locations,
May 2010
Lower Ley Creek-BERA
Syracuse, New York



Map Creation Date: February 2011

Coordinate system: New York State Plane, Central
FIPS 3102
Datum :NAD83
Units: Feet

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Data: g:\ArcViewprojects\SERAS01\00-007
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Legend

- Upstream Edible and Forage Fish
- Middle (Forage Fish Only)
- Downstream Edible and Forage Fish

500 0 500 1,000 Feet

U.S EPA Environmental Response Team
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W.A.# 0-007

Figure 3
Fish Sampling Locations
Lower Ley Creek-BERA
Syracuse, New York

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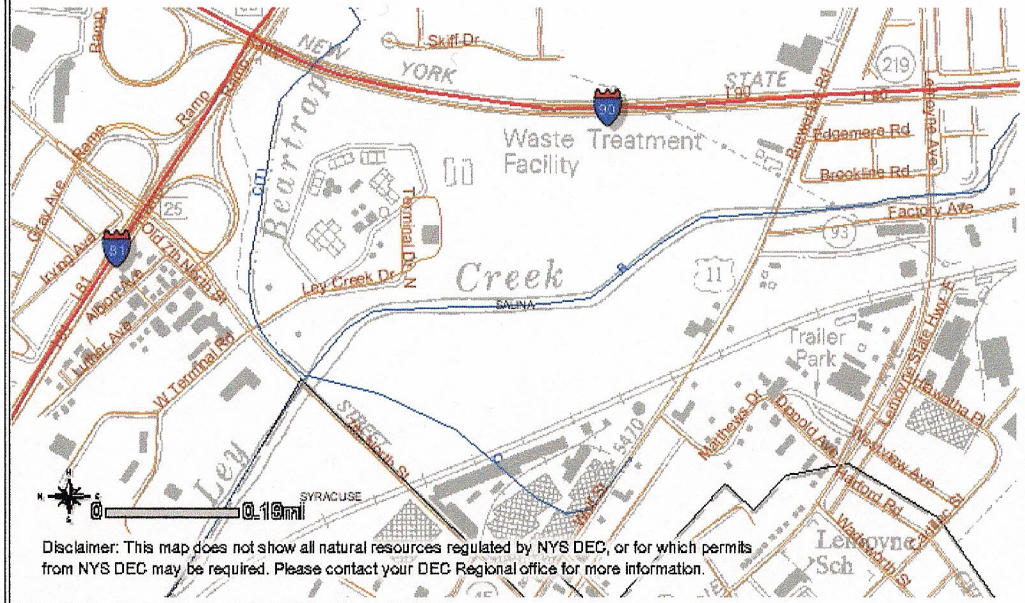
Please set your printer orientation to "Landscape".

Ley Creek NYSDEC Stream Classification



Disclaimer: This map does not show all natural resources regulated by NYS DEC, or for which permits from NYS DEC may be required. Please contact your DEC Regional office for more information.

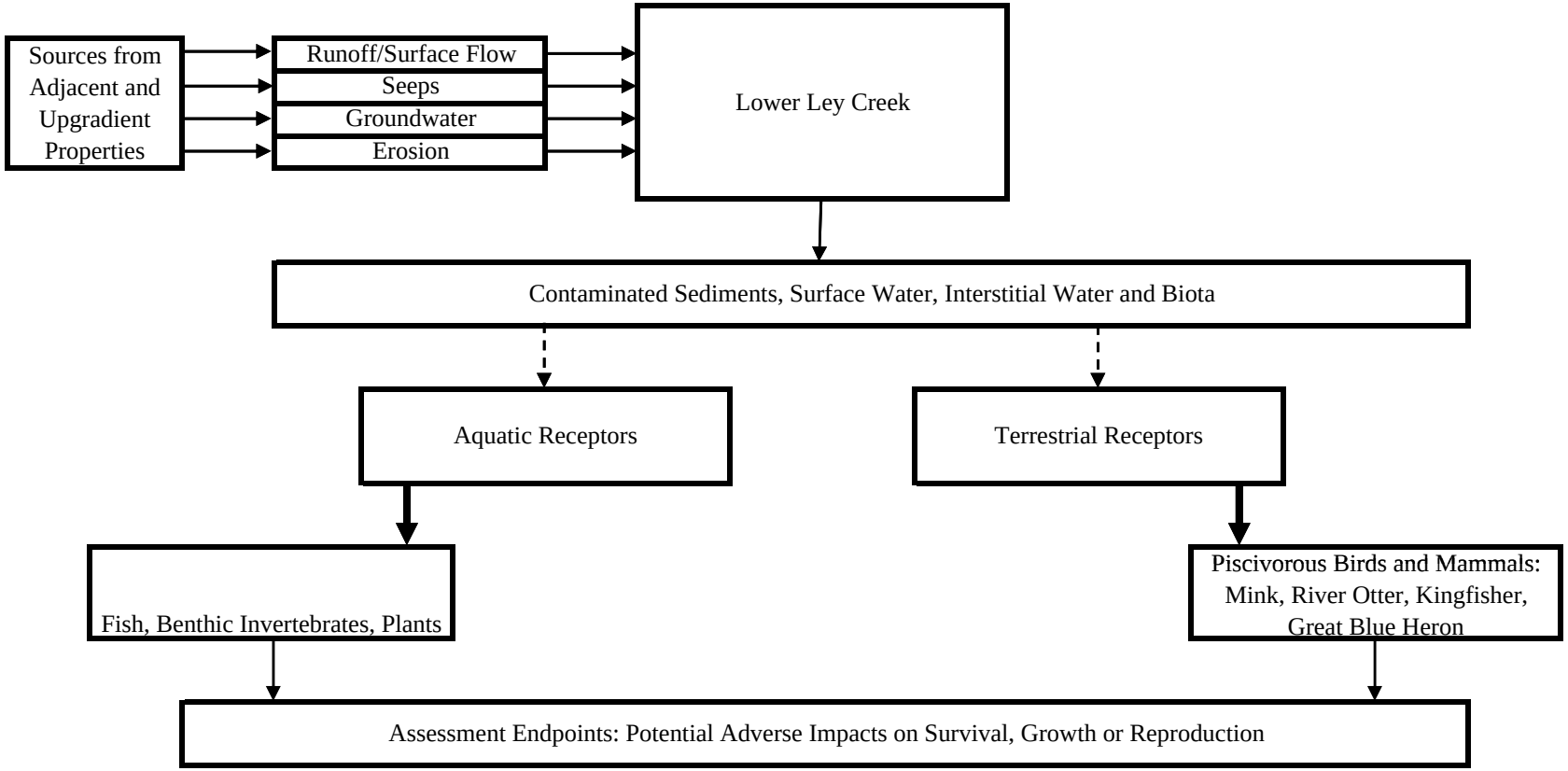
MinX: 404726, MaxX: 407002, MinY: 4771638, MaxY: 4770803

Visible Layers

- Classified Streams
- Classified Ponds
- Rare Plants and Rare Animals
- Interstate Highways
- Adirondack Park Boundary
- Counties

Disclaimer:This map was prepared by the New York State Department of Environmental Conservation using the most current data available. It is deemed accurate but is not guaranteed. NYS DEC is not responsible for any inaccuracies in the data and does not necessarily endorse any interpretations or products derived from the data.

Figure 5. Conceptual Site Model
Lower Ley Creek Baseline Ecological Risk Assessment
Syracuse, NY



APPENDIX A.
Toxicity Reference Values
Lower Ley Creek Site
Syracuse, New York

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A.0 Derivation of Toxicity Reference Values

A toxicity reference value (TRV) is a contaminant dose level that is compared with a predicted exposure dose level, calculated based on site-specific data, to assess the presence and degree of risk to a receptor or group of receptors from that contaminant. A TRV is generally based on data from laboratory toxicological evaluations. Usually, two TRVs are used to predict ecological risk, a no observable adverse effect level (NOAEL) and a lowest observable adverse effect level (LOAEL). The NOAEL is the highest dose at which adverse effects are not expected to occur, and the LOAEL is the lowest dose at which adverse effects are expected to occur.

To derive TRVs, a comprehensive literature search was performed to identify studies on the toxicity of contaminants of concern (COCs) to ecological receptors. A variety of databases were searched, including Biological Abstracts, Applied Ecology Abstracts, Chemical Abstract Services, Medline, Toxline, BIOSIS, ENVIROLINE, Current Contents, Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), Integrated Risk Information System (IRIS), and the Aquatic Toxicity Information Retrieval Database (AQUIRE).

In addition, a number of secondary literature sources provided summaries or reviews of toxicological literature related to a variety of contaminants. These documents were not used directly to derive TRVs because they do not capture the details of the toxicological methods needed for the selection of technically defensible TRVs. However, these summary documents provided an excellent source for locating original studies that may have been overlooked in the database searches. Examples of such summary documents include Agency for Toxic Substances and Disease Registry (ATSDR) documents, United States (U.S.) Fish and Wildlife Service Contaminant Hazard Reviews, U.S. Environmental Protection Agency (EPA) Great Lakes Water Quality Initiative documents, and U.S. EPA Ambient Water Quality Criteria documents.

Studies that were obviously not useful or appropriate for deriving a TRV were eliminated. A number of criteria were considered when evaluating the appropriateness of using a particular study for deriving a TRV. The most important consideration was the suitability of the test result for evaluating the assessment endpoint. A number of additional criteria were also considered. For example, studies were selected in which the test organism was in as similar a taxonomic grouping as possible to the measurement endpoint species. Exposure doses had to be quantified and effects measured and reported. The exposure duration was preferably either chronic, sub-chronic, or involved a sensitive life stage; multi generational studies were also appropriate. For laboratory studies, the likelihood that a similar result would be obtained if the test were repeated was an additional consideration. Sample sizes had to be adequate and the treatment groups must have been compared to appropriate control groups. At the very least, a negative control had to be included in the study design. In addition, the measured endpoints of the study had to be ecologically relevant. For the purposes of deriving a TRV for an ecological risk assessment, an ecologically relevant endpoint is one which is closely tied to the survival and viability of a population in the field. Usually, the endpoints measured for this purpose were survival, growth, and reproduction. In addition, appropriate statistical analyses must have been performed and the statistical significance reported. Finally, the study design preferably included at least three treatments in addition to any controls which may have been selected.

The selected TRVs were based preferably on high-quality studies which satisfy many or all of the above requirements. From these studies, the lowest concentration that was associated with adverse ecological effects on the test organism was selected as the LOAEL. Studies which reported both a LOAEL and NOAEL were selected over studies which reported only one effect level, due to the uncertainty associated with an unbounded effect level. If only a LOAEL could be identified from the studies, an uncertainty factor of 10 was used to calculate a NOAEL (Dourson and Stara 1983; U.S. EPA 1989; Amdur and Klaasen 1996; Sample et al. 1996). If a LOAEL could not be located for a COC, a median lethal dose (LD_{50}) was selected; a factor of 10 was then used to calculate a LOAEL and a factor of 100 was used to calculate a NOAEL. Professional judgement was used in some cases to select the most appropriate TRV. Additionally, TRVs were selected to be consistent with TRVS utilized in the Onondaga Lake and Nine Mile Creek/Geddes Brook ERAs, two aquatic systems that are within the Lower Ley Creek watershed.

The studies that were reviewed to derive toxicity reference values for this risk assessment are described below. Concentrations selected to be used as TRVs are summarized in Table 11.

A.1 Antimony

A.1.1 Toxicity to Mammals

A mammalian TRV was derived for antimony according to Eco-SSL guidance (U. S. EPA 2003).A literature search was conducted, and NOAEL and LOAEL values reported for exposure of mammals to antimony were compiled. To derive a wildlife TRV, a minimum of three NOAEL or LOAEL results reported for a least two test species for either growth, reproduction, or survival effects are required. A geometric mean is calculated if three NOAEL results are available for either growth or reproduction effect groups. For antimony, the geometric mean of the NOAEL values for growth and reproduction was higher than the lowest bounded LOAEL for effects on reproduction, growth or survival. The Eco-SSL TRV for mammals was identified as the highest bounded NOAEL below the lowest bounded LOAEL; a NOAEL value of 0.059 mg/kgBW/day was identified.

Diets containing antimony trioxide were fed to male and female Wistar rats at concentrations of 0, 84, 421 and 1686 mg/kgBW/day for 90 days (Hext et al. 1999). No effects on growth, growth rate, food consumption or clinical signs were observed at any exposure dose. Minor changes in hematology and urine biochemistry were observed. The ecological significance of changes in blood chemistry is not known; therefore, this experiment was not used to derive a TRV for Sb.

Field voles (Microtus agrestis) were exposed to antimony trioxide in their diets at concentrations of 0, 500, or 6,700 mg/kg for 21 days, 0 or 500 mg/kg for 60 days, or 0 or 20,000 mg/kg for 12 days (Ainsworth et al. 1991). Dietary exposure to Sb produced elevated concentrations of Sb in liver, lung and kidney tissue; however, no histopathological changes were observed in liver or kidney tissue even at the highest exposure concentration. An equilibrium between uptake and excretion of Sb was rapidly established, and progressive increases in organ concentration as exposure duration increased were not observed. A NOAEL of 500 mg/kg was observed in this experiment.

Male Wistar rats were exposed to Sb in their diet at concentrations of 0, 0.1 and 1.0 % (wet weight) metal Sb and 1.0 % antimony trioxide for 12 weeks (Hiraoka 1986). Following exposure, rats were fed Sb-free diets for the following 12 weeks. Behavior and general appearance of the rats were not affected by exposure to Sb at any concentration tested in this experiment. Liver weights were significantly higher and heart weights were significantly lower in rats exposed to 1.0 % dietary Sb as compared to controls; however, heart and liver function were not measured. An ingestion rate of 0.027 kg/day and a body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to unit of mg/kgBW/day. A NOAEL of 771 mg/kgBW/day was calculated based on the results of this experiment.

Antimony trioxide in aqueous suspension was administered to male Swiss albino mice by gavage at concentrations of 0, 400, 666.7 and 1,000 mg/kgBW/day for 21 days (Gurnani et al. 1992). Mice of both sexes were exposed to three doses at the above concentrations to evaluate effects of acute exposure. Antimony did not induce chromosomal aberrations following the acute exposures. Following chronic exposure, the frequency of chromosomal aberrations increased proportionately with dose, and was significantly different from controls at all dose levels. The frequency of abnormal sperm did not differ between treated and control mice. The highest dose was lethal on day 20 of treatment. Based on the observed chromosomal aberrations, a LOAEL of 400 mg/kgBW/day and an estimated NOAEL of 40 mg/kgBW/day were identified from this experiment.
Mice were exposed to Sb (as antimony potassium tartrate) in drinking water at an exposure concentration of 5 mg/L throughout their lifespan (Schroeder et al. 1968). The endpoint measured was longevity. Median lifespan of female mice was significantly reduced as compared to control mice. A LOAEL of 1.25 mg/kgBW/day and an estimated NOAEL of 0.125 mg/kgBW/day were calculated based on the results of this experiment. Based on the ecological significance of the endpoint (survival), the TRV values from this study will be used to evaluate the risk posed by Sb to mammalian receptors.

Ainsworth, N., J. A. Cooke and M. S. Johnson (1991). Behavior and toxicity of antimony in the short-tailed field vole (Microtus agrestis). Ecotoxicol. Environ. Safety 21: 165-170.

Gurnani, N., A. K. Sharma and G. Talukder (1992). Comparison of the clastogenic effects of antimony trioxide on mice in vivo following acute and chronic exposure. Biometals 5: 47-50.

Hext, P. M., P. J. Pinto and B. A. Rimmel (1999). Subchronic feeding study of antimony trioxide in rats. J. Appl. Toxicol. 19(3): 205-209.

Hiraoka, N. (1986). The toxicity and organ distribution of antimony after chronic administration to rats. J. Kyoto Prefect. Univ. Med. 95: 997-1017.

Schroeder, H. A., M. Mitchener, J. J. Balassa, M. Kanisawa and A. P. Nason (1968). Zirconium, niobium, antimony and fluorine in mice: effects on growth, survival and tissue levels. J. Nutr 95: 95-101.

Schroeder, H. A., M. Mitchener and A. P. Nason (1970). Zirconium, niobium, antium, antimony, vanadium and lead in rats: Life term studies. J. Nutr 100(1): 59-68.

U. S. EPA 2003. Attachment 4-5, Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs). Eco-SSL Standard Operating Procedure (SOP) #6: Derivation of Wildlife Toxicity Reference Value (TRV). OSWER Directive 92857-55.

U.S. EPA 1988. Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.1.2 Toxicity to Birds

No studies were located that evaluated toxicity of antimony (Sb) to birds.

A.2 Arsenic

A.2.1 Toxicity to Mammals

Wildlife TRVs were derived for arsenic according to Eco-SSL guidance (U. S. EPA 2003). A literature search was conducted, and NOAEL and LOAEL values reported for exposure of mammals to antimony were compiled. To derive a wildlife TRV, a minimum of three NOAEL or LOAEL results reported for a least two test species for either growth, reproduction, or survival effects are required. A geometric mean is calculated if three NOAEL results are available for either growth or reproduction effect groups. For arsenic, the geometric mean of the NOAEL values for growth and reproduction was higher than the lowest bounded LOAEL for effects on reproduction, growth or survival. The Eco-SSL mammalian TRV for arsenic was identified as the highest bounded NOAEL below the lowest bounded LOAEL; a NOAEL value of 1.04 mg/kgBW/day was identified.

Dogs were fed diets containing sodium arsenite at concentrations of 0, 1, 2, and 4 mg/kgBW/day for 59 days (Neiger and Osweiller 1989). On day 59, the dosage was doubled for the rest of the experiment, which ended on Day 183. Significant dose-dependent decreases in food consumption and body weight were observed in dogs exposed to As at concentrations of 4 and 8 mg/kgBW/day. Weight loss was due to decreased food consumption, not to any direct effect of As. No gross or microscopic lesions were found in livers of any group. The effect concentrations observed in this experiment were not used to derive a TRV for this risk assessment, as a decrease in body weight is not considered an ecologically relevant effect.

Arsenic trioxide was administered to adult female rats (strain Crl:CD®(SD)BR) at concentrations of 0, 1, 2.5, 5 and 10 mg/kgBW/day in conformance with good laboratory practice (GLP) regulations (Stump et al. 1998). Arsenic exposure began 14 days prior to mating and continued until gestation day 19. Reduced body weight and food intake were observed in rats exposed at a concentration of 10 mg/kgBW/day; stomach abnormalities such as eroded areas and adhesions were also observed. Fetal weights were reduced and the incidence of two skeletal developmental variations were also increased in the high dose group. The fetal effects were attributed to the effect on fetal weight and were not indicative of teratogenicity. Other intrauterine parameters measured (numbers of corpora lutea, implantation sites, resorptions and viable fetuses) were not affected by treatment. Arsenic-related malformations were not observed at any exposure concentration.

Mice were fed diets containing As (as H_3AsO_4; 52.76% As) at concentrations of 0, 20, 100 or 500 mg/kg (As

concentrations of 0, 10.55, 52.76 and 263.8 mg/kg, respectively) for two generations (Hazelton Laboratories 1990). Exposure started 14 weeks pre-mating for the first generation and continued through weaning of pups from the F_1 females. At the highest exposure concentration, lower birth weights, postnatal growth retardation and increased postnatal mortality were observed. Dam survival and weight gain were also affected at this exposure concentration. Growth of second generation males and females was significantly reduced at an exposure concentration of 100 mg/kg. With the exception of a few weeks (66 weeks of food consumption measurements, total), food consumption did not differ significantly between the 20 and 100 mg/kg exposure groups and control animals. Body weights of 31.9 g and 33.72 g, and food ingestion rates of 5.82 g/day and 6.11 g/day (reported for the 100 mg/kg and 20 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. Based on the observed growth effects, a LOAEL of 9.63 mg/kgBW/day and a NOAEL of 1.91mg/kgBW/day were identified.

Mice were administered an oral dose of 5 ppm arsenite in drinking water for three generations (Schroeder and Mitchener 1971). No effects on number of litters, age at first litter, interval between litters, dead litters, offspring deaths or runts were observed. Average litter size was smaller, and an increase in ratio of males to females were noted. A LOAEL of 1.26 mg/kgBW/day and an estimated NOAEL of 0.0126 mg/kg BW/day were calculated based on the results of this experiment.

Hazelton Laboratories 1990. Two-Generation Dietary Reproduction Study with Arsenic Acid in Mice. Hazelton Laboratories, Inc. Report HLA 6120-138.

Neiger, R. D. and G. D. Osweiller (1989). Effect of subacute low level dietary sodium arsenite on dogs. Fundam. Appl. Toxicol. 13: 439-451.

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.

Stump, D. G., K. J. Clevidence, J. F. Knapp, J. F. Holson and C. H. Farr (1998). An oral developmental toxicity study of arsenic trioxide in rats. Teratology 57: 216-217.

U. S. EPA 2003. Attachment 4-5, Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs). Eco-SSL Standard Operating Procedure (SOP) #6: Derivation of Wildlife Toxicity Reference Value (TRV). OSWER Directive 92857-55.

U.S. EPA 1988. Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.2.2 Toxicity to Birds

Wildlife TRVs were derived for arsenic according to Eco-SSL guidance (U. S. EPA 2003). A literature search was conducted, and NOAEL and LOAEL values reported for exposure of birds to arsenic were compiled. A geometric mean of the NOAEL values for growth and reproduction could not be calculated, as only two NOAEL values were available. The NOAEL TRV identified for avian wildlife is 2.24 mg/kgBW/day, the lowest reported NOAEL value for reproduction, growth or survival.

Adult mallard ducks were exposed to four concentrations [0, 25, 100 and 400 mg sodium arsenate/kg diet; 51.35% arsenic (As)] for 115 to 128 days (Stanley et al. 1994). Ducklings were placed on the same diet as their parents for 14 days after hatching. At the levels tested in this experiment, dietary As did not affect hatching success or embryo deformities. Duckling production (number of ducklings alive at day 14 for nests producing more than one duckling) was significantly decreased in birds exposed to 400 mg/kg sodium arsenate (205.4 mg/kg As); this exposure concentration was identified as the LOAEL. An ingestion rate of 0.139 kg/day and adult body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the mg/kg diet concentrations to units of 5.7 mg/kgBW/day (NOAEL) and 22.8 mg/kgBW/day (LOAEL).

Mallard ducklings were fed diets containing As (as sodium arsenate) at concentrations of 0, 30, 100 or 300 mg/kg for 10 weeks (Camardese et al. 1990). Dietary exposure to As at a concentration of 300 mg/kg reduced growth in

male ducklings. Growth of female ducklings was significantly different from controls at all dietary exposure concentrations. Based on the ecological significance of the observed effect (reduced growth in female ducklings), an exposure concentration of 30 mg/kg was selected as the LOAEL from this experiment. Body weights of 0.81 and 0.62 kg and ingestion rates of 0.094 and 0.080 kg/day (cited by authors for control and 30 mg/kg 5-week old female ducklings, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 3.9 mg/kgBW/day and an estimated NOAEL of 0.39 mg/kgBW/day were calculated based on results from this experiment.

Thirty-two week old White leghorn hens were fed diets containing arsenic (As from Roxarsone) at nominal concentrations of 0, 11, 22, 44 or 88 mg/kg for four weeks (Chiou et al. 1997). Measured As concentrations in the diet were 1.1, 9.1, 21.2, 43.0 and 85.1 mg/kg. Egg production improved in hens fed diets containing As at a concentration of 9.1 mg/kg. Egg production, egg weight, and food intake were significantly reduced in hens exposed to As at concentrations of 43.0 and 85.1 mg/kg. Hens in the 85.1 mg/kg group ceased to produce eggs after two weeks of exposure. Based on the ecological significance of the observed effect (reproduction), the 43.0 mg/kg exposure concentration was identified as the LOAEL. Body weights of 1.439 and 1.49 kg and ingestion rates of 0.087 and 0.114 kg/day (cited by authors for the 43.0 and 21.2 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgbW/day. A LOAEL of 2.3 mg/kgBW/day and a NOAEL of 1.6 mg/kgBW/day were calculated based on results of this experiment.

Young male cowbirds were fed a diet containing paris green (copper aceto-arsenite) at concentrations of 0, 25, 75, 225 or 625 mg/kg for seven months (U.S. FWS 1969). All of the birds at the two highest exposure concentrations died. Twenty percent of the birds in the 75 mg/kg (33.26 mg/kg As) exposure group died, but no mortality was observed in the 25 mg/kg (11.09 mg/kg As) exposure group. An ingestion rate of 0.01087 kg/day and body weight of 0.049 kg were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 7.38 mg/kgBW/day and a NOAEL of 2.46 mg/kgBW/day were calculated based on results of this experiment. This study was selected to derive a TRV for birds exposed to arsenic for this risk assessment.

Camardese, M. B., D. J. Hoffman, L. LeCaptain and G. W. Pendleton (1990). Effects of arsenate on growth and physiology in mallard ducklings. Environ. Toxicol. Chem. 9(6): 785-796.

Chiou, P. W., K. Chen and B. Yu (1997). Effects of roxarsone on performance, toxicity, tissue accumulation and residue of eggs and excreta in laying hens. Journal of the Science of Food and Agriculture 74(2): 229-236.

Piccirillo, V. J. and R. P. Quesenberry (1980). Reproductive capacities of control mallard ducks (Anas platyrhynchos) during a one-generation reproduction study. Journal of Environmental Pathology and Toxicology 4: 133-139.

Stanley, T. R., Jr.,, J. W. Spann, G. J. Smith and R. Roscoe (1994). Main and interactive effects of arsenic and selenium on mallard reproduction and duckling growth and survival. Arch Environ. Contam. Toxicol. 26: 444-451.

U. S. EPA 2003. Attachment 4-5, Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs). Eco-SSL Standard Operating Procedure (SOP) #6: Derivation of Wildlife Toxicity Reference Value (TRV). OSWER Directive 92857-55.

A.3 Barium

A.3.1 Toxicity to Mammals

Toxicity of barium (Ba) to rats was evaluated by exposing weanling Long-Evans rats to 5 mg/kg Ba as barium acetate in drinking water for life (Schroeder and Mitchener 1975). No adverse effects were observed at this exposure concentration; effects measured included median life-span, longevity, incidence of tumors, serum cholesterol, glucose and uric acid. A slight enhancement of growth was observed. A water ingestion rate of 0.053 L/day and body weight of 0.43 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 0.062 mg/kgBW/day was calculated based on the results of this experiment.

Tardiff et al. (1980) exposed 4-week old Charles River rats to Ba (as barium chloride) in drinking water at

concentrations of 0, 10, 50 or 250 mg/L (0, 1.9, 8.9, and 41.9 mg/kgBW/day, as reported by authors) for 13 weeks. Barium concentration in food was 6.6 micrograms per kilogram (□g/kg), Ba was not detectable in control water, and measured Ba concentrations in water solutions deviated less than 2 % of calculated concentrations. No effects on food consumption, body weight, hematologic parameters, serum ions, serum enzymes, gross pathology or histopathology were observed. The rats exposed to 250 mg/L consumed less water and had a significant decrease in relative adrenal weight when compared to control animals. Based on the lack of ecologically significant effects, the highest exposure dose tested in this experiment was considered a NOAEL (41.9 mg/kgBW/day) in deriving the TRV for Ba.

Perry et al. (1989) evaluated toxicity of Ba in drinking water to female Long-Evans rats exposed to concentrations of 0, 1, 10 or 100 mg/L for 16 months. Rats exposed to 100 mg/L Ba exhibited significant increases in systolic pressure, depressed rates of cardiac contraction, depressed electrical excitability, and lower ATP content in the heart. The ecological significance of the observed effects is not known; therefore, this dose was considered a NOAEL. A water ingestion rate of 0.022 L/day and body weight of 0.435 kg (Perry et al. 1983) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 5.1 mg/kgBW/day was observed in this experiment.

Forty-two day old B6C3F1 mice and Fischer 344/N rats were exposed to barium (as barium chloride dihydrate) in drinking water at concentrations of 0, 125, 500, 1000, 2000 or 4000 mg/L for 92 days (Dietz et al. 1992). No adverse effects were observed in animals exposed at concentrations of 2000 mg/L or less. Mortality was observed in both mice and rats at an exposure concentration of 4000 mg/L, ranging from 60 to 70% in mice and 10 to 30% in rats. Mortality in mice was attributed to treatment-related renal toxicity; renal lesions in rats were much less severe. The daily exposure dose for mice in the 4000 mg/L exposure group ranged from 436 to 562 mg/kgBW/day, while daily exposure doses for rats in the 4000 mg/L exposure group ranged from 121 to 282 mg/kgBW/day (reported by authors). Based on the observed mortality, the lowest measured daily dose within the mice and rats exposed to the 4000 mg/L concentration was selected as the LOAEL (121 mg/kgBW/day for male rats). The observed NOAEL for male rats was 61.1 mg/kgBW/day. These values will be used to evaluate toxicity of barium to mammals in this risk assessment.

Rats were exposed to barium (as barium chloride dehydrate) in drinking water at concentrations of 0, 500, 1250 or 2500 mg/L (equivalent to a dose of 0, 15, 45 or 75 mg/kgBW/day) for 104 weeks (males) or 105 weeks (females) (National Toxicology Program (NTP) 1994). Increased relative kidney weight was observed in females exposed to barium at a concentration of 2500 mg/L. A LOAEL of 75 mg/kgBW/day and a NOAEL of 45 mg/kgBW/day were selected as the mammal TRVs, based on the observed renal effects.

Dietz, D.D., Elwell, M.R., Davis, W.E. Jr., and E.F. Meirhenry. 1992. Subchronic toxicity of barium chloride dihydrate administered to rats and mice in the drinking water. Fund. Appl. Toxicol. 19:527-537.

Perry, H.M., E.F. Perry, M.N. Erlanger and S.J. Koop. 1983. Cardiovascular effects of chronic barium ingestion. In: Proc. 17th Ann. Conf. Trace Substances in Environ. Health, Vol. 17. U. of Missouri Press, Columbia, MO.

Perry, H.M. Jr., S.J. Koop, Perry, E.F. and M.W. Erlanger. 1989. Hypertension and associated cardiovascular abnormalities induced by chronic barium feeding. J. Toxicol. Environ. Health. 28:373-388.

Schroeder, H.A. and M. Mitchener. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105:421-427.

Tardiff, T.G., M. Robinson and N.S. Ulmer. 1980. Subchronic oral toxicity of $BaCl_2$ in rats. J. Env. Path. Tox. 4:267-275.

A.3.2 Toxicity to Birds

One-day old chicks were exposed to barium hydroxide in their diet for four weeks; concentrations evaluated were 250, 500, 1,000, 2,000, 4,000, 8,000, 16,000 and 32,000 mg/kg (Johnson et al. 1960). No mortality was observed in birds exposed at dietary concentrations up to 2,000 mg/kg. Mortality was observed in all groups exposed to concentrations of 4,000 mg/kg as barium (Ba) and higher. Half the birds in the 8,000 mg/kg exposure group died,

and all of the birds in the two highest exposure groups died. Growth was significantly depressed in chicks fed barium at dietary concentrations of 4,000 and 8,000 mg/kg. A LOAEL of 41.7 mg/kgBW/day (4,000 mg/kg) and a NOAEL of 20.8 mg/kgBW/day (2,000 mg/kg) were calculated based on results of this experiment.

Johnson, D. Jr., A.L. Mehring, Jr. and H.W. Titus. 1960. Tolerance of chickens for barium. Proc. Soc. Exp. Biol. Med. 104:436-438.

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. EPA

A.4 Cadmium

A.4.1 Toxicity to Mammals

Male and female weanling brown rats were fed a control diet, a diet containing 5 mg/kg of Cd as cadmium chloride ($CdCl_2$), or fed the control diet and given drinking water which contained Cd at a concentration of 5 mg/L for 18 months (Pribble and Weswig 1973). Growth was measured for 10 weeks following exposure to Cd. Growth rate of male rats exposed to Cd via drinking water was significantly less control animals; no effects on growth were observed in females or males exposed via diet. Exposure concentrations were converted to units of mg/kgBW/day using a food ingestion rate of 0.016 kg/day, a water ingestion rate of 0.0245 L/day, and a body weight of 0.152 kg (U.S. EPA 1988). A LOAEL of 0.8 mg/kgBW/day was calculated for water exposure, and a NOAEL of 0.53 mg/kgBW/day was calculated for exposure via diet based on results of this experiment. This study was not used to derive a TRV, as growth was the only parameter measured and growth is not considered and ecologically relevant endpoint.

Female mice were fed diets containing Cd at concentrations of 0.25, 5, or 50 mg/kg for 252 days (Bhattacharyya et al. 1988). One-half of the females were bred for 6 consecutive rounds of pregnancy/lactation, while the remaining females served as non-pregnant controls. Significant decreases in body weight, femur Ca content, and femur Ca/dry weight ratio were observed in the pregnant mice fed Cd at 50 mg/kg. An ingestion rate of 0.0058 kg/day and adult body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 8.8 mg/kgBW/day and a NOAEL of 0.88 were calculated based on the results of this experiment.

Male bank voles were fed diets containing cadmium (as $CdCl_2$) at concentrations of 0.27 (control), 15 or 40 mg/kg for six months (Swiergosz et al. 1998). No effects on body or organ weights were observed at any exposure concentration. High mortality was observed in both exposure groups; 54% in the 15 mg/kg group and 40% in the 40 mg/kg group (compared to 14% in the control group). Kidneys from both cadmium exposed groups showed degeneration of proximal tubules, fenestration, and albuminuria. The number of mature spermatozoa was lower in both cadmium exposed groups. Based on the observed mortality, an exposure concentration of 15 mg/kg was selected as the LOAEL in the experiment. A body weight of 0.035 kg (cited by authors) and a food ingestion rate of 0.0055 kg/day (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.36 mg/kgBW/day and an estimated NOAEL of 0.236 mg/kgBW/day were calculated based on results of this experiment.

Male Wistar rats were fed diets containing Cd [as either cadmium chloride or cadmium-metallothionein; (CdMt)] at concentrations of 0, 0.3, 3, 30 or 90 mg/kg for 10 months (Groten et al. 1994). In rats fed cadmium chloride, kidney Cd concentrations were higher than in rats fed the same doses as CdMt. Histopathological effects (Glomerulonephrosis and basophilic tubules) were observed in rats fed cadmium chloride at concentrations of 30 and 90 mg/kg, and CdMt at a concentration of 90 mg/kg. Nephrotoxicity was mainly related to total renal Cd concentration. An ingestion rate of 0.027 kg/day and adult body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.3 mg/kgBW/day and a NOAEL of 0.23 were calculated based on the results of this experiment.

Cadmium was toxic to mice administered an oral dose of 10 ppm Cd in drinking water (Schroeder and Mitchener 1971). Numerous abnormalities were observed in offspring, and 60 % of the second generation breeding pairs failed to breed. A water ingestion rate of 0.0075 L/day and adult body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.3 mg/kg BW/day and an estimated

NOAEL of 0.23 mg/kg BW/day were calculated based on the results of this experiment. Based on the ecological significance of the endpoint (reproduction) and because the LOAEL is the lowest cited adverse effect level for mammals, the TRV values from this study will be used to evaluate the risk posed by Cd to mammalian receptors.

Weanling white Charles River mice were given cadmium in drinking water at a concentration of 5 mg/L for 400 days (Schroeder et al. 1963). Cadmium had no effect on growth, mature weights, or survival up to 18 months of age. At 21 months of age, mortality of cadmium-exposed males was significantly higher than mortality of control mice. A body weight of 0.0515 kg (cited by authors) and a water ingestion rate of 0.0069 L/day (calculated using allometric equation from (Calder and Braun 1983) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 0.67 mg/kgBW/day was calculated based on results of this experiment.

Sprague-Dawley rats were administered Cd (as $CdCl_2$) orally at concentrations of 0, 0.1, 1.0 and 10 mg/kgBW/day for 6 weeks (Sutou et al. 1980a; Sutou et al. 1980b). After this, the animals were mated for 3 weeks. Females were administered Cd during gestation and sacrificed on day 20 for fetal examination. Number of total implantations and live fetuses decreased significantly, and number of resorbed fetuses increased significantly in the group exposed to Cd at a concentration of 10 mg/kgBW/day. No significant effects were observed in the group exposed to 1.0 mg Cd/kgBW/day. A LOAEL of 10 and a NOAEL of 1.0 were identified from this experiment. This study was selected to derive a TRV for this risk assessment.

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A.4.2 Toxicity to Birds

Juvenile mallard drakes were fed diets containing 0, 50, 150, or 450 mg/kg of cadmium (Cd) for 42 days (Di Giulio and Scanlon 1985). An ingestion rate of 0.0578 kg/day and a body weight of 0.936 kg (Sugden et al. 1981) were used to convert the exposure concentrations to units of mg/kgBW/day. Significant metabolic effects were seen only in the 450 mg/kg (27.8 mg/kg BW/day) treatment group. Birds exposed to Cd at this concentration exhibited 20.3 % decrease in body weight, 26 % decrease in liver weight, 15 % increase in kidney weight, 21 % decrease in liver aldolase activity, 46 % increase in plasma uric acid concentrations, 74 % decrease in plasma triiodothyronine concentrations, 28 % increase in adrenal weights, and 31 % increase in adrenal cortisone concentrations. Ducks in the 150 mg/kg (9.3 mg/kg BW/day) treatment group also exhibited 12 % increase in kidney weight and 23 % increase in adrenal weight. No adverse effects were observed at a dietary concentration of 50 mg/kg (3.1 mg/kg BW/day).

Mallard ducklings were fed 0, 5, 10, or 20 mg/kg of Cd in their diet from 1 day of age to 12 weeks of age (Cain et al. 1983). An ingestion rate of 0.0578 kg/day and a body weight of 0.936 kg (Sugden et al. 1981) were used to convert the exposure concentrations to units of mg/kgBW/day. Ducklings receiving 20 mg/kg (1.2 mg/kg BW/day) Cd exhibited an 8 % decrease in packed cell volume, a 6 % reduction in hemoglobin concentration, and a 52 % increase in serum glutamic pyruvic transaminase activity, all of which were statistically significant at the 0.05 level. Necropsies of these specimens revealed mild to severe kidney lesions, and whole body Cd concentrations ranged from 58.30 to 65.30 mg/kg, dry weight. No significant adverse effects were noted at a dietary Cd level of 10 mg/kg (0.7 mg/kg BW/day).

Male Japanese quail that were fed a diet containing 75 mg/kg (11.3 mg/kg BW/day) of Cd as cadmium chloride for four weeks exhibited a 62 % decrease in testis size, a lack of spermatogenesis, damage to small intestine mucosa, and severe anemia (Richardson et al. 1974). These responses were associated with a mean liver Cd concentration of 42 mg/kg. An ingestion rate of 0.018 kg/day and a body weight of 0.12 kg (Varghese 2000) were used to convert the exposure concentration to units of mg/kgBW/day. A five day exposure/three day post-exposure LC_{50} of 1,496 mg/kg Cd was calculated for this species (Hill and Camardese 1986).

Domestic hens fed a diet containing 50 mg/kg (3.6 mg/kg BW/day) and 100 mg/kg (7.1 mg/kg BW/day) Cd also exhibited reduced egg production and egg weight (Anke et al. 1970). A concentration of 200 mg/kg (14.2 mg/kg BW/day) Cd resulted in ceased egg production within two days. An ingestion rate of 0.103 kg/day and a body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day.

Juvenile (two week old) male Leghorn chickens were fed a diet ad libitum containing various concentrations of Cd in two separate experiments (Pritzl et al. 1974). The first experiment used Cd concentrations of 0, 400, 600, 800, and 1,000 mg/kg for 20 days; whereas, the second experiment used Cd concentrations of 0 and 700 mg/kg for 20 days. A significant reduction in growth rate and feed consumption was noted at a dietary concentration of 400 mg/kg (39.4 mg/kgBW/day). An LD_{50} of 565 mg/kg (55.6 mg/kg BW/day) was calculated from the second experiment. An ingestion rate of 0.019 kg/day and a body weight of 0.193 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day.

Leghorn chickens were fed a diet containing cadmium sulfate for a period of 48 weeks. Egg production and eggshell thickness decreased significantly in hens exposed to a dietary concentration of 48 mg/kg, but were not affected at an exposure concentration of 12 mg/kg (Leach et al. 1979). This study was used to develop the LOAEL and NOAEL values because of the long exposure period and the ecological significance of the endpoints. An ingestion rate of 0.103 kg/day and adult body weight of 1.45 kg (U.S. EPA 1988) were used to convert the mg/kg diet concentrations to units of mg/kgBW/day. A LOAEL of 3.4 mg/kg BW/day and a NOAEL of 0.85 mg/kg BW/day Cd were calculated based on the results of this experiment.

Adult (one year old) male and female mallard ducks were fed a diet containing 0.08, 1.6, 15.2, and 210 mg/kg, wet weight of Cd as cadmium chloride ad libitum for 90 days (White and Finley 1978). Male testis weight, male kidney weights, and egg production by females were significantly less in the 210 mg/kg treatment when compared with the controls. No adverse effects were observed at dietary concentrations of 15.2 mg/kg or less. A LOAEL of 20 mg/kgBW/day and a NOAEL of 1.45 mg/kgBW/day were calculated based on results of this experiment. This study was selected to derive a TRV for this risk assessment.

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A.5 Chromium

A.5.1 Toxicity to Mammals

Trivalent Chromium

Rats (BD strain) were fed diets containing chromic oxide (Cr_2O_3; 68.42% Cr) at concentrations of 0, 1%, 2% or 5% for two years (Ivankovic and Preussmann 1975). Groups of 60 male and female rats were fed chromic oxide baked in bread five days per week for two years; they received the control diet with a vegetable supplement each weekend. Measurement endpoints were mortality, longevity, organ weight, and type and incidence of tumors. No significant differences in mortality, longevity, or type and frequency of tumors were observed at any exposure concentration. A dose-dependant reduction in liver and spleen weight was observed; however, no macroscopic or histological changes were observed in these organs. Rats exposed at a concentration of 5% consumed an average of 1,800 g Cr_2O_3/kgBW over the 600-day exposure period (reported by authors); an exposure concentration of 3 g Cr_2O_3/kgBW/day or 2.053 g Cr/kgBW/day was calculated. A NOAEL of 2,053 mg/kgBW/day was identified from this experiment.

A mammalian TRV was derived for trivalent chromium according to Eco-SSL guidance (U. S. EPA 2008).A literature search was conducted, and NOAEL and LOAEL values reported for exposure of mammals to chromium were compiled. To derive a wildlife TRV, a minimum of three NOAEL or LOAEL results reported for a least two test species for either growth, reproduction, or survival effects are required. A geometric mean is calculated if three NOAEL results are available for either growth or reproduction effect groups. For chromium, the geometric mean of the NOAEL values for growth and reproduction was identified as the Eco-SSL TRV for mammals. A NOAEL value of 2.40 mg/kgBW/day was identified.

Hexavalent Chromium

A mammalian TRV was derived for hexavalent chromium according to Eco-SSL guidance (U. S. EPA 2008).A literature search was conducted, and NOAEL and LOAEL values reported for exposure of mammals to chromium

were compiled. To derive a wildlife TRV, a minimum of three NOAEL or LOAEL results reported for a least two test species for either growth, reproduction, or survival effects are required. A geometric mean is calculated if three NOAEL results are available for either growth or reproduction effect groups. For chromium, the geometric mean of the NOAEL values for growth and reproduction was identified as the Eco-SSL TRV for mammals. A NOAEL value of 9.24 mg/kgBW/day was identified.

Adult albino Swiss female mice were exposed to Cr^{+6} in drinking water at concentrations of 0, 250, 500 and 750 mg/L during days 14 to 19 of pregnancy (Junaid et al. 1995). Gestational weight gains and skeletal ossification in fetuses were significantly less for mice in the two highest exposure concentration groups than in control mice. Post-implantation losses and fetal abnormalities were significantly higher in the 500 and 750 mg/L exposure groups. An ingestion rate of 0.0075 L/day and a body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 114 mg/kgBW/day and a NOAEL of 56.8 were calculated based on the results of this study.

Adult female mice were administered Cr^{+6} in drinking water at concentrations of 0, 250, 500 and 750 mg/L during organogenesis (days 6 to 14 of gestation; Junaid et al. 1996). Maternal weight gain, number of fetuses per litter, and fetal weight were significantly lower than controls in the two highest exposure groups; number of resorption sites were significantly higher. A mean body weight of 0.038 kg (cited by authors) and water ingestion rate of 0.0075 L/day (500 mg/L exposure group; U.S. EPA 1988) and 0.0073 L/day (250 mg/L exposure group; cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 98.7 mg/kgBW/day and a NOAEL of 48.0 mg/kgBW/day were calculated based on the results of this study.

Adult female mice were exposed to Cr^{+6} in drinking water at concentrations of 0, 250, 500 and 1,000 mg/L throughout their entire gestation period (Trivedi et al. 1989). Resorptions and postimplantation losses were significantly higher in the 250 mg/L exposure group as compared to controls; a reduction in cranial ossification was also observed. Significant skeletal abnormalities and embryotoxic effects were observed in the 500 mg/L exposure group; no implantation sites were found in the highest exposure group. Based on the reproductive effects observed at an exposure concentration of 250 mg/l, this dose was identified as a LOAEL. An ingestion rate of 0.0075 L/day and a body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 56.8 mg/kgBW/day and an estimated NOAEL of 5.68 were calculated based on the results of this study. Based on the ecological significance of the endpoint (reproduction) and because the LOAEL is the lowest cited adverse effect level for mammals, the TRV values from this study will be used to evaluate the risk posed by Cr to mammalian receptors.

Sprague-Dawley albino rats were exposed to chromium (as potassium chromate) in drinking water at concentrations of 0, 0.45, 2.2, 4.5, 7.7, 11 or 25 mg/L for one year (Mackenzie et al. 1958). No differences in food consumption, weight gain, mortality, or pathological tissue changes were observed in the Cr-exposed group. A water ingestion rate of 0.058 L/day and body weight of 0.48 kg (U.S. EPA 1988) was used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 3.02 mg/kgBW/day was calculated based on this experiment.

The LOAEL TRV for exposure of mammals to chromium selected for use in this risk assessment is based on a study cited in Sample et al. (1996). Rats were exposed to hexavalent chromium at concentrations of 0, 134, or 1000 mg/L for three months. Increased mortality was observed in the group exposed to 1000 mg/kg chromium, which was considered a subchronic LOAEL. A chronic LOAEL was calculated using a subchronic-chronic uncertainty factor of 0.1. A water ingestion rate of 0.046 L/day and body weight of 0.35 kg (U.S. EPA 1988) was used to convert the exposure concentrations to units of mg/kgBW/day, resulting in a LOAEL TRV of 13.14 mg/kgBW/day.

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A.5.2 Toxicity to Birds

Organic Chromium

One-week old broiler chicks were fed diets containing organic chromium (as either Cr picolinate or as yeast Cr) at concentrations of 0, 200 or 400 μg/kg for 5 weeks (Motozono et al. 1998). Growth of chicks fed yeast Cr at a concentration of 400 μg/kg was significantly depressed compared to growth of chicks fed the control diet. Body weights of 1.007 kg and 0.9706 kg and ingestion rates of 0.103 kg/day and 0.1026 kg/day (cited by authors for the 200 and 400 μg/kg yeast Cr groups, respectively) were used to convert the mg/kg diet concentrations to units of mg/kgBW/day. A LOAEL of 0.042 mg/kgBW/day and a NOAEL of 0.021 mg/kgBW/day were calculated based on results of this experiment.

Trivalent Chromium

An avian TRV was derived for trivalent chromium according to Eco-SSL guidance (U. S. EPA 2008).A literature search was conducted, and NOAEL and LOAEL values reported for exposure of birds to chromium were compiled. To derive a wildlife TRV, a minimum of three NOAEL or LOAEL results reported for a least two test species for either growth, reproduction, or survival effects are required. A geometric mean is calculated if three NOAEL results are available for either growth or reproduction effect groups. For chromium, the geometric mean of the NOAEL values for growth and reproduction was was identified as the Eco-SSL TRV value; a NOAEL value of 2.66 mg/kgBW/day was identified.

Heinz and Haseltine (1981) exposed two to three year old breeding pairs of black ducks (Anas rubripes) to a diet containing 0, 20, or 200 mg/kg, wet weight, (0, 2, or 20 mg/kg BW/day) of Cr^{+3} as chromium potassium sulfate for a period of approximately five months (until the onset of egg-laying by the females). Hatched ducklings were then fed a mash diet containing the same concentrations as their parents. Seven-day old chicks were tested for avoidance behavior in response to a fright stimulus; none of the Cr concentrations resulted in alteration of avoidance behavior. An ingestion rate of 0.125 kg/day (Heinz et al. 1989) and adult body weight of 1.25 kg (Dunning 1993) were used to convert the mg/kg diet concentrations to units of mg/kgBW/day. A NOAEL of 20 mg/kgBW/day was identified from this experiment.

Chung et al. (1985) evaluated the dietary toxicity of Cr^{+3} to young chickens, and the effects of manganese (Mn) and molybdenum (Mo) on the toxicity of chromium (Cr). Day-old broiler chicks were exposed to Cr^{+3} as chromium sulfate at concentrations of 0 and 4,000 mg/kg for 2 weeks. No mortality was observed in chicks fed Cr^{+3}, however growth was significantly depressed (LOAEL of 4,000 mg/kg Cr^{+3}). An ingestion rate of 0.0075 kg/day and body weight of 0.066 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day[1]. A LOAEL of 455 mg/kgBW/day and an estimated NOAEL of 45.5 mg/kgBW/day were calculated for Cr^{+3} based on the results of this study.

[1] An ingestion rate and body weight for 7-day old chicks was used to estimate contaminant exposure. While this will over- and under estimate food consumption by younger and older chicks, it was assumed to approximate food consumption throughout the 2 week study.

Chicks were fed diets containing trivalent chromium at concentrations of 0, 500, 1500, 2500 or 4000 mg/kg for five weeks (Kang et al. 1980)[2]. Growth of chicks exposed to Cr^{+3} at concentrations of 1500 mg/kg and higher was significantly lower than growth of chicks fed the control diet. An ingestion rate of 0.0075 kg/day and body weight of 0.066 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day[3]. A LOAEL of 170.5 mg/kgBW/day and a NOAEL of 56.8 mg/kgBW/day were calculated for Cr^{+3} based on the results of this study.

Hexavalent Chromium

Chung et al. (1985) evaluated the dietary toxicity of Cr^{+6} to young chickens, and the effects of manganese (Mn) and molybdenum (Mo) on the toxicity of chromium (Cr). Day-old broiler chicks were exposed to Cr^{+6} as potassium chromate in the diet at concentrations of 0, 900, 1,200 and 1,500 mg/kg for 2 weeks. Mortality was significantly greater and growth was depressed in all chicks exposed to Cr^{+6} (LOAEL of 900 mg/kg Cr^{+6}). Addition of Mn or Mo at 500 mg/kg significantly reduced the mortality caused by the Cr^{+6}, but did not prevent the growth depression observed at the two highest Cr doses. Tissue concentrations of Cr were higher in chicks fed diets containing only Cr^{+6}, indicating Mn and Mo may interfere with Cr absorption. An ingestion rate of 0.0075 kg/day and body weight of 0.066 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day[4]. A LOAEL of 102.3 mg/kgBW/day and an estimated NOAEL of 10.2 mg/kgBW/day were calculated for Cr^{+6} based on the results of this study.

Chicks were fed diets containing hexavalent chromium (as potassium chromate) at concentrations of 0, 200, 400 or 600 mg/kg for eight weeks (Kang et al. 1980)[5]. Growth and mortality of chicks exposed to Cr^{+6} at concentrations of 400 and 600 mg/kg was significantly lower than growth of chicks fed the control diet. An ingestion rate of 0.025 kg/day and body weight of 0.27 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day[6]. A LOAEL of 37 mg/kgBW/day and a NOAEL of 18.5 mg/kgBW/day were calculated for Cr^{+6} based on the results of this study.

Chicks were fed diets containing hexavalent chromium at concentrations of 0, 300, 600 or 900 mg/kg for five weeks (Choi et al. 1981)[7]. Growth of chicks exposed to Cr^{+6} at any concentration was significantly lower than growth of chicks fed the control diet. Mortality was significantly higher in chicks fed diets containing Cr^{+6} at concentrations of 600 and 900 mg/kg. An ingestion rate of 0.0075 kg/day and body weight of 0.066 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day[8]. A LOAEL of 34.1 mg/kgBW/day and an estimated NOAEL of 3.41 mg/kgBW/day were calculated for Cr^{+6} based on the results of this study.

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[2] Most of this article was written in Korean. The abstract and Tables were in English.

[3] An ingestion rate and body weight for 21-day old chicks was used to estimate contaminant exposure. While this will over- and under estimate food consumption by younger and older chicks, it was assumed to approximate food consumption throughout the 5 week study.

[4] An ingestion rate and body weight for 7-day old chicks was used to estimate contaminant exposure. While this will over- and under estimate food consumption by younger and older chicks, it was assumed to approximate food consumption throughout the 2 week study.

[5] Most of this article was written in Korean. The abstract and Tables were in English.

[6] An ingestion rate and body weight for 28-day old chicks was used to estimate contaminant exposure. While this will over- and under estimate food consumption by younger and older chicks, it was assumed to approximate food consumption throughout the 8 week study.

[7] Most of this article was written in Korean. The abstract and Tables were in English.

[8] An ingestion rate and body weight for 21-day old chicks was used to estimate contaminant exposure. While this will over- and under estimate food consumption by younger and older chicks, it was assumed to approximate food consumption throughout the 5 week study.

Mature male Japanese quail were fed diets containing hexavalent chromium (as potassium dichromate) at concentrations of 0 or 142 mg/kg for 12 weeks (Butkauskas and Sruoga 2004). Hatchability of eggs fertilized by Cr-exposed males decreased 14%, and early embryo mortality doubled. An ingestion rate of 0.018 kg/day and body weight of 0.12 kg (Varghese 2000) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 21.3 mg/kgBW/day and an estimated NOAEL of 2.13 mg/kgBW/day were calculated based on results of this experiment.

Haseltine et al. (1985), in an unpublished study reported by Eisler (1986), fed black ducks diets containing 10 or 50 mg/kg anionic Cr^{+3} as Cr $K(SO_4)_2 \cdot 12H_20$ for five months. No effects were observed on survival, reproduction and blood chemistry. Ducklings produced by the treated groups were fed diets containing Cr at the original parental dosages; there was a significant reduction in survival in the 50 mg/kg exposure group. An ingestion rate of 0.125 kg/day (Heinz et al. 1989) and adult body weight of 1.25 kg (Dunning 1993) were used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 1 mg/kg BW/day and LOAEL of 5 mg/kg BW/day were calculated based on the results of this experiment. Based on the ecological significance of the endpoint (survival) and because the LOAEL is the lowest cited adverse effect level for birds, the TRV values from this study will be used to evaluate the risk posed by Cr to avian receptors.

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A.6 Copper

A.6.1 Toxicity to Mammals

Lecyk (1980) exposed mice to six dietary levels of copper sulfate [0, 0.5, 1.0, 1.5, 2.0, 3.0, and 4.0 grams per kilogram (g/kg)] for one month prior to mating. Mice were maintained on diets throughout day 19 of pregnancy, at which time females were killed and embryos were examined. At dietary exposure levels as high as 2 g/kg diet, no adverse effects on percent survival, litter size, fetal weight or number of abnormalities were found. Adverse effects on all measured endpoints were observed at a dietary exposure concentration of 3 g/kg. An ingestion rate of 0.0058 kg/day and adult body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 527 mg/kg BW/day and a NOAEL of 352 mg/kg BW/day were calculated based on the results of this study.

Mink were fed diets supplemented with 0, 25, 50, 100 or 200 mg/kg Cu (as $CuSO_4 \cdot 5H_20$) for 357 days; the control diet contained 60.5 mg/kg Cu (Aulerich et al. 1982). The Cu supplemented diets had no observed toxic effects on the adult animals. Percent mortality of kits from birth to four weeks of age was significantly greater in kits from female mink fed 100 or 200 mg/kg supplemental Cu. Because of the ecological significance of the endpoints and the exposure duration, results from this study were used to develop the LOAEL and NOAEL values used in this risk assessment. Because of the ecological significance of the endpoints and the exposure duration, results from this study were used to develop the LOAEL and NOAEL values used in this risk assessment. A LOAEL of 15.14 mg/kg BW/day and a NOAEL of 11.7 mg/kg BW/day were calculated based on results of this experiment.

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A.6.2 Toxicity to Birds

Mayo et al. (1956) evaluated Cu tolerance of young chickens. Growth, mortality and occurrence of muscular dystrophy were measured. In one experiment, a dietary concentration of 324 mg/kg resulted in muscular dystrophy and a significant inhibition of growth at 4 weeks of age. In another experiment, dietary Cu at a concentration of 520 mg/kg caused reduced growth at 4 weeks of age, but body weights of Cu-exposed birds were equal to controls at 8 weeks of age. When Cu was added as copper-bound casein instead of as copper sulfate, growth depression was not observed, but the incidence of muscular dystrophy remained the same. Significant mortality was observed in chicks fed diets containing Cu at a concentration of 1,270 mg/kg. This paper was only presented as an abstract, and it was not clear whether the chicks in the two experiments where growth inhibition was observed received different diets or different forms of supplemental Cu. No methods were presented to evaluate sample size, experimental design, or statistical analysis, therefore this study was not used to derive a TRV for Cu.

A flock of 51-week old leghorn hens experienced a 16% decrease in egg production in a single week (Gilbert et al. 1996). Analysis of the feed showed a Cu concentration of 1,477 mg/kg. Severe oral ulcers were present in the pharynx of the hens. To confirm that the observed ulcers and decrease in egg production were due to excess dietary Cu, a group of 35 hens were fed diets containing Cu (as copper sulfate) at a concentration of 1,437 mg/kg diet for 2 weeks; the control group received a basal diet with a Cu concentration of 78 mg/kg. Egg production and food consumption were significantly lower in hens fed the high-Cu diet than in hens fed the basal diet. Pharyngeal lesions and gizzard erosions were significantly increased in hens fed the high Cu diet. A body weight of 1.45 kg (U.S. EPA 1988) and food ingestion rates of 0.129 kg/day (basal diet group) and 0.060 kg/day (1,437 mg/kg group, cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 59.5 mg/kgBW/day and a NOAEL of 6.9 mg/kgBW/day were calculated based on the results of this experiment.

Jackson (1977) exposed adult hens to dietary concentrations of Cu ranging from16 mg/kg (control diet) to 1,936 mg/kg for 35 days. No effects on body weight were observed at an exposure concentration of 256 mg/kg, while exposure to Cu at a concentration of 496 mg/kg caused marked body-weight loss. Egg production was not affected at an exposure concentration of 496 mg/kg, but it significantly decreased at an exposure concentration of 976 mg/kg. Because of the ecological significance of the reproductive endpoint (egg production), these values were used to develop a NOAEL (496 mg/kg) and LOAEL (976 mg/kg) in this assessment. Ingestion rates and body weights for each treatment group cited by the author were used to convert the exposure concentrations to units of mg/kgBW/day (0.101 kg/day and 1.73 kg, and 0.082 kg/day and 1.62 kg, respectively). A LOAEL of 49.4 mg/kgBW/day and a NOAEL of 28.9 mg/kg BW/day were calculated based on the results of this experiment.

White leghorn layers were fed diets supplemented with 0, 200, 400, 600, or 800 mg/kg copper sulfate for four weeks (Chiou et al. 1997). Measured dietary Cu concentrations were 27, 195, 405, 598 and 758 mg/kg. Food intake, egg production, liver function, and Cu residues in the liver, egg and excreta were measured. Food intake and egg production were significantly lower than that measured for control birds for the 598 and 758 mg/kg exposure groups. Liver enzyme activities were significantly higher in birds exposed at dietary concentrations of 598 and 758 mg/kg. Histological examination of livers showed bile duct proliferation and lymphocyte infiltration in livers of birds exposed at a concentration of 758 mg/kg. Copper residues in liver and excreta increased significantly as exposure concentration increased; egg concentrations peaked at an exposure concentration of 405 mg/kg. Based on reduced egg production, the exposure concentration of 598 mg/kg was selected as the LOAEL for this experiment. Food ingestion rates of 0.097 and 0.077 kg/day and body weights of 1.461 and 1.393 kg (cited by authors for the 405 and 598 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 33.2 mg/kg BW/day and a NOAEL of 26.9 mg/kg BW/day were calculated. Based on the ecological significance of the endpoint (reproduction) and because the LOAEL is the lowest cited adverse effect level for birds, the TRV values from this study will be used to evaluate the risk posed by Cu to avian receptors.

One-day old New Hampshire chicks were fed diets containing supplemental Cu (as copper oxide) at nominal concentrations of 0, 10.8, 26, 47.5, 78, 121.1, 182, 268.1, 390, 562.2, 806, 1,150.4 mg/kg diet for 10 weeks (Mehring et al. 1960). The basal diet contained 26 mg Cu/kg, and the four highest exposure diets were analyzed and found to contain Cu at concentrations of 403, 570, 749 and 1,180 mg/kg. The average live weight of chicks that received the diets containing 570, 749 and 1,180 mg/kg were 94.0%, 70.1% and 51.0% of the live weight of chicks fed the basal diet, respectively. Mortality rates of 15 and 40% were observed in the two highest exposure groups. After six weeks of feeding on the basal diet, the average live weight of chicks in the three highest exposure groups were 100.5%, 86.4%, and 83% of the live weights of chicks that received the basal diet for the duration of the experiment. Based on decreased growth (70.1% of control bird weight after 10 week exposure), an exposure concentration of 749 mg/kg diet was identified as the LOAEL in this experiment. A LOAEL of 61.7 mg/kgBW/day and a NOAEL of 47 mg/kgBW/day were identified based on the results of this experiment. This study was selected to derive a TRV for this risk assessment.

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A.7 Lead

A.7.1 Toxicity to Mammals

Adult raccoons (one pair per exposure concentration) were administered oral doses of lead acetate at concentrations of 0, 1, 2 or 4 mg/kgBW/day, five days per week, for eight weeks (Hamir et al. 1999). No clinical signs of lead toxicosis were observed at any exposure concentration. Renal tubular degeneration and acid-fast intranuclear inclusions were observed in raccoons exposed at concentrations of 2 and 4 mg/kgBW/day. This study was not used to derive a TRV for this risk assessment, as the ecological significance of the kidney abnormalities observed is not known.

Male CF-1 mice were given lead acetate in their drinking water at concentrations of 0, 2500 mg/L, or 5000 mg/L for six weeks (Wadi and Ahmad 1999). Exposure to lead at a concentration of 5000 mg/L significantly decreased seminal vessicle and epididymis weight, the number of sperm within the epididymis, and the percent of sperm that were motile. A significant increase in the number of abnormal sperm within the epididymis was also observed. A water ingestion rate of 0.0089 L/day (U.S. EPA 1988) and body weight of 0.038 kg (cited by authors) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 1,171 mg/kgBW/day and a NOAEL of 586 mg/kgBW/day were calculated based on the results of this experiment.

Female Sprague-Dawley rats were given lead acetate in their drinking water at concentrations of 0 (control), 0 (group 2) or 7000 (group 1) mg/L starting on day 1 of gestation (Thoreux-Manlay et al. 1995). At birth, the prenatally exposed litters from the group 1 females were cross-fostered by the untreated group 2 females. Pups from the group 2 females were cross-fostered by the group 1 dams, which continued to receive lead in their drinking water. Growth of pups exposed to lead prenatally or via lactation was significantly reduced at 22 days of age, but did not differ from controls at 97 days of age. Sertoli cell function (number of spermatozoa) was decreased in both groups of lead-exposed rats, but fertility was not significantly affected. The adverse effect on growth of young was identified as an ecologically significant effect. A water ingestion rate of 0.058 L/day and body weight of 0.48 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 846 mg/kgBW/day and an estimated NOAEL of 84.6 mg/kgBW/day were calculated based on the results of this experiment.

Female mice were fed diets containing lead at concentrations of 0, 0.125, 0.25, 0.5 or 1% from the day of the vaginal plug for 16 to 18 days (Leonard et al. 1982). At an exposure concentration of 0.5%, the number of pregnancies (defined as the number of females having at least one implant) decreased and the number of postimplantation deaths increased significantly. A food ingestion rate of 0.0064 kg/day and body weight of 0.038 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. . A LOAEL of 842 mg/kgBW/day and a NOAEL of 421 mg/kgBW/day were calculated based on the results of this experiment.

Sprague-Dawley rats were given lead acetate in their drinking water at a concentration of 0 or 6000 mg/L from gestation day 5 through weaning (Ronis et al. 1996). Nineteen percent of the lead-exposed pups were stillborn, and pup weight at five days of age was significantly lower than weight of control pups. Prepubertal growth was suppressed in male and female lead-exposed rats. Pubertal growth rate was significantly decreased in males, however postpubertal growth was not different from growth of control rats. The adverse effect on reproduction and growth of pups were identified as ecologically significant effects. A water ingestion rate of 0.058 L/day and body weight of 0.48 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 725 mg/kgBW/day and an estimated NOAEL of 72.5 mg/kgBW/day were calculated based on the results of this experiment.

Adult pregnant mice (C57Bl strain) were fed a diet containing Pb at concentrations of 0, 0.125, 0.25, 0.5, or 1 % for 48 hours following observation of the presence of a vaginal plug (Jacquet et al. 1976). Dietary Pb concentrations of 0.125 %, 0.25 %, and 0.5 % resulted in an increase in the number of embryos in the four-cell stage versus the eight-cell stage. At a dietary exposure level of 1 %, an increase in the number of undivided embryos was observed. In normal mouse embryo development, the embryo is in the eight-cell stage after 48 hours and is placed near the end of the oviduct ready to be discharged to the uterus. Effects of delayed cleavage on embryo loss prior to implantation is not known. An ingestion rate of 0.0058 kg/day and adult body weight of 0.033 kg (U.S. EPA 1988) were used to

convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 220 mg/kg BW/day (1,250 mg/kg), and an estimated NOAEL of 22 mg/kg BW/day were calculated based on the results of this experiment.

Pregnant female mice were given lead acetate in their drinking water at concentrations of 0, 500, 750 and 1,000 mg/L starting on gestation day 12 and continuing to four weeks postpartum (Waalkes et al. 1995). Offspring were weaned and received Pb in their drinking water after weaning for 112 weeks. Renal lesions (atypical tubular hyperplasia or tumors) occurred rarely in control male mice (4 %) and increased in dose related fashion for Pb exposed male offspring: 500 ppm, 16 %; 750 ppm, 24 %; and 1,000 ppm, 48 %. The number of lesions in the 1,000 mg/L group was significantly higher than for the control group. Lead-treated females also developed renal lesions, but at much lower rates. An ingestion rate of 0.0058 kg/day and adult body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 176 mg/kg BW/day (1000 mg/kg), and a NOAEL of 132 mg/kg BW/day were calculated based on results of this study. This study was not used to derive a TRV for this risk assessment, as the ecological significance of the kidney abnormalities observed is not known.

To evaluate the effect of exposure to lead on the development of the male reproductive system, female Wistar rats were given lead acetate in their drinking water at concentrations of 0 or 300 mg/L starting day 1 of pregnancy and continuing through lactation until pups were 21 days of age (Corpas et al. 2002). Exposure to lead resulted in a significant decrease in testis and seminal vesicle weights of male pups. The diameter of seminiferous tubules, and the number of spermatogonia and spermatocytes were also significantly reduced in lead-exposed pups compared to controls. The in utero exposure concentration was converted to units of mg/kgBW/day using a body weight of 0.275 kg (cited by authors) and a water ingestion rate of 0.0435 mg/day (U.S. EPA 1988). A LOAEL of 47.5 mg/kgBW/day was and an estimated NOAEL of 4.75 mg/kgBW/day were calculated for this experiment. The biological significance of a decrease in number of sperm in rats is not known. Rats produce large numbers of sperm, and a decrease in number has not been shown to affect fertility.

Lead was administered in drinking water to two different age groups of albino mice at concentrations of 0 or 800 mg/L for 11 weeks before mating (Sharma and Kanwar 1985). One group of mice were adults, the other were just-weaned young mice. Female mice continued to receive lead-dosed water during mating and until weaning of new born pups. The number of pups per litter was significantly lower in lead-exposed mice; 26% lower in the adult treated group and 36% lower in the postnatally treated young mice. Percent survival of young to 4 weeks of age was also significantly lower in both lead-exposed groups compared to controls. Using body weights cited by the authors (28 g, adult group and 15 g, postnatal group) and measured amount of lead consumed by the two groups (0.8 mg/day, adult group and 0.75 mg/day, postnatal group), the exposure concentration was converted to units of mg/kgBW/day. A LOAEL value of 28.6 mg/kgBW/day and 50 mg/kgBW/day were calculated for the adult and postnatal mice, respectively. A LOAEL of 28.6 and as estimated NOAEL of 2.86 were calculated based on this experiment.

Lead was administered to rats at six dietary levels (1, 10, 50, 100, 1,000 and 2,000) for three generations and measured changes in reproduction and growth (Azar et al. 1973). No effects on number of pregnancies, number of pups born alive, fertility index, viability index or lactation index were observed at any exposure levels. An exposure concentration of 1,000 mg/kg resulted in reduced offspring weight and kidney damage in the young. An ingestion rate of 0.027 kg/day and adult body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 80 mg/kg BW/day, and a NOAEL of 8 mg/kg BW/day were calculated. The TRV values from this study will be used to evaluate the risk posed by Pb to mammalian receptors.

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A.7.2 Toxicity to Birds

The gastric motility of adult male and female red-tailed hawks (Buteo jamaicensis) fed 0.82 and 1.64 mg/kg BW/day (mg/kgBW/day concentration reported by authors) for three weeks was evaluated through the use of surgically implanted transducers. Neither concentration had any effect on gastric contractions or egestion of undigested material pellets (Lawler et al. 1991). The highest NOAEL observed in this experiment was 1.64 mg/kgBW/day.

The effect of Pb on survival of American kestrels was evaluated by feeding the birds either a control diet, or a diet containing mallard ducks which had died of Pb poisoning (mean Pb concentration was 29.3 mg/kg) for 60 days (Stendell 1980). No kestrels died or exhibited visible signs of Pb poisoning during the 60-day exposure period. An ingestion rate of 0.0307 kg/day (Barrett and Mackey 1975) and a body weight of 0.111 kg (Dunning 1993) were used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 8.1 mg/kgBW/day was calculated based on the results of this experiment.

Ringed turtle doves received 0 or 100 micrograms per milliliter (μg/ml) Pb in their drinking water from two weeks prior to breeding throughout a breeding cycle (Kendall and Scanlon 1981). Exposure to Pb did not increase the time required to produce eggs, and no adverse effects on egg production or fertility were observed. Bone Pb concentrations in adult birds and bone and liver Pb concentrations in juveniles were higher than in control birds or progeny of control birds. A water ingestion rate of 0.017 L/day (calculated using an allometric equation from (Calder and Braun 1983) and a body weight of 0.16 kg (Schwarzbach et al. 1991) was used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 10.6 mg/kgBW/day was calculated based on the results of this experiment.

American kestrels were fed diets containing biologically incorporated lead at concentrations of 0.5 (control), 120, 212, or 448 mg/kg dry weight for 60 days (Custer et al. 1984). Weight loss, anemia, or clinical signs of lead poisoning were not observed at any exposure concentration; no other effects were measured. A body weight of 120 g and food ingestion rate of 25 g/day (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 93.3 mg/kgBW/day was observed in this experiment.

Bobwhite quail were fed diets supplemented with Pb (as lead acetate) at concentrations of 0, 500, 1,000, 1,500,

2,000 and 3,000 mg/kg for 6 weeks (Damron and Wilson 1975). Weight gain and food consumption were significantly decreased in birds receiving the two highest exposure concentrations. Mortality of birds receiving 3,000 mg/kg Pb was 46.7 %, much greater than any other exposure group; however, it was not statistically significant due to large variability among replicate pens. In another experiment, male bobwhite were fed diets containing 0, 500, 1,000 or 1,500 mg/kg Pb (as lead acetate) for eight weeks. Mortality, food consumption, sperm concentration and sperm viability were measured; no effects were observed at any exposure concentration. A food ingestion rate of 0.0143 kg/day and adult body weight of 0.168 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day; 2,000 mg/kg was selected as the NOAEL level. A NOAEL of 128 mg/kgBW/day (exposure concentration of 1,500 mg/kg, endpoint measured sperm concentration and viability) was observed in this experiment.

Adult male and female red-tailed hawks were administered lead acetate by gavage at a concentration of 0.82 mg/kg BW/day for three weeks (Redig et al. 1991). Compared to control birds, there was an 83 % decrease in delta-aminolevulinic acid dehydratase (ALAD) activity and a 74 % increase in the levels of free porphyrins circulating in the blood of experimental birds. Immune function (as measured by antibody titers to foreign red blood cells or mitogenic stimulation of T-lymphocytes) was not significantly affected at this exposure level. This study was not used to derive a TRV for this risk assessment, as the ecological significance of the measured effects (ALAD, free porphyrins) is not known.

Nine raptors (five red-tailed hawks, three rough-legged hawks and one golden eagle) were administered 3 mg/kgBW Pb daily in the form of a lead acetate trihydrate solution by mouth for 30 weeks (Reiser and Temple 1980). Control birds (six red-tailed hawks, one Swainsons hawk) were dosed with a sodium acetate solution by mouth. Clinical signs of lead toxicosis (anorexia, green bile-stained feces and anemia) were observed in eight of the nine experimental birds. Three birds died three to four weeks following the onset of clinical symptoms. This study was not used to derive the TRVs for this risk assessment because dosing was via solution rather than dietary, and because different species were included within the experimental group.

(Beyer et al. 1988) fed red-winged blackbirds, brown-headed cowbirds, common grackles, northern bobwhites and eastern screech owls diets containing lead acetate. The dietary concentration was increased by 60 % weekly until half of the birds in each treatment group died. Because the exposure concentrations changed throughout the experiment, this study was not used to derive TRVs for this risk assessment.

One-day old American kestrel chicks were dosed orally with metallic Pb at concentrations of 0, 25, 125 or 625 mg/kgBW/day for 10 days (Hoffman et al. 1985a; Hoffman et al. 1985b). Forty percent of the birds in the highest dose group died after six days of exposure. Growth rates of birds which received Pb at concentrations of 125 or 625 mg/kgBW/day were significantly lower than the growth rates of control birds.
Day-old Japanese quail were fed diets containing Pb (as lead acetate) at concentrations of 0, 1, 10, 100, 500 or 1,000 mg/kg for five weeks (Morgan et al. 1975). Body weight, packed cell volume, and hemoglobin were significantly reduced in birds that received 1,000 mg/kg lead. At five weeks of age, testes size was also significantly reduced in the highest exposure group; this exposure concentration was identified as the LOAEL. Mean body weights of the 500 and 1,000 mg/kg exposure groups at three weeks were 65 and 55 g. Ingestion rates were calculated as a percent of the adult ingestion rate of 18 g/day (body weight of 0.12 kg; (Varghese 2000)), resulting in ingestion rates of 9.8 and 8.3 g/day, respectively. A LOAEL of 151 mg/kgBW/day and a NOAEL of 75.4 mg/kgBW/day were calculated based on the results of this experiment.

Day-old mallard ducklings were fed diets supplemented with Pb-contaminated sediment at concentrations of 1.9 (control diet), 414 and 828 μg/g for six weeks (Hoffman et al. 2000b). A clean sediment-supplemented control (24 % sediment) and a positive control diet containing lead acetate at a concentration equivalent to the 828 μg/g Pb-contaminated sediment diet were included in the experimental design. Mortality was observed only in the lead acetate group (7 %), but was not significantly different from the control group. Hematocrit and hemoglobin were significantly lower in ducklings which received lead acetate. Blood ALAD activity levels were significantly lower and protoporphyrin levels were higher in both groups which received Pb-contaminated sediment and the ducklings which received lead acetate. Acid-fast renal tubular inclusion bodies and nephrosis are abnormalities associated with Pb poisoning; inclusion bodies were observed in 50 % and tubular nephrosis was observed in 75 % of ducklings fed lead acetate. Renal inclusion bodies were observed in two of nine ducklings from the 414 μg/g group, and in 4 of 9 ducklings from the 828 μg/g group. Growth was affected only in ducklings fed lead acetate. Based on

the reduced growth observed in ducklings exposed to lead acetate at a concentration of 828 μg/g, this concentration was selected as the LOAEL for this experiment. A food ingestion rate of 0.0645 kg/day and body weight of 0.379 kg (cited by (Sugden et al. 1981) for three-week old mallard ducklings) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 140.9 mg/kgBW/day and an estimated NOAEL of 14.1 mg/kgBW/day were calculated based on results of this experiment.

Day-old mallard ducklings were fed diets containing either a clean sediment (24%) supplemented control diet, sediment (3449 mg/kg lead) supplemented diets at 12% or 24% (414 and 828 mg/kg lead, respectively) or a positive control diet supplemented with 828 mg/kg lead acetate for 6 weeks (Douglas-Stroebel et al. 2004). Brain growth was significantly decreased in ducklings fed the 24% lead sediment supplemented diet, and the lead acetate supplemented diet. Duckling growth was significantly decreased in the group fed the lead acetate supplemented diet. Histopathological brain lesions were observed in ducklings fed the 24% lead sediment supplemented diet and a nutritionally suboptimum diet. Based on the growth effects observed in the ducklings fed the lead acetate-supplemented diet, this concentration was identified as the LOAEL. A food ingestion rate of 0.0645 kg/day and body weight of 0.379 kg (cited by (Sugden et al. 1981) for three-week old mallard ducklings) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 140.9 mg/kgBW/day and an estimated NOAEL of 14.1 mg/kgBW/day were calculated based on results of this experiment.

Day-old Canada geese were fed diets supplemented with Pb-contaminated sediment concentrations of 1.9 (control diet), 414, 828 and 1,656 μg/g for six weeks (Hoffman et al. 2000a). Mortality was observed only in the highest exposure group (22 %), but it was not significantly different from the control group. Hematocrit, hemoglobin, and ALAD activity were significant lower and protoporphyrin levels were higher in the two highest exposure groups. Renal tubular degeneration was observed in one gosling from the 1,656 μg/g group, but histopathologic lesions most commonly associated with Pb poisoning in waterfowl were not observed in other geese. Growth was decreased in goslings from the highest exposure group. Based on the reduced growth observed in goslings exposed to lead at a concentration of 1,656 μg/g, this concentration was selected as the LOAEL for this experiment. A food ingestion rate of 0.093 kg/day and body weight of 1.44 kg (cited in (National Research Council 1994) for three-week old geese) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 107 mg/kgBW/day and an estimated NOAEL of 53.5 mg/kgBW/day were calculated based on results of this experiment.

Heinz et al. (1999) studied the bioavailability and toxicity of Pb-contaminated sediment to adult mallards. In the first experiment, ducks were fed a pelleted commercial duck diet containing 0, 3, 6, 12 or 24 % Pb-contaminated sediment (103, 207, 414 and 828 μg/g lead, respectively) for five weeks. Ducks fed the 24 % Pb-contaminated sediment exhibited atrophy of the breast muscles, green staining of the feathers around the vent, viscous bile, green staining of the gizzard lining, and renal tubular intranuclear inclusion bodies; one of 10 birds died. In the second experiment, the dietary concentration of the Pb-contaminated sediment was increased to 48 %, but only about 20 % was actually ingested due to food washing by the birds. Duration of this experiment was also five weeks. Protophyrin levels were elevated, and all of the Pb-exposed birds had renal tubular intranuclear inclusion bodies. A third experiment was conducted to determine if the effects of Pb were greater when birds were fed a nutritionally deficient diet. Ducks were fed a control diet, a commercial duck mash with 24 % Pb-contaminated sediment, or a ground corn diet with 24 % Pb-contaminated sediment for 15 weeks. Food washing was again observed; actual ingestion rates were 17 and 14 % for the Pb-contaminated duck mash and ground corn diets, respectively. Mortality occurred in four of five birds fed the Pb-contaminated ground corn diet. At necropsy, all birds fed the Pb-contaminated ground corn diet were emaciated, had renal tubular intranuclear inclusion bodies, and blackish-green bile. Based on the clinical signs of Pb poisoning observed in the first experiment, an exposure concentration of 828 μg/g Pb was selected as the LOAEL from this experiment. An ingestion rate of 0.139 kg/day and body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 92 mg/kgBW/day and a NOAEL of 46 mg/kgBW/day were calculated based on the results of this experiment.

Captive mute swans were fed either a commercial avian maintenance diet or ground rice containing 0, 12 (no rice group), or 24% highly contaminated (3,950 mg/kg lead) sediment or 24% reference sediment (9.7 mg/kg lead) for 6 weeks (Day et al. 2003). None of the swans died at any exposure concentration. A 24% decrease in mean body weight was observed in the swans fed the rice diet containing the highly contaminated sediment. Acid-fast renal tubular inclusion bodies and nephrosis are abnormalities associated with Pb poisoning; inclusion bodies and tubular nephrosis were observed in all of the swans in this treatment group. Inclusion bodies were also observed in all of

the swans fed the commercial feed and 24% sediment diet. Measured lead concentrations were 702 mg/kg in the rice/24% sediment diet, and 850 mg/kg in the commercial feed/24% sediment diet. Based on the reduced body weight, renal tubular inclusion bodies, and nephrosis observed in the swans fed the rice/24% sediment diet (all measurements indicative of lead poisoning), this exposure concentration (702 mg/kg) was identified as a LOAEL. The measured lead concentration in the reference sediment/rice diet was 4.4 mg/kg; this concentration was identified as the NOAEL. Body weights of 8.1 and 7.45 kg and ingestion rates of 0.249 and 0.203 kg/day for the 24% reference and 24% highly contaminated sediment groups, respectively (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 19.1 mg/kgBW/day and a NOAEL of 0.14 mg/kgBW/day were calculated based on results of this experiment. This study was not used to derive a TRV for this risk assessment, as adult body weight changes are not ecologically significant, and the ecological significance of the observed kidney effects is not known.

Japanese quail were exposed to four dietary concentrations of lead acetate (1, 10, 100 or 1,000 mg/kg) for a period of 12 weeks (Edens 1976). Percent hatch of setable eggs was significantly decreased in hens exposed to 100 mg/kg Pb. Dietary Pb at a concentration of 1,000 mg/kg almost completely suppressed egg production. The results from this experiment will be used to develop the NOAEL and LOAEL values because of the ecological significance of the endpoints and the method and duration of exposure. A LOAEL of 11.8 mg/kg BW/day (100 mg/kg) and a NOAEL of 1.18 mg/kg BW/day were calculated based on results of this experiment. Based on the ecological significance of the endpoint (reproduction) and because the LOAEL is the lowest cited adverse effect level for birds, the TRV values from this study will be used to evaluate the risk posed by Pb to avian receptors.

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A.8 Manganese

A.8.1 Toxicity to Mammals

Female timed-pregnant Long Evans rats were randomly assigned to four groups (Rehnberg et al. 1980). Pups were delivered naturally, and litters were normalized to six pups (3 male and 3 female) on day one. Neonates were dosed via intubation with MnO4 at concentrations of 0, 21, 71 or 214 g/g/day for 21 days. Growth was significantly decreased in pups exposed to Mn at concentrations of 71 or 214 g/g/day, and significant mortality was observed at the highest exposure concentration.

Pregnant female Long-Evans rats were exposed to normal Fe or low Fe diets containing manganese oxide at concentrations of 50 (basal diet), 400, 1,100 and 3,550 mg/kg from day one of gestation through 224 days of age of

the offspring (Laskey et al. 1982). The offspring began feeding on the Mn-treated diets at 14 to 15 days of age. Mortality of all animals on the low-Fe diet with 3,550 mg/kg Mn exceeded 90 % by day 50; no mortality was observed in any other treatment group. At 90 to 100 days of age, non-littermate males and females from each dose group were caged for two weeks. Pregnancy percentage was significantly reduced in F_1 female rats which received a normal-Fe diet which contained Mn at a concentration of 3,550 mg/kg. Reproductive development (decreased testes weight, sperm count and testosterone concentration) was affected in males which received the normal-Fe diet and Mn at a concentration of 3,550 mg/kg. A LOAEL of 284 mg/kg BW/day, and a NOAEL of 88 mg/kg BW/day were calculated. The TRV values from this study will be used to evaluate the risk posed by Mn to mammalian receptors.

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A.8.2 Toxicity to Birds

Male Japanese quail were exposed to basal diets (56 mg/kg Mn) supplemented with 5,000 mg/kg manganese oxide for 75 days (Laskey and Edens 1985). No reduction in growth was observed, and aggressive behavior was reduced relative to control birds. Reduced aggressive behavior was not considered an adverse effect. The reported exposure concentration of 977 mg/kgBW/day was used as the NOAEL for this risk assessment.

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A.9 Mercury (inorganic)

A.9.1 Toxicity to Mammals

Male and female ICR mice were exposed to cinnabar (a naturally occurring mercuric sulfide) by gavage at a concentration of 0 or 10 milligrams per kilogram body weight per day (mg/kgBW/d) for 11 weeks (Huang et al. 2007). Significant absorption in the gastrointestinal tract and transport to the cerebral cortex was observed. Marked hypoactivity was observed in male mice after six weeks of exposure to cinnabar. Motor equilibrium performance was significantly decreased after six weeks (males) and seven weeks (females) of exposure compared with performance of control mice. Both sexes exhibited prolongation of pentobarbitol-induced sleeping times. Because all the effects measured in this experiment were behavioral and the ecological significance of behavioral impacts is not known, this study was not used to derive a TRV for inorgnaic mercury.

Adult male Wistar rats were exposed to inorganic mercury (as mercuric chloride) at concentrations of 0, 50 or 100 ppm in drinking water for 90 days (Boujbiha et al. 2009). Treated males were paired with untreated females. At an exposure concentration of 50 ppm, there was a significant decrease in epididymal sperm number, epididymal sperm motility, and the number of viable embryos per litter compared with control rats. A body weight of 0.289 kg and water ingestion rate of 0.0354 liters per day were used to convert the exposure concentration to dosage units of mg/kgBW/d (U.S. EPA 1988). A LOAEL of 6.12 mg/kgBW/d and an estimated NOAEL of 0.612 mg/kgBW/d were calculated based on results of this experiment.

Thirty-day old female Sprague-Dawley rats were exposed to mercuric chloride via gavage at concentrations of 0, 1 or 2 mg/kgBW/d for 60 days (Heath et al. 2009). At 90 days of age, the females were mated with unexposed males. Dosing continued until approximately day 13 of gestation. In females exposed at a concentration of 2 mg/kgBW/d, there were significantly fewer implantations, and significantly more non-viable implantations compared to controls. A LOAEL of 2 mg/kgBW/d and a NOAEL of 1 mg/kgBW/d were identified based on this experiment.

Effects of mercuric chloride on the reproductive performance of two generations of male and female Sprague-Dawley rats were evaluated (Atkinson et al. 2001). Parental generation (F_o) rats were exposed to concentrations of 0:0 (males:females), 0.5:0.75,1.0:1.5, or 1.5:2.5 mg/kgBW/d. Dosing of males began during the pre-mating period (60 days or one complete cycle of spermatogenesis) and continued throughout mating (21 days). Dosing of females began during the pre-mating period (16 days or one complete cycle of oogenesis for females) and continued through lactation (63 days). For the F_o generation, the number of pregnant females, number of live pups per litter, the fertility index, and the live birth index were significantly decreased at an exposure concentration of 0.5:0.75 (male:female) mg/kgBW/d. Exposure to mercuric chloride did not affect fertility or litter size in the F_1 generation, but the live birth index and 4-day survival index were significantly decreased at an exposure concentration of 1.0:1.5 (male:female) mg/kgBW/d. Based on the reproductive effects, a concentration of 0.5 mg/kgBW/d was identified as the LOAEL, and a NOAEL of 0.05 mg/kgBW/d was estimated.

Mice were exposed to inorganic mercury (as mercuric chloride) via gavage at concentrations of 0, 0.25, 0.50, or 1.0 mg/kgBW/d (Khan et al. 2004). Dosing of males began during the pre-mating period (40 days or one complete cycle of spermatogenesis) and continued throughout mating (21 days). Dosing of females began during the pre-mating period (16 days or one complete cycle of oogenesis for females) and continued through lactation (63 days). Doses were adjusted weekly according to body weight changes. Viability of offspring from mice exposed at a concentration of 1.0 mg/kgBW/d was significantly reduced compared with control mice. Fertility indices of all mice exposed to mercuric chloride were significantly lower than for control mice. Based on the ecological significance of the observed effect (reproduction), an exposure concentration of 0.25 mg/kgBW/d was identified as the LOAEL in this experiment, and a NOAEL of 0.025 mg/kgBW/d was estimated using a factor of 10.

No adverse effects on survival or reproduction mink were observed in mink administered10 μg/g inorganic mercuric chloride for five months (Aulerich et al. 1974). A NOAEL of 1 mg/kgBW/day and an estimated LOAEL of 10 mg/kgBW/day were identified for inorganic Hg based on the results of this experiment. These values were selected for use as TRVs in this risk assessment.

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A.9.2 Toxicity to Birds

Japanese quail were fed diets containing 0, 2, 4, 8, 16 or 32 mg/kg mercury (as mercuric chloride) from hatching until one year of age (Hill and Shaffner 1976). No effects on food consumption or growth were observed in any group. Egg production increased as dietary mercuric chloride increased. Egg fertilization rates were depressed at mercury exposure concentrations of 4 mg/kg and higher, and percent hatchability of all eggs was significantly lower

than controls for birds exposed at a concentration of 8 mg/kg. A LOAEL of 0.9 mg/kgBW/day and a NOAEL of 0.45 mg/kgBW/day were calculated based on results of this experiment.

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A.10 Mercury (methylmercury)

A.10.1 Toxicity to Mammals

Albanus et al. (1972) fed cats a diet composed of pike collected in a Hg-contaminated lake in Sweden. The methylmercury concentration of the diet was 6 mg/kg, or 0.45 mg/kgBW/day (cited by authors). Behavioral changes such as aggressiveness and stiff, waddling gaits were noted four to 11 days before the onset of convulsions, which developed between 60 and 83 days after the start of the experiment.

Five groups of cats were tested by Eaton et al. (1980). Three groups were fed ringed seal liver at concentrations of 4.8, 10.2, or 19.8 mg/kgBW/day. Only 3 % of the Hg in the seal liver was in the organic form methylmercury. Another group was fed beef liver spiked with methylmercuric chloride, at a dose of 0.25 mg/kgBW/day. A fifth group was fed uncontaminated beef liver. Evidence of Hg intoxication appeared only in the group of cats fed methylmercury-spiked beef liver. Animals began showing convulsions after 68 days, and mean survival period for the animals was 78 days.

Pregnant female cats were dosed with methylmercuric chloride administered in corn oil suspensions in gelatin capsules at concentrations of 0. 0.03, 0.083, and 0.25 mg/kgBW/day during days 10 through 58 of gestation (Khera 1973). At oral levels of 0.25 mg/kgBW/day, there was an increased incidence of abortion, fetal anomalies, and decreased cell density in the external granular layer of the cerebellum. Minimal or no embryopathic effects were observed at lower concentrations.

Cats were fed diets containing uncontaminated fish, methylmercury-contaminated fish at levels of 0.25 mg/kgBW/day, or dosed orally with gelatin capsules containing methylmercuric chloride dissolved in corn oil at a concentration of 0.25 mg/kgBW/day (Charbonneau et al. 1974). Clinical signs of Hg intoxication including ataxia, tremor and convulsions developed after 55 to 96 days of exposure in both treated groups. There was no significant difference in time to onset of symptoms of Hg intoxication, blood or tissue Hg concentrations, or time to death between cats receiving methylmercury-contaminated fish and those receiving methylmercuric chloride. Blood and brain Hg concentrations at which clinical signs of Hg intoxication appeared were 10 mg/kg and 10 mg/kg, respectively. No pathological changes were observed in the spinal cord or peripheral nerves, and no abnormalities were observed during chromosome studies of terminal bone marrow samples.

A commercial mink diet containing 5 μg/g methylmercury was lethal to adult mink in 30 days, while mink fed 10 μg/g inorganic mercuric chloride showed no adverse effects on survival or reproduction after five months (Aulerich et al. 1974). The exposure concentration was converted to units of mg/kgBW/day using an ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A LOAEL of 1.1 mg/kgBW/day was calculated for methylmercury, and a NOAEL of 2.2 mg/kgBW/day was calculated for inorganic Hg based on the results of this experiment.

Jernalov et al. (1976) fed adult female mink a diet containing Hg-contaminated pike for a 100-day period. The concentration of methylmercury in the pike was 5.7 mg/kg, and pike comprised 40 % of the total diet by weight. Based on an adult body weight of 1.13 kg and ingestion rate of 0.249 kg/day, the mink were exposed to a dietary level of 0.5 mg/kgBW/day. The mink showed no symptoms of poisoning and their behavior was unchanged throughout the experimental period.

It is difficult to determine the reason for the apparent discrepancy in levels of Hg which produced toxic effects in the above studies. Actual Hg concentration in the diet was not analyzed in the first two studies, and only the pike portion of the diet was analyzed by Jernalov et al. (1976). The portion of diet comprised of fish differed in all three studies, and Se levels were only analyzed by Jernalov et al. (1976). Fish tissue is generally high in Se, which protects organisms from Hg toxicity (Cuvin-Aralar and Furness 1991, Stoewsand et al. 1974), and also reduces

assimilation of Hg from food by 5 to 10 % (Turner and Swick 1983).

Female mink were fed diets containing 0.1, 0.5 and 1.0 mg/kg total Hg (Dansereau et al. 1999). Piscivorous and nonpiscivorous fish naturally contaminated with organic Hg were used to prepare the experimental diets. No negative control was used in this study due to the inability to find a freshwater fish diet uncontaminated by Hg. First generation females (G1) were exposed to the diets for approximately 400 days, and their female offspring (G2) were exposed to the diets for approximately 300 days. All females were mated to males that were fed the diet containing 0.1 mg/kg Hg for 60 days prior to the mating season. Mercury exposure did not affect length of gestation period, number of kits, survival or growth of neonatal kits. There was an inverse relationship between whelping proportion and exposure concentration, but this was not statistically significant. High mortality was observed in G1 females (60 %) and G2 females (86 %) fed the 1.0 mg/kg Hg diet. The exposure concentrations were converted to units of mg/kgBW/day using an ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A LOAEL of 0.22 mg/kgBW/day and a NOAEL of 0.11 mg/kgBW/day were calculated based on the results of this experiment.

Methylmercury was added to diets of male river otters at levels of 2, 4, and 8 mg/kg Hg (O'Conner and Nielson 1980). Control animals were healthy for the entire study period, but otters exposed to Hg in the diet had mean survival times of 184, 117 and 57 days, respectively. Tissue concentrations at time of death were similar, although dietary levels differed. The exposure concentrations were converted to units of mg/kgBW/day (0.25, 0.50 and 1.00 mg/kgBW/day, respectively) using an ingestion rate of 0.8 kg/day and body weight of 6.35 kg (Harris 1968). A LOAEL of 0.25 mg/kgBW/day and an estimated NOAEL of 0.025 mg/kgBW/day were calculated based on the results of this experiment.

Rats were exposed to dietary methylmercuric chloride at concentrations of 0.1, 0.5 and 2.5 mg/kg for three generations (Verschurren et al. 1976). Pup viability was significantly reduced in the group exposed to a concentration of 2.5 mg/kg. An ingestion rate of 0.028 kg/day and body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.16 mg/kgBW/day and a NOAEL of 0.032 mg/kgBW/day were calculated.

Adult female mink were fed commercial diets containing 1.1, 1.8, 4.8, 8.3 and 15.0 μg/g Hg as methylmercuric chloride for a 93-day period (Wobeser et al. 1976; Wobeser et al. 1976). Signs of Hg intoxication were seen in mink fed 1.8 μg/g mercuric chloride and greater. Signs of intoxication were anorexia, weight loss, ataxia, splaying of the hind legs, irregular vocalization and convulsions. The rapidity of the onset of clinical intoxication was directly related to the Hg content of the diet. A LOAEL of 0.025 mg/kgBW/day and a NOAEL of 0.015 mg/kgBW/day were calculated based on the results of this experiment.

Ranch-bred mink were maintained in outside cages and fed diets containing methylmercury at concentrations of 0 or 1.0 μg/g for 8 months (Wren et al. 1987a; Wren et al. 1987b). Fertility of adult mink, percentage of females whelped, number of kits per female, growth rate of kits, and kit survival to weaning were not affected by Hg exposure at this concentration. Unexpected mortality of mink was observed during a cold snap; clinical signs elicited by the animals prior to death were consistent with methylmercury intoxication (weakness, splaying of the hind legs, tremors and loss of appetite). Although mortality had not been observed in previous experiments at this exposure concentration, the authors attributed the observed mortality to a combination of cold stress and Hg exposure. Based on these results, a NOAEL using the 1.1 mg/kg dose from the Wobeser et al. (1976) study was not considered to be protective, as increased mortality was observed at a lower dose when combined with natural stressors present in field conditions. Therefore, an uncertainty factor of 0.1 was applied to the LOAEL of 0.025 mg/kgBW/day TRV to derive a NOAEL of 0.0025 mg/kgBW/day.

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A.10.2 Toxicity to Birds

Post-fledging white ibises were exposed to diets containing methylmercury (MeHg) to evaluate the relationship between MeHg exposure and endocrine development (Adams et al. 2009). This study was not summarized or used to derive a toxicity reference value (TRV), as the units presented for the dietary dose are milligrams per kilogram (mg/kg) wet weight per day (w.w./day) in the Abstract, and mg/kg diet in the Methods Section.

Pre-fledgling white ibises were exposed to diets containing MeHg to evaluate the relationship between MeHg exposure and foraging behavior and efficiency (Adams and Frederick 2008). This study was not summarized or used to derive a TRV, as the units presented for the dietary dose are mg/kg w.w./day in the Abstract, and mg/kg diet in the Methods Section.

American kestrels were fed diets containing methyl mercury (as methyl mercuric chloride) at concentrations of 0, 3, 6 or 12 mg/kg w.w. from eight weeks prior to the onset of laying until five days prior to the anticipated hatch date (Bennett et al. 2009). Only two pairs were tested at each exposure concentration. All birds fed 12 mg/kg MeHg died before breeding. One pair fed 6 mg/kg MeHg laid four infertile eggs, and one bird died after 75 days of exposure. It was noted that mortality may have been observed in the 6 mg/kg exposure group in the above experiment, had the exposure period been longer than 59 days. One pair fed 3 mg/kg MeHg failed to lay eggs. Total mercury (Hg) measured in eggs from the birds fed 6 mg/kg MeHg was 21.5 and 16.4 mg/kg w.w., and 8.34 mg/kg w.w in one pair fed 3 mg/kg MeHg. Due to the small samples size tested, this study was not used to derive a TRV for exposure of birds to MeHg.

Liquid mercury was historically used to process gold and silver ore mined from Virginia City, Nevada (Henny et al. 2002). A study was conducted on double-crested cormorants, snowy egrets, and black-crowned night-herons nesting along the Lower Carson River System. Most eggs had MeHg concentrations below 0.8 mg/kg w.w., and no effects on hatchability were observed. After hatching, young birds were fed diets averaging 0.36 to 1.18 mg MeHg/kg w.w. through fledging. Peripheral nerve damage was evident in young night-herons and cormorants, and was correlated with tissue Hg concentrations. Evidence of toxicity was also observed in the immune (spleen, thymus and bursa) and detoxicating (liver, kidney) systems. The lowest reported body weight for adult cormorants (1.54 kg) (Hartman 1961) and food ingestion rate (0.282 kg/day) (Gremillet and Pios 1994) were used to convert the exposure concentrations to units of mg/kgBW/d. An exposure concentration ranging from 0.066 mg/kgBW/d to 0.216 mg/kgBW/d was calculated. This study was not used to derive a TRV, as the ecological significance of the observed histopathological damage is uncertain.

Goshawks (Accipiter gentilis) were fed a diet of chickens which had been fed methylmercury-dressed wheat (0.4 to 0.5 mg/kgBW/d) for five to six weeks and sacrificed (Borg et al. 1970). All chickens were clinically healthy at the end of the feeding period. The average Hg level in the chicken feed was 8 mg/kg, and in skeletal muscle of the chickens was 10 mg/kg. Muscle and liver from the chickens was fed to goshawks. Intake of Hg by the goshawks was 0.7 to 1.2 mg/kgBW/d (as reported by authors). Clinical symptoms of Hg poisoning appeared after two weeks. All birds were dead 47 days after the start of the experiment. Muscle Hg levels of the goshawks averaged 40 to 50 mg/kg, while brain Hg levels in the dead goshawks ranged from 30 to 40 mg/kg. This study was not used to derive a TRV for this risk assessment due to the small sample size of birds tested (two controls, four experimental).

Effects of Hg on a free-living breeding population of common loons in western Maine and New Hampshire were evaluated using behavioral, physiological, survivorship, and reproductive endpoints (Evers et al. 2008). Biological matrices that could be non-lethally collected (blood, eggs and feathers) were used to identify adverse effect levels. The reproductive success of 178 territories on 80 lakes was monitored for 6 to 10 years. A significant negative correlation between number of chicks fledged and Hg concentrations in female loons was observed. An adverse effect threshold for reproduction of 3.0 µg/g in adult blood was identified in this study. Dietary exposure concentrations were not measured, therefore this study could not be used to derived a TRV.

Total Hg concentrations in prey items fed to nestling wood storks were measured in three colonies in Georgia (Gariboldi et al. 1998). The food items collected generally came from nestlings three to seven weeks old. Mean dietary Hg concentrations were 0.28, 0.10 and 0.19 mg/kg for an inland and two coastal colonies, respectively. Freshwater prey species had higher Hg concentrations, and the diet of the inland colony birds were entirely

comprised of freshwater species. Mean nest success for the three colonies was 1.9, 2.58 and 2.5 fledged young per nest for the inland and coastal colonies, respectively. Because other factors that may influence reproductive success (e.g. differences in abundance of prey items) were not measured in this study, the authors could not conclude that dietary Hg exposure resulted in the lower reproductive success observed at the inland colony.

Juvenile common loons were fed diets containing methyl mercury (as methyl mercuric chloride) at concentrations of no added MeHg (less than [<] 0.03 micrograms per gram [µg/g] w.w.), 0.1, 0.5, or 1.5 µg/g w.w. for 105 days (Kenow et al. 2003). No overt signs of mercury toxicosis (behavioral abnormalities or ataxia) were observed. No significant effects on growth or food consumption were observed at any exposure concentration. Chicks hatching from eggs collected from low pH lakes reached an asymptotic mass 165 grams (g) smaller than chicks from eggs collected from neutral pH lakes. A no-observed-effect-level (NOAEL) of 1.5 mg/kg was observed in this experiment. A food ingestion rate of 0.306 kilograms per day (kg/day) and body weight (BW) of 0.994 kilograms (kg) (Lindo 1997) were used to convert the exposure concentration to units of mg/kgBW/d. A NOAEL of 0.461 mg/kgBW/d was observed in this experiment. The authors cautioned against extrapolation of results beyond the 105-day trial period. Pharmacokinetics of MeHg absorption and elimination in young loons have been described (Kenow et al. 2007); very rapid elimination rates were observed during feather growth, and rates slowed dramatically following feather development. They noted that chicks may be more vulnerable to MeHg exposure after fledging, when feather growth ceases and tissue mercury exposure may increase considerably.

Chickens were fed diets containing mercury (Hg) (from herring meal) at concentrations of 0, 0.014 and 0.018 mg/kg for 52 weeks (March et al. 1974). Adult mortality and body weight were not affected by dietary Hg at the concentrations tested. Egg fertility, hatchability and quality (e.g., egg weight or shell thickness) were not affected by Hg. An ingestion rate of 0.103 kg/day and body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/d. A NOAEL of 0.0012 mg/kgBW/d was calculated based on the results of this experiment.

Japanese quail were fed diets containing methylmercury at concentrations of 0 or 30 mg/kg (Welsh and Soares 1976). All birds fed the diet containing 30 mg/kg were dead within two weeks. Addition of Se (0.05, 0.1, or 0.6 mg/kg) or vitamin E [10 or 500 International Units (IU)] markedly decreased the lethal effects of Hg. Except the birds which received 0.6 mg/kg Se, all experimental birds were dead at the end of the experimental period (34 days). An ingestion rate of 0.018 kg/day and body weight of 0.12 kg (Varghese 2000) were used to covert the exposure concentration to units of mg/kgBW/d. A LOAEL of 4.5 mg/kgBW/d and NOAEL of 0.45 mg/kgBW/d were calculated based on the results of this experiment.

Laying hens were fed diets containing Hg (as phenyl mercuric chloride) at concentrations of 0, 5, and 30 mg/kg for two months (Pribilincova et al. 1996). Egg production and egg weight were significantly reduced in the group exposed to Hg at a concentration of 30 mg/kg. Fertility and hatchability were not affected at any exposure concentration. An ingestion rate of 0.12 kg/day (cited by authors) and body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/d. A LOAEL of 2.5 mg/kgBW/d and a NOAEL of 0.41 mg/kgBW/d were calculated based on the results of this experiment.

American kestrels were fed diets containing methyl mercury (as methyl mercuric chloride) at concentrations of 0, 3, 6 or 12 mg/kg w.w. for 59 days (Bennett et al. 2009). All birds at the highest exposure concentration died or were euthanized in less than 50 days. None of the birds in the 6 mg/kg exposure group died, but signs of neurotoxicity were observed in seven of nine kestrels. No overt signs of toxicity were observed in kestrels fed 3 mg/kg MeHg. Histological lesions in the cerebellum were observed in birds from the 12 mg/kg group. Even though motor signs of toxicity were observed in birds fed 6 mg/kg MeHg, neural lesions were not found. A lowest-observed-adverse-effect-level (LOAEL) of 6 mg/kg was identified, based on the neurotoxicity symptoms observed. An ingestion rate of 0.0302 kg/day and BW of 0.1 kg (Barrett and Mackey 1975) were used to convert the exposure concentration to units of mg/kgBW/d. A LOAEL of 1.81 mg/kgBW/d and a NOAEL 0.906 mg/kgBW/d were identified based on this experiment.

Effects of dietary methylmercury on zebra finches (seed eaters) were evaluated by Scheuhammer (1988). Four groups of birds were fed diets containing 0, 1.0, 2.5 or 5.0 μg/g methylmercury. A dietary level of 5 μg/g caused significant neurological impairment and death in zebra finches. No symptoms were noted in the group fed levels of 2.5 μg/g. An ingestion rate of 0.0049 kg/day and body weight of 0.014 kg (cited by author) were used to convert the

exposure concentrations to units of mg/kgBW/d. A LOAEL of 1.75 mg/kgBW/d and a NOAEL of 0.88 mg/kgBW/d were calculated based on the results of this experiment.

Adult mallards were fed a control diet or a diet containing 9.2 mg/kg Hg (measured concentration; as methylmercury chloride) for 11 weeks (Heinz and Hoffman 1998). One of 12 males exposed to Hg died, and leg paralysis was observed in eight others. The percent hatch of fertile eggs and number of seven day old ducklings produced per female was significantly lower in Hg-exposed birds. An ingestion rate of 0.139 kg/day (Piccirillo and Quesenberry 1980) and a body weight of 1.21 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/d. A LOAEL of 1.06 mg/kgBW/d and an estimated NOAEL of 0.11 mg/kgBW/d were calculated based on the results of this experiment.

Red-tailed hawks (Buteo jamaicensis) were fed chicks contaminated with methylmercury (Fimreite and Karstad 1971). The chicks were fed diets containing Panogen 15, a commercial seed treatment containing 2.5 % methylmercury dicyandiamide (MMD) at rates of 6, 12 and 18 mg/kg MMD for three weeks. Mercury levels measured in chick livers were 3.9, 7.2 and 10.0 mg/kg, respectively. Mean estimated intake of Hg by the three groups of hawks over the 12-week exposure period was 0.575 mg/day; 1.12 mg Hg/day; and 1.46 mg/day, respectively. Mortality occurred in hawks receiving the most contaminated diet (1.12 mg/kgBW/d) after an exposure period of one month or more. Exposure concentrations were converted to units of mg/kgBW/d using a red-tailed hawk body weight of 1.1 kg (Dunning 1993). Pathological changes noted in all hawks which received the highest Hg doses (1.12 and 1.46 mg/day, or 1.02 and 1.33 mg/kgBW/d, respectively) included swelling of axons of myelinated nerves in the spinal cord, and dilatation of myelin sheaths and loss of myelin. No adverse effects were observed in hawks which received 0.575 mg/day (0.52 mg/kgBW/d). A LOAEL of 1.02 mg/kgBW/d and a NOAEL of 0.52 mg/kgBW/d were calculated based on the results of this experiment.

Juvenile great egrets were fed diets containing MeHg (as methylmercury chloride) at concentrations of 0, 0.5, or 5 mg/kg from 12 to 105 days of age (Bouton et al. 1999). At an exposure concentration of 0.5 mg/kg, significant effects on activity, tendency to seek shade, and motivation to hunt prey were observed. However, time taken to capture fish and efficiency of capture were not different from control birds. Birds in the 5 mg/kg exposure group became so sick they were euthanized before the end of the experiment. Although behavioral effects were observed at an exposure concentration of 0.5 mg/kg, significant impacts on hunting ability were not observed, which would be considered an ecologically relevant effect. A LOAEL of 5.0 mg/kg was identified, however severe effects were observed at this exposure concentration. A food ingestion rate of 0.131 kg/day (Frederick et al. 1999) and body weight of 1 kg (Gross et al.) were used to convert the exposure concentration to units of mg/kgBW/d. A LOAEL of 0.625 mg/kgBW/d and a NOAEL of 0.0625 mg/kgBW/d were identified from this experiment.

Black ducks were fed diets containing methyl mercury at concentrations of zero or three mg/kg for 28 weeks (beginning 12 weeks before laying) during two consecutive breeding seasons (Finley and Stendall 1978). Dietary Hg exposure did not affect adult body weights or mortality. Hatchability of eggs and duckling survival were significantly lower for experimental birds in both breeding seasons. An ingestion rate of 0.125 kg/day (Heinz et al. 1989) and body weight of 1.25 kg (Dunning 1993) were used to convert the exposure concentration to units of mg/kgBW/d. A LOAEL of 0.3 mg/kgBW/d and an estimated NOAEL of 0.03 mg/kgBW/d were calculated based on the results of this experiment.

Kidney lesions were found in juvenile starlings (Sturnus vulgaris; omnivores) that consumed a commercial diet contaminated with 1.1 mg/kg Hg (Nicholson and Osborn 1984). The exposure concentration was converted to units of mg/kgBW/d using a body weight of 63.8 g (Terres 1980) and ingestion rate of 11% of body weight per day (7 g/day; Kenaga 1973), resulting in a LOAEL of 0.12 mg/kgBW/d and an estimated NOAEL of 0.012 mg/kgBW/d.

Barr (1986) conducted a field study of common loons (Gavia immer) nesting on the Wabigoon-English River systems, areas affected by unpredictable water level fluctuations and Hg contamination. It was noted that nesting success of loons in this area was suppressed. Water level fluctuations due to the dams were ruled out as a causative factor, as decreased nesting success was observed in lakes experiencing only natural water level changes as well. A strong negative correlation was found between the successful use of territories by breeding loons and Hg contamination. A reduction in egg laying, and nest site and territorial fidelity were associated with mean Hg concentrations ranging from 0.3 to 0.4 mg/kg in prey, and from 2 to 3 mg/kg in adult brain tissue and eggs. Loons established few territories, laid only one egg, and raised no young where mean Hg in prey species exceeded 0.4

mg/kg. Non-Hg toxicants were found in loons and prey items at low levels, and were discounted as a major factor in the failure of loon reproduction. An ingestion rate of 1.5 kg/day and body weight of 4.5 kg (Alexander 1977) were used to convert the exposure concentration to units of mg/kgBW/d (LOAEL of 0.1 mg/kgBW/d).

Breeding American Kestrels were exposed to MeHg (as methylmercury chloride) at measured concentrations of 0.0059 (control), 0.274, 0.759, 1.19, 1.68, or 2.2 mg/kg wet weight from March 11 to day 23 of the incubation period (duration ranged from 77 to 113 days) (Albers et al. 2007). Birds were then switched to a clean diet. A significant decrease in egg production, incubation performance, and the number of eggs hatched was observed in birds exposed at a concentration of 1.68 mg/kg (0.51 mg/kgBW/d). Number of fledglings and percent (%) of nestlings fledged were markedly reduced at an exposure concentration of 0.274 mg/kg. An ingestion rate of 0.0302 kg/day and body weight of 0.1 kg (Barrett and Mackey 1975) were used to convert the exposure concentration to units of mg/kgBW/d. A LOAEL of 0.0827 and an estimated NOAEL of 0.00827 were identified based on this experiment.

Lake pH, mercury concentrations in small fish, blood Hg concentrations in adult male, female, and juvenile common loons, and loon productivity were measured at 120 lakes in Wisconsin, USA and New Brunswick and Nova Scotia, Canada (Burgess and Meyer 2008). Significant differences in loon tissue Hg concentrations, body mass, fish Hg concentrations, and lake pH were observed between the two study areas. Body masses of adult loons were 22% higher in Canada. Average Hg concentrations in small fish were higher in Canadian lakes, and average lake pH was lower. Mercury concentrations in small fish were higher in more acidic lakes in both regions. Blood Hg concentrations in loons were higher in lakes with lower pH, and lakes with elevated fish Hg concentrations. Loon productivity decreased with two measures of higher loon Hg exposure: Hg concentration in adult female blood, and Hg concentration in small fish. Although low loon productivity was observed at lakes where Hg exposure was low, loon productivity was never high if Hg exposure was high. Loon productivity was reduced 50% when fish Hg concentrations were 0.21 µg/g w.w., and no reproduction was observed when prey fish Hg concentrations were 0.41 µg/g w.w. or higher. Based on the reproductive effects, a concentration of 0.21 µg/g was identified as the LOAEL. An ingestion rate of 1.5 kg/day and body weight of 4.5 kg (Alexander 1977) were used to convert the exposure concentration to units of mg/kgBW/d. A LOAEL of 0.07 mg/kgBW/d and an estimated NOAEL of 0.007 mg/kgBW/d were estimated based on this study.

Mallard ducks were fed diets containing methylmercury at concentrations of 0, 0.5 and 3 mg/kg for 12 months (Heinz 1974). The dry-mash concentrations of 0.5 and 3 mg/kg were equivalent to approximately 0.1 and 0.6 mg/kg, respectively, in a natural succulent duck diet. No treatment-related mortality was observed in adult birds. Number of eggs laid and hatching success were lower for birds exposed to Hg at a concentration of 3 mg/kg. The number of ducklings that died in the first week of life was also significantly higher for this group; histopathological analysis indicated the ducklings died from damage to nervous tissue in the cerebellum. An ingestion rate of 0.139 kg/day and body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/d. A LOAEL of 0.066 mg/kgBW/d and a NOAEL of 0.01 mg/kgBW/d were calculated based on the results of this experiment.

For two consecutive years, mallard hens were fed diets containing methylmercury at concentrations of 0, 0.5 and 3 mg/kg (approximately 0.1 and 0.6 mg/kg on the basis of a natural succulent diet; Heinz 1976). No significant differences in egg production, number of eggs laid outside the nest box, or hatching success were observed. The percentage of normal ducklings that survived for one week was significantly lower for hens fed 3 mg/kg Hg. Brain lesions observed in dead ducklings at necropsy included demyelination, neuron shrinkage and necrosis. In avoidance tests, ducklings whose parents were fed 3 mg/kg Hg were hyper-responsive compared with controls and ducklings from parents fed 0.5 mg/kg Hg. An ingestion rate of 0.139 kg/day and body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/d. A LOAEL of 0.066 mg/kgBW/d and a NOAEL of 0.01 mg/kgBW/d were calculated based on the results of this experiment.

Juvenile great egrets were fed diets containing MeHg as methylmercury chloride at concentrations of 0, 0.5, or 5 mg/kg from 1 to 14 weeks of age (Spalding et al. 2000a). A significant decrease in appetite and growth was observed in both mercury-exposed groups. Growth of chicks fed diets containing 0.5 mg/kg MeHg was significantly lower than controls at week 11, and persisted until the end of the experimental period. Neural damage was not observed in great egret chicks fed MeHg at a concentration of 0.5 mg/kg, but was observed in birds exposed at a concentration of 5 mg/kg (Spalding et al. 2000b). A LOAEL of 0.5 mg/kg was identified, based on the effects

on growth. The authors noted that MeHg consumption ranged from a high of 0.135 mg/kgBW/d during week 3 to a low of 0.048 mg/kgBW/d. A food ingestion rate of 0.15 kg/kg body weight (presented by authors for the final weeks of the experiment, when reduced growth was observed) was used to convert the exposure concentration to units of mg/kgBW/d. This LOAEL of 0.075 mg/kgBW/d for methylmercury chloride was multiplied by 0.8 (molecular weight of mercury divided by the molecular weight of methylmercury chloride) to calculate a LOAEL of 0.06 mg/kgBW/d for methylmercury. It should be noted that the authors caution use of the 0.5 mg/kg exposure concentration as a LOAEL, as selenium availability was high in the test system. In a system where selenium is less available, adverse effects may be observed at a lower exposure concentration.

Three generations of mallard ducks were fed a diet containing MeHg as methylmercury dicyandiamide at a concentration of 0.5 mg/kg (approximately 0.1mg/kg on the basis of a natural succulent diet) or a control diet (Heinz 1979). Mercury (Hg) had no effect on adult body weight or weight changes during reproduction. Females exposed to dietary Hg laid a greater percentage of eggs outside the nest box, laid fewer sound eggs and produced fewer ducklings. Ducklings from Hg-exposed parents were hyper-responsive to a frightening stimulus in avoidance tests. Based on the reproductive effects, an exposure concentration of 0.5 mg/kg was identified as the lowest observed adverse effect level (LOAEL). A LOAEL of 0.064 mg/kgBW/d and an estimated NOAEL of 0.0064 mg/kgBW/d were calculated. These values were selected as TRVs to evaluate toxicity of methylmercury to birds for this risk assessment.

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A.11 Nickel

A.11.1 Toxicity to Mammals

Female B6C3F1 mice were exposed to nickel sulfate in drinking water at concentrations of 0, 1,000, 5,000 and 10,000 mg/L for 180 days (0, 115.7, 285.7 and 395.7 mg/kgBW/day; Dieter et al. 1988). Minimal to mild nephrosis was observed in mice from the two highest exposure concentrations, and thymic atrophy was found in all Ni-exposed mice. A LOAEL of 115.7 mg.kgBW/day and an estimated NOAEL of 11.6 mg/kgBW/day were calculated based on the results of this experiment.

Wistar rats were exposed to dietary Ni (as nickel sulfate hexahydrate) at concentrations of 0, 250, 500, 1,000 or 2,500 mg/kg for three generations (Ambrose et al. 1976). No adverse effects on fertility, gestation, viability and lactation indices were observed at any dietary concentration of Ni tested. Number of siblings cast per litter and weaned per litter decreased with increasing Ni concentration; however, no statistical analysis was conducted by the authors for these endpoints. Offspring growth was significantly reduced in rats exposed at concentrations of 1,000 and 2,500 mg/kg. An ingestion rate of 0.027 kg/day and body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 77 mg/kgBW/day (1,000 mg/kg) and a NOAEL of 39 mg/kgBW/day (500 mg/kg) were calculated based on the results of this experiment.

Nickel chloride was administered in drinking water to pregnant mice (CD-1 strain) from the 2nd through the 17th day of gestation at concentrations of 0, 500 or 1,000 mg/L (0, 80 and 160 mg/kgBW/day, respectively; as cited by authors; U.S. EPA 1983). Exposure to Ni at a concentration of 1,000 mg/L resulted in significantly fewer pregnancies, lower fetal body weight, and lower maternal body weights. No fetal or maternal effects were observed

in the 500 mg/L group. A LOAEL of 160 mg/kgBW/day and a NOAEL of 80 mg/kgBW/day were calculated based on the results of this experiment.

Rats were exposed to nickel chloride in drinking water at concentrations of 0, 50, 250 and 500 mg/L for two generations (Kimmel et al. 1986). At an exposure concentration of 500 mg/L, adult and pup body weights were reduced, and the number of live pups per litter throughout lacatation was reduced. No effects on number of pregnancies, fertility, or number of live litters were observed at any exposure concentration. A greater effect on reduced pup weight and number of live pups through lacatation was observed in the second generation. A water ingestion rate of 0.0435 L/day and a body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 62 mg/kgBW/day and a NOAEL of 31 mg/kgBW/day were calculated based on the results of this experiment.

Nickel chloride was administered to CD rats in drinking water at concentrations of 0, 50, 250 or 500 mg/L for two generations (Price et al. 1988). Among pregnant females, reduced body weight and increased mortality were observed at the higher exposure concentrations during late gestation, parturition and lactation. Water intake was also significantly reduced, making it difficult to separate direct effects of Ni on dams from indirect effects associated with reduced water intake. Adverse effects on offspring were observed during the perinatal and postnatal periods, rather than during gestation. Significant reductions in live litter size and postnatal body weight of the F_2 litters were observed in the group exposed to Ni at a concentration of 500 mg/L. Nickel consumption during gestation of the F2 litter was 23.1 and 42.1 mg/kgBW/day for the 250 and 500 mg/L exposure groups, respectively (reported by authors). A LOAEL of 42.1 mg/kgBW/day and a NOAEL of 23.1 mg/kgBW/day were calculated based on the results of this experiment.

Nickel (0, 10, 30, or 100 mg/L as nickel chloride) was added to drinking water to determine its effects on reproduction of Wistar rats (Kakela et al. 1999). Females, males, or both were exposed to nickel from before copulation until day 21 of lactation. When males were exposed at a concentration of 30 mg/L, the number of pregnancies and the number of pups born were significantly reduced. Shrinkage of the seminiferous tubules and a decrease in the number of basal spermatogonia were observed. When females or both parents were exposed at a concentration of 100 mg/L, pup mortality during lactation was high. A water ingestion rate of 0.0435 L/day and body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 3.73 mg/kgBW/day and a NOAEL of 1.24 mg/kgBW/day were calculated based on the results of this experiment.

Female Long-Evans rats were exposed to nickel chloride in drinking water at concentrations of 0, 10, 50 or 250 mg/L for 11 weeks prior to mating and then during 2 successive gestation and lactation periods (Smith et al. 1993). Maternal body weight and indices of reproductive performance (litter size, sex ratio at birth, initial body weight and weight gain during lactation) were comparable across treatment groups. Perinatal mortality was significantly higher for the 250 mg/L exposure group for the first lactation period; the proportion of dead pups per litter was significantly higher for all nickel-exposed groups in the second lactation period. A LOAEL of 1.33 mg/kgBW/day (10 mg/L group; mg/kgBW/day conversion cited by authors) and an estimated NOAEL of 0.13 mg/kgBW/day were calculated based on the results of this experiment.

Wistar rats were exposed to dietary Ni (as nickel sulfate hexahydrate) at concentrations of 0, 250, 500, 1,000 or 2,500 mg/kg for three generations (Ambrose et al. 1976). No adverse effects on fertility, gestation, viability and lactation indices were observed at any dietary concentration of Ni tested. Number of siblings cast per litter and weaned per litter decreased with increasing Ni concentration; however, no statistical analysis was conducted by the authors for these endpoints. Offspring growth was significantly reduced in rats exposed at concentrations of 1,000 and 2,500 mg/kg. A LOAEL of 80 mg/kgBW/day (1,000 mg/kg) and a NOAEL of 40 mg/kgBW/day (500 mg/kg) were calculated based on the results of this experiment. These values were selected as TRVs to evaluate toxicity of nickel to mammals for this risk assessment.

Ambrose, A. M., P. S. Larson, J. F. Borzelleca and G. R. J. Hennigar (1976). "Long-term toxicologic assessment of nickel in rats and dogs." J. Food Sci. Tech. 13: 181-187.

Dieter, M. P., C. W. Jameson, A. N. Tucker, M. I. Luster, J. E. French, H. L. Hong and G. A. Boorman (1988). "Evaluation of tissue disposition, myelopoietic and immunologic responses in mice after long-term exposure to

nickel sulfate in the drinking water." J. Toxicol. Environ. Health 24: 357-372.

Kakela, R., A. Kakela and H. Hyyarinen (1999). "Effects of nickel chloride on reproduction of the rat and possible antagonistic role of selenium." Comp. Biochem. Physiol. Part c 123: 27-37.

Kimmel, G. L., C. J. Price, B. R. Sonawane, R. Rubenstein and J. S. Bellin (1986). "The effect of nickel chloride in drinking water on reproductive and developmental parameters." Teratology 33(3): 90C.

Price, C. J., J. D. George, M. C. Marr, P. E. Sanderson, R. Rubenstein, G. L. Kimmel, B. R. Sonawane, A. Bathija and C. de Rosa. 1988. Two-Generation Reproduction and Fertility Study of Nickel Chloride Administered to CD Rats in the Drinking Water. Fertility and Reproductive Performance of the F1 Generation. Final Study Report (III of III). RTI Master Protocol Number RTI-182/472U-3228-07. Report to Office of Solid Waste Management, U.S. EPA by Research Triangle Institute. pp.

Smith, M. K., E. L. George, J. A. Stober, H. A. Feng and G. L. Kimmel (1993). "Perinatal toxicity associated with nickel chloride exposure." Environ. Res. 61: 200-211.

U.S. EPA. 1983. Fetotoxic Effects of Nickel in Drinking Water in Mice. EPA/600/1-83-007. U.S. Environmental Protection Agency. pp.

U.S. EPA. 1988. Recommendations For and Documentation of Biological Values for use in Risk Assessment. EPA/600/6-87-008. United States Environmental Protection Agency.

A.11.2 Toxicity to Birds

Day-old Hubbard broiler chicks were fed diets containing nickel (Ni) (as nickel sulfate or nickel acetate) at concentrations of 0, 100, 300, 500, 700, 900, 1,100 and 1,300 mg/kg for four weeks (Weber and Reid 1967). Growth of chicks was significantly reduced at dietary concentrations of 700 mg/kg and greater; this concentration was selected as the LOAEL as growth reduction is an ecologically significant effect. A food ingestion rate of 0.0126 kg/day and body weight of 0.121 kg (cited for 14-day old chicks, U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 72.9 mg/kgBW/day and a NOAEL of 52 mg/kgBW/day were calculated based on the results of this experiment.

Mallard ducklings were exposed to 0, 176, 774 and 1,069 mg/kg Ni diet for 90 days (Cain and Paifford 1981). No effects on growth or mortality were observed in ducklings exposed to up to 774 mg/kg Ni as nickel sulfate. Seventy percent mortality was observed in the group of ducklings which received the diet containing 1,069 mg/kg. A LOAEL of 107 mg/kgBW/day and a NOAEL of 77.4 mg/kgBW/day were calculated. These values were selected as TRVs to evaluate toxicity of nickel to birds for this risk assessment.

Cain, B.W. and E.A. Paifford. 1981. Effects of dietary nickel on survival and growth of mallard ducklings. Arch. Environ. Contam. Toxicol. 10:737-745.

Sugden, L.G., E.A. Driver and M.C.S. Kingsley. 1981. Growth and energy consumption by captive mallards. Can. J. Zool. 59:1567-1570.

Weber, C.W. and B.L. Reid. 1967. Nickel toxicity in growing chicks. J. Nutrition. 95:612-616.

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. EPA/600/6-87-008.

A.12 Selenium

A.12.1 Toxicity to Mammals

Young male Sprague-Dawley rats were fed diets containing Se (either as sodium selenate or seleniferous wheat) at concentrations of 0, 1.6, 3.2, 4.8, 6.4, 8.0, 9.6 or 11.2 mg/kg for 6 weeks {Halverson, 1966 #1286}. Weekly weight

gain and survival were measured. Significant growth depression was observed in rats fed diets containing Se at concentrations of 6.4 mg/kg or higher. Mortality was observed in groups fed Se at concentrations of 8.0 mg/kg or higher; one hundred% mortality was observed in the group fed Se at a concentration of 11.2 mg/kg. Based on the reduced growth observed at an exposure concentration of 6.4 mg/kg, a LOAEL of 6.4 mg/kg and a NOAEL of 4.8 mg/kg were identified. Body weights of 0.108 kg and 0.117 kg and food ingestion rates of 0.0113 kg/day and 0.0134 kg/day cited by the authors for the 6.4 mg/kg and 4.8 mg/kg exposure groups were used to convert the exposure concentrations to units on mg/kgBW/day. A LOAEL of 0.67 mg/kgBW/day and a NOAEL of 0.55 mg/kgBW/day were calculated based on results of this experiment.

Male house rats (Rattus rattus) were fed diets containing Se (as sodium selenite) at concentrations of 0, 2 and 4 mg/kg for 5 weeks (Kaur and Parshad 1994). Ingestion of a diet containing Se at 4 mg/kg caused a significant decrease in sperm concentration, motility, the percentage of live spermatozoa, and testicular and cauda epididymal weight. A dose-dependant effect of Se on sperm morphology was observed; sperm from rats fed 2 mg/kg and 4 mg/kg dietary Se had three and 20 times more abnormalities than sperm from control rats, respectively. An ingestion rate of 0.016 kg/day (U.S. EPA 1988; value cited for 150 g Fischer 344 rats) and body weights of 0.14 and 0.15 kg (cited by authors for rats from the 4 and 2 mg/kg groups, respectively) were used to convert the exposure units to mg/kgBW/day. A LOAEL of 0.46 mg/kgBW/day and a NOAEL of 0.21 mg/kgBW/day were calculated based on the results of this experiment.

Long-Evans rats were given drinking water containing Se (either as sodium selenite or sodium selenate) at concentrations of 0 or 2 g/ml for 180 days (Schroeder 1967). Mice (Charles River CD strain) were given selenite in drinking water at a concentration of 0 or 2 g/ml for 360 days. Increased mortality was observed in rats given selenite in drinking water (58 and 30 % after two months for males and females, respectively). Livers of rats that died were grossly abnormal, with fatty infiltration and degeneration, and cellular atrophy. No adverse effects were observed in mice. A water ingestion rate of 0.053 L/day and body weight of 0.43 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. Based on the mortality observed in rats, a LOAEL of 0.25 mg/kgBW/day and an estimated NOAEL of 0.025 mg/kgBW/day were calculated based on the results of this experiment.

Rosenfeld and Beath (1954) evaluated toxicity of Se in drinking water to rats. Rats were exposed to potassium selenate at concentrations of 1.5, 2.5 and 7.5 mg/L for one year. No adverse effects on reproduction were observed among rats exposed to 1.5 m/L Se, but the number of second generation young was reduced by 50 % in the group exposed to 2.5 mg/L. An ingestion rate of 0.046 L/day and body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.33 mg/kgBW/day and a NOAEL of 0.20 mg/kgBW/day were calculated based on the results of this experiment. The TRV values from this study will be used to evaluate the risk posed by Se to mammalian receptors.

Kaur, R. and V.R. Parshad. 1994. Effects of dietary selenium on differentiation, morphology and functions of spermatozoa of the house rat, Rattus rattus. Mutat. Res. 309(1):29-35.

Rosenfeld, I. And O.A. Beath. 1954. Effect of selenium on reproduction in rats. Proc. Soc. Exp. Biol. Med. 87:295-297.

Schroeder, H.A. 1967. Effects of selenate, selenite and tellurite on the growth and early survival of mice and rats. J. Nutr. 92:334-338.

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. EPA/600/6-87/008.

A.12.2 Toxicity to Birds

Black-crowned night herons were fed diets containing Se as selenomethionine at concentrations of 0 and 10 mg/kg for 13 days prior to egg laying (Smith et al. 1988). Hatching success, organ weights, hemoglobin concentration, hematocrit and eggshell thickness did not differ between controls and experimental birds. Developmental malformations commonly associated with Se exposure were not observed in heron embryos or hatchlings. An ingestion rate of 0.161 kg/day (Kushlan 1978) and body weight of 0.883 kg (Dunning 1993) were used to convert

the exposure concentrations to units of mg/kgBW/day. A NOAEL of 1.8 mg/kgBW/day was calculated based on the results of this experiment.

A feeding study with mallard ducks was conducted to identify diagnostic criteria for Se toxicosis in birds (Albers et al. 1996). One-year old male mallards were fed diets containing 0, 10, 20, 40 or 80 mg/kg Se as seleno-DL-methionine for 16 weeks. All ducks receiving diets containing 80 mg/kg died; 15 % of the birds fed 40 mg/kg Se died. Food consumption and body weight were significantly decreased in birds that received the 40 mg/kg Se diet; muscular atrophy, delayed molt, sloughed or broken claws and loss of feathers from the head and neck were also observed in this group. Testis weights were significantly decreased in the males which received the 20 mg/kg diet. Proposed diagnostic criteria for non-fatal chronic selenosis were low body weight due mostly to loss of breast muscle mass, poor plumage, delayed molt, a liver Se concentration that exceeds 66 mg/kg dry weight, reduced hatching success or an increased number of musculoskeletal abnormalities in embryos, or eggs that have a concentration of Se exceeding 10 mg/kg dry weight. An ingestion rate of 0.139 kg/day and adult body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.2 mg/kgBW/day (20 mg/kg; effects on testis) and a NOAEL of 1.1 mg/kgBW/day (10 mg/kg) were calculated based on the results of this experiment.

American kestrels were fed diets containing Se (as selenomethionine) at concentrations of 0, 6 or 12 mg/kg (dry weight) for 11 weeks (Santolo et al. 1999). No differences in egg production, hatchability, or incidence of embryonic malformations were observed in any treatment group. Fertility was significantly lower in birds fed diets containing 12 mg/kg Se as compared to control birds. To convert the dietary concentration from dry to wet weight, a percent moisture content of 32 % (mean water content for small mammals; Sample and Suter 1994) was assumed, resulting in dietary exposure concentrations of 4.08 and 8.16 mg/kg. An ingestion rate of 0.0307 kg/day (Barrett and Mackey 1975) and body weight of 0.111 kg (Dunning 1993) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.26 mg/kgBW/day and a NOAEL of 1.13 mg/kgBW/day were calculated based on the results of this experiment.

Wiemeyer and Hoffman (1996) evaluated dietary toxicity of Se (as selenomethionine) to Eastern screech owls. Owls were fed diets containing 0, 4.4 or 13.2 mg/kg Se (wet weight). Laboratory analysis of the diets confirmed the following exposure concentrations: not detected (ND) to 0.13 mg/kg for the control group, and 3.53 and 12 mg/kg for the two exposure groups. Adult body weight, number of eggs laid per pair, number of eggs hatched per pair, and number of nestlings surviving to five days were significantly lower for birds which received the highest dose. Control and low dosage birds did not differ in adult body weight, food consumption, or reproductive parameters. An ingestion rate of 0.025 kg/day (Pattee et al. 1988) and adult body weight of 0.185 kg (Dunning 1993) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.62 mg/kgBW/day and a NOAEL of 0.48 mg/kgBW/day were derived based on the results of this study.

Mallard ducks were fed diets containing Se as selenomethionine at concentrations of 0 and 10 mg/kg for 41 days prior to egg laying (Heinz et al. 1987). Birds exposed to dietary Se produced fewer young and had a higher incidence of abnormal embyros than controls. An ingestion rate of 0.139 kg/day and body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.11 mg/kgBW/day and an estimated NOAEL of 0.11 mg/kgBW/day were calculated based on the results of this experiment.

Heinz et al. (1989) evaluated dietary toxicity of organic Se as selenomethionine to mallard ducks. Ducks were exposed to diets containing 0, 1, 2, 4, 8 or 16 mg/kg Se diet (wet weight) for 100 days. Reduced duckling growth and survival was observed in groups where parents were fed diets containing 8 or 16 mg/kg Se. Diets containing 8 and 16 mg/kg Se caused malformations in 6.8 and 67.9 %, respectively, of unhatched eggs compared with 0.6 % for controls. An ingestion rate of 0.10 kg/day and body weight of 1.0 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.8 mg/kgBW/day and a NOAEL of 0.4 mg/kgBW/day were calculated. Based on the ecological significance of the endpoint (survival) and because the LOAEL is the lowest cited adverse effect level for birds, the TRV values from this study will be used to evaluate the risk posed by Se to avian receptors.

Albers, P.H., D.E. Green and C.J. Sanderson. 1996. Diagnostic criteria for selenium toxicosis in aquatic birds: Dietary exposure, tissue concentrations and macroscopic effects. J. Wildl. Diseases. 32(3):468-485.

Barrett, G.W. and C.V. Mackey. 1975. Prey selection and caloric ingestion rate of captive American kestrels. Wilson Bull. 87(4):514-519.

Dunning, J.B. Jr. 1993. CRC Handbook of Avian Body Masses. CRC Press, Boca Raton, FL.

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.

Heinz, G.H., D.J. Hoffman and L.G. Gold. 1989. Impaired reproduction of mallards fed an organic form of selenium. J. Wildl. Manage. 53:418-428.

Kushlan, J.A. 1978. "Feeding ecology of wading birds." In: Wading Birds. Sprunt, A., J.C. Ogden and S. Winckler (eds.). National Audubon Society Research Report No. 7. p. 249-297.

Pattee, O.H., S.N. Wiemeyer and D.M. Swineford. 1988. Effects of dietary fluoride on reproduction in Eastern screech-owls. Arch. Environ. Contam. Toxicol. 17:213-218.

Piccirillo, V.J. and R.P. Quesenberry. 1980. Reproductive capacities of control mallard ducks (Anas platyrhynchos) during a one-generation reproduction study. J. Environ. Path. Toxicol. 4:133-139.

Sample, B.E. and G.W. Suter II. 1994. Estimating exposure of terrestrial wildlife to contaminants. ES/ER/TM-125. Oak Ridge National Laboratory, Oak Ridge, TN.

Santolo, G.M., J.T. Yamamoto, J.M. Pisenti and B.W. Wilson. 1999. Selenium accumulation and effects on reproduction in captive American kestrels fed selenomethionine. J. Wildl. Manage. 63(2):502-511.

Smith, G.J., G.H. Heinz, D.J. Hoffman, J.W. Spann and A.J. Krynitsky. 1988. Reproduction in black-crowned night herons fed selenium. Lake and Reservoir Management. 4(2):175-180.

Wiemeyer, S.N. and D.J. Hoffman. 1996. Reproduction in Eastern screech owls fed selenium. J. Wildl. Manage. 60(2): 332-341.

A.13 Thallium

A.13.1 Toxicity to Mammals

Thallium (as thallium chloride and thallium acetate) was administered orally to female NMRI mice and Wistar rats at concentrations of 0, 3 and 6 mg/kgBW/day from day 6 to day 15 of pregnancy (Roll and Matthiaschk 1981). In mice, no malformations of the skeleton or internal organs were observed in offspring from females exposed to either form of Tl at any concentration. A slight increase in postimplantive loss and a decrease in weight at birth was observed at an exposure concentration of 6 mg/kgBW/day thallium chloride. Exposure to either compound at a concentration of 3 mg/kgBW/day did not cause any embryotoxic effects in rats. Maternal toxicity was observed in rats exposed to the highest concentration of either compound. Based on the effects observed in rats, a LOAEL of 6 mg/kgBW/day and a NOAEL of 3 mg/kgBW/day were the results of this experiment.

Rats were exposed to Tl (as thallium sulfate) in drinking water at an exposure concentration of 10 mg/L for 60 days (Formigli et al. 1986). There was a significant reduction in sperm motility in rats exposed to Tl compared to a control group. Degenerative changes in Sertoli cells (cells that are the main regulators of normal spermatogenesis) were observed in Tl-treated rats, but not in control animals. A mean daily intake of 270 g Tl/rat and body weight of 0.365 kg (cited by authors) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 0.74 mg/kgBW/day and an estimated NOAEL of 0.074 mg/kgBW/day were calculated based on the results of this experiment. Based on the ecological significance of the endpoint (reproduction) and because the LOAEL is the lowest cited adverse effect level for mammals, the TRV values from this study will be used to evaluate the risk posed by Tl to avian receptors.

Formigli, L., R. Scelsi, P. Poggi, C. Gregotti, A. DiNucci, E. Sabbioni, L. Gottardi and L. Manzo. 1986. Thallium-induced testicular toxicity in the rat. Environ. Res. 40:531-539.

Roll, R. and G. Matthiaschk. 1981. Investigations on embryotoxic effects of thallium chloride and thallium acetate in mice and rats. Teratology. 24(2):46A-47A.

A.13.2 Toxicity to Birds

No studies evaluating chronic dietary toxicity of thallium to birds were located.

A.14 Vanadium

A.14.1 Toxicity to Mammals

Male Swiss Webster mice were exposed to V in drinking water at concentrations of 0, 20, 40, 60 and 80 mg/kgBW/day for 64 days (Llobet et al. 1993). Males were mated with untreated females for four days. A significant decrease in the pregnancy rate was observed in females mated to males which received 60 or 80 mg/kgBW/day. Sperm count was significantly reduced in males exposed to V at concentrations of 40, 60 or 80 mg/kgBW/day, but sperm motility and testes weight were not affected. However, since fertility was not affected in the 40 mg/kg group, a LOAEL of 60 mg/kgBW/day was selected by the authors.

Sprague-Dawley albino rats were exposed to V via oral intubation at concentrations of 0, 5, 10 or 20 mg/kg-day (Domingo et al. 1986). Males were administered V (as sodium metavanadate) for 60 days prior to mating with females which received the same doses for 14 days prior to mating. The females continued to receive daily doses during gestation and lactation. No significant differences in number of corpora lutea, implantations, live and dead fetuses, and resorptions were observed at any exposure concentration. An increase in the number of dead fetuses and number of resorptions was observed in rats exposed to V at concentrations of 10 and 20 mg/kgBW/day, but these increases were not statistically significant when compared to the control group (n = approximately 10 per treatment group). Significant differences in growth (body length and weight) of offspring nursed by V-treated mothers for 21 days after birth were observed at all dose levels. A LOAELof 2.1 mg/kgBW/day and an estimated NOAEL of 0.21 mg/kgBW/day will be used to evaluate risk from exposure to V for mammals.

Domingo, J.L., J.L. Paternain, J.M. Llobet and J. Corbella. 1986. Effects of vanadium on reproduction, gestation, parturition and lactation in rats upon oral administration. Life. Sci. 39:819-824.

Llobet, J.M., Colomina, M.T., J.J. Sirvent, J.L. Domingo and J. Corbella. 1993. Reproductive toxicity evaluation of vanadium in male mice. Toxicology. 80(2,3):199-206.

A.14.2 Toxicity to Birds

Adult female Japanese quail were fed diets containing V at concentrations of 0, 50, 100 or 300 mg/kg for one month (Hafez and Kratzer 1976). No adverse effects on egg production or egg weight were observed at any exposure concentration. A body weight 0.182 kg and food ingestion rate of 0.0244 kg/day (cited by (Hussein et al. 1988) for laying females) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 40.2 mg/kgBW/day was observed in this experiment.

Day-old cockerels were fed a diet supplemented with V (as ammonium vanadate) at concentrations of 0 or 5 g/g for 32 days (Nielsen et al. 1980). No adverse effects on growth, hematocrit, kidney (Na,K) ATPase, liver weight/body weight ratio or gall bladder size were observed. The addition of 500 g/g chromium to the V-supplemented diet resulted in significantly depressed growth and hematocrit, enlarged livers, distended gall bladders and elevated plasma cholesterol. Because no adverse effects were observed when cockerels were only exposed to V, the 5 g/g exposure concentration was considered a NOAEL. A food ingestion rate of 0.0126 kg/day and body weight of 0.121 kg cited for 14-day old chicks (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 0.5 mg/kgBW/day was observed in this experiment.

Day-old male White leghorn chicks were fed diets containing V (as calcium orthovanadate) at concentrations of 0,

12.5 or 25 mg/kg for four weeks (Kubena et al. 1986). No mortality was observed at any exposure concentration. No effects on growth were observed in vanadium-treated chicks at 1, 2, 3 or 4 weeks of age. Because no adverse effects were observed, the exposure concentration of 25 mg/kg was identified as a NOAEL from this experiment. A body weight of 0.123 kg (cited for 14-day old male white Leghorn chicks, U.S. EPA 1988) and an ingestion rate of 0.0128 kg/day (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 2.6 mg/kgBW/day was observed in this experiment.

Twenty-five week old White Leghorn hens were fed diets containing V (from dicalcium phosphate) at concentrations of 0, 3 or 6 mg/kg for four weeks (Sell et al. 1986). Albumen quality was slightly affected in hens fed V at 3 mg/kg; it was significantly decreased in eggs produced by hens fed V at a concentration of 6 mg/kg. No effect on egg production or egg weight was observed at either exposure dose. Because albumen quality is not a biologically significant effect, the 6 mg/kg exposure concentration was identified as a NOAEL. A body weight of 1.45 kg (U.S.EPA 1988) and an ingestion rate of 0.111 kg/day (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 0.46 mg/kgBW/day was observed in this experiment.

Seventy-two week old White Leghorn hens were fed diets containing V (as ammonium metavanadate) at concentrations of 0 or 5 mg/kg for six weeks (Benabdeljelil and Jensen 1989). V-treated hens laid eggs with reduced albumen quality. No significant effects were observed on egg production rates or body weights. The ecological significance of reduced albumen quality is not known. Because no ecologically significant adverse effects were observed, the exposure concentration of 5 mg/kg was identified as a NOAEL from this experiment. A body weight of 1.45 kg and an ingestion rate of 0.117 kg/day (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 0.4 mg/kgBW/day was observed in this experiment.

Twenty-five week old White Leghorn hens were fed diets containing V (as ammonium metavanadate) at concentrations of 0 or 5 mg/kg for eight weeks (Benabdeljelil and Jensen 1989). Albumen quality and hen body weights were significantly lower in the V-exposed group. No significant effects were observed on egg production. The ecological significance of reduced albumen quality is not known. Because no ecologically significant adverse effects were observed, the exposure concentration of 5 mg/kg was identified as a NOAEL from this experiment. A body weight of 1.45 kg and an ingestion rate of 0.102 kg/day (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 0.35 mg/kgBW/day was observed in this experiment.

The interaction between dietary phosphorus (P) and V was evaluated by Qureshi et al. (1999). Day-old broiler chicks were fed P-deficient (0.05% P) or P-supplemented (0.60% P) diets containing V as ammonium vanadate at concentrations of 0, 10, 25 or 50 mg/kg for 21 days. Mortality was observed in all P-deficient treatment groups. Growth inhibition was observed in chicks fed diets containing V at concentrations of 25 or 50 mg/kg, however no statistical analysis was conducted on this endpoint. Immunological responses were significantly increased in chicks exposed to V at all exposure concentrations. The biological significance of a stimulated immune response is not known. Although growth inhibition is considered an ecologically relevant endpoint, due to the lack of statistical analysis on this endpoint this study was not used to derive a TRV.

Twenty-eight week old White Leghorn hens were fed diets containing V at concentrations of 0, 100 or 300 mg/kg for one month (Hafez and Kratzer 1976). Egg production was significantly decreased in hens fed diets containing V at a concentration of 300 mg/kg. Because of the ecological significance of the reduced egg production (reproductive effect), an exposure concentration of 300 mg/kg was identified as the LOAEL. A body weight 1.45 kg and food ingestion rate of 0.103 kg/day (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 21.3 mg/kgBW/day and a NOAEL of 7.1 mg/kgBW/day were observed in this experiment.

Day-old male broiler chicks were fed practical diets or semi-purified diets containing V (as ammonium meta vanadate) at concentrations of 0, 50, 100 or 200 mg/kg for four weeks (Hafez and Kratzer 1976). In the first experiment, growth was significantly reduced in chicks fed diets containing V at a concentration of 100 mg/kg. No mortality was observed at this exposure concentration. In chicks fed the semi-purified diet, significant growth reduction and 30% mortality was observed in chicks fed diets containing V at a concentration of 50 mg/kg. Due to the reduced survival and growth observed in the second experiment, 50 mg/kg was identified as the LOAEL. A body weight of 0.344 kg (cited in U.S. EPA 1988 for 14-day old broiler chicks) and a food ingestion rate of 0.0304 kg/day (calculated using allometric food consumption equation from U.S.EPA 1988) were used to convert the

exposure concentrations to units of mg/kgBW/day. A LOAEL of 4.4 mg/kgBW/day and an estimated NOAEL of 0.44 mg/kgBW/day were calculated based on results of this experiment.

Day-old female broiler chicks were fed diets containing V (as vanadyl chloride) at concentrations of 0, 10, 20 or 40 mg/kg for three weeks (Blalock and Hill 1987). Growth was significantly reduced in birds fed V, however statistical analysis was not conducted to evaluate at which exposure concentration growth was significantly different from growth in control birds. Significant mortality was observed in chicks fed diets containing 40 mg/kg V. Because of the ecological significance of the observed effect (reduced survival), an exposure concentration of 40 mg/kg was identified as the LOAEL. Body weights of 0.152 kg and 0.237 kg (cited by authors for the 40 and 20 mg/kg exposure groups, respectively) and ingestion rates of 0.0153 and 0.0222 kg/day (calculated using allometric food consumption equation from U.S.EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 4.03 mg/kgBW/day and a NOAEL of 1.87 mg/kgBW/day was observed in this experiment.

One-day old female laying strain chickens (Hyline W-36) were fed diets containing V (as calcium orthovanadate) at calculated concentrations of 0, 25, 50 or 100 mg/kg for 15 months (Phillips et al. 1982). Exposure to V at concentrations of 50 or 100 mg/kg caused a 15 to 30% growth depression at the onset of egg production (25 weeks of age) and delayed sexual maturity. The data were not presented in this paper, and the statistical significance of the observed effects was not discussed. Based on the growth and reproductive effects observed at an exposure concentration of 50 mg/kg, this dose was identified as the LOAEL. A food ingestion rate of 0.103 kg/day and adult body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 3.6 mg/kgBW/day and a NOAEL of 1.8 mg/kgBW/day were observed in this experiment.

Calcium orthovanadate was fed to 29-week old female Leghorn chickens at nominal V concentrations of 0, 12.5, 25, 50 or 100 mg/kg for 20 weeks (Kubena and Phillips 1982). Diets were analyzed, and were within 10% of the calculated values. Mortality was only observed in hens fed the 100 mg/kg diet. Egg production and hen body weights were significantly lower in hens exposed to V at concentrations of 50 and 100 mg/kg. Because of the ecological significance of the reduced egg production (reproductive effect), an exposure concentration of 50 mg/kg was identified as the LOAEL. Body weights of 1.461 kg and 1.554 kg (cited by authors for the 50 and 25 mg/kg exposure groups, respectively) and ingestion rates of 0.103 and 0.109 kg/day (calculated using allometric food consumption equation from U.S.EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 3.5 mg/kgBW/day and a NOAEL of 1.8 mg/kgBW/day was observed in this experiment.

Fifty-week old White Leghorn hens were fed diets containing V (as ammonium vanadate) at concentrations of 0 or 40 mg/kg for seven weeks (Ousterholt and Berg 1981). Dietary V resulted in a significant decrease in albumin quality, egg production, egg weight, body weight, food consumption and shell quality. Because of the ecological significance of the reduced egg production (reproductive effect), an exposure concentration of 40 mg/kg was identified as the LOAEL. A body weight of 1.45 kg (U.S. EPA 1988) and an ingestion rate of 0.112 kg/day and (cited by authors) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 3.1 mg/kgBW/day and an estimated NOAEL of 0.31 mg/kgBW/day were calculated based on results from this experiment.

Day-old White Plymouth Rock hens were fed diets containing V (as NH_4VO_3) at concentrations of 0, 10 or 25 mg/kg for 2 weeks (Hathcock et al. 1964). Growth was significantly reduced in chicks fed V at 25 mg/kg, and 95% mortality was also observed in this treatment group. No adverse effects were observed in chicks fed V at a concentration of 10 mg/kg. Due to the reduced survival, the 25 mg/kg exposure concentration was identified as the LOAEL. A body weight of 0.073 kg and ingestion rate of 0.0082 kg/day were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.8 mg/kgBW/day and a NOAEL of 1.1 mg/kgBW/day were observed in this experiment.

Seven-day old broiler chicks were fed diets containing V (as $Ca_3[VO_4]_2$) at concentrations of 0, 10, 15, 20, 30 or 40 mg/kg for three weeks (Romoser et al. 1961). Growth was significantly depressed in chicks fed diets containing V at concentrations of 30 and 40 mg/kg. Due to the reduced growth, the 30 mg/kg exposure concentration was identified as the LOAEL. A body weight of 0.344 kg (cited in U.S. EPA 1988 for 14-day old broiler chicks) and a

food ingestion rate of 0.0304 kg/day (calculated using allometric food consumption equation from U.S.EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.65 mg/kgBW/day and a NOAEL of 1.76 mg/kgBW/day were observed in this experiment.

Forty-three week old Single Comb White Leghorn hens were fed diets containing V (as ammonium metavanadate) at concentrations of 0, 10, 30, or 100 mg/kg for four weeks (Benabdeljelil and Jensen 1990). At 10 mg/kg, albumin quality was significantly reduced. Egg production was significantly decreased in hens exposed to V at concentrations of 30 and 100 mg/kg. Body weight and food intake were significantly reduced in hens fed the 100 mg/kg diet. Because of the ecological significance of the reduced egg production (reproductive effect), an exposure concentration of 30 mg/kg was identified as the LOAEL. A body weight of 1.45 kg (U.S. EPA 1988) and ingestion rates of 0.098 kg/day and 0.107 kg/day (cited by authors for the 30 and 10 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.03 mg/kgBW/day and a NOAEL of 0.74 mg/kgBW/day was observed in this experiment.

Day-old White Plymouth Rock cross chicks were fed diets containing V (as $NaVO_3$) at concentrations of 0, 5, 10, 15, 20 or 25 mg/kg for two weeks (Hill 1974). Growth was significantly reduced at all exposure concentrations. A body weight of 0.066 kg and an ingestion rate of 0.0075 kg/day (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.57 mg/kgBW/day and an estimated NOAEL of 0.057 mg/kgBW/day were calculated based on the results of this experiment. Reduced growth is considered an ecologically relevant effect, however due to the short exposure duration this study was not used to derive the TRV for vanadium for birds.

Sell et al. (1982) evaluated the effects of dietary vanadium from two commercial sources of dicalcium phosphate (Dical A or B) or from the commercial diets supplemented with reagent grade ammonium vanadate on egg production and quality. Forty-one week old White Leghorn hens were fed diets containing V at measured concentrations of 1.4 (Dical A), 10.2 (Dical B), 27.5 (Dical A plus NH_4VO_3) or 30.5 mg/kg (Dical B plus NH_4VO_3) for 4 weeks (Sell et al. 1982). Albumen quality was significantly decreased at exposure concentrations of 10.2, 27.5 and 30.5 mg/kg. Egg production was significantly decreased at exposure concentrations of 27.5 and 30.5 mg/kg.. Because of the ecological significance of the reduced egg production (reproductive effect), an exposure concentration of 27.5 mg/kg was identified as the LOAEL. A body weight 1.45 kg (U.S. EPA 1988) and ingestion rates of 0.106 and 0.116 kg/day (cited by authors for the 27.5 and 10.2 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.0 mg/kgBW/day and a NOAEL of 0.82 mg/kgBW/day was observed in this experiment; this value will be used to evaluate toxicity of vanadium to avian receptors in this risk assessment.

Mallard ducks were exposed to vanadium (V) (as vanadyl sulfate) at dietary concentrations of 2.84, 10.36 or 110 mg/kg for 12 weeks (White and Dieter 1978). No effects on body weight, blood chemistry or mortality were observed at any exposure concentration. An ingestion rate of 0.121 kg/day and body weight of 1.17 kg (reported by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 11.4 mg/kgBW/day was observed in this experiment. A LOAEL was calculated using a factor of 10 (114 mg/kgBW/day). To be consistent with the Onondaga Lake ERA, these values will be used to evaluate risk to birds from exposure to V in this risk assessment.

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A.15 Zinc

A.15.1 Toxicity to Mammals

European ferrets were fed diets containing Zn at concentrations of 500, 1,500 and 3,000 mg/kg; the control diet contained 27 mg/kg Zn (Straube et al. 1980). The groups fed the two highest concentrations showed severe signs of toxicity after one to two weeks of exposure, and 100 % mortality was observed in the 3,000 mg/kg group in less than two weeks. Ferrets fed a diet containing 1500 mg/kg Zn lost 40 to 50 % of their body weight, and the animals had soft, enlarged kidneys. A diffuse nephrosis and glomerular damage were observed upon histopathologic examination. No significant adverse effects were observed in the 500 mg/kg group. A food ingestion rate of 0.17 kg/day and body weight of 0.6 kg were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 425 mg/kgBW/day and a NOAEL of 142 mg/kgBW/day were calculated based on the results of this experiment.

Adult mink were exposed to a diet supplemented with 1,000 mg/kg Zn (as $ZnSO_4 \cdot 7H_20$) for 22 weeks; the control diet contained 20.2 mg/kg Zn (Bleavins et al. 1983). Kits were maintained on the same diet as their parents for 12 weeks following their birth. Gestation length, litter size, kit birth weight and kit mortality to weaning were not significantly different between the Zn-treated and control groups. Reduced growth rate, alopecia, achromatrichia and profound immunosuppression were observed in the offspring produced by the Zn-treated females. Based on the reduced growth observed in offspring, an exposure concentration of 1,020.2 mg/kg was selected as the LOAEL. An ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987) was used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 225 mg/kg BW/day and an estimated NOAEL of 22.5 mg/kg BW/day were calculated.

Schlicker and Cox (1968) exposed Sprague-Dawley rats for 37 days during mating and gestation period to two dietary dose levels of zinc oxide (2,000 and 4,000 mg/kg); a control group was included in the experimental design. No adverse effects were observed at an exposure concentration of 2,000 mg/kg. Increased rates of fetal resorption and reduced fetal growth rates were observed in rats exposed to Zn at a concentration of 4,000 mg/kg. A LOAEL of 320 mg/kg BW/day and a NOAEL of 160 mg/kg BW/day were calculated based on results of this experiment. The TRV values from this study will be used to evaluate the risk posed by Zn to mammalian receptors.

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A.15.2 Toxicity to Birds

White leghorn hens were fed diets containing Zn (as zinc oxide) at concentrations of 0 or 20,000 mg/kg for five days (Palafox and Ho-A 1980). All birds were fed the basal diet from 5 days to 12 weeks. Fertility and hatchability of eggs laid by Zn-treated hens collected 14 to 28 days after the 5-day feeding period were significantly decreased compared to those laid by control birds. Egg production and body weight of Zn-treated birds were also significantly depressed from 0 to 4 weeks. An ingestion rate of 0.103 kg/day and body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 1,420 and an estimated NOAEL of 142 mg/kgBW/day were calculated based on the results of this experiment.

Mallard ducks were fed diets containing supplemental Zn (as zinc carbonate) at concentrations of 0, 3,000, 6,000, 9,000 or 12,000 mg/kg for 60 days (Gasaway and Buss 1972). The Zn concentration of the basal diet, a pelleted chicken developer-turkey finisher, was 250 mg/kg. To convert the exposure concentrations to a wet weight basis, a 7.5 percent moisture content cited for a chick developer diet (Street 1978) was used, resulting in exposure concentrations of 231 (control), 3,006, 5,550, 8,556 and 11,331 mg/kg. Food consumption of treated birds decreased immediately after being placed on the Zn-treated pellets; food consumption was inversely related to the concentration of Zn in the diet. In all birds fed Zn-supplemented diets, leg paralysis, diarrhea and weight loss were observed within 10 days. The gonads of Zn-treated birds were so reduced in size reproductive function may have been lost. Two of six birds fed Zn at a concentration of 3,000 mg/kg survived to the end of the experiment; all other experimental birds died. An ingestion rate of 0.139 kg/day and body weight of 1.25 kg (Piccirillo and Quesenberry 1980) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 938 mg/kgBW/day and an estimated NOAEL of 93.8 mg/kgBW/day were calculated based on the results of this experiment.

Hussein et al. (1988) evaluated the effects of exposure to high dietary zinc on egg production in Japanese quail (Coturnix coturnix japonica) laying hens. Laying hens were fed a basal diet (control) or a diet containing Zn at a concentration of 1.5% (as ZnO) for seven days. Food intake, egg production, and egg shell breaking strength were significantly reduced by exposure to Zn at this concentration (15,000 mg/kg). Hens were returned to the basal diet after seven days; food intake and egg production returned to levels similar to that of controls by the third week post-treatment. Based on the decrease in egg production observed at an exposure concentration of 15,000 mg/kg, this concentration was identified as a LOAEL. A body weight of 0.156 kg and food ingestion rate of 0.00843 kg/day (cited by authors) were used to convert the exposure concentration in mg/kg to units of mg/kgBW/day. A LOAEL of 811 mg/kgBW/day and an estimated NOAEL of 81.1 mg/kgBW/day were identified from this experiment.

Day-old broiler chicks were pair-fed diets containing Zn as (ZnO) at measured concentrations of 5280 mg/kg (zinc-exposed) or 73 mg/kg (pair-fed control) for 4 weeks (Dean et al. 1991). Growth was significantly depressed in chicks fed high diets containing Zn at a concentration of 5280 mg/kg compared to pair-fed control birds despite equivalent food consumption. Altered thyroid histology (follicular cell hyperplasia and reduced follicle area) and a significant decrease in concentrations of circulating thyroid hormones (serum GH, T4 and T3) were observed in birds fed the high zinc diet, indicating that the impaired growth may be due in part to reduced thyroid function. Based on the reduced growth, the exposure concentration of 5280 mg/kg was identified as a LOAEL. A body weight of 0.121 and a food ingestion rate of 0.0126 (cited for 14 day-old chicks in U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 550 mg/kgBW/day and an estimated NOAEL of 55 mg/kgBW/day were calculated from results of this experiment.

Three-day old mallard ducklings were fed diets containing Zn (as zinc sulfate) at concentrations of 0 or 2,500 mg/kg for 56 days (Kazacos and Van Vleet 1989). An additional control group was fed measured amounts of basal diet based on the lowest average daily food intake of ducklings in the Zn group. Ducklings were removed from the

experimental groups and pancreatic tissue was examined to evaluate Zn-related pancreatic alterations over time. Ultrastructural features of the pancreas from the inanition control group were similar to those of the control group. Pancreatic atrophy due to decreased number and size of acinar cells was observed in Zn-treated ducklings. An ingestion rate of 0.0748 kg/day and body weight of 0.572 kg (Sugden et al. 1981; value cited for 4-week old ducklings) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 327 mg/kgBW/day and an estimated NOAEL of 32.7 mg/kgBW/day were calculated based on the results of this experiment.

Fourteen-day old female broiler chicks were fed diets containing supplemental Zn (as zinc oxide) at concentrations of 0, 2,000, 4,000 or 6,000 mg/kg for 42 days (Dewar et al. 1983). The basal diet contained 101 mg/kg Zn. Mortality was high in chicks fed the high Zn diet. Growth was significantly depressed in all chicks fed the Zn supplemented diets, and gizzard erosion and pancreatic lesions were observed in many chicks upon histopathological examination. Due to excessive food spillage, food consumption could not be measured accurately in this experiment. In a second experiment, 1-day old chicks were fed diets containing 0, 1,000, 2,000 or 4,000 mg/kg supplemental Zn for 28 days. High mortality was observed in the 4,000 mg/kg group, and growth was significantly decreased in the chicks fed Zn at concentrations of 2,000 or 4,000 mg/kg. Pancreatic lesions were found in all chicks fed Zn supplemented diets during this experiment, and gizzard erosion was observed in the two higher concentration groups. Based on adverse effects on growth, a LOAEL of 2,000 mg/kg and a NOAEL of 1,000 mg/kg supplemental Zn were identified. Ingestion rates of 0.04 and 0.044 kg/day and body weights of 0.308 and 0.335 kg (cited by authors for the 2,000 and 1,000 mg/kg experimental groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 273 mg/kgBW/day and a NOAEL of 145 mg/kgBW/day were calculated based on the results of this experiment.

Hisex laying hens were fed diets containing supplemental zinc (as ZnO) at concentrations of 0 (control, basal diet contained Zn at a concentration of 56.0 mg/kg), 4, 8, 12, 16, and 20 g/kg for three weeks (Jackson et al. 1986). Due to severe reduction of food intake, birds fed diets containing Zn at a concentration of 20 g/kg were removed from the experiment. Feather loss was observed in birids fed diets containing Zn at concentrations of 12, 16 and 20 g/kg. Damage of the gizzard lining (erosion and rupture) was observed in approximately 20% of birds fed supplemental zinc at a concentration of 16 g/kg. Food intake, body weight and egg production were significantly reduced at all Zn exposure concentrations. Based on the reduced egg production observed at the lowest exposure concentration (4 g/kg), this concentration was identified as a LOAEL. A body weight of 1.73 kg (cited by authors) and an ingestion rate of 0.103 kg/day (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 238 mg/kgBW/day and an estimated NOAEL of 8.5 mg/kgBW/day were identified from this experiment.

Berg and Martinson (1972) evaluated the effects of diet composition on toxicity of Zn to chicks. Chicks were fed four different diets containing 0 (control) or 2000 mg/kg zinc from one day to two weeks of age. Zinc added to a corn-soybean diet only depressed growth by 5.5% as compared to controls. Moderate growth depression was observed when Zn was added to corn-fish meal or sucrose-soybean meal diets (14.8 and 11.3 %, respectively), and severe growth depression was observed when 2000 mg/kg Zn was added to a sucrose-fish meal diet (44.3 %). Based on the reduced growth observed at an exposure concentration of 2000 mg/kg, this concentration was identified as a LOAEL. A body weight of 0.066 kg and food ingestion rate of 0.0075 kg/day cited for 7 day-old chicks (U.S.EPA 1988) was used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 227 mg/kgBW/day and an estimated NOAEL of 22.7 mg/kgBW/day were identified from this experiment.

One-day old chicks (New Hampshire X Single Comb White Leghorn) were fed diets containing Zn at concentrations of 37 (control), 103, or 2,183 mg/kg for 21 days (Stahl et al. 1989). A significant decrease in growth was observed in chicks exposed to dietary Zn at a concentration of 2,183 mg/kg for 21 days; no effect on growth was observed at 103 mg/kg. In addition to growth, excretion, immune response, and Fe and Cu utilization were measured. Chicks fed the two experimental diets excreted more Zn than control birds. Tissue concentrations of Zn were similar to control birds in the 103 mg/kg exposure group, while the highest exposure group accumulated Zn in their tissues. Immune responses were not affected by Zn exposure. Iron and Cu utilization were affected in birds fed the highest Zn diet; however, effects were also seen in pair-fed birds and may have been due to reduced food intake or growth. The average food ingestion rates and body weights cited in U.S. EPA 1988 for 7, 14, and 21-day old chicks (0.0075, 0.0126 and 0.019 kg/day and 0.066, 0.121 and 0.193 kg) were used to convert the concentration at which decreased growth was observed to units of mg/kgBW/day (0.013 kg/day and 0.127 kg). A LOAEL of 223.5 and a NOAEL of 10.5 were calculated.

White leghorn chicks were fed sucrose-fish meal (22% protein), corn-fish meal (28% protein) or corn-fish meal (22% protein) basal diets containing Zn (as ZnO) at concentrations of 0 (control), 200, 400, 800, 1200, 1600 or 2000 mg/kg from one-day to two weeks of age (Berg and Martinson 1972). An additional group of chicks was fed a sucrose-soybean meal diet containing Zn at concentrations of 0 (control), 400, 1200 or 2000 mg/kg. Growth and bone ash concentration were measured. With the sucrose-fish meal diet, growth and bone ash were significantly depressed at an exposure concentration of 800 mg/kg. In chicks fed the high protein corn-fish meal diet, no effect on growth were observed, but bone ash was significantly decreased at an exposure concentration of 2000 mg/kg. No significant effects on growth or bone ash were observed in chicks fed the low protein corn-fish meal or the sucrose-soybean meal basal diets at any exposure concentration. Based on the reduced growth observed in chicks fed the sucrose-fish meal diet at an exposure concentration of 800 mg/kg, this concentration was identified as a LOAEL. A body weight of 0.066 kg and food ingestion rate of 0.0075 kg/day cited for 7 day-old chicks (U.S.EPA 1988) was used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 91 mg/kgBW/day and a NOAEL of 45.5 mg/kgBW/day (400 mg/kg) were identified from this experiment. These values were not used to derive TRVs for this risk assessment due to the short duration of the exposure period.

The effects of dietary Zn on pancreatic function were evaluated in day-old chicks (Lu and Combs 1988). Chicks were fed a purified basal diet supplemented with 0, 500 or 1,000 mg/kg Zn (as zinc oxide) for nine days. Actual measured Zn concentrations in the diet were 87, 630 and 1,060 mg/kg. The purified diet was Se-deficient (0.01 mg/kg); Se is important in maintenance of pancreatic exocrine function in the chick. Growth rate, food intake and food utilization efficiency were significantly decreased in chicks fed Zn at a concentration of 1,000 mg/kg. Activities of pancreatic exportable enzymes were decreased, and these reductions were associated with reductions in digestibility of dietary starch. Similar effects were not observed in chicks fed a non-purified diet supplemented with 2,000 mg/kg for 20 days. The experiments demonstrated that the pancreas is a target organ of Zn toxicity in the chick, and that dietary factors that increase the bioavailability of Zn affect the concentration at which toxic effects are observed. Food ingestion rates of 5.5 and 5.9 g/day and body weights of 46.7 and 48.5 g (cited by authors for 1,060 and 630 mg/kg exposure groups, respectively) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 125 mg/kgBW/day and a NOAEL of 77 mg/kgBW/day were calculated based on the results of this experiment. This experiment was not used to derive TRVs for this risk assessment, because adverse growth effects were only observed when chicks were fed Se-deficient diets.

Hisex laying hens were fed diets containing supplemental zinc (as ZnO) at concentrations of 0 (control, basal diet contained Zn at a concentration of 56.0 mg/kg), 1, 2, 3, 4,5 and 6 g/kg for 140 days (Jackson et al. 1986). Significant mortality was observed in birds offered diets containing 5 or 6 g/kg added Zn (56 and 86% mortality, respectively). Significant linear reductions were observed in egg number, total egg weight, and food conversion efficiency in response to increasing dietary ZnO. Based on the significantly reduced egg production observed at an exposure concentration 2 g/kg, this concentration was identified as a LOAEL. Body weights of 1.74 and 1.86 kg (cited by authors for the 2 and 1 g/kg exposure groups, respectively) and an ingestion rate of 0.103 kg/day (U.S. EPA 1988) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 118.4 mg/kgBW/day and a NOAEL of 55.4 mg/kgBW/day were identified from this experiment.

Stahl et al. (1990) exposed white leghorn hens to a basal diet (28 mg/kg Zn) supplemented with 20, 200 or 2,000 mg/kg Zn as zinc sulfate for 44 weeks. No significant effects on fertility, progeny growth to three weeks of age, or Zn-related feather fraying were observed at any exposure concentration. Egg hatchability was less than 20 percent of controls among hens consuming 2,028 mg/kg zinc. A LOAEL of 131 mg/kgBW/day and a NOAEL of 14.5 mg/kgBW/day were calculated based on the results of this experiment. The TRV values from this study will be used to evaluate the risk posed by Zn to avian receptors.

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A.16 Bis(2-ethylhexyl)phthalate

A.16.1 Toxicity to Mammals

No studies were located that evaluated toxicity of bis(2-ethylhexyl)phthalate to mammals.

A.16.2 Toxicity to Birds

Ring doves were fed diets containing bis-2-ethylhexylphthalate at a concentration of 0 or 10 mg/kg for four weeks (Peakall 1974). No significant effect on eggshell thickness or permeability was observed. A NOAEL of 1.1 mg/kgBW/day was observed in this experiment.

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A.17 Chlordane

A.17.1 Toxicity to Mammals

Timed-pregnant Sprague Dawley rats were orally gavaged daily with chlordane at concentrations of 0, 0.25, 0.5, 1, 2, 4, 8, 16, 32 or 64 mg/kg body weight from gestational day 6 to 19 (St. Omer et al 2001). Significant mortality was observed at all exposure concentration of 8 mg/kg/day or higher. Chlordane exposure at 8 mg/kgBW/day or less did not affect fetal sex ratio or litter size in utero. Based on the observed effect (60% mortality), the exposure concentration of 8 mg/kgBW/day was identified as the effect concentration. A factor of 10 was used to convert this to a LOAEL of 0.8 mg/kgBW/day, with an estimated NOAEL of 0.08 mg/kgBW/day.

Mice were fed diets containing technical chlordane at concentrations of 0, 1, 5 or 12.5 mg/kg for 104 weeks (Khasawinah and Grutsch 1989). Hematology, biochemistry, urinalysis, organ weights, and histopathology of major tissues and organs were assessed on all animals that died during the study and on all survivors at week 104. A significant increase in hepatic necrosis were observed in animals exposed to chlordane at a concentration of 5 mg/kg (LOAEL of 0.75 mg/kgBW/day). A NOAEL concentration of 1 mg/kg (0.15 mg/kgBW/day) was identified in this experiment and will be used in this risk assessment.

A.17.2 Toxicity to Birds

Japanese quail were fed diets containing 25 mg/kg chlordane for eight days and untreated feed for six days (Kreitzer and Heinz 1974). Their avoidance response to a moving silhouette was measured daily for fourteen days. Significantly fewer chlordane-treated birds responded to the stimulus than control birds during the eight days of exposure. Partial recovery was observed after six days of consumption of untreated food. An ingestion rate of 0.018 kg/day and body weight of 0.12 kg (<www.feathersite.com>) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 3.75 and an estimated NOAEL of 0.375 were calculated based on the results of this experiment. Because behavioral effects are not considered ecologically relevant, this study was not used to derive the TRV for effects of chlordane on birds.

Red-winged blackbirds were fed diets containing 10, 50 and 100 mg Chlordane/kg diet for 84 days (Stickel et al. 1983). No mortality was observed in the group that received the 10 mg/kg exposure concentration; 26 and 24 percent mortality was observed in the 50 and 100 mg/kg groups, respectively. An ingestion rate of 0.0159 kg/day (calculated using an allometric equation from Nagy 1987) and body weight of 0.064 kg (cited by the authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 12.4 mg/kgBW/day and NOAEL of 2.48 mg/kg/day will be used to evaluate the risk from chlordane to avian receptors.

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Nagy, K.A. 1987. "Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds." Ecol. Mongr. 57:111-128.

Stickel, L.F., W.H. Stickel, R.A. Dyrland and D.L. Hughes. 1983. Oxychlordane, HCS-3260 and nonachlor in birds: lethal residues and loss rates. J. Toxicol Environ. Health. 12:611-622.

A.18 DDT and metabolites

A.18.1 Toxicity to Mammals

Cats were fed a single dose of DDT incorporated into the diet at levels of 300 to 500 mg/kg. Within 24 hours, generalized tremors and marked ataxia were noted in most animals (Pollock and Wang 1953). Electroencephalograph readings showed a markedly increased amplitude of cerebellar activity over that seen in normal control animals. Based on an average adult cat bodyweight of 3.6 kg (U.S.EPA 1988), a single oral dose of 300 to 500 mg/kg is equivalent to a dose of 83 to 139 mg/kgBW.

Deichmann et al. (1971) reported subnormal reproduction in Beagles fed 12 mg/kgBW/day p,p'-DDT five days a week for 14 months. Effects noted were delayed estrous, reduction in libido, stillbirths, lack of mammary

development, reduced milk production, and high mortality in offspring. The experimental design makes the reported results difficult to interpret. Only four females were tested in this experiment. Two of the females did not become pregnant after copulation, and one never went into heat. In addition, females fed only DDT were mated with a male that had been fed a mixture of DDT and aldrin.

In another study, dogs were fed gelatin capsules containing 4 mg/kgBW/day of pure p,p'-DDD or technical DDD for four days (Cueto et al., 1958). The number of dogs which received a particular dosage level of a compound ranged from one to three. No effects were noted in the dogs fed pure p,p'-DDD. Symptoms noted in dogs which received the technical DDD included anorexia, apathy, weakness, and inability to stand. At autopsy, atrophy of the adrenal cortex was noted. Pure o,p'-DDD was prepared from the technical material. This isomer produced massive necrosis and atrophy of the adrenal glands at a dose of 4 mg/kgBW/day.

Nelson and Woodard (1949) also reported severe adrenal cortical atrophy in dogs fed DDD. Eleven adult dogs were fed DDD dissolved in corn oil at levels of 50 to 200 mg/kgBW/day for periods of 1 to 33 months. Seven of the dogs died by the end of the experiment, including two of the four that received the lowest dosage (after 20 and 21 months of exposure). The only symptoms noted prior to death were weakness and anorexia. Based on the ecological significance of the effect (mortality of 50% of the test animals), an exposure concentration of 50 was selected as the dose. A factor of 10was used to convert the lethal concentration to a LOAEL. A LOAEL of 5 mg/kgBW/day and an estimated NOAEL of 0.5 will be used to evaluate toxicity of p,p-DDD to mammals.

Effects of oral exposure to 1, 5 and 10 mg of technical DDT/kg/day were studied through three generations of beagle dogs (Ottoboni et al. 1977). There were a total of 135 adult female and 63 adult male dogs in the experiment, and 650 pups were produced. DDT-treated females had their first estrous cycles two to three months earlier than control females. The were no statistically significant differences among control and DDT dogs in any other reproductive parameter measured. The liver/body weight ratio was also higher in some DDT-treated animals. No histological changes were noted in the adrenal glands of DDT-treated dogs.

Gilbert (1969) conducted feeding experiments with mink for a period of 310 days. During this period, DDE contaminated fish were incorporated into standard mink diets. Over the 310 day experimental period, the mink received 0.087 mg DDE/kgBW/day for 102 days, 0.063 mg DDE/kgBW/day for 115 days, and uncontaminated feed for 93 days. Mink fed on the experimental diet had significantly heavier spleen and adrenal weights than controls at the end of the experimental period. In addition, in utero kit loss and overall kit loss were significantly higher in experimental females. This study was not used to develop TRVs for this risk assessment due to the inconsistency in the dietary level of DDE, interrupted feeding of the material, and the possibility that other contaminants may have been present in fish tissues.

Mice were fed diets containing technical DDT at concentrations of 0, 2, 20 or 250 mg/kg for 4 generations (Terracini et al. 1973). A significant increase in the number of females with liver cell tumors was observed in the group exposed to 250 mg/kg. Reproductive endpoints were not measured in this experiment. An ingestion rate of 0.0058 kg/day and body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 44 mg/kgBW/day and a NOAEL of 3.5 mg/kgBW/day were calculated based on the results of this experiment.

Mice were fed diets containing DDT at concentrations of 0, 25, 100 and 250 mg/kg for three generations (Keplinger et al. 1968). Significant effects on fertility were observed at dietary concentrations of 100 and 250 mg/kg DDT. An ingestion rate of 0.0058 kg/day and body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 17.8 mg/kgBW/day and a NOAEL of 4.4 mg/kgBW/day were calculated based on the results of this experiment.

Mice were fed diets containing technical DDT at concentrations of 0, 5, 10, and 20 mg/kg for 86 days, or concentrations of 0, 30, 60 and 120 mg/kg for 130 days (Ledoux et al. 1977). Breeding parameters measured included time of first vaginal plug; date of littering; number of live and dead pups at birth, day 15, and day 30; and weight of live pups on days 1, 15 and 30. Mice fed DDT at concentrations of 5, 10 or 20 mg/kg produced larger litters and weaned more pups than control mice. Mice receiving 30 or 120 mg/kg DDT had significantly fewer pups born, fewer alive on days 1, 15 and 30, and significantly lower pup weights on day 30. A decrease in measured parameters was also observed in mice fed 60 mg/kg DDTcompared to controls, but the decrease was not statistically

significant. An ingestion rate of 0.0058 kg/day and body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 5.3 mg/kgBW/day and a NOAEL of 3.2 mg/kgBW/day were calculated based on the results of this experiment.

Adult female NMRI mice were orally exposed to p,p'-DDT at concentrations of 0 or 0.05 mg/day for 28 days (Lundberg 1974). The animals were sacrificed on day 10 to 12 of pregnancy, and the number of implanted an resorbed fetuses were counted. The frequency of implanted ova was significantly decreased in the p,p-DDT exposeure group compared to the control group. A body weight of 0.033 kg was used to convert the exposure concentration to units of mg/kgBW/day (U.S. EPA 1988). A LOAEL of 1.52 mg/kgBW/day and an estimated NOAEL of 0.152 mg/kgBW/day were calculated.

Fitzhugh (1948) fed rats diets containing DDT at concentrations of 0, 10, 50, 100 and 600 mg/kg for 2 years. The number of young produced was significantly decreased in all rats exposed to 50 mg DDT/kg or greater. A LOAEL of 4.0 mg/kgBW/day and a NOAEL of 0.8 mg/kg/day were calculated based on results of this experiment and will be used to evaluate risk from exposure to DDT for mammals.

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A.18.2 Toxicity to Birds

Mallards were fed diets containing 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane (DDD) (technical formulation, 64% p,p'-isomer, 20% o,p'-isomer, 16% impurities) at concentrations of 0, 10 or 40 mg/kg {Heath, 1969 #3689}. Birds received the treated diets from several weeks prior to the onset of the first laying season until the second year of

laying. Exposure to dietary DDD did not affect eggshell thickness. Duckling production per hen was significantly reduced in birds receiving diets containing 40 mg/kg DDD the first laying season, and in both exposure groups the second layingseason. Based on the ecological significance of the observed effects (reproduction), the 10 mg/kg exposure concentration was identified as the LOAEL. An adult body weight of 1.25 kg and ingestion rate of 0.139 kg/day {Piccirillo, 1980 #2556}were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.11 mg/kgBW/day and an estimated NOAEL of 0.11 mg/kgBW/day were calculated based on results from this experiment.

Mallards were fed diets containing 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) (p,p'-isomer) at concentrations of 0, 10 or 40 mg/kg {Heath, 1969 #3689}. Birds received the treated diets from several weeks prior to the onset of the first laying season until the second year of laying. Exposure to dietary DDE severely impaired reproductive success; effects of both exposure concentrations were similar, but were initiated more rapidly at the 40 mg/kg exposure concentration. Shells laid by mallards in the 10 mg/kg exposure group were 13% thinner than controls in the second laying season. Shells laid by mallards in the 40 mg/kg exposure group were 13% thinner than controls both laying seasons. Hatchability of eggs and duckling production per hen were significantly reduced in both exposure groups. Based on the ecological significance of the observed effects (reproduction), the 10 mg/kg exposure concentration was identified as the LOAEL. An adult body weight of 1.25 kg and ingestion rate of 0.139 kg/day {Piccirillo, 1980 #2556}were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.11 mg/kgBW/day and an estimated NOAEL of 0.11 mg/kgBW/day were calculated based on results from this experiment.

Mallards were fed diets containing 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) (p,p'-isomer) at concentrations of 0, 2.5, 10 or 40 mg/kg {Heath, 1969 #3689}. The highest exposure concentration was reduced to 25 mg/kg after the first laying season due to observed mortality. Birds received the treated diets from several weeks prior to the onset of the first laying season until the second year of laying. Shells laid by mallards in the 25 mg/kg exposure group were 13% thinner than controls in the second laying season. Duckling survival was significantly reduced in the 25 mg/kg exposure group. Based on the ecological significance of the observed effects (reproduction), the 25 mg/kg exposure concentration was identified as the LOAEL. An adult body weight of 1.25 kg and ingestion rate of 0.139 kg/day {Piccirillo, 1980 #2556}were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.8 mg/kgBW/day and a NOAEL of 1.1 mg/kgBW/day were calculated based on results from this experiment.

Robins were fed earthworms injected with a DDT peanut oil solution (Bernard 1963). Dietary exposure of robins to DDT was 110 mg/kgBW/day. One hundred percent mortality (n = 8) was noted within 14 days.

Three groups of 15 house sparrows were fed diets containing DDT added to chick starter mash at levels of 100, 200 and 300 mg/kg (Bernard 1963). The exposure concentrations were converted to units of mg/kgBW/day using an average house sparrow body weight of 25.2 g (Kenaga 1973) and the reported daily ingestion rate of 6 g (Bernard 1963); 23.8, 47.6 and 71.4 mg/kgBW/day, respectively. All of the birds fed the highest dose (except three which were sacrificed) were dead within 29 days. Forty-nine days of exposure to 200 mg/kg DDT resulted in 100 percent mortality. After 90 days of exposure, all but two of the birds receiving the lowest dose were dead. Levels of DDT measured in brain tissue from dead birds were similar, regardless of the dietary level ingested.

Ingestion of diets containing 8 mg/kg DDT (22 pairs) or 4 mg/kg DDE (15 pairs) reduced fertility, hatchability, and fledging success in Bengalese Finches (Lonchura striata; Jefferies 1971). Birds were fed the contaminated diet from 6 weeks prior to pairing until fledging of the young had occurred. The exposure concentrations were converted to units of mg/kgBW/day using the reported average body weight of 14.13 g and ingestion rate of 4.401 g/day; 2.49 and 1.25 mg/kgBW/day, respectively.

One hundred percent mortality was noted in cowbirds (Molothrus ater) fed diets containing 500 mg/kg p,p'-DDT dissolved in cottonseed oil mixed with turkey starter crumbles (Stickel and Stickel 1969). All birds were dead within 11 days. Time to mortality for individual birds was correlated with weight at the beginning of the experiment, with heaviest birds surviving longest. The exposure concentration was converted to units of mg/kgBW/day using an average adult cowbird body weight of 49 g (Dunning 1993) and ingestion rate of 12.6 g/day (Nagy 1987), resulting in an exposure concentration of 128.6 mg/kgBW/day.

Brown (1978) reported that high residues of DDT in fat may be metabolized and result in adverse effects if birds are starved or activity increases. Cowbirds which had survived a diet containing 40 mg/kg DDT (10.3 mg/kgBW/day, using the conversion factors cited above) for 2 months suffered tremors if they were unusually disturbed by workers handling their cages.

Twelve pairs of American kestrels were fed a diet containing 2.8 mg/kg p,p'-DDE on a wet weight basis (Porter and Weimeyer 1972). Two of the males died after 14 and 16 months on the diet. DDE residues in brain tissue were high, and fat reserves were depleted. The kestrels died at a time when the birds lose weight and deplete fat reserves due to reproduction and molt. An ingestion rate of 0.0307 kg/day (Barrett and Mackey 1975) and a body weight of 0.111 kg (Dunning 1993) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 0.77 mg/kgBW/day and an estimated NOAEL of 0.077 mg/kgBW/day were calculated based on the results of this experiment.

American kestrels were fed diets containing 2.8 mg/kg p,p'-DDE on a wet weight basis (0.77 mg/kgBW/day, calculated using conversion factors cited above) for 2 years (Weimeyer and Porter 1970). The DDE was dissolved in cottonseed oil and added to ground meat. The first egg laid by each pair was collected in 1968 and again in 1969. Levels of p,p'-DDE measured in eggs ranged from 1.05 to 5.89 mg/kg in 1968, and from 17.4 to 44.2 mg/kg in 1969. Average eggshell thickness was 10 percent less in 1969 than in 1968.

Lincer (1975) conducted laboratory feeding studies with American kestrels in which females were maintained on diets containing 0.3, 3.0, 6.0, and 10.0 mg/kg DDE. An ingestion rate of 0.0307 kg/day (Barrett and Mackey 1975) and body weight of 0.111 kg (Dunning 1993) were used to convert the exposure concentrations to units of mg/kgBW/day (0.083, 0.83, 1.66 and 2.77 mg/kgBW/day, respectively). Animals on the 0.3 mg/kg diet produced eggs with shells which were not significantly different in thickness from controls, and 6 percent thinner than archived eggshells collected prior to widespread use of DDT. Birds maintained on the 3.0 and 6.0 mg/kg diets produced eggshells which were 14.0 and 17.4 percent thinner than experimental controls, and 25.5 and 28.3 percent thinner than pre-DDT eggshells, respectively. Eggshell thinning of greater than 20 percent results in decreased nesting success due to eggshell breakage (Anderson and Hickey 1972, Dilworth et al. 1972). Eggshell thinning to the extent observed at the exposure concentration of 3.0 mg/kg may be expected to affect breeding success of American kestrels. A LOAEL of 0.83 mg/kgBW/day and a NOAEL of 0.083 mg/kgBW/day were calculated based on results of this experiment.

Anderson et al. (1975) studied the reproductive success of brown pelicans off the southern California coast from 1969 to 1975. Concentrations of DDTr in anchovies (the major food source of the pelican colony) and pelican eggs were measured. Over the five year monitoring period, concentrations of DDTr in anchovies steadily declined from 4.27 mg/kg wet weight in 1969 to 0.15 mg/kg in 1974. Eggshell thickness gradually increased over the 5-year period. Productivity at the pelican colony increased from a total of four young fledged in 1969 to 1185 fledged in 1974. At prey tissue levels of 0.15 mg/kg DDTr in anchovies, the fledging rate was 30 percent below the estimated rate necessary to maintain a stationary population, therefore this concentration was identified as a LOAEL. A LOAEL of 0.028 mg/kgBW/day and an estimated NOAEL of 0.0028 mg/kgBW/day were calculated based on results of this experiment.

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A.19 Dichlorobenzenes

A.19.1 Toxicity to Mammals

No studies were located that evaluated oral toxicity of dichlorobenzenes to mammals.

A.19.2 Toxicity to Birds

Geese were fed diets containing p-dichlorobenzene at a concentration of 0 or 500 mg/kg for five weeks (Hollingsworth et al. 1956). Based on measured intake rate, an exposure dose of 600 mg/kgBW/day was estimated. A reduction in growth was observed in dichlorobenzene-exposed birds, and a 30% of the birds in this group died. This was considered to be a sub-chronic LOAEL TRV, which was converted to a chronic LOAEL using a factor of 10. A LOAEL of 60 mg/kgBW/day and a NOAEL of 6 mg/kgBW/day were identified based on this experiment.

Hollingsworth, R. L., V. K. Rowe, F. Oyen, H. R. Hoyle and H. C. Spencer (1956). "Toxicity of paradichlorobenzene determination on experimental animals and human subjects." A.M.A. Arch. Ind. Health 14: 138-147.

A.20 Dieldrin

A.20.1 Toxicity to Mammals

Pregnant CD-1 mice and CD rats were administered dieldrin by gastric intubation at concentrations of 0, 1.5, 3.0 and 6.0 mg/kgBW/day on days 7 to 16 of gestation (Chernoff et al. 1975). The highest dose of dieldrin induced significant increases in liver to body weight ratios and reduced weight gain in adult females, and resulted in 41 percent mortality in rats. Teratogenic effects were not observed. A LOAEL of 6.0 mg/kgBW/day and NOAEL of

3.0 mg/kgBW/day were derived as the result of this experiment.

Laboratory mice were fed diets containing dieldrin at concentrations of 0 or 5 mg/kg for 120 days, beginning 30 days prior to mating (Good and Ware 1969). Effects measured were parent mortality, fertility, fecundity, effects on first litter and effects on all litters. Exposure to dieldrin significantly reduced the size of all litters. An ingestion rate of 0.0058 kg/day and body weight of 0.033 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.88 mg/kgBW/day and an estimated NOAEL of 0.088 mg/kgBW/day were calculated based on the results of this experiment.

Rats were exposed to dietary concentrations of dieldrin (0, 2.5, 12.5, and 25 mg/kg) for three generations (Treon and Cleveland 1955). The number of pregnancies in females was reduced at the lowest exposure concentration compared to the number observed in the control group. An ingestion rate of 0.027 kg/day and body weight of 0.35 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.19 mg/kgBW/day and an estimated NOAEL of 0.019 mg/kg/day were calculated based on the results of this experiment.

The effect of dietary dieldrin on the fertility and fecundity of captive raccoons was evaluated by Morris (1973). Raccoons were fed diets containing 0 or 2 mg/kg dieldrin for 22 months. Young male raccoons on the dieldrin-treated diet showed delayed sexual maturation and reduced fertility. After 7 months of exposure to dieldrin, active spermatogenesis was observed in 5 of 10 control males but only 1 of 10 treated males, and the percent of viable sperm was significantly higher in control males. Treated females exhibited abnormal estrous cycles, reduced ovulation rates, increased resorption of embryos and higher adult mortality. An ingestion rate of 0.37 kg/day {Conover, 1989 #654; Teubner, 1983 #3187} and body weight of 6.9 kg (Merritt 1987) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.14 mg/kgBW/day and an estimated NOAEL of 0.014 mg/kgBW/day were calculated based on the results of this experiment.

Rats (strain OSU-Wistar) were exposed to dieldrin in their food at concentrations of 0, 0.08, 0.16,0.31,0.63, 1.25, 2.5, 5.0, 10.0, 20.0 and 40.0 mg/kg for 3 generations (Harr et al. 1970). The reproductive values measured were dam survival, conception rate, born litter size and weaned litter size. Dieldrin was not toxic to females fed concentrations of 2.5 to 10 mg/kg, but significant mortality was observed in nursing pups. When females were fed diets containing 0.31 mg/kg dieldrin, there was a significant decrease in pup survival in the second litter and in the number of third generation female rats that conceived. Based on the ecological significance of the observed effects (reproduction), an exposure concentration of 0.31 mg/kg was selected as the LOAEL. A LOAEL of 0.018 mg/kgBW/day and a NOAEL of 0.009 mg/kgBW/day will be used to evaluate risk from exposure to dieldrin for mammalian receptors.

Chernoff, N., R.J. Kavlock, J.R. Kathrein, J.M. Dunn and J.K. Haseman. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. Toxicol. Appl. Pharmacol. 31:302-308.

Good, E.E. and G.W. Ware. 1969. Effects of insecticides on reproduction in the laboratory mouse. IV. Endrin and dieldrin. Toxicol. Appl. Pharmacol. 14:201-203.

Harr, J.R., R.R. Claeys, J.F. Bone and T.W. McCorcle. 1970. Dieldrin toxicosis: Rat reproduction. Am. J. Vet. Res. 31(1): 181-189.

Merritt, J.F. 1987. Guide to the Mammals of Pennsylvania. Pittsburgh, PA: University of Pittsburgh Press. 408p.

Morris, J.E. 1973. Effects of dieldrin on reproduction in raccoons. PhD Thesis, University of Missouri-Columbia. 112 pages.

A.20.2 Toxicity to Birds

Adult Gray partridge were fed diets containing dieldrin at concentrations of 0 or 3 mg/kg technical dieldrin prior to and during the breeding season (Neill et al. 1971). Egg production, fertility, hatchability, chick growth and survival to six weeks of age were measured. Dieldrin exposed birds produced an average of 4.3 eggs per pair less than controls, but within pair variance was high and not statistically significant. Hatchability of fertile eggs was 81.8%

for controls and 73.5% for dieldrin-treated birds. These values approached but did not attain statistical significance. Mortality levels were low and weight gains were comparable in the two groups of chicks.

Yearling mallards were fed diets containing dieldrin at concentrations of 0 or 4 mg/kg for 30 prior to and 60 days following the onset of egg production (Muller and Lockman 1972). Egg production, fertility, hatchability and progeny development to six weeks of age were measured. Hatchability and embryo survival were significantly decreased in mallards that were exposed to dieldrin in their diets. A body weight of 1.25 kg and food ingestion rate of 0.139 kg/day (Piccirillo and Quesenberry 1980) were used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 0.445 mg/kgBW/day and an estimated NOAEL of 0.0445 mg/kgBW/day were calculated based on results of this experiment.

Barn owls (Tyto alba) were fed diets containing dieldrin at concentrations of 0 or 0.58 mg/kg for two years (Mendenhall et al. 1983). Dieldrin exposure produced a slight but statistically significant reduction in eggshell thickness. No significant effects were observed in number of eggs laid per pair, number of eggs hatched per pair, percent eggs broken, embryo mortality, or nestling mortality. This exposure dose was identified as a chronic NOAEL. A body weight of 466 g and a food ingestion rate of 62.5 g/day were used to convert the exposure dose to units of mg/kgBW/day. A NOAEL of 0.077 mg/kgBW/day and an estimated LOAEL of 0.77 mg/kgBW/day were identified from this experiment. To be consistent with the Onondaga Lake ERA, these values were selected for use in this risk assessment.

Mendenhall, V. M., E. E. Klass and M. A. R. McLane (1983). "Breeding success of barn owls (Tyto alba) fed low levels of DDE and dieldrin." Arch Environ. Contam. Toxicol. 12: 235-240.

Muller, H. D. and D. C. Lockman (1972). Fecundity and progeny growth following subacute insecticide ingestion by the mallard. Poult. Sci. 51: 239-241.

Neill, D. D., H.D. Muller and J.V. Schutze . (1971). Pesticide effects on the fecundity of the gray partridge. Bull Environm. Contam. Toxicol. 6(6): 546-551.

Piccirillo, V. J. and R. P. Quesenberry (1980). Reproductive capacities of control mallard ducks (Anas platyrhynchos) during a one-generation reproduction study. Journal of Environmental Pathology and Toxicology 4: 133-139.

A.21 Endrin

A.21. 1 Toxicity to Mammals

Laboratory mice were fed diets containing endrin at concentrations of 0 or 5 mg/kg for 120 days, beginning 30 days prior to mating (Good and Ware 1969). Effects measured were parent mortality, fertility, fecundity, effects on first litter and effects on all litters. Exposure to endrin at a concentration of 5 mg/kg resulted in significant adult mortality, a reduction in litter size and number of young produced per day, and a significant reduction in litter size. A LOAEL of 0.92 mg/kgBW/day and an estimated NOAEL of 0.092 mg/kgBW/day were calculated based on the results of this experiment.

Good, E.E. and G.W. Ware. 1969. Effects of insecticides on reproduction in the laboratory mouse. IV. Endrin and dieldrin. Toxicol. Appl. Pharmacol. 14:201-203.

A.21.2 Toxicity to Birds

Experienced breeding pairs of screech owls (Otus asio) were fed diets containing 0 or 0.75 mg/kg endrin from mid-February until each pair completed incubation (Fleming et al. 1982). Owls fed diets containing endrin laid fewer eggs per day per laying female, had fewer eggs hatched per incubated clutch, and produced fewer fledglings than owls fed the control diet. Based on the ecological significance of the observed effect (reproduction), the exposure concentration of 0.75 mg/kg was identified as the LOAEL. A body weight of 0.185 kg (Dunning 1993)and an ingestion rate of 0.025 kg/day (Pattee et al. 1988)were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.1 mg/kgBW/day and an estimated NOAEL of 0.01 mg/kgBW/day will be used to

estimated risk from exposure to endrin for birds.

Dunning, J., Barnard, Jr. (1993). Avian Body Masses. Boca Raton, CRC Press.

Fleming, W. J., M. A. Ross McLane and M. A. Haegele (1982). Endrin decreases screech owl productivity. J. Wild. Manage. 46: 462-468.

Pattee, O. H., S. N. Wiemeyer and D. M. Swineford (1988). Effects of dietarty fluoride on reproduction in eastern screech-owls. Arch Environ. Contam. Toxicol. 17: 213-218.

A.22 Hexachlorobenzene

A.22.1 Toxicity to Mammals

Hexachlorobenzene (HCB) was administered daily in gelatin capsules to beagles at doses of 1, 10, 100 and 1000 mg/day for 12 months (Gralla et al. 1977). Reduced food consumption, weight loss and mortality were observed in dogs receiving HCB at a concentration of 100 mg/day. A body weight of 8.3 kg (mean weight of dogs reported by authors) was used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 12.1 mg/kgBW/day and a NOAEL of 1.2 mg/kgBW/day were calculated.

Sprague-Dawley rats were fed diets containing 0.0, 0.32, 1.6, 8.0 or 40.0 mg analytical-grade hexachlorobenzene/kg from weaning through the lifespan of the second generation (130 weeks; Arnold et al. 1985). There were no treatment-related effects on growth, food consumption, haematological parameters, fertility or adult survival. Pup viability was significantly reduced in the group which received a dose of 40 mg/kg. An ingestion rate of 0.034 kg/day and adult body weight of 0.48 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 2.8 mg/kgBW/day and a NOAEL of 0.57 mg/kgBW/day were calculated.

Mink and ferrets were fed diets containing 0, 1, 5, 25, 125 or 625 mg/kg hexachlorobenzene (HCB) prior to and throughout reproduction (331 days) to evaluate the effects of dietary HCB exposure on reproduction and offspring viability (Bleavins et al. 1984). One hundred percent mortality was observed in mink and ferrets fed the diet containing 625 mg/kg HCB. None of the mink fed the 125 mg/kg diet whelped, and none of the ferrets exposed at this concentration went into estrus. Kit mortality was greater in offspring produced by female mink exposed to 1, 5 and 25 mg/kg dietary HCB than by control mink; kit mortality was greater for ferrets exposed to 25 mg/kg dietary HCB. Kit body weight at birth was significantly lower for all HCB-exposed mink, and for kits produced by ferrets exposed to 5 or 25 mg/kg. In general, mink were more sensitive to HCB exposure than were ferrets. A LOAEL of 0.14 mg/kg BW/day (1mg/kg) and an estimated NOAEL of 0.014 mg/kg BW/day will be used to evaluate the risk posed by HCB to mammalian receptors.

Arnold, D.L., C.A. Moodie, S.M. Charbonneau, H.C. Grice, P.F. McGuire, F.R. Bryce, B.T. Collins, Z.Z.Zawidzka, D.R. Krewski, E.A. Nera and I.C. Munro. 1985. Long-term toxicity of hexachlorobenzene in the rat and the effect of dietary vitamin A. Fd. Chem. Toxic. 23(9):779-793.

Bleavins, M.R., R.J. Aulerich and R.K. Ringer. 1984. Effects of chronic dietary hexachlorobenzene exposure on the reproductive performance and survivability of mink and European ferrets. Arch. Environ. Contam. Toxicol. 13:357-365.

Gralla, E.J., R.W. Fleischman, Y.K. Luthra. M. Hagopian, J.R. Baker, H. Esber and W. Marcus. 1977. Toxic effects of hexachlorobenzene after daily administration to beagle dogs for one year. Toxicology and Applied Pharmacology. 40:227-239.

Merritt, J.F. 1987. Guide to the Mammals of Pennsylvania. Pittsburgh, PA: University of Pittsburgh Press. 408 p.

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. EPA/600/6-87-008.

U.S. EPA. 1993. Wildlife Exposure Factors Handbook, Volume I of II. United States Environmental Protection

Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/187a.

A.22.2 Toxicity to Birds

Hexachlorobenzene (HCB) was fed to five groups of black Australorp laying pullets at dietary levels of 0, 0.1, 1, 10 and 100 mg/kg for 6 months (Avrahami and Steele 1972). No adverse effects on adult birds, egg production, fertility or hatchability were observed at any exposure concentration tested. After 6 months concentrations of HCB in body fat were 21 to 31 times the concentration in the feed. An ingestion rate of 0.103 kg/day and a body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 7.1 mg/kg BW/day was calculated based on the results of this experiment.

Adult Japanese quail were fed diets containing HCB at concentrations of 0, 1, 5, 20, or 80 mg/kg for 90 days (Vos et al. 1971). Tremors were observed in the group exposed to HCB at a concentration of 80 mg/kg, and 33 percent of the birds in this group died. A significant decrease in hatchability and egg volume was also observed in the highest exposure group. An ingestion rate of 0.018 kg/day and a body weight of 0.12 kg (Varghese 2000) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 12 mg/kg BW/day and a NOAEL of 3 mg/kg BW/day were observed in this experiment.

Adult Japanese quail were fed diets containing 20 mg/kg of HCB for 90 days (Schwetz et al. 1974). Exposure at this concentration resulted in decreased survival of chicks and increased liver weight of adult birds. A LOAEL of 2.25 mg/kg BW/day and an estimated NOAEL of 0.225 mg/kg BW/day will be used to evaluate the risk posed by HCB to avian receptors.

Avrahami, M. and R.T. Steele. 1972. Hexachlorobenzene. II. Residues in laying pullets fed HCB in their diet and the effects on egg production, egg hatchability, and on chickens. N.Z. Journal of Agricultural Research. 15:482-488.

Schwetz, B.A., J.M. Norris, R.J. Kociba, P.A. Keeler, R.F. Cornier and P.J. Gehring. 1974. Reproduction study in Japanese quail fed hexachlorobutadiene for 90 days. Toxicol. Appl. Pharmacol. 30:255-265.

U.S. EPA. 1988. Recommendations for and documentation of biological values for use in risk assessment. EPA/600/6-87-008.

A.23 Hexachlorocyclohexanes

A.23.1 Toxicity to Mammals

Mink were fed a diet containing lindane at a concentration of 0 or 1 mg/kgBW/day from before breeding (30 and 60 days prior, first and second experiment, respectively) until pups were weaned (Beard et al. 1997). In the first experiment, lindane caused a decrease in the number of females accepting a second mating. A decrease in whelping rate was observed. Because mink that only mated once had a lower whelping rate than those that mated twice, it could not be determined whether the lower rates were due to lack of a second mating or to increased embryo loss. In a second experiment, whelping rate was again decreased in mink exposed to lindane; mating response at the second mating was not affected. The proportion of embryos lost after implantation (implantation scars not represented by kits at whelping) was increased following lindane exposure, indicating the lindane effect was a result of embryo mortality after implantation. A LOAEL of 1 mg/kgBW/day and an estimated NOAEL of 0.1 mg/kgBW/day were calculated based on the results of this experiment.

The effect of long-term exposure to lindane on reproduction was evaluated in mink exposed to dietary lindane (at concentrations of 0 or 1 mg/kgBW/day) for three generations (Beard and Rawlings 1998). First generation mink were exposed to lindane-treated food beginning at the time they were weaned. No overt signs of toxicity were observed in adults or offspring. Exposure to lindane at a concentrations of 1 mg/kgBW/day significantly reduced the proportion of mated mink that whelped, and the litter size of mink that whelped. Testis size was significantly reduced in third generation lindane-treated males. Overall, lindane exposure resulted in a 60 percent reduction in number of kits born. A LOAEL of 1 mg/kgBW/day and an estimated NOAEL of 0.1 mg/kgBW/day will be used to evaluate risk to mammals from exposure to lindane.

Beard, A.P., A.C. McRae and N.C. Rawlings. 1997. Reproductive efficiency in mink (Mustela vison) treated with the pesticides lindane, carbofuran and pentachlorophenol. J. Reprod. Fert. 111:21-28.

Beard, A.P. and N.C. Rawlings. 1998. Reproductive effects in mink (Mustela vison) exposed to the pesticides Lindane, carbofuran and pentachlorophenol in a multigeneration study. J. Reprod. Fert. 113:95-104.

A.23.2 Toxicity to Birds

Thirty week old Leghorn hens were administered gelatin capsules containing lindane at concentrations of 0 or 100 mg/kg daily for six weeks (Whitehead et al. 1972). Egg shell thickness, egg and yolk weight, and hatchability were not affected by lindane exposure. Egg production was significantly decreased at the 100 mg/kg exposure concentration; production did not recover once exposure to lindane stopped. An adult body weight of 1.45 kg (U.S. EPA 1988)used convert the exposure dose to units of mg/kgBW/day. A LOAEL of 69 mg/kgBW/day and an estimated NOAEL of 6.9 mg/kgBW/day were calculated based on results of this experiment.

Lindane was dissolved in the drinking water of captive Japanese quail at concentrations of 0, 1, 3 or 9 mg/L for seven days during reproduction (Jansen 1996). A significant decrease in fertility and hatchability was observed in birds exposed to lindane at a concentration of 3 mg/kg. An ingestion rate of 0.0169 kg/day (Nagy 1987) and a body weight of 0.15 kg (Vos et al. 1971) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.34 mg/kg BW/day and a NOAEL of 0.11 mg/kg BW/day were observed in this experiment.

U.S. EPA 1988. Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

Whitehead, C. C., A. G. Downing and R. J. Pettigrew (1972). The effecs of lindane on laying hens. Br. Poult. Sci. 13(3): 293-299.

A.24 Polycyclic Aromatic Hydrocarbons

A.24.1 Toxicity to Mammals

MacKenzie and Angevine (1981) exposed mice to benzo(a)pyrene via oral intubation at concentrations of 0, 10, 40 and 60 mg/kgBW/day during days 7 to 16 of gestation. Exposure at the highest concentration significantly reduced pregnancy rates and number of viable litters produced by exposed females. Total sterility was observed in 97 percent of offspring from mice exposed to concentrations of 40 and 60 mg/kgBW/day. Fertility (number and size of litters produced in a 6-month breeding study) was impaired in offspring from mice exposed to a concentration of 10 mg/kgBW/day. A LOAEL of 10 mg/kgBW/day and an estimated NOAEL of 1 mg/kgBW/day will be used to evaluate risk from exposure to HMWPAHs to mammals for this risk assessment.

MacKenzie, K. M. and D. M. Angevine (1981). "Infertility in mice exposed in utero to benzo(a)pyrene." Biology of Reproduction 24: 183-191.

A.24.2 Toxicity to Birds

White Carneau (WC) and Show Racer (SR) Pigeons were injected once weekly, intramuscularly, with vehicle (corn oil alone),or with benzo(a)pyrene (BaP) or benzo(e)pyrene (BeP) at a concentration of 10 mg/kg body weight (Hough et al. 1993). Pigeons from each of the three groups were killed after three or five months of treatment. Exposure to BaP resulted in complete infertility in female birds; there were grossly visible changes in ovarian appearance. A LOAEL of 1.43 mg/kgBW/day and an estimated NOAEL of 0.143 mg/kgBW/day were calculated based on results of this experiment.

Hough, J. L., M. B. Baird, G. T. Sfeir, C. S. Pacini, D. Darrow and C. Wheelock (1993). "Benzo(a)pyrene enhances atherrosclerosis in White Carneau and Show Racer pigeons." Arterioscler. thromb. 13(12): 1721-1727.

A.25 Trichlorobenzenes

A.25.1 Toxicity to Mammals

Rats were continuously exposed to 0, 25, 100, or 400 ppm 1,2,4-trichlorobenzene in drinking water, starting with the birth of the F_0 generation and continuing through the weaning of the F_2 generation (Robinson et al. 1981). No effect on fertility, growth or survival were observed at any exposure concentrations. Adrenal enlargement was observed in the highest exposure group. The LOAEL for this risk assessment was derived based on the increase in adrenal gland weight observed in the 400 mg/kg (53.6 mg/kgBW/day) group. The NOAEL was considered to be the 100 ppm (14.8 mg/kgBW/day) dose.

Robinson, K. S., R. J. Kavlock, N. Chernoff and E. Gray (1981). "Multigeneration study of 1,2,4-trichlorobenzene in rats." J. Toxicol. Environ. Health 8: 489-500.

A.25.2 Toxicity to Birds

No studies were located that evaluated dietary toxicity of trichlorobenzenes to birds.

A.26 Xylenes

A.26.1 Toxicity to Mammals

Mice were exposed to mixed xylenes at concentrations of 0. 0.5, 1.0, 2.1, 2.6, 3.1, or 4.1 mg/kgBW/day via gavage from days 6 to 15 of gestation (Marks et al. 1982). Xylene exposure at concentrations of 2.6 mg/kgBW/day or greater significantly reduced fetal weights and increased the incidence of fetal malformations. A LOAEL of 2.6 mg/kgBW/day and a NOAEL of 2.1 mg/kgBW/day were identified from this experiment.

Marks, T. A., T. A. Ledoux and J. A. Moore (1982). "Teratogenicity of a commercial xylene mixture in the mouse." J. Toxicol. Environ. Health 9: 97-105.

A.26.2 Toxicity to Birds

No studies were located that evaluated toxicity of xylene to birds.

A.27 Dioxins/furans

A.27.1 Toxicity to Mammals

Mature female dark mink were fed diets containing 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at measured concentrations of 0.0006 (control), 0.016, 0.053, 0.180 or 1.4 μg/kg for 131 to 132 days (Hochstein et al. 2001). Due to subnormal breeding in control females (probably due to indoor housing conditions), definitive effects of TCDD on reproduction could not be evaluated. However, there were dose-dependant decreases in kit birth weight and survival to 3 weeks of age in the groups that did reproduce. Females in the 0.016 and 1.4 μg/kg exposure groups failed to whelp. Final mink body weights were inversely proportional to dietary TCDD concentrations; mink the in highest exposure group lost 26% of their body weight during the study. Consumption of the 1.4 μg/kg diet resulted in lethargy, bloody stools and 16.7% mortality. A body weight of 1.13 kg and food ingestion rate of 0.249 kg/day (U.S. EPA 1993a) were used to convert the exposure concentrations to units of mg/kgBW/day. Based on the observed mortality at an exposure concentration of 1.4 μg/kg, a LOAEL of 0.31 μg/kgBW/day and a NOAEL of 0.04 μg/kgBW/day were identified from this experiment.

Adult female mink were fed diets containing TCDD at nominal concentrations of 0, 0.001, 0.01, 0.1, 1 10 or 100 μg/kg for up to 125 days (Hochstein et al. 1998). There was a dose-dependant decrease in food consumption and body weight. By Day 28, mortality was 12.5, 62.5 and 100% in the 1.0, 10 and 100 μg/kg exposure groups, respectively. At Day 125, mortality had increased to 62.5 and 100% in the 1.0 and 10 μg/kg groups. Calculated LC_{50} values were 0.264 μg/kgBW/day and 0.047 μg/kgBW/day for the 28- and 125-day exposure periods, respectively.

Sprague-Dawley rats were fed diets containing TCDD at concentrations of 0, 0.001, 0.01 or 0.1 μg/kgBW/day for three generations (Murray et al. 1977). At an exposure concentration of 0.01 μg/kgBW/day, fertility was significantly decreased in the f_1 and f_2 generations. In the f_2 and f_3 generations, litter size, survival, and growth of pups were significantly decreased compared to control rats. Based on the observed reproductive effects, a LOAEL of 0.00001 mg/kgBW/day and a NOAEL of 0.000001 mg/kgBW/day were identified from this experiment.

Hochstein, J. R., S. J. Bursian and R. J. Aulerich. 1998. Effects of dietary exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in adult female mink (Mustela vison). Arch Environ. Contam. Toxicol. 35(2): 348-353.

Hochstein, M. S., Jr.,, J. A. Render, S. J. Bursian and R. J. Aulerich. 2001. Chronic toxicity of dietary 2,3,7,8-tetrachlorodibenzo-p-dioxin to mink. Vet. Human Toxicol. 43(3): 134-139.

Murray, F. J., F. A. Smith, K. D. Nitschke, C. G. Humiston, R. J. Kociba and B. A. Schwetz. 1977. Three-generation reproduction study of rats ingesting 2,3,7,8-tetrachlorodibenzo-p-dioxin. Toxicol. Appl. Pharmacol. 41(1): 200.

U.S. EPA. 1993a. Wildlife Exposure Factors Handbook, Volume I of II. Washington, DC. EPA/600/4-93/187a., United States Environmental Protection Agency, Office of Research and Development.

A.27.2 Toxicity to Birds

White Leghorn chickens were exposed through their diet to soil contaminated with a mixture of 2,3,7,8-substituted polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/PCDFs) at concentrations of less than 0.5 pg/g International Toxic Equivalent Quantity (I-TEQ; control), 42 pg/g I-TEQ, and 460 pg/g I-TEQ for 178 days (Stephens et al. 1995). All chickens remained in good health throughout the study and no gross abnormalities were observed at necropsy. Egg production was similar for all exposure groups. The authors reported daily intakes of 2.5 ng I-TEQ/kg/day and 0.3 ng I-TEQ/kg/day for the high and low exposure groups, respectively. Because no adverse effects were observed at the highest exposure concentration, a NOAEL of 0.0000025 mg/kg/day was identified from this experiment.

Matsushita et al. (2002) evaluated the effects of maternal exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on second generation chicks. TCDD was administered intramuscularly to Barred Plymouth Rock hens at a concentration of 0 or 200 ng/kg once a week from 14 to 23 weeks of age. Egg production by TCDD-exposed hens started at 20 weeks of age, two weeks earlier than in controls. Egg production by the exposed hens ceased between 23 and 24 weeks of age, and did not start again until two weeks after exposure stopped. Hatchability of the eggs produced by the TCDD-exposed hens was lower than for the control group, and the sex ratio (percent [%] males) was slightly higher than for controls. Based on the effects on egg production, a LOAEL of 0.000029 mg/kg/day (0.0002 mg/kg/week) and an estimated NOAEL of 0.0000029 mg/kgBW/day were identified from this experiment.

Sexually mature (40 to 45 week old) hen pheasants (Phasianus colchicus) were administered intraperitoneal (ip) injections of TCDD at concentrations of 0, 0.01, 0.1 or 1.0 micrograms per kilogram (µg/kg) one time per week for 10 weeks (Nosek et al. 1992; Nosek et al. 1992). No mortality was observed in any treatment group during the 10 week dosing period. Mortality was observed in birds exposed at a concentration of 1.0 µg/kg; 57% of the birds in this group died between weeks 15 and 24. Egg production by birds in the highest dose group was significantly lower, and mortality of embryos from fertilized eggs was significantly higher than in control birds ($p < 0.05$, one-way ANOVA, followed by least-significant differences tests). Greater than 98% embryo mortality was observed in eggs laid by hens exposed at a concentration of 1.0 µg/kg. Based on the observed mortality, reduced egg production, and lower embryo survival at an exposure dose of 1.0 µg/kg/week, a LOAEL of 0.00014 mg/kg/day and an estimated NOAEL of 0.000014 mg/kgBW/day were calculated.

Matsushita, S., M. Ikeda, M. Tamura, K. Setani, J. Yamashita, C. Suzuki, M. Ikeya and T. Tomita. 2002. Effects of maternal exposure to 2,3,7,8-tetrachloro-p-dioxin (TCDD) on second-generation chickens. Environmental Sciences: an International Journal of Environmental Physiology and Toxicology 9(2-3): 94.

Nosek, J. A., S. R. Craven, J. R. Sullivan, S. S. Hurley and R. E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin in ring-necked pheasant hens. J. Toxicol. Environ. Health 35(3): 187-198.

Stephens, R. D., M. X. Petreas and D. G. Hayward. 1995. Biotransfer and bioaccumulation of dioxins and furans from soil: chickens as a model for foraging animals. Science of the Total Environment 1975: 253-273.

A.27.3 Toxicity to Fish

Adult female lake trout (Salvelinus namaycush) were exposed to diets containing TCDD for 11 weeks prior to spawning (Walker et al. 1994). Egg TCDD concentrations between 233 and 387 pg/g egg ww resulted in non-viable oocytes. Egg concentrations between 50 and 152 pg/g resulted in a dose-related increase in sac fry mortality associated with yolk sac edema, craniofacial alterations, and arrested fry development. An LD_{50} of 58 pg/g egg was calculated. An egg concentration of 50 pg/g (0.00005 mg/kg) was identified as the LOAEL. No adverse effects were observed at an egg concentration of 23 pg/g (0.000023 mg/kg).

Walker, M.K., P.M. Cook, A.R. Batterman, B.C. Butterworth, C. Bernini, J.J. Libal, L.C. Hufnagle and R.E. Peterson. 1994. Translocation of 2,3,7,8-tetrachlorodibenzo-p-dioxin from adult female lake trout (Salvelinus namaycush) to oocytes: Effects on early life stage development and sac fry survival. Can. J. Fish. Aquat. Sci. 51:1410-1419.

A.28 Total PCBs

A.28.1 Toxicity to Mammals

A.28.1.1 Acute Toxicity

In a preliminary study to determine the cause of reproductive complications in mink fed Great Lakes fish, adult breeder mink were fed a basal diet supplemented with 30 mg/kg of PCBs for six months (181 days). However, all of the mink died, emaciated, by the end of the experimental period (Aulerich and Ringer 1977). The dose was converted to daily exposure concentration by multiplying it by a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and dividing by a body weight of 1.13 kg (Merritt 1987). An acute exposure concentration of 6.61 mg/kgBW/day was calculated based on this experiment.

Mink were fed diets containing Aroclor 1254 at concentrations of 0, 5 or 15 mg/kg for four months (Aulerich and Ringer 1977). Fifteen mg/kg of Aroclor 1254 in the diet resulted in a complete inhibition of reproduction and 31 percent adult mortality, compared to 6 percent mortality in the controls. Five mg/kg of Aroclor 1254 resulted in a 95 percent reduction in the number of kits born live; the ratio of live kits to female adults was reduced by 87 percent. The 5 and 15 mg/kg doses were converted to daily exposure concentrations using the conversion factors cited above, resulting in exposure concentrations of 1.1 and 3.3 mg/kg BW/day, respectively.

In another experiment, mink were fed diets containing Aroclor 1254 at concentrations of 0, 5, or 10 mg/kg for ten months (Aulerich and Ringer 1977). All mink fed the PCB-supplemented diets failed to produce offspring. Seventy-one percent mortality was observed in the group fed Aroclor 1254 at a concentration of 10 mg/kg. The dose was converted to daily exposure concentration by multiplying it by a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and dividing by a body weight of 1.13 kg (Merritt 1987). The 5 and 10 mg/kg doses were converted to daily exposure concentrations using the conversion factors cited above, resulting in exposure concentrations of 1.1 and 2.2 mg/kg BW/day, respectively.

Aulerich, R. J. and R. K. Ringer (1977). Current status of PCB toxicity, incl;uding reproduction in mink. Arch Environ. Contam. Toxicol. 6: 279-292

A.28.11.2 Chronic Toxicity

Several studies were found pertaining to the dietary toxicity of PCBs to mink, most of which examined effects on reproduction, growth and survival. Since the mink is the most sensitive mammal species tested to date, these studies were the only studies reviewed to derive a TRV for mammals for this risk assessment.

In a preliminary study to determine the cause of reproductive complications in mink fed Great Lakes fish, adult breeder mink were fed a basal diet supplemented with 30 mg/kg of PCBs for six months (181 days). However, all of the mink died, emaciated, by the end of the experimental period (Aulerich and Ringer 1977). As a result of the preliminary study, a long-term study was conducted to determine the effects of long-term, low-level consumption of PCBs on growth. Mink were fed a basal diet supplemented with 5 and 10 mg/kg of PCBs for a period of approximately 8.5 months. The basal diet plus 10 mg/kg of PCBs resulted in a significant 56 percent decrease in body weight gain after a period of 4 months. Body weight gain was reduced by 39 percent in the 5 mg/kg treatment group, but this reduction was not statistically significant. Both the 5 and 10 mg/kg treatment groups failed to produce offspring; the control group produced 17 live and 8 dead kits. Various degrees of embryotoxicity were observed during necropsy of the treated animals (Aulerich and Ringer 1977). The 5 and 10 mg/kg doses were converted to daily exposure concentrations by multiplying them by a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and dividing by a body weight of 1.13 kg (Merritt 1987). This yielded exposure concentrations of 1.1 and 2.2 mg/kg BW/day for the 5 and 10 mg/kg treatment groups, respectively.

Based on the results of the above experiment, another experiment was conducted to determine the effects of long-term consumption of low-level PCBs on reproduction. Fifteen mg/kg of Aroclor 1254 in the diet resulted in a complete inhibition of reproduction and 31 percent adult mortality, compared to 6 percent mortality in the controls. Five mg/kg of Aroclor 1254 resulted in a 95 percent reduction in the number of kits born live; the ratio of live kits to female adults was reduced by 87 percent. In an effort to determine the persistence of the impaired reproductive condition, 11 adult females that received 5 mg/kg of Aroclor 1254 for a period of six months were placed on a control diet for one year. The results indicate that the impaired reproductive performance of these females was not a permanent condition (Aulerich and Ringer 1977). The 5 and 15 mg/kg doses were converted to daily exposure concentrations using the conversion factors cited above, resulting in exposure concentrations of 1.1 and 3.3 mg/kg BW/day, respectively.

In another experiment, mink were fed one of four diets supplemented with 2 mg/kg of a different Aroclor (1016, 1221, 1242 or 1254) or a control ration for nine months prior to whelping until kits were four weeks old (Aulerich and Ringer 1977). Aroclor 1254 was the only PCB that adversely affected reproduction. Only two of seven mated females whelped, and only one live kit was produced (compared to eight of eight whelped and 28 live kits produced by females fed the control diet). The single kit produced by the Aroclor 1254 exposed mink weighed considerably less that the kits produced by the control female. The exposure concentration was converted to units of mg/kgBW/day using a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A LOAEL of 0.44 mg/kgBW/day and an estimated NOAEL of 0.044 mg/kgBW/day were calculated based on results of this experiment.

Female mink were fed diets containing control fish or fish collected from the Housatonic River at measured concentrations of 0.03 (control), 0.34, 0.61, 0.96, 1.6, or 3.7 µg total PCBs/g feed wet weight from 11 weeks prior to mating through weaning of kits (Bursian et al. 2006). Twelve kits from each treatment group were maintained on their respective diets for an additional 180 days. Dietary PCB exposure had no effect on number of offspring produced, gestation period, breeding success, or whelping success. Percent survival of kits exposed at a concentration of 3.7 µg/g was significantly lower than control survival at six weeks of age; growth in this group was significantly lower at three weeks of age. A LOAEL of 0.414 mg/kgBW/day (3.7 µg/g) and a NOAEL of 0.169 mg/kgBW/day (1.6 µg/g; dose in units of mg/kgBW/day cited by authors) were observed in this experiment.

Eight month old mink fed a basal diet containing 1.0 mg/kg of Aroclor 1254 for a period of approximately six months exhibited no mortality or any significant changes in the thyroid, pituitary, adrenal glands, or serum T3 and T4 levels (Wren et al 1987a). Reproduction and kit development was evaluated under the same test conditions in a separate study (Wren et al. 1987b) by the same investigators. Male fertility and female offspring production were not affected by the 1.0 mg/kg Aroclor 1254 diet. However, growth rate of kits nursed by exposed mothers was significantly reduced. The investigators estimated the daily exposure concentrations to be 0.10 mg/kg BW/day for males and 0.18 mg/kg BW/day for females. Based on the growth reduction, an exposure concentration of 0.10 mg/kgBW/day was identified as a LOAEL, and a NOAEL of 0.01 mg/kgBW/day was estimated using a factor of 10.

When Kubiak and Best (1991) fed mink a liver diet contaminated with PCBs , a concentration of 1.0 mg/kg PCBs resulted in reproductive impairment and a concentration of 5 mg/kg resulted in mortality. This dose was converted to a daily exposure concentration by multiplying it by an ingestion rate of 0.249 kg/day (U.S. EPA 1993) and dividing by a body weight of 1.13 kg (Merritt 1987). This resulting in a LOAEL of 0.22 mg/kg BW/day.

In another study, one-year-old mink were fed a diet of beef and cereal prepared from cows which had been given 10 consecutive daily oral doses of 1 and 10 mg/kg of Aroclor 1254 dissolved in an olive oil and dairy concentrate (Platanow and Karstad 1973). The cows did not exhibit any clinical, gross, or histopathological signs of PCB toxicity. The cows were killed 24 hours following the last dose, and the musculature, liver, and kidneys ground and mixed with commercial mink food cereal at a level of 24 percent cereal. The resulting rations containing 0.64 and 3.57 mg/kg of total PCB were fed to mink for a period of 160 days. The mink were fed this diet ad libitum 2 months prior to the breeding season and continued for 160 days. All 16 mink that were fed 3.57 mg/kg of PCBs died by day 105. Two of the 16 mink that were fed 0.64 mg/kg died by days 122 and 129. The mink exhibited poor appetites, lethargy, and weakness before dying. Some passed tarry feces, indicating gastrointestinal hemorrhaging. At both treatment levels, males survived longer than females. An ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987) were used to convert the exposure concentrations to units of mg/kgBW/day. This yielded exposure concentrations of 0.14 and 0.78 mg/kg BW/day for the 0.64 and 3.57 mg/kg doses, respectively.

In another study, male and female ranch-bred mink were acclimated to a diet consisting of ocean fish scraps, commercial mink cereal, and meat by-products. Ocean fish scraps made up 40 percent of this diet. Dietary treatment levels were prepared by substituting 10, 20, and 40 percent of the ocean fish scraps with PCB-contaminated carp from Saginaw Bay, Lake Huron. The mean dietary PCB concentrations were 0.015 mg/kg (control), 0.72 mg/kg (10 percent carp), 1.53 mg/kg (20 percent carp), and 2.56 mg/kg (40 percent carp). Groups of 15 mink (3 males, 12 females) were assigned to one of the four treatment groups for a period of 12 weeks. Mink receiving the highest PCB-containing diet (40 percent carp or 0.32 mg/kg BW/day, as reported by the investigators) exhibited a 42 percent reduction in mean litter size, 86 percent fewer live kits at birth, and no kits surviving beyond 24-hours post-partum. Even mink receiving the 10 percent carp diet (or 0.13 mg/kg BW/day, as reported by the investigators) exhibited a 67 percent reduction in kits surviving three to six weeks relative to the control (Heaton et al. 1995). This study cited a LOAEL of 0.13 mg/kgBW/day and a NOAEL of 0.004 mg/kgBW/day for exposure of mink to PCBs.

Female mink were exposed to a technical PCB (Clophen A50; A50), one fraction of A50 containing the non- and mono-ortho-chlorinated congeners, another fraction of A50 containing the congeners with two to four ortho-chlorines, of an organic extract from Baltic gray seal blubber. Mink were exposed at concentrations of 0, 0.1, or 0.3 mg/animal/day for 18 months, over two reproductive seasons (Brunstrom et al. 2001). No toxicity was observed in adult females at any exposure concentration. Exposure to the 2-4 ortho-congeners and to seal blubber extract did not affect kit production of growth. At the end of the first reproductive season, kit birth weight was significantly decreased in females exposed to A50 at a concentration of 0.3 mg/animal/day; whelping frequency and litter size were not significantly affected. At the end of the second season, effects on reproduction were more pronounced and dose-dependent. Exposure of females to the 0-1 ortho-fraction strongly reduced the number of kits per mated female, two-week survival, and growth of kits In the group exposed to A50 at a concentration of 0.3 mg/animal/day, only 39% of the females produced offspring, compared with 93% of control females. Newborn kits from

these females weighed 25% less than control kits, and 100% mortality of kits was observed within 24 hours. Females exposed to A50 at a concentration of 0.1 mg/mink/day produced fewer kits per mated female, and two-week kit survival was only 36% compared with 73% survival of control kits.. Based on the reproductive effects observed in the second reproductive season, an exposure concentration of 0.1 mg/mink/day was identified as the LOAEL. A body weight of 1.231 kg (cited by authors for this exposure group) was used to convert the exposure concentration to units of mg/kgBW/day. A LOAEL of 0.08 mg/kgBW/day and an estimated NOAEL of 0.008 mg/kgBW/day were calculated based on results from this experiment.

In a study on multigenerational effects in mink fed Saginaw Bay PCB-contaminated carp, Restum et al. (1998) observed a significant reduction in kit body weights after parental exposure to 0.25 mg/kg, wet weight of PCBs in fish (0.05 mg/kgBW/day as reported by authors). A significant reduction in kit survival was observed at a parental exposure concentration of 0.5 mg/kg (0.1 mg/kgBW/day). Of note in this study was that adverse effects on kit survival were observed even several months after the parents had been placed on the control diet. The inference was that long-term effects on mink can be observed even after short exposure periods to a PCB-contaminated diet. The concentration of 0.25 mg/kg was selected as the LOAEL for this risk assessment, as effects on growth, reproduction or survival were targeted as ecologically significant effects. A LOAEL of 0.034 mg/kgBW/day and a NOAEL of 0.0034 mg/kgBW/day will be used to evaluate risk from exposure to PCBs for mammals.

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A.28.2 Toxicity to Birds

A dietary concentration of 1500 mg/kg (dry weight) was administered to red-winged blackbirds for six days, by which time 50 percent of the birds had died (Stickel et al. 1984). Due to the acute nature of this study (short duration and high mortality), it was not used to assess the chronic effects of PCBs to birds in this risk assessment.

Adult mallards were fed diets containing measured concentrations of 0 or 161 mg/kg Aroclor 1242 for 12 weeks (Haseltine and Prouty 1980). No difference in time taken to lay a clutch, fertility, embryo mortality, hatch success, chick survival, or chick weight gain to three weeks of age was observed between the two groups. Eggshell thickness decreased 8.9% in eggs produced by PCB-exposed hens. The mean PCB concentration in eggs was 105 mg/kg ww (range 70 to 153 mg/kg ww). An ingestion rate of 0.139 kg/day and a body weight of 1.25 kg (Piccirillo and Quesenberry 1980)were used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 1.25 mg/kgBW/day was observed in this experiment.

White Leghorn hens were fed diets containing either Aroclor 1221, 1268 or 5542 at concentrations of 0 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility, egg production, hatchability, progeny growth or adult body weight were observed. Ingestion rates of 0.127 kg/day (Aroclor 1221), 0.125 kg/day (Aroclor 1268), or 0.122 kg/day (Aroclor 5542) and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 1.3 mg/kgBW/day (Aroclor 1221), 1.28 mg/kgBW/day (Aroclor 1268), and 1.25 mg/kgBW/day were calculated based on results of this experiment.

When Aroclor 1254 was fed to 9 month-old mallard hens at a concentration of 25 mg/kg, dry weight, in the diet for at least one month prior to egg laying, no detrimental effects on reproduction or nest attentiveness were observed (Custer and Heinz 1980). Assuming that the diet was one-third solids, this equates to a wet weight concentration of approximately 8.3 mg/kg. To convert this dosage to units of mg/kg BW/day, the dose was first multiplied by the food ingestion rate for the mallard duck of 0.139 kg/day, then divided by an adult body weight of 1.24 kg (Piccirillo and Quesenberry 1980) to yield a NOAEL of 0.92 mg/kg BW/day.

When screech owls were fed Aroclor 1248 in their diet at a concentration of 0 or 3 mg/kg for two breeding seasons, the number of eggs per clutch, hatchability, chick malformations, survival, and eggshell thickness were not affected (McLane and Hughes 1980). Mean PCB concentration in eggs was 3.32 mg/kg ww the first breeding season (range = 2.24 to 4.84 mg/kg ww)and 7.12 mg/kg ww (range = 3.98 to 17.8 mg/kg) the second breeding season. To convert to units of mg/kg BW/day, this value was divided by the reported mean body weight of 0.185 kg for screech owls (Dunning 1993) and multiplied by a food ingestion rate of 0.025 kg/day (Pattee et al. 1988). This resulted in a dietary exposure concentration (NOAEL) of 0.41 mg/kg BW/day.

Mallard ducklings over 9 weeks of age, were fed a PCB-treated diet for 5 days, followed by 3 days of an untreated diet. The 8-day LC50s ranged from 1,975 mg/kg for Aroclor 1260 to 3,182 mg/kg for Aroclor 1242 (Heath et al. 1972). The lowest LC50 value was converted to a LOAEL of 197.5 mg/kg using an accepted conversion factor of 10. In order to express this value in units of mg/kg BW/day, a food ingestion rate of 0.0578 kg/day and a body weight of 0.936 kg reported for 8-week old mallard ducks were used (Sugden et al. 1981). This resulted in a LOAEL concentration of 12.2 mg/kg BW/day.

Yearling male American kestrels were fed prey items (day-old cockerels) containing approximately 33 mg/kg, wet weight, of Aroclor 1254 for 62 to 69 days (Bird et al. 1983). This dose was converted by the investigators to a daily exposure concentration of 9 to 10 mg/kg BW/day. Kestrels receiving the treated diet exhibited a significant 22 to 27 percent reduction in sperm concentrations. This response was associated with a muscle PCB concentration of 107 mg/kg, lipid normalized, and a testes concentration of 128 mg/kg, lipid normalized. A LOAEL of 9 mg/kgBW/day and an estimated NOAEL of 0.9 mg/kgBW/day were derived from this experiment.

Captive American kestrels were fed PCB-spiked food (Aroclor 1248:1254:1260) at concentrations of 0 and 7 mg/kgBW/day for 100 days prior to and during breeding (Fernie et al. 2001a). Effects were evaluated during the breeding season dietary exposure occurred, and well as during the subsequent breeding season without further dietary PCB exposure. In the first year, PCB-exposed birds delayed clutch initiation, and had smaller clutches, higher incidence of complete clutch infertility, and poorer hatching and fledging success. There were no effects on adult body weight or reproductive measures in either group in the breeding season following the PCB-exposure season. Based on the observed reproductive effects, a LOAEL of 7 mg/kgBW/day and an estimated NOAEL of 0.7 mg/kgBW/day were identified from this experiment.

To determine whether PCB exposure of adult birds affects the size and composition of their eggs, captive American kestrels were fed diets containing PCBs at concentrations of 0 and 7 mg/kgBW/day for 100 days, from 1 month prior to pairing to until the anticipated hatching day of the eggs (Fernie et al. 2000). Total PCB concentrations were 0.01 and 34.1 mg/kg wet weight for control and PCB-treated birds, respectively. Variation in egg size within PCB clutches was significantly greater than within control clutches. Yolks in the PCB-contaminated eggs were heavier, with less wet and dry albumin relative to control eggs. Water content and shell thickness were not affected by PCB exposure. Changes in egg composition were not associated with egg size, lay date, ambient temperature, humidity or precipitation, other factors known to affect these variables in bird eggs. Eggs from the PCB-treated birds have relatively more lipid and less protein available for embryonic development; this may impact the growth and viability of nestlings. These changes in egg composition may be a possible mechanism contributing to the reduced reproductive performance observed in PCB-exposed birds. A LOAEL of 7 mg/kgBW/day and an estimated NOAEL of 0.7 mg/kgBW/day were identified from this experiment.

Captive American kestrels were hatched from clutches with eggs containing environmentally relevant levels of total PCBs (34.1 mg/kg whole egg wet weight versus 0 mg/kg for controls; (Fernie et al. 2001b). Parent birds had been fed diets containing PCBs at a concentration of 7 mg/kgBW/day for 100 days, from 1 month prior to pairing to hatching of the eggs. The second generation PCB birds were paired with unexposed kestrels with previous reproductive experience. In ovo PCB exposure suppressed egg laying completely in 25 percent of PCB females, time between pairing and egg laying was increased, and clutch size was smaller. Clutch initiation was delayed in PCB male pairs, clutch size was smaller, and percent of broods with complete mortality was significantly higher than control pairs. Changes in reproductive success for birds exposed to PCBs in ovo are similar to effects observed in kestrels directly exposed to dietary PCBs. Based on the observed reproductive effects, a LOAEL of 7 mg/kgBW/day and an estimated NOAEL of 0.7 mg/kgBW/day were identified from this experiment.

White Leghorn hens were fed diets containing either Aroclor 1221, 1232, 1268 or 5542 at concentrations of 0 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility or adult body weight were observed at any exposure concentration. A significant decrease in egg production, progeny growth, and hatchability were observed in hens exposed to Aroclor 1232 at a concentration of 20 mg/kg. An ingestion rate of 0.131 kg/day and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.34 mg/kgBW/day and an estimated NOAEL of 0.134 mg/kgBW/day were calculated for Aroclor 1232 based on results of this experiment.

White Leghorn hens were fed diets containing Aroclor 1242 at concentrations of 0, 2 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility or adult body weight were observed at any exposure concentration. A significant decrease in egg production, hatchability and chick growth were observed in hens exposed to Aroclor 1242 at a concentration of 20 mg/kg. An ingestion rate of 0.118 kg/day and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.21 mg/kgBW/day and a NOAEL of 0.121 mg/kgBW/day were calculated for Aroclor 1242 based on results of this experiment.

Another study investigated the behavioral component of reproduction in mourning doves given dietary supplements of 0, 10, or 40 mg/kg Aroclor 1254 (Tori and Peterle 1983). The exposure concentrations were converted to units of mg/kgBW/day using an ingestion rate of 0.0112 kg/day and a body weight of 0.1 kg (Kenaga 1973), resulting in doses of 0, 1.12 mg/kg BW/day, and 4.48 mg/kg BW/day. Control doves displayed normal courtship behaviors and patterns. Doves that were fed at the 10 ppm (1.12 mg/kg BW/day) level spent twice as much time in the courtship phase as the control birds, with only 50% completing courtship and nesting. Of the 50% that did nest and incubate eggs, nest initiation was significantly delayed, resulting in a delay in egg laying as well. None of the doves on the 40 ppm dietary supplement completed the nesting process. The authors hypothesized that the decline of reproductive activity was induced by the degradation of estrogen and androgen in the birds, which is presumably a result of increased hepatic microsomal enzyme activity as a response to the presence of PCBs. A LOAEL of 1.12 mg/kgBW/day and an estimated NOAEL of 0.112 mg/kgBW/day were identified from this experiment.

The effect of dietary PCBs on circulating progesterone levels (plasma concentration of this steroid is a critical component in hormonal control of avian ovulation) was investigated by (Koval et al. 1987). Mourning doves were exposed to PCBs in their diet at concentrations of 0 and 10 mg/kg for 28 days prior to mating. Although progesterone levels in control and PCB-treated birds were similar at many points in the reproductive cycle, levels in control birds were always greater than those in treated birds. Only 50 percent of treated birds laid eggs compared to 77 percent of the control birds. The time interval between occupation of the nest by the female and egg laying was significantly longer for PCB- treated birds (11.4 versus 3.1 days). This study provided additional evidence for delayed reproduction in PCB-treated birds. As above, the exposure concentration was converted to units of mg/kgBW/day using an ingestion rate of 0.0112 kg/day and a body weight of 0.1 kg (Kenaga 1973), resulting in a LOAEL of 1.12 mg/kgBW/day.

Peakall and Peakall (1973) maintained ring doves on a diet that contained 10 mg/kg Aroclor 1254. They found that reproductive success was dependent on exposure of the female to the PCB compound. Females fed PCB-spiked food were less attentive to their nest and had erratic nesting behaviors which interfered with egg development. Artificial incubation greatly increased the breeding success for these birds. The food concentration of 10 mg/kg was converted to units of mg/kg BW/day using an ingestion rate of 0.015 kg/day and a body weight of 0.160 kg (Schwarzbach et al. 1991), resulting in a LOAEL of 0.94 mg Aroclor 1254 /kgBW/day. Similar values were obtained by Peakall et al. (1972) for the ringed turtle dove, in which a dietary Aroclor 1254 concentration of 10 mg/kg adversely affected hatching success due to heavy embryonic mortality . A LOAEL of 0.94 mg/kgBW/day and an estimated NOAEL of 0.094 mg/kgBW/day were calculated based on results of these experiments.

Male and female pairs of American kestrels were fed diets containing 3 mg/kg, wet weight, of Aroclor 1248 incorporated into a commercial diet for approximately 20 weeks. Eggs were collected from the pairs 2 to 4 days after egg-laying was complete. The eggs collected from the treated pairs of birds exhibited a five percent reduction in eggshell thickness. This response was associated with a parent muscle tissue PCB concentration of 18.5$\pm$5.1 mg/kg, wet weight (Lowe and Stendell 1991). An ingestion rate of 0.0307 kg/day (Barrett and Mackey 1975) and a body weight of 0.111 kg (Dunning 1993) were used to convert the exposure concentration to units of mg/kgBW/day, resulting in a LOAEL of 0.83 mg/kgBW/day. A recent summary paper by Peakall and Lincer (1996) indicates that PCBs do not cause eggshell thinning except at very high doses that are likely to cause other reproductive toxicological effects as well. Therefore, the LOAEL based on this study (0.83 mg/kgBW/day) was not used to derive a TRV for evaluating the dietary toxicity of PCBs in birds.

White Leghorn hens were fed diets containing Aroclor 1242 at concentrations of 0, 5, 10, 20, 40 or 80 mg/kg for six weeks followed by six weeks of feeding a PCB-free diet (Britton and Huston 1973). Hatchability of eggs laid by hens exposed to Aroclor 1242 was significantly decreased at exposure concentrations of 10 mg/kg and higher. Recovery of hatchability was not complete in hens exposed to 20, 40 or 80 mg/kg after six weeks of consuming a PCB-free diet. An ingestion rate of 0.103 kg/day and a body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.71 mg/kgBW/day and a NOAEL of 0.36 mg/kgBW/day were calculated based on results of this experiment.

White Leghorn hens were fed diets containing either Aroclor 1232, 1242, 1248, 1254 or 1016 at concentrations of 0, 5, 10 or 20 mg/kg for eight weeks, followed by eight weeks of consuming the control diet (Lillie et al. 1975). No adverse effects on egg production, egg weight, egg shell thickness, food consumption or adult body weight were

observed at any exposure concentration. There was a significant decrease in hatchability of fertile eggs when hens were exposed to Aroclor 1232, 1242 or 1248 at a dietary concentration of 10 mg/kg. Growth of chicks produced by hens exposed to Aroclor 1242 and 1248 at a concentration of 10 mg/kg was significantly decreased compared to growth of chicks produced by control hens. Based on the decreased growth and reproductive effects, a LOAEL of 10 mg/kg was identified for Aroclors 1232, 1242 and 1248. An ingestion rate of 0.103 kg/day (U.S. EPA 1988) and a body weight of 1.66 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.62 mg/kgBW/day and a NOAEL of 0.31 mg/kgBW/day were calculated based on results of this experiment.

White Leghorn chickens were fed diets containing 0, 5 or 50 mg/kg of Aroclor 1254 for up to 39 weeks (Platonow and Reinhart 1973). Due to a drastic decline in egg production and hatchability of fertile eggs, the 50 mg/kg exposure group was placed on a clean diet at 14 weeks and recovery was measured. A significant decrease in egg production was also observed in hens fed diets containing 5 mg/kg Aroclor 1254, however hatchability of fertile eggs was not affected in this group. At an egg concentration of 15 mg/kg, a significant increase in embryo mortality and a decrease in hatchability were observed. No effect on hatchability was observed at an egg concentration of 5 mg/kg. An ingestion rate of 0.103 kg/day and a body weight of 1.45 kg (U.S. EPA 1988) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.36 mg/kgBW/day and an estimated NOAEL of 0.036 mg/kgBW/day were calculated based on results of this experiment.

Nestling kestrels were dosed orally with the planar PCB 126 at concentrations of 0, 50, 250 or 1000 ng/gBW/day from day 1 after hatching to day 10 after hatching (Hoffman et al. 1996). Notable liver enlargement was observed in all PCB treatment groups relative to controls. Significant adverse effects including reduced growth, liver enlargement and necrosis, lymphocyte depletion with atrophy of associated organs (spleen, bursa of Fabricius), and histopathological alterations of the thyroid in the intermediate exposure group. A LOAEL of 0.05 mg/kgBW/day (50 ng/gBW/day) was identified in this experiment. This study was not used to derive a TRV for this risk assessment due to the exposure method (oral intubation of PCB 126 dissolved in corn oil); exposure via this method may result in greater availability and toxicity than dietary exposure.

Summer et al. (1996a) exposed white Leghorn hens for eight weeks with commercial diets mixed with contaminated carp from Saginaw Bay, Lake Huron. The concentrations of PCBs in the resulting diets, measured as the sum of Aroclors 1242, 1248, 1254, and 1260, were 0.3 mg/kg (control), 0.8 mg/kg, and 6.6 mg/kg, wet weight. Hens were artificially inseminated weekly, and food consumption, body weights, and egg production were monitored daily. Food consumption initially declined in all the treatment groups but was greatest in the high dose group by the end of the study. Body weights were greater in the control and the low dose groups by the end of the study. Finally, egg production initially decreased during the acclimation period prior to the study, but egg production in the high dose group returned to pre-trial levels by the end of the study while egg production in the control and the low dose group remained significantly lower. The decreased egg production, as well as the increased body weights, in the control and the low dose group were explained by the authors as effects of fatty liver hemorrhagic syndrome (FLHS), with which the necropsy results were consistent. It was hypothesized that the PCBs in the high dose group provided a protective mechanism against FLHS, thus resulting in the higher egg production, since this protective mechanism had been observed in other studies. In a second phase of this experiment (Summer et al. 1996b), eggs were allowed to develop through day 25 of incubation, and hatching and deformity rates were observed and noted. Rates of deformities correlated with concentrations of PCBs in food, and both treatments (0.8 and 6.6 mg/kg, wet weight, in the diet) produced significantly higher rates of deformities (24% and 40%, respectively) compared to the control (17%). To convert the lower PCB treatment concentration (0.8 mg/kg, wet weight) to units of mg/kg BW/day, the average daily PCB consumption of hens in this treatment group reported by the authors (Summer et al. 1996a) for the 8-week duration of the study (67.1 ug/day) was divided by the corresponding average body weight (1620 g) to obtain a LOAEL of 0.0414 mg/kg BW/day. To convert the control PCB concentration (0.3 mg/kg, wet weight) to units of mg/kg BW/day, the average daily PCB consumption of hens in this treatment group reported by the authors (Summer et al. 1996a) for the 8-week duration of the study (26.75 ug/day) was divided by the corresponding average body weight (1690 g) to obtain a NOAEL of 0.0158 mg/kg BW/day. Although this study provided the lowest LOAEL and NOAEL of the studies presented here, these values were not selected for use in this risk assessment because the food source for the study came from an area that is known to contain a variety of pollutants in addition to PCBs, and the contribution of these other contaminants to the effects observed in this study are unknown.

White Leghorn hens were fed diets containing either Aroclor 1248 or 1254 at concentrations of 0, 2 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility or adult body weight were observed at any exposure concentration. A significant decrease in egg production and hatchability were observed when hens were exposed to Aroclor 1248 or 1254 at a concentration of 20 mg/kg. Chick growth was significantly decreased when hens were fed diets containing 2 mg/kg Aroclor 1248 or 1254. There was a significant increase in progeny mortality when hens were fed diets containing Aroclor 1248 at a concentration of 20 mg/kg. A LOAEL of 2 mg/kg was identified for Aroclor 1248 and 1254 based on the observed impact on progeny growth). An ingestion rate of 0.119 kg/day and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.122 mg/kgBW/day and a NOAEL of 0.012 mg/kgBW/day were calculated for Aroclors 1248 and 1254 based on results of this experiment. The results of this study were selected for use in this risk assessment due to the ecological significance of the endpoint (growth) and the specificity of the test chemical (PCBs only). Therefore, a LOAEL of 0.122 mg/kg BW/day and an estimated NOAEL of 0.012 mg/kg BW/day will be used in this risk assessment to evaluate the risk from PCBs to avian species.

Hen pheasants were administered weekly doses of 0, 12.5, or 50 mg Aroclor 1254 in gelatin capsules weekly for 16 weeks (Dahlgren et al. 1972). Hatchability and the number of fertile eggs pipped but not hatched were significantly lower in both PCB-exposed groups. A LOAEL TRV of 1.8 mg/kgBW/day and an estimated NOAEL of 0.18 mg/kgBW/day were calculated based on results of this experiment. These values will be used for this risk assessment to be consistent with other ERAs that have been conducted for aquatic systems within this watershed.

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U.S. EPA 1988. Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.28.3 Toxicity of PCBs to Fish

Meador et al. (2002) compiled data from studies that identified adverse effects in salmonids and tissue concentrations of PCBs at which the adverse effects were observed. For each study, the lowest tissue concentration of total PCBs associated with a biological response was identified. The tissue concentration identified as the 10th percentile of the adverse effect concentrations reported in the compiled studies was selected as the residue effect threshold (RET) above which juvenile salmonids would be expected to exhibit adverse sublethal effects from accumulated PCBs. A tissue concentration of 0.14 mg/kg w.w. was identified as an RET protective of salmonids. In the original version of this report, because the studies identified by Meador et al (2002) included studies utilizing injection as the route of exposure and some of the adverse effects identified were not ecologically relevant, the above RET was recalculated using only studies where exposure was via water or diet, and where the observed adverse effects were either growth or survival. The tissue concentration identified with the 10th percentile of the adverse effect level reported in these studies was 0.23 mg/kg w.w.

For this risk assessment, a literature search was conducted to locate additional studies that identified adverse effects in fish and reported tissue concentrations at which those effects were observed. Only studies where exposure was via water or diet, and where the observed adverse effects were either on growth, reproduction or survival were summarized. After review of the original paper, several studies cited in Meador et al. (2002) were not included in this analysis due to questions regarding whether the cited tissue concentration was wet or dry weight, or because the tissue concentration was not cited (it was estimated in the Meador et al. 2002 paper using based on absorption efficiencies reported in other studies). As was done by Meador et al. (2002), the tissue concentration identified as the 10th percentile of the adverse effect concentrations reported in the compiled studies was selected as the residue effect threshold. The tissue concentrations identified with the 10th percentile of the LOECs and NOECs reported in these studies were 1.44 mg/kg w.w. and 0.91 mg/kg w.w., respectively.

Meador, J.P., T.K. Collier and J.E. Stein. 2002. Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act. Aquatic Conserv. Mar. Freshw. Ecosyts. 12:493-516.

A.29 Aroclor 1016

A.29.1 Toxicity to Mammals

Mink were fed a diet supplemented with 2 mg/kg of Aroclor 1016 or a control ration for nine months prior to whelping until kits were four weeks old (Aulerich and Ringer 1977). No effects on reproduction were observed at this exposure concentration. The exposure concentration was converted to units of mg/kgBW/day using a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A NOAEL of 0.44 mg/kgBW/day was calculated for Aroclor 1016 based on results of this experiment.

Aulerich, R. J. and R. K. Ringer (1977). "Current status of PCB toxicity, incl;uding reproduction in mink." Arch Environ. Contam. Toxicol. 6: 279.

Merritt, J. F. (1987). Guide to the Mammals of Pennsylvania. Pittsburgh, PA, University of Pittsburgh Press.

U.S. EPA (1993). Wildlife Exposure Factors Handbook, Volume I of II. Washington, DC. EPA/600/4-93/187a., United States Environmental Protection Agency, Office of Research and Development.

A.29.2 Toxicity to Birds

White Leghorn hens were fed diets containing Aroclor 1016 at concentrations of 0, 5, 10 or 20 mg/kg for eight weeks, followed by eight weeks of consuming the control diet (Lillie et al. 1975). No adverse effects on egg production, egg weight, egg shell thickness, food consumption or adult body weight were observed at any exposure concentration. An ingestion rate of 0.103 kg/day (U.S. EPA 1988) and a body weight of 1.66 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 1.24 mg/kgBW/day was calculated based on results of this experiment.

Lillie, R. J., H. C. Cecil, J. Bitman, G. F. Fries and J. Verrett (1975). "Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens." Poult. Sci. 54: 1550-1555.

U.S. EPA. (1988). Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.30 Aroclor 1221

A.30.1 Toxicity to Mammals

Mink were fed a diet supplemented with 2 mg/kg of Aroclor 1221 or a control ration for nine months prior to whelping until kits were four weeks old (Aulerich and Ringer 1977). No effects on reproduction were observed at this exposure concentration. The exposure concentration was converted to units of mg/kgBW/day using a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A NOAEL of 0.44 mg/kgBW/day was calculated for Aroclor 1221 based on results of this experiment.

Aulerich, R. J. and R. K. Ringer (1977). "Current status of PCB toxicity, incl;uding reproduction in mink." Arch Environ. Contam. Toxicol. 6: 279.

Merritt, J. F. (1987). Guide to the Mammals of Pennsylvania. Pittsburgh, PA, University of Pittsburgh Press.

U.S. EPA (1993). Wildlife Exposure Factors Handbook, Volume I of II. Washington, DC. EPA/600/4-93/187a., United States Environmental Protection Agency, Office of Research and Development.

A.30.2 Toxicity to Birds

White Leghorn hens were fed diets containing Aroclor 1221 at concentrations of 0 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility, egg production, hatchability, progeny growth or adult body weight were observed. An ingestion rate of 0.127 kg/day (Aroclor 1221) and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A NOAEL of 1.3 mg/kgBW/day was calculated based on results of this experiment.

Lillie, R. J., H. C. Cecil, J. Bitman, G. F. Fries and J. Verrett (1975). "Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens." Poult. Sci. 54: 1550-1555.

U.S. EPA. (1988). Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.31 Aroclor 1232

A.31.1 Toxicity to Mammals

No studies were located that evaluated toxicity of Aroclor 1232 to mammals.

A.31.2 Toxicity to Birds

White Leghorn hens were fed diets containing Aroclor 1232 at concentrations of 0 or 20 mg/kg for nine weeks, followed by seven weeks of consuming the control diet (Lillie et al. 1974). No adverse effects on egg weight, egg shell thickness, survival, fertility or adult body weight were observed at any exposure concentration. A significant decrease in egg production, progeny growth, and hatchability were observed in hens exposed to Aroclor 1232 at a concentration of 20 mg/kg. An ingestion rate of 0.131 kg/day and a body weight of 1.95 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 1.34 mg/kgBW/day and an estimated NOAEL of 0.134 mg/kgBW/day were calculated for Aroclor 1232 based on results of this experiment.

White Leghorn hens were fed diets containing Aroclor 1232 at concentrations of 0, 5, 10 or 20 mg/kg for eight weeks, followed by eight weeks of consuming the control diet (Lillie et al. 1975). No adverse effects on egg production, egg weight, egg shell thickness, food consumption or adult body weight were observed at any exposure concentration. There was a significant decrease in hatchability of fertile eggs when hens were exposed to Aroclor 1232 at a dietary concentration of 10 mg/kg. Based on the reproductive effects, a LOAEL of 10 mg/kg was identified for Aroclors 1232. An ingestion rate of 0.103 kg/day (U.S. EPA 1988) and a body weight of 1.66 kg (cited by authors) were used to convert the exposure concentrations to units of mg/kgBW/day. A LOAEL of 0.62 mg/kgBW/day and a NOAEL of 0.31 mg/kgBW/day were calculated based on results of this experiment.

Lillie, R. J., H. C. Cecil, J. Bitman, G. F. Fries and J. Verrett (1975). "Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens." Poult. Sci. 54: 1550-1555.

U.S. EPA. (1988). Recommendations For and Documentation of Biological Values for use in Risk Assessment. United States Environmental Protection Agency. EPA/600/6-87-008.

A.32 Aroclor 1242

A.32.1 Toxicity to Mammals

Mink were fed a diet supplemented with 2 mg/kg of Aroclor 1242 or a control ration for nine months prior to whelping until kits were four weeks old (Aulerich and Ringer 1977). No effects on reproduction were observed at this exposure concentration. The exposure concentration was converted to units of mg/kgBW/day using a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A NOAEL of 0.44 mg/kgBW/day was calculated for Aroclor 1242 based on results of this experiment.

Aulerich, R. J. and R. K. Ringer (1977). "Current status of PCB toxicity, incl;uding reproduction in mink." Arch Environ. Contam. Toxicol. 6: 279.

Merritt, J. F. (1987). Guide to the Mammals of Pennsylvania. Pittsburgh, PA, University of Pittsburgh Press.

U.S. EPA (1993). Wildlife Exposure Factors Handbook, Volume I of II. Washington, DC. EPA/600/4-93/187a., United States Environmental Protection Agency, Office of Research and Development.

A.32.2 Toxicity to Birds

Adult mallards were fed diets containing measured concentrations of 0 or 161 mg/kg Aroclor 1242 for 12 weeks (Haseltine and Prouty 1980). No difference in time taken to lay a clutch, fertility, embryo mortality, hatch success, chick survival, or chick weight gain to three weeks of age was observed between the two groups. Eggshell thickness decreased 8.9% in eggs produced by PCB-exposed hens. The mean PCB concentration in eggs was 105 mg/kg ww (range 70 to 153 mg/kg ww). An ingestion rate of 0.139 kg/day and a body weight of 1.25 kg (Piccirillo and Quesenberry 1980)were used to convert the exposure concentration to units of mg/kgBW/day. A NOAEL of 1.25 mg/kgBW/day was observed in this experiment.

Mallard ducklings over 9 weeks of age, were fed a PCB-treated diet for 5 days, followed by 3 days of an untreated diet. The 8-day LC50s ranged from 1,975 mg/kg for Aroclor 1260 to 3,182 mg/kg for Aroclor 1242 (Heath et al.

1972). The lowest LC50 value was converted to a LOAEL of 197.5 mg/kg using an accepted conversion factor of 10. In order to express this value in units of mg/kg BW/day, a food ingestion rate of 0.0578 kg/day and a body weight of 0.936 kg reported for 8-week old mallard ducks were used (Sugden et al. 1981). This resulted in a LOAEL concentration of 12.2 mg/kg BW/day, and an estimated NOAEL of 1.22 mg/kgBW/day.

Haseltine, S. D. and R. M. Prouty (1980). Aroclor 1242 and reproductive success of adult mallards (Anas platyrhynchos). Environ. Res. 23: 29-34.

Heath, R. G., J. W. Spann, E. F. Hill and J. F. Kreitzer 1972. Comparative dietary toxicities of pesticides to birds. U.S. Fish and Wildlife Service. Special Scientific Report - Wildlife No. 152.

Piccirillo, V. J. and R. P. Quesenberry (1980). Reproductive capacities of control mallard ducks (Anas platyrhynchos) during a one-generation reproduction study. Journal of Environmental Pathology and Toxicology 4: 133-139.

Sugden, L. G., E. A. Driver and M. C. S. Kingsley (1981). Growth and energy consumption by captive mallards. Can. J. Zool. 59: 1567-1570.

A.33 Aroclor 1248

No studies were located that evaluated toxicity of Aroclor 1248 to birds or mammals.

A.34 Aroclor 1254

A.34.1 Toxicity to Mammals

An experiment was conducted to determine the effects of long-term consumption of low-level PCBs on reproduction in mink. Fifteen mg/kg of Aroclor 1254 in the diet resulted in a complete inhibition of reproduction and 31 percent adult mortality, compared to 6 percent mortality in the controls. Five mg/kg of Aroclor 1254 resulted in a 95 percent reduction in the number of kits born live; the ratio of live kits to female adults was reduced by 87 percent. In an effort to determine the persistence of the impaired reproductive condition, 11 adult females that received 5 mg/kg of Aroclor 1254 for a period of six months were placed on a control diet for one year. The results indicate that the impaired reproductive performance of these females was not a permanent condition (Aulerich and Ringer 1977). The 5 and 15 mg/kg doses were converted to daily exposure concentrations using the conversion factors cited above, resulting in exposure concentrations of 1.1 and 3.3 mg/kg BW/day, respectively.

In another study, one-year-old mink were fed a diet of beef and cereal prepared from cows which had been given 10 consecutive daily oral doses of 1 and 10 mg/kg of Aroclor 1254 dissolved in an olive oil and dairy concentrate (Platanow and Karstad 1973). The cows did not exhibit any clinical, gross, or histopathological signs of PCB toxicity. The cows were killed 24 hours following the last dose, and the musculature, liver, and kidneys ground and mixed with commercial mink food cereal at a level of 24 percent cereal. The resulting rations containing 0.64 and 3.57 mg/kg of total PCB were fed to mink for a period of 160 days. The mink were fed this diet ad libitum 2 months prior to the breeding season and continued for 160 days. All 16 mink that were fed 3.57 mg/kg of PCBs died by day 105. Two of the 16 mink that were fed 0.64 mg/kg died by days 122 and 129. The mink exhibited poor appetites, lethargy, and weakness before dying. Some passed tarry feces, indicating gastrointestinal hemorrhaging. At both treatment levels, males survived longer than females. An ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987) were used to convert the exposure concentrations to units of mg/kgBW/day. This yielded exposure concentrations of 0.14 and 0.78 mg/kg BW/day for the 0.64 and 3.57 mg/kg doses, respectively.

In another experiment, mink were fed one of four diets supplemented with 2 mg/kg of a different Aroclor (1016, 1221, 1242 or 1254) or a control ration for nine months prior to whelping until kits were four weeks old (Aulerich and Ringer 1977). Aroclor 1254 was the only PCB that adversely affected reproduction. Only two of seven mated females whelped, and only one live kit was produced (compared to eight of eight whelped and 28 live kits produced by females fed the control diet). The single kit produced by the Aroclor 1254 exposed mink weighed considerably less that the kits produced by the control female. The exposure concentration was converted to units of

mg/kgBW/day using a food ingestion rate of 0.249 kg/day (U.S. EPA 1993) and a body weight of 1.13 kg (Merritt 1987). A LOAEL of 0.44 mg/kgBW/day and an estimated NOAEL of 0.044 mg/kgBW/day were calculated based on results of this experiment.

Eight month old mink fed a basal diet containing 1.0 mg/kg of Aroclor 1254 for a period of approximately six months exhibited no mortality or any significant changes in the thyroid, pituitary, adrenal glands, or serum T3 and T4 levels (Wren et al 1987a). Reproduction and kit development was evaluated under the same test conditions in a separate study (Wren et al. 1987b) by the same investigators. Male fertility and female offspring production were not affected by the 1.0 mg/kg Aroclor 1254 diet. However, growth rate of kits nursed by exposed mothers was significantly reduced. The investigators estimated the daily exposure concentrations to be 0.10 mg/kg BW/day for males and 0.18 mg/kg BW/day for females. Based on the growth reduction, an exposure concentration of 0.10 mg/kgBW/day was identified as a LOAEL, and a NOAEL of 0.01 mg/kgBW/day was estimated using a factor of 10.

Aulerich, R. J. and R. K. Ringer (1977). "Current status of PCB toxicity, incl;uding reproduction in mink." Arch Environ. Contam. Toxicol. 6: 279.

Merritt, J. F. (1987). Guide to the Mammals of Pennsylvania. Pittsburgh, PA, University of Pittsburgh Press.

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Wren, C. D., D. B. Hunter, J. F. Leatherland and P. M. Stokes (1987b). "The effects of polychlorinated biphenyls and methylmercury singly and in combination, on mink. II. Reproduction and kit development." Arch. Environ. Contam. Toxicol. 16: 449-454.

A.34.2 Toxicity to Birds

A study was conducted to evaluate the behavioral component of reproduction in mourning doves given dietary supplements of 0, 10, or 40 mg/kg Aroclor 1254 (Tori and Peterle 1983). The exposure concentrations were converted to units of mg/kgBW/day using an ingestion rate of 0.0112 kg/day and a body weight of 0.1 kg (Kenaga 1973), resulting in doses of 0, 1.12 mg/kg BW/day, and 4.48 mg/kg BW/day. Control doves displayed normal courtship behaviors and patterns. Doves that were fed at the 10 ppm (1.12 mg/kg BW/day) level spent twice as much time in the courtship phase as the control birds, with only 50% completing courtship and nesting. Of the 50% that did nest and incubate eggs, nest initiation was significantly delayed, resulting in a delay in egg laying as well. None of the doves on the 40 ppm dietary supplement completed the nesting process. The authors hypothesized that the decline of reproductive activity was induced by the degradation of estrogen and androgen in the birds, which is presumably a result of increased hepatic microsomal enzyme activity as a response to the presence of PCBs. A LOAEL of 1.12 mg/kgBW/day and an estimated NOAEL of 0.112 mg/kgBW/day were identified from this experiment.

Tori, G. M. and T. J. Peterle (1983). "Effects of PCBs on mourning dove courtship behavior." Bull Environ. Contam. Toxicol. 30: 44-49.

A.35 Aroclor 1260

No studies were located that evaluated toxicity of Aroclor 1260 to birds or mammals.

APPENDIX B
Receptor Life History Profiles
Lower Ley Creek Site
Syracuse, New York

BELTED KINGFISHER (*Ceryle alcyon*)

Life History

The belted kingfisher is a pigeon-sized, territorial bird that is the only kingfisher present throughout most of North America (Bull and Farrand 1977; NGS 1987). During the breeding season, kingfishers occur throughout much of the United States and Canada, excepting high elevation areas. Kingfishers overwinter throughout much of their breeding range, but will migrate if ice accumulation limits food availability. Migrating kingfishers may travel as far south as northern South America (Hamas 1994).

The kingfisher is characterized by a blue-gray back and head with a white collar and stout bill. The male can be identified from the female by a blue-gray band across its breast, whereas the female has a second, rust-colored band across her belly. Kingfishers grow to 11 to 14 inches long from head to tail for both males and females. Rattling calls identify and help the kingfisher aggressively defend its territory (Cassidy 1990).

Belted kingfishers utilize a variety of freshwater and coastal habitats, including ponds, lakes, rivers, streams, reservoirs, large vernal pools, estuaries, and harbors. Kingfishers prefer calm, clear waters free of floating debris and dense aquatic plants. They also require foraging perches along water bodies from which they can hunt (Hamas 1994).

Food items include primarily shallow water fish, although crayfish, frogs, small snakes, salamanders, insects, crabs, and even mice may be consumed (Hayward 1885; Bull and Farrand 1977; Landrum *et al.* 1993). Dives for fish are quick, vertical, and loud. Belted kingfishers are reported to consume fish ranging in size from 2.45 to 17.78 centimeters (cm) in length, with the average size of fish consumed being less than 7.6 cm total length (Salyer and Lagler 1946). Most fish are caught by kingfishers within 60 cm of the water surface (Hamas 1994). It is estimated that a pair of kingfishers with nearly fledged young require approximately 90 fish per day to feed their offspring and themselves (Landrum *et al.* 1993). Vessel (1978) determined that a nestling can consume 11.2 fish per day during its maximum growth stage.

This species is solitary with the exception of the nesting season. Breeding times for this species vary with locale. Unseasonably mild weather may initiate early nesting in the lower United States. The presence of herbaceous cover and good fishing habitat are the basis for the selection of breeding areas and nest sites. Nests consist of stream bank or shoreline burrows, and vary in length depending upon the soil texture. Burrows are typically three to seven feet long (White 1953). Although usually near water, nests have been found up to 1.6 kilometers (km) away from water. A clutch of five to eight eggs are usually laid between early April and mid-June. Incubation lasts for 25 days with nest occupation for an additional 23 days. Both parents incubate the eggs (Bent 1940; Cassidy 1990; Landrum *et al.* 1993). Chicks grow rapidly during the first ten days, reach a maximum weight near the end of the third week of the nestling period, but lose weight before fledging (Vessel 1978; Albano 2000). Young leave the nest after 33 to 38 days. Young are able to capture live prey about a week after fledging. Fledglings remain near the nest and juveniles disperse by mid-summer (Bent 1940; Cassidy 1990; Landrum *et al.* 1993).

Males generally do not readily leave their territories and will remain there throughout the winter as long as ice does not impede fishing. Females typically migrate southward and return to the same mate and nesting site every year. The likelihood of migration for both males and females appears to depend on the severity of the winter (Landrum *et al.* 1993; Hamas 1994; DeGraaf and Yamasaki 2001).

Predators include raptors, especially accipters and hawks. Belted kingfishers escape predators by diving into the water. Nest predators include mammals and snakes, typically skunks, raccoons, and mink (White 1953; Hamas 1994). Belted kingfishers are relatively short-lived compared to most piscivorous birds. Juvenile mortality is high. If a bird survives to breed in its first year, it might be expected to live to the age of 2 to 3 years. The oldest age recorded by U.S. Fish and Wildlife Service banding records is 3 years, 8 months (Albano 2000; Lane *et al.* 2004). Albano (2000) reported one female that lived to at least 4 years of age.

Exposure Profile

The belted kingfisher was selected as a surrogate to model the effects of contaminants on an upper trophic level aquatic bird species. The belted kingfisher is highly adaptable and the presence of man-induced disturbances would not minimize use of the site by this species.

Reported body weights for adult belted kingfishers range from 104.3 to 188.8 grams (g) (Baldwin and Kendeigh 1938; Poole 1938; Hartman 1946; Salyer and Lagler 1946; Murray and Jehl 1964; Alexander 1977; Clench and Leberman 1978; Vessel 1978; Brooks and Davis 1987; Fry and Fry 1992; Hamas 1994; Albano 2000). Body weight varies seasonally. Males are heaviest prior to pair formation, and they lose weight during burrow excavation. Females are heaviest just prior to egg-laying. Both sexes lose weight during the period where they are providing food to nestlings; weight is lowest during the late nestling stage (Albano 2000). An average body weight of 136 g based on a Pennsylvania population (Brooks and Davis 1987) was used in this risk assessment.

Belted kingfishers feed mainly on shallow water fish, although crayfish, frogs, small snakes, salamanders, insects, crabs, and even mice may also be consumed (Hayward 1885; White 1936; White 1938; Salyer and Lagler 1946; White 1953; Eipper 1956; Alexander 1977). Crayfish have been reported as a substantial component of the kingfisher diet in several studies (Salyer and Lagler 1946; Eipper 1956; Alexander 1977; Steinmetz *et al.* 2003). For this risk assessment, it will be assumed that the diet of a belted kingfisher is comprised of 100 percent (%) fish.

Food ingestion rates reported for adult belted kingfishers range from 0.031 to 0.1009 kilograms per day (kg/day) wet weight (Alexander 1977; Vessel 1978; Kelly 1998). A higher food ingestion rate was reported for a nestling (0.1984 kg/day; White 1936), however only one bird was observed and there is no information on whether the bird was food limited prior to observation; therefore this study was not included. The allometric equation of Nagy (1987) was used to estimate the bioenergetics for the belted kingfisher. Based on a daily metabolic rate of 444 kcal/kg body weight-day, an average food intake rate of 0.448 kg/wet weight/kg body weight/day was used for this risk assessment.

The algorithm developed by (Calder and Braun 1983) was used to estimate the water ingestion rate using the equation: water ingestion (liters per day; L/day) = $0.059\ Wt^{0.67}$, where Wt. is BW in kg). The drinking water intake rate used for this risk assessment is 0.114 L/kg body weight/day.

Incidental sediment ingestion (as may occur during nest building and grooming) was assumed to be 1% of total dietary prey intake; the incidental sediment ingestion rate used for this risk assessment is 0.0045 kg/kg body weight-day.

Belted kingfishers defend two types of territories: the breeding territory and the foraging territory. The size of the breeding territory and fledging success appear to be related to the concentration of

prey (Davis 1982). The home range of this species varies seasonally and is usually reported as meters of shoreline (Landrum *et al.* 1993). Home range sizes ranging from 389.3 to 8045 m of shoreline have been reported for this species (Salyer and Lagler 1946; Cornwell 1963; Davis 1982; Brooks and Davis 1987; Mazeika *et al.* 2006). Resident kingfishers were considered to rely solely on Lower Ley Creek as their foraging habitat (and each of the three individual reaches as well).

In summary, the food chain model parameters for the belted kingfisher are as follows:

BW:	0.136 kg
Food ingestion:	0.448 kg/kg body weight-day w.w.
Water ingestion:	0.114 L/kg body weight-day
Sediment ingestion:	0.0045 kg/kg body weight-day
HR:	Lower Ley Creek

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GREAT BLUE HERON (*Ardea herodias*)

Life History

The great blue heron is the largest and most widely distributed American heron, inhabiting lakes, ponds, rivers, marshes and occasionally newly-plowed fields and meadows (Bull and Farrand 1977; Eckert 1987). The great blue heron is migratory in the northernmost portion of its range. Lingering birds usually fall prey to severe weather (Bull and Farrand 1977). Southward migration begins in early October; northward migration begins in March or early April (Eckert 1987). The great blue heron has a blue-grey back with blackish sides and a white and grey striped belly. The head has a white crown, cheeks and throat. A black stripe on the side of the crown merges into a long occipital crest. The bill is yellow and legs are a greenish-brown . The sexes have similar plumage, but females are smaller. With the exception of its breeding season, this species is solitary in its habits (Hancock and Kushlan 1984).

Great blue herons are primarily diurnal, but nocturnal foraging is common in tidal habitats (Hancock and Kushlan 1984). They feed anywhere they can locate prey (terrestrial or aquatic), but they primarily forage in shallow water less than 50 centimeters (cm) deep (Bent 1926; Bayer 1978). The great blue heron typically feeds either by standing motionless in the water waiting for prey, or by searching stealthily with a slow and careful walk. Their primary food item is fish, although frogs, small turtles, crustaceans, mice, voles, shrews, snakes, and ground-nesting birds are also consumed. Great blue herons generally capture fish ranging from 5 to 40 cm in length, although larger fish are occasionally consumed (Kirkpatrick 1940; Willard 1977). Almost without exception, the great blue heron will shake its bill in the water immediately after swallowing prey, perhaps to wash off debris. Although the digestive fluids of the heron are acidic enough to dissolve bone rapidly, an occasional undigested pellet of feathers and fur is regurgitated (Eckert 1987; Butler 1992).

Courtship occurs soon after the spring migration, with copulation usually occurring on the ground. Great blue herons are mostly monogamous, with new mates selected each year (Butler 1992). Colonial nests are placed on the uppermost branches of trees or shrubs. Occasionally a ground nest will be built if a secluded area is available. Successful nesting areas are usually returned to year after year. Three to seven pale greenish-blue eggs are incubated equally by both sexes for about 28 days (Eckert 1987). The chicks fledge at about 2 months of age. Nesting success depends on food supplies, and most nestling loss is due to starvation (Quinney 1982). Heron chicks weigh approximately 50 grams (g) at hatching, and will attain about 86 percent (%) of their adult weight by 45 days of age (Quinney 1982). Fish fed to chicks range in size from 7.8 cm to 22.8 cm (Hoffman 1978). Great blue herons do not reach sexual maturity until 2 years of age (DeGraaf and Yamasaki 2001).

Very few birds or animals will attack an adult great blue heron. Adult mortality due to attempting to swallow large fish, starvation, or entanglement in utility wires has been reported (Terres 1980). Crows, ravens, owls, eagles, gulls, bears and raccoons prey on the eggs and nestlings (Bent 1926; Hancock and Kushlan 1984). Mortality rates are 69% in the first year, decreasing thereafter (Bayer 1978). The maximum reported lifespans for great blue herons in the wild are 23 years, 3 months (Clapp *et al.* 1982) and 24 years, 6 months (Klimkiewicz 2008).

Exposure Profile

Reported body weights for adult great blue herons range from 1.905 to 3.64 kg (Esten 1931; Poole 1938; Hartman 1961; Palmer 1962; Eckert 1976; Alexander 1977; Hoffman 1978; Quinney

1982; Schramm *et al.* 1987; Wolf and Jones 1989; Butler 1992; Environment Canada 1998; Glahn *et al.* 2000). Males tend to be slightly larger than females. An average body weight for a female great blue heron of 2.20 kg was selected for this risk assessment based on Dunning (1993).

Great blue herons are opportunistic predators, and will consume whatever prey are available. Fish are the preferred food item for this species. Based on stomach contents, Palmer 1962 reported a diet comprised of 71.6% fish, 8.2% insects, 8.9% crustaceans, 4.3% herptiles, and 4.7% small mammals. Cottam and Uhler (1945) reported a dietary composition of 43% non-game fish, 25% game or commercial fish, 8% insects, 8% crustaceans, 4% herptiles, and 5% small mammals. Kirkpatrick (1940) reported a dietary composition of 92% fish, 1.7% insects, 2% crustaceans, 4% herptiles and 0.3% small mammals delivered to nestlings by adults. Alexander (1977) reported a dietary composition of 94% fish, 1% crustaceans, 3% amphibians, and 1% bird and mammal, percentage by weight of stomach contents. For this risk assessment, it was assumed that great blue herons consume 100% fish.

Food ingestion rates ranging from 0.202 kilograms per day (kg/d) to 0.477 kg/d wet weight (ww) have been reported for this species (Powell 1983; Schramm *et al.* 1987; Hoy *et al.* 1989; Littauer 1990; Butler 1993; Stickley *et al.* 1995; U.S. Department of Agriculture 1997; Glahn *et al.* 1999; Glahn *et al.* 2000). Using the bioenergetic algorithm of Nagy (1987), a daily food ingestion rate of 0.189 kg/kg body weight/d wet weight was used in this risk assessment.

A species-specific water ingestion rate could not be located for the great blue heron. Water ingestion rates were calculated using the allometric equation developed by (Calder and Braun 1983): WI (liters per day [L/d]) = $0.059\ Wt^{0.67}$, where WI is the daily water ingestion rate and Wt is the BW in kg. Using this equation, a water ingestion rate of 0.045 L/kg body weight/day was derived and used in this risk assessment.

An incidental sediment ingestion rate for the great blue heron was not located in the literature. An incidental ingestion rate for sediment was assumed to be approximately 1% of the total daily food intake, equivalent to 0.0019 kg/kg body weight/day, was used in this risk assessment.

Feeding territory sizes have been reported for this species in terms of kilometers (km) shoreline length and area (in hectares [ha]). In addition, foraging distances from nesting colonies have been measured. Foraging distances ranging between 1.8 to 34.1 km from nesting colonies have been reported (Mathisen and Richards 1978; Thompson 1978; Peifer 1979; Dowd and Flake 1985). Dietary exposure to contaminants will occur within a feeding territory; the distance traveled to a feeding territory is not a factor. Actively defended foraging areas ranging from 0.129 to 4.1 km of shoreline length have been reported for this species (Bayer 1978; Peifer 1979; Dowd and Flake 1985). Measured mean feeding territory sizes of 0.6 and 8.4 ha were reported by Bayer 1978. Resident great blue herons were considered to rely solely on Lower Ley Creek as their foraging habitat (and each of the three individual reaches as well).

In summary, the food chain model parameters for the great blue heron are as follows:

BW:	2.20 kg
Food ingestion:	0.189 kg/kg body weight/d wet weight
Water ingestion:	0.045 L/kg body weight/day
Sediment ingestion:	0.0019 kg/kg body weight/day
HR:	Lower Ley Creek

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RIVER OTTER (*Lutra canadensis*)

Life History

River otters are mustelids that inhabit lakes, streams, and estuarine environments. They are long-bodied, with dark reddish-brown fur and lighter underparts. They have short muzzles, short, stocky legs, and a long, thick tail. Their webbed feet attest to their primarily aquatic existence. River otters require food-rich areas having little impact from humans. Historically, river otters occurred throughout most of the U.S. except for the arid southwest. However, populations have been extirpated from or are on the decline throughout much of the Midwest, although some states have imported otters in an attempt to reestablish populations (Toweill and Tabor 1982). Along the coastal United States, populations are stable or increasing.

The river otter's home range varies by habitat and incorporates areas for foraging and reproduction. Each part of the home range is not used equally; food supply has the greatest influence on use of an area. Males usually have larger home ranges than females. Home ranges are often somewhat linear along rivers and coasts, and on a yearly basis may include up to 100 miles of shoreline (Schwartz and Schwartz 1981). On a seasonal basis, the home range is usually much smaller (Schwartz and Schwartz 1981; U.S. EPA 1993a, b).

River otters may be active day and night, though most activity is in early- to mid-morning and evening. In the U.S., they are thought to be active year-round (Jones and Birney 1988; Toweill and Tabor 1982). Otters generally live in family groups, although adult males may be solitary, or be excluded from the nest for a few months when the young are nursing (Merritt 1987; Toweill and Tabor 1982). Dens are usually in the previously abandoned burrows of animals such as woodchucks, in old muskrat or beaver lodges, or in natural shelters such as hollow trees or log jams (Jones and Birney 1988; Toweill and Tabor 1982).

River otters are mainly carnivorous. Although fish comprise the bulk of the diet, otters are opportunistic feeders and do not do well on a diet of fish alone (Schwartz and Schwartz 1981; Toweill and Tabor 1982). Other food sources include frogs, invertebrates (including crayfish, stonefly nymphs, clams, crabs, earthworms, snails, and beetles), turtles, snakes, muskrats, young beavers, shrews, voles, rabbits, moles, and birds (Hoffmeister 1989; Wayre 1979; Jones and Birney 1988; Schwartz and Schwartz 1981).

Breeding occurs in late winter or early spring. In the U.S., otters show delayed implantation, so that the gestation period is about 9-12 months in length (Rue 1981; Hoffmeister 1989; Toweill and Tabor 1982). Most litters are born in February through April. Litters contain from 1-6 (average 2-3) young (Hoffmeister 1989, Toweill and Tabor 1982; Nowak 1991). Females usually breed soon after parturition (Rue 1981; Merritt 1987). Young are weaned at about 3 months of age and disperse a few months after that (U.S. EPA 1993a). Females may breed at 2 years; males usually don't mate successfully until 4-7 years of age (Davis and Schmidly 1994; Merritt 1987; Toweill and Tabor 1982). Females may give birth every year, but more often reproduce bi-annually or every three years (Toweill and Tabor 1982).

The greatest threat to river otters is from hunters and trappers. Dogs, bobcats, coyotes, foxes, alligators, mountain lions, wolves, black bears, and large raptors may also occasionally kill otters (Rue 1981; Toweill and Tabor 1982). River otters may live up to 15 years in wild and up to 23 years in captivity (Rue 1981; Nowak 1991).

Exposure Profile

Adult river otters weigh from 3 to 14 kg (6.6-31 pounds) (Nowak 1991). Males are larger than females (Jones and Birney 1988; Rue 1981). In New York State, the average weight is about 5.45 kg (NYSDEC 2000), which was selected for this risk assessment. Resident otters were considered to rely solely on Lower Ley Creek as their foraging habitat.

Estimates of food ingestion for the otter range from 0.12 to 0.13 kg/kg BW/day (Wayre 1979; Harris 1968; Newell 1987). Using Nagy's (1987) allometric equation to estimate food ingestion based on metabolism, a food ingestion rate of 0.128 kg/kg body weight/day w.w. was calculated for this risk assessment.

A water ingestion rate for the river otter was not available from the literature. Therefore, the water ingestion rate was estimated using the allometric equation developed by Calder and Braun (1983). A water ingestion rate of 0.084 L/kg body weight/day was used in this risk assessment.

An incidental sediment ingestion rate for the river otter was not located in the literature. The incidental sediment rate of river otters was assumed to be approximately 1% of total daily food intake. An incidental sediment ingestion rate of 0.0013 kg/kg body weight/day was used in this risk assessment.

In summary, the food chain model parameters for the river otter are as follows:

BW:	5.45 kg
Food ingestion:	0.128 kg/kg body weight/day w.w.
Water ingestion:	0.084 L/kg body weight/day
Sediment ingestion:	0.0013 kg/kg body weight/day
HR:	Lower Ley Creek

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MINK (*Mustela vison*)

Life History

Mink are distributed over much of boreal North America, southward throughout the eastern United States, and in the west to California, New Mexico, and Texas. They can be found in virtually any habitat containing permanent water but are not commonly found in upland areas (Jones and Birney 1988). Although primarily nocturnal, their activity often extends into midday (Barbour and Davis 1974; Baker 1983; Hoffmeister 1989).

Mink are characterized by dark chestnut brown fur with tails comprising one-third to one-half of their 46 to 70 centimeter (cm) length. The coat is thick and dense, with an oily underfur overlaid by long, coarse guard hairs. Males may weigh twice as much as females. A long neck and thin body are supported by short sturdy legs. An occasional white spot appears on the undersides of the animals, and the ears are short and rounded, lying close to the head. The feet have five webbed toes used for swimming and capturing fish, and anal scent glands are well-developed (Godin 1977; Linscombe et al. 1982).

Dens are always near water, usually either in an old muskrat burrow or constructed by the mink (Jones and Birney 1988). Males tend to live in their own burrows that are less elaborate than ones occupied by females (Barbour and Davis 1974). The mink is a constant wanderer, and home ranges (HR) tend to be linear since mink often follow a shoreline (Jones and Birney 1988). Male HRs are notably larger areas than females, with male HRs overlapping one or more female's (Baker 1983). Mink are solitary and mark their territories by spraying (Merritt 1987).

Mink are opportunistic predators, and their diet reflects the availability of seasonally and annually variable food sources (Barbour and Davis 1974). Their ability to feed on a variety of aquatic, semi-aquatic, and terrestrial prey items allow them to adapt to a wide variety of habitat. Their diets may consist of crayfish, muskrats, frogs, fish, snakes, rodents, rabbits, birds, and plants, among other items (Schwartz and Schwartz 1981; Jones and Birney 1988). Crayfish and muskrats are a major portion of the summer diet in many regions of North America (Barbour and Davis 1974; Merritt 1987; Jones and Birney 1988; Fleskes and Klaas 1993); however in the South, frogs, fish, crayfish, insects and clams are more prevalent in the mink's diet (Sealander 1979). Mink typically prey on fish less than 15 centimeters (cm) in length (Heggenes and Borgstrom 1988). They kill larger prey items by biting them on the neck. Females have a difficult time handling larger prey (i.e. muskrats), therefore their diet is usually more limited.

Breeding seasons vary regionally, ranging from January to early April, with gestation periods ranging from 39 to 76 days (Soper 1973; Barbour and Davis 1974; Schwartz and Schwartz 1981; Merritt 1987). A single annual litter, highly variable in size (consisting of from 1 to 17 young), may be produced (Schwartz and Schwartz 1981). Average litter sizes vary among regions (Barbour and Davis 1974; Schwartz and Schwartz 1981; Merritt 1987; Jones and Birney 1988; Hoffmeister 1989). Young are weaned between five to six weeks of age, leave the nest at six to eight weeks, and are sexually mature by ten months of age (Schwartz and Schwartz 1981; Merritt 1987).

The mink has several predators. Great horned owls, foxes, lynxes, coyotes, bobcats, wolves and dogs are known to prey on mink (Schwartz and Schwartz 1981; Merritt 1987) and in the Deep South alligators (Soper 1973). Human-caused mortality, especially trapping, is also a significant mortality factor. There have been records of some mink individuals living up to six years, but mink seldom live longer than two years in the wild (Schwartz and Schwartz 1981).

Exposure Profile

The mink was selected as a surrogate to model the effects of contaminants on a mammalian piscivore. It is recognized that mink may not actually occur within the study area, however the site is well within the recorded range for mink, and mink are known to use similar habitat for feeding (Linscombe et al 1982). Mink have been documented to utilize suitable habitat even if it occurs in urbanized areas (Mech 2003). Resident mink were considered to rely solely on Lower Ley Creek as their foraging habitat.

Reported adult mink body weights range from 0.454 to 2.31 kilograms (kg) (Harding 1934; Marshall 1936; Jackson 1961; Mitchell 1961; Soper 1973; Barbour and Davis 1974; Haley 1975; Alexander 1977; Godin 1977; Sealander 1979; Bleavins and Aulerich 1981; Rue 1981; Schwartz and Schwartz 1981; Linscombe *et al.* 1982; Mumford and Whitaker 1982; Baker 1983; Hornshaw *et al.* 1983; Arnold and Fritzell 1987; Merritt 1987; Jones and Birney 1988; Aulerich *et al.* 1990; Wamberg *et al.* 1996; Tauson *et al.* 1998; Bursian *et al.* 2006). For this risk assessment, an average body weight of 0.600 kg was used, based on the average adult female weight provided by Mitchell (1961).

Mink are opportunistic predators that hunt principally along shorelines and emergent vegetation (U.S. EPA 1993). Seasonal availability and regional preferences govern the primary constituents of the minks diet. Mammals, fish and crayfish are usually the most abundant prey items, but amphibians, and young birds are also taken (Burgess and Bider 1980; Linscombe *et al.* 1982; Merritt 1987). (Arnold and Fritzell 1987) reported high consumption of avian species when birds were limited in mobility by incubation, brood rearing, or molting. Overall, based on stomach content analysis, the dietary composition of the mink consists of 33 percent (%) fish, 38 % mammals, 4% birds, 9% crustaceans, 15% amphibians, 0.5% insects, 0.4% vegetation, and 3% other or unidentified matter (Korschgen 1952; Korschgen 1958; Alexander 1977; Schwartz and Schwartz 1981). For this risk assessment, mink are assumed to be 100% piscivorous.

Food ingestion rates have been measured in several studies (Bleavins and Aulerich 1981; Aulerich *et al.* 1986; Zielinski 1986; Aulerich *et al.* 1990; U.S. EPA 1993; Heaton *et al.* 1995; Wamberg *et al.* 1996; Bursian *et al.* 2006). Reported food ingestion rates range from 0.128 to 0.409 kilograms per day (kg/day) wet weight. Using Nagy's (1987) allometric equation to estimate food ingestion rates based on metabolism, a food ingestion rate of 0.198 kg/kg body weight/day w.w. was calculated for this risk assessment.

Water ingestion rates ranging from 49 to 84 milliliters per day (ml/day) have been reported for mink (Wamberg et al. 1996; Tauson et al. 1998). A water intake rate of 0.133 milliliters per gram body weight (ml/g BW) was reported by (Farrel and Wood 1968), however this value included drinking water and water in food, therefore this value was not included. For this risk assessment, a water ingestion rate for mink of 0.104 L/kg body weight/day was estimated using the allometric equation developed by Calder and Braun (1983).

An incidental sediment ingestion rate for mink was not located in the literature. The incidental sediment ingestion rate by mink was assumed to be approximately 1% of total daily food intake. An incidental sediment ingestion rate of 0.002 kg/kg body weight/day was used in this risk assessment.

Reported HR sizes for mink vary from 7.7 to 6,475 hectares (ha) (Marshall 1936; Mitchell 1961; Rue 1981; Schwartz and Schwartz 1981; Arnold and Fritzell 1987; Arnold and Fritzell 1987; Merritt 1987; Jones and Birney 1988; Arnold and Fritzell 1990). For this risk assessment, it was assumed that mink forage exclusively in Lower Ley Creek.

In summary, the food chain model parameters for the mink are as follows:

BW:	0.600 kg
Food ingestion:	0.198 kg/kg body weight/day w.w.
Water ingestion:	0.104 L/kg body weight/day
Sediment ingestion:	0.002 kg/kg body weight/day
HR:	Lower Ley Creek

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