U.S. Army
Corps of Engineers

New England District
Concord, Massachusetts

U.S. Environmental
Protection Agency

New England Region
Boston, Massachusetts

ECOLOGICAL RISK ASSESSMENT FOR GENERAL ELECTRIC (GE)/HOUSATONIC RIVER SITE, REST OF RIVER

Volume 6
Appendix H: Assessment Endpoint - Piscivorous Birds
Appendix I: Assessment Endpoint - Piscivorous Mammals
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DCN: GE-070703-ABRC

July 2003

Environmental Remediation Contract
GE/Housatonic River Project
Pittsfield, Massachusetts

Contract No. DACW33-00-D-0006

03P-0966-6

Task Order 0003

APPENDIX H

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS BIRDS

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LIST OF ACRONYMS

BW	body weight
CL	confidence limit
COC	contaminant of concern
COPC	contaminant of potential concern
DL	detection limit
EROD	ethoxyresorufin O-deethylase
FIR	food intake rate
GE	General Electric Company
LOAEL	lowest observed adverse effect level
NOAEL	no observed adverse effect level
PAHs	polycyclic aromatic hydrocarbons
PCB-126	3,3',4,4',5-pentachlorobiphenyl
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-p-dioxin
PCDF	polychlorinated dibenzofuran
PSA	Primary Study Area
TCDD	tetrachlorodibenzo-p-dioxin
TDI	total daily intake
TEF	toxic equivalency factor
TEQ	toxic equivalence
tPCBs	total PCBs
UCL	upper confidence limit
USFWS	U.S. Fish and Wildlife Service
USGS	U.S. Geological Survey
WOE	weight-of-evidence

APPENDIX H

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS BIRDS

H.1 INTRODUCTION

The purpose of this appendix is to characterize and quantify the current and potential risks posed to piscivorous birds exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain, from total PCBs (tPCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The river is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters. The Primary Study Area (PSA) includes the river and 10-year floodplain from the confluence of the East and West Branches of the Housatonic River, downstream of the GE facility, to Woods Pond Dam.

A Pre-ERA was conducted to narrow the scope of the ERA by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A three-tiered deterministic approach was used to screen COPCs. The deterministic assessments compared potential conservative estimates of exposure with conservative adverse effects benchmarks to identify contaminants of potential concern to piscivorous birds in the Housatonic River. A hazard quotient (total daily intake/effect benchmark) for piscivorous birds greater than one in the Housatonic River area resulted in the COPC being screened through to the probabilistic ERA. Subsequent to the Pre-ERA, several other COPCs (primarily organochlorine pesticides) were screened out because their actual concentrations in the PSA were likely much lower than the measured values due to laboratory interference (see Section 2.4). In summary, the COPCs that were retained for the probabilistic risk assessment for piscivorous birds were tPCBs and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxic equivalence (TEQ).

Total PCBs detected in Housatonic River media samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4 and Appendix C.10).

H.1.1 Overview of Approach

A step-wise approach was used to assess the risks of tPCBs and TEQ to piscivorous birds in the Housatonic River watershed. The four main steps in this process include:

1. Derivation of a conceptual model (Figure H.1-1).
2. Assessment of exposure of piscivorous birds to COCs (Figure H.1-2).
3. Assessment of the effects of COCs on birds (Figure H.1-3).
4. Characterization of risks to piscivorous birds (Figure H.1-4).

H.1.2 Conceptual Model

The conceptual model presented in Figure H.1-1 illustrates the exposure pathways for piscivorous birds exposed to tPCBs and TEQ in the PSA.

Total PCBs and TEQ are persistent, lipophilic, and hydrophobic. Therefore, they are bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001). Piscivorous birds that reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally through diet and trophic transfer. Other routes of exposure, considered to be less important to overall exposure, include inhalation, water consumption, and sediment ingestion (Moore et al. 1999).

The belted kingfisher (*Ceryle alcyon*) and osprey (*Pandion haliaetus*) were selected as the representative species for piscivorous birds in the PSA. Kingfishers have been observed nesting and breeding in the PSA; however, observations of ospreys suggest that birds foraging in the PSA are transients. The PSA contains suitable habitat for ospreys, with abundant prey, so there is a high likelihood that as the Massachusetts and Connecticut osprey population continues to expand, they may nest in the PSA. Great blue heron were also considered when selecting the representative species, but were not included because although productivity data exist for herons in the vicinity of the PSA (MDFW 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986a,b, 1987, 1989, 1991, 1996), only a few of the birds from the rookery forage in the Housatonic River. Estimating exposure for these birds, and also estimating the exposure of other herons in the area, would have been difficult. Therefore, effects, or lack thereof, on productivity observed in the field could not be attributable to COCs.

Life history profiles for belted kingfishers and ospreys are summarized in the following text boxes. Additional life history information on these species is provided in Sections H.2.1.1.1 and H.2.1.2.1, respectively.

Life History of Belted Kingfisher

The belted kingfisher is a pigeon-sized member of the Alcedinidae family and is a common bird in North America, excluding the far north and the higher elevations of the Rocky Mountains.

Habitat - Prefer foraging areas with clear water and visibility unobstructed by turbidity or aquatic vegetation. Size of territory depends upon the availability of prey, ranging from 0.5 to 1.36 miles (0.8 to 2.2 km) of shoreline.

Diet - Principal prey is fish, but may also feed on berries and other small animals, including mollusks, crustaceans, insects, amphibians, reptiles, young birds, and small mammals.

Life History of Osprey

Ospreys, also known as fish hawks or fishing eagles, are the only species in the family Pandionidae. The range of these raptorial birds covers almost all of North America, except for the extreme north.

Habitat - Use both fresh and saltwater ecosystems, but primarily the latter. Osprey are tree nesters, but have also adapted to man-made structures. Foraging ranges from 1.7 km to 15 km, depending on prey availability.

Diet - Almost exclusively piscivorous, preferring medium-sized fish (13 to 40 cm). On rare occasions, osprey will take dead fish or prey on small mammals, reptiles, and crustaceans.

The assessment endpoint that is the subject of this appendix is the survival, growth, and reproduction of piscivorous birds in the Housatonic River PSA. The measurement endpoints used to evaluate the assessment endpoint were based on the determination of the extent to which the concentrations of PCBs and TEQ ingested in the diet will impact the survival, reproduction, or growth of piscivorous birds. This was carried out by an in situ investigation of kingfisher reproductive success and by comparisons of modeled exposure to doses reported in the literature to cause adverse effects.

H.1.3 Exposure Assessment

The exposure assessment estimates the exposure of piscivorous birds to tPCBs and TEQ in the Housatonic River PSA (Figure H.1-2). It begins with a description of the exposure model. Input variables for the exposure model were parameterized using life history information on the representative species and concentrations of tPCBs and TEQ in prey collected in the PSA. Monte Carlo and probability bounds analyses were then conducted to estimate exposure for each COC.

H.1.4 Effects Assessment

The effects assessment provides an overview of the literature on the effects of tPCBs and TEQ to survival, growth, and reproduction of representative bird species (Figure H.1-3). An extensive literature search was conducted to locate studies with data and information on the survival, growth, and reproduction of avian species exposed to the COCs. Each study was evaluated using defined acceptability criteria. The studies were then selected and used to derive the most appropriate effects metrics.

H.1.5 Risk Characterization

The risk characterization evaluates the likelihood that adverse effects may occur as a result of wildlife exposure to tPCBs and TEQ (Figure H.1-4). Two lines of evidence were available to characterize risks to piscivorous birds from exposure to tPCBs and TEQ:

- **Modeled Exposure and Effects.** The purpose was to determine the extent to which the concentrations of tPCBs and TEQ ingested in the diet would cause adverse effects to the survival, reproduction, or growth of piscivorous birds. A probabilistic food web model was used to estimate exposure of representative piscivorous bird species to tPCBs and TEQ. The total daily intakes for kingfishers and ospreys in the PSA were calculated using equations from the Wildlife Exposure Factors Handbook (EPA 1993) and related publications. These estimated exposures were compared to the results of toxicological studies reported in the literature to determine if the avian species were being exposed to tPCBs and TEQ at concentrations likely to induce adverse effects.

- **Field Study - Belted Kingfisher.** A belted kingfisher reproduction study was performed in the PSA during the 2002 breeding season by GE (Henning 2002). The study evaluated the reproductive output and relationship between reproductive output of kingfishers and exposure of adult and nestling kingfishers to tPCBs.

A weight-of-evidence (WOE) assessment was conducted to combine the results from each line of evidence. The section concludes with a discussion of sources of uncertainty in the assessment of risks of COCs to piscivorous birds and the conclusions of the risk characterization.

H.1.6 Organization

This appendix is organized as follows:

- Section H.2 describes the exposure model, input parameters, and uncertainty propagation techniques. Also presented in this section are the input data and exposure results for the belted kingfisher and osprey.
- Section H.3 describes the effects to birds exposed to tPCBs and TEQ.
- Section H.4 describes the risk characterization for the two lines of evidence. In addition, a discussion of the sources of uncertainty regarding risk estimates is provided, as well as the conclusions regarding risk for piscivorous birds exposed to tPCBs and TEQ in the Housatonic River.

H.2 CHARACTERIZATION OF EXPOSURE

The representative species selected for piscivorous birds are the belted kingfisher (*Ceryle alcyon*) and osprey (*Pandion haliaetus*). These birds are native to the Housatonic River area and feed on prey exposed to the COCs directly and through trophic transfer. Exposure of these species to tPCBs and TEQ was estimated using a total daily intake model adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993).

Total PCBs and TEQ are considerably more toxic to developing embryos and young birds than to adults (Peterson et al. 1993). Eggs are hatched and young are raised during the spring and summer months; therefore, the exposure assessment was performed for this period.

This assessment focuses on Reaches 5 and 6 (Woods Pond), also referred to as the Primary Study Area (PSA). Where possible, exposure assessments were also conducted for two reference locations: East Branch of the Housatonic River, upstream of Dalton (termed "upstream reference area" hereafter), and Threemile Pond in Sheffield, MA.

This section begins with a description of the general model used for the two representative species. Subsequent sections describe the inputs used in the exposure analyses for each representative species. The section concludes with a description of the Monte Carlo and probability bounds analyses conducted to estimate total daily intake of tPCBs and TEQ by each of the representative piscivorous bird species in the Housatonic River PSA and reference areas.

H.2.1 Exposure Model for Piscivorous Birds

The model used to estimate exposure of piscivorous birds to tPCBs and TEQ focuses on ingestion of these contaminants through the diet. Other exposure routes (e.g., water) were considered to be of much less importance for tPCBs and TEQ. The model used in the exposure analysis was:

$$TDI = FT \times FIR \sum_{i=1}^{n} C_i \times P_i \qquad \text{(Eq. 1)}$$

where

TDI = Total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ).

FT = Foraging time in the PSA (unitless).

FIR = Normalized food intake rate (kg/kg bw/d).

C_i = Concentration in ith prey item (mg/kg for tPCBs, ng/kg for TEQ).

P_i = Proportion of the ith prey item in the diet (unitless).

***Choosing Exposure Distributions*—**Input distributions were generally assigned as follows:

- Lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue for tree swallows).
- Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
- Point estimates for minor variables or variables with low coefficients of variation.

In certain situations (e.g., poor fit to the data), other distributions were fit to the data or other approaches were used. These latter situations are described in detail where they occurred in the following exposure analysis sections.

***Incorporation of Spatial Averaging in Derivation of Estimated Prey Concentrations*—** Concentrations of tPCBs and TEQ vary spatially in prey. Many wildlife species forage over distances ranging from tens of meters to greater than 10 km. An individual integrates the spatial variation in tissue concentrations of their prey. Therefore, estimates of the central tendency were used in the exposure model as an expression of the spatial and temporal averaging of concentrations of COCs in prey tissue. In the probabilistic exposure analyses, it was assumed that the spatially and temporally averaged exposure estimate did not vary among individuals foraging in the same area.

To account for uncertainty due to sample size, the measure of centrality in the Monte Carlo analysis was the minimum of: (1) the 95% upper confidence limit (UCL) calculated using the Land H-statistic (assuming the data are lognormally distributed), or (2) the maximum concentration measured. The Land H-statistic has the underlying assumption that data are lognormally distributed. Testing of data for lognormality is discussed in Section 6.3. In the

probability bounds analyses, however, the uncertainty regarding the arithmetic mean was accounted for with a different procedure. The procedure generally involved using the Land H-statistic to estimate the lower and upper 95% confidence limits on the mean (Gilbert 1987), and then using these lower and upper confidence limits to derive bounds on all possible distributions that exist within this range. This approach results in an expression of the uncertainty about the true value of the arithmetic mean that arises due to the limited sample size. In cases where the 95% UCL could not be estimated or exceeded the measured maximum concentration, other techniques were used to derive the bounds on the mean. These techniques and criteria for their use are described in Section 6.3 and Appendix C.5 of the ERA.

Techniques for Propagating Uncertainty—In this assessment, two types of probabilistic analyses were performed: Monte Carlo analysis and probability bounds analysis. The former is the most common probabilistic method employed in ERAs, and guidance for its use in Superfund assessments is available (EPA 1997). While Monte Carlo methods are appropriate for the determination of exposure risks when input distributions are known precisely, they do not adequately represent the effects of uncertainty around the input distributions (Ferson 1996). In many ERAs, the available data are limited; consequently, the input distributions used to calculate risks are uncertain. Probability bounds analysis is a tool for separating variability and uncertainty to obtain bounds on the result that explicitly account for the uncertainty about the input distributions. As in Monte Carlo analysis, the overall slopes of the bounds indicate how much variability exists in the system. The distance between the bounds is an indication of the uncertainty that exists due to lack of knowledge. More detailed descriptions of Monte Carlo analysis and probability bounds analysis can be found in Appendix C.4.

For each Monte Carlo analysis, a sensitivity analysis was performed to determine the relative influence each input variable had on the output exposure distribution. This was done for each input variable by determining the correlation between the randomly chosen input values and the corresponding output exposure estimates. Input variables that have a strong influence on the output exposure estimate tend to have high correlation coefficients, although this interpretation becomes problematic when there are dependencies between input variables. The results of the sensitivity analyses are included in this exposure assessment.

The exposure models for the ERA contain multiple variables, some of which may be correlated. The assumption of independence can be inappropriate, because dependencies can affect the estimated exposure. If correlations are not accounted for, the variance and the tails of the exposure distribution may be poorly estimated. This assessment uses several approaches to address correlations between variables. These approaches include simulation of observed correlations, assumption of perfect covariance (e.g., when the diet consists of two prey items, the proportion of one item in the diet is equal to one minus the other item), or no assumptions at all about dependencies (all possible relationships between two variables can occur). The specific approach used depends on the type of data and the application. In cases where independence of variables seemed intuitively obvious (e.g., COC concentration in the prey item and proportion of that item in the diet), independence was assumed.

Treatment of Non-Detects—The approach for generating summary statistics or distributions when the data set includes samples with COC concentrations below the detection limit is described in detail in Section 6.4 and Appendix C.2. In summary, for a data set with non-detects, summary statistics were generated assuming that the non-detect contaminant concentrations were zero (ND = 0), and assuming that the non-detect contaminant concentrations were equal to the detection limit (ND = DL). If the ratio between the statistic of interest (e.g., mean) calculated assuming ND = DL and the statistic assuming ND = 0 was less than or equal to 1.3, the analysis was performed assuming that all non-detected chemical concentrations were equal to half the detection limit (ND = 1/2 DL).[1] If the ratio was greater than 1.3 and the sample size and detection frequency were adequate (i.e., n ≥5, detection frequency ≥25%), the probability plot method described in Appendix C.2 was used to estimate concentrations. If sample size and detection frequency were not adequate, then the exposure calculations were repeated assuming ND = 0, ND = 1/2 DL, and ND = DL for the Monte Carlo analysis, and ND = 0 and ND = DL for the probability bounds analysis. For a more detailed discussion of the treatment of non-detects, see Section 6.4 (Figure 6.4-2) of the ERA and Appendix C.2.

[1] This decision criterion supplements the procedures described in Appendix C.2.

H.2.1.1 Belted Kingfisher

Belted kingfishers were selected as one of the representative species for piscivorous birds because the habitat within the study area is suitable for kingfishers and is within the normal range of the species during the breeding season. Sightings of kingfishers were common in the PSA and they were observed nesting at several locations (Appendix A). Kingfishers feed primarily on fish, which are relatively high in the food chain and are susceptible to accumulation of lipophilic substances such as tPCBs. Many species of piscivorous birds, including terns, herons, and cormorants, have been shown to accumulate tPCBs to significant concentrations (Bosveld and Van den Berg 1994), and it is expected that kingfishers would similarly accumulate tPCBs and TEQ.

H.2.1.1.1 Life History

General Description

The belted kingfisher is a pigeon-sized member of the Alcedinidae family and is a common bird in North America. Body size for adult birds ranges from 125 to 215 g with little difference between males and females (Hamas 1994). The breeding range spans the majority of the continent, excluding the far north and the higher elevations of the Rocky Mountains (Hamas 1994; DeGraaf and Yamasaki 2001). Nest construction begins in late April (Ellison 1985), with egg dates ranging from May 14 to June 6 (Veit and Peterson 1993). In the northern setting of the PSA, kingfishers will generally have only one brood per year (Hamas 1994), with an average of 6 to 7 eggs per clutch (DeGraaf and Yamasaki 2001).

Habitat

Kingfishers prefer foraging areas with clear water and visibility unobstructed by turbidity or aquatic vegetation (Hamas 1994). Kingfishers typically nest in excavated burrows in earthen banks (Brooks and Davis 1987) near their foraging territory (DeGraaf and Yamasaki 2001). In the Housatonic River PSA, sightings were made over the length of the river from the confluence to Woods Pond (Appendix A).

Dietary Habits

The principal prey of kingfishers is fish, but they also feed on berries and a variety of other small animals, including mollusks, crustaceans, insects, amphibians, reptiles, young birds, and small mammals (Hamas 1994). Most fish caught by the kingfisher are small, typically less than 10 cm in length (Prose 1985; Imhof 1962; Salyer and Lagler 1946), and as small as 2.5 cm (Salyer and Lagler 1946). Maximum prey lengths have been observed to reach 14 cm (Davis 1982) and 17.8 cm (Salyer and Lagler 1946) in different locations. Fish prey species are those that typically live in shallow water or near the surface (Hamas 1994) and include trout, salmon, suckers, perch, minnows, killifish, sticklebacks, and others (EPA 1993).

H.2.1.1.2 Exposure Model Input Distributions

The input variable parameterizations used in the Monte Carlo and probability bounds analyses to estimate exposure of belted kingfishers to tPCBs and 2,3,7,8-TCDD TEQ are summarized in Tables H.2-1 and H.2-2. Exposure analyses were conducted separately for Reaches 5 and 6 (Woods Pond). Two reference areas (upstream reference area and Threemile Pond) were also included for comparison.

Foraging Time

Selection and defense of territories by kingfishers depends on season and prey availability. They aggressively defend their territories to protect nests during the breeding season (Hamas 1994). The size of a kingfisher's territory depends on the richness of prey, but is generally compact. The range expressed in terms of shoreline length is 0.5 to 1.36 miles (0.8 to 2.2 km) (Salyer and Lagler 1946; Brooks and Davis 1987). At a Minnesota lake, territories stretched up to 5.0 linear miles (8.0 km) from the nest site (Cornwell 1963). Given the small size of the foraging range, it was assumed that some kingfishers forage exclusively within the PSA.

Body Weight (BW)

Body weights of belted kingfishers vary only slightly with sex (Hamas 1994). Dunning (1993) reported a body weight range of 125 to 215 g with a mean of 148 g. Mean body weights have been reported in this range by other investigators (Alexander 1977; Salyer and Lagler 1946).

Brooks and Davis (1987) calculated mean body weights of 136 g for birds in Pennsylvania and 158 g for kingfishers in Ohio. Hamas (1994) recorded body weights of male and female kingfishers in Minnesota in spring and found females to be slightly heavier than males, 152 and 144 g, respectively. Salyer and Lagler (1946) reported a mean body mass of 170 g for birds in Michigan.

In the Monte Carlo analysis, body weight was assumed to be normally distributed with a mean of 150 g and a standard deviation of 14.7. The mean was derived from available body weight data from the literature and the standard deviation was determined using an average coefficient of variation of 9.77% from body weight studies that presented standard deviation (Brooks and Davis 1987; Dunning 1993). The uncertainty in this variable is likely due to variability, rather than lack of knowledge or data. Thus, the same distribution was used in the probability bounds analysis for this input variable.

Food Intake Rate (FIR)

The food intake rate of belted kingfishers has not been well characterized. Food ingestion rate data are available in the EPA *Wildlife Exposure Factors Handbook* (EPA 1993); however, this information was not appropriate for use in this exposure model. These data lacked body weight information, lacked statistical analyses, were estimates themselves, and/or were collected from young or captive birds. The field-based estimate of the daily food intake rate of free-living adult kingfishers (0.50 g/g-day, Alexander 1977) was close to the 30th percentile of the modeled food intake rate (see below) for these birds. Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic rate of free-living birds using the following general equation:

$$FMR\ (kJ/day) = a \times BW(g)^{b} \qquad \text{(Eq. 4)}$$

For both the Monte Carlo and the probability bounds analyses, *FMR* for kingfishers was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (body weight [*BW*], *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999), assuming an underlying normal distribution for each. There were insufficient data to generate an allometric equation for Coraciiformes, of which

belted kingfishers are members, so the equation for all birds was used. The slope term *log a* had a mean of 1.02 and a standard error of 0.0393 in log units, and the slope term *b* had a mean of 0.681 and a standard error of 0.0182 (Nagy et al. 1999). The body weight (*BW*) distribution was described above. The results of the calculation were then converted to kcal/kg bw/d.

Food intake rate is derived from *FMR* using the following equation:

$$FIR(kg/kg\ bw/d) = FMR / \sum_{i=1}^{n} (AE_i \times GE_i) \qquad \text{(Eq. 5)}$$

where AE_i is the assimilation efficiency of the *i*th food item (unitless) by kingfishers and GE_i is the gross energy of the *i*th food item (kcal/g). For kingfishers, mean assimilation efficiencies were 77% for aquatic invertebrates and 79% for fish. The mean gross energies were 1.1 kcal/g wet weight for aquatic invertebrates and 1.2 kcal/g wet weight for fish (EPA 1993). The assimilation efficiency and gross energy variables have small coefficients of variation, and are thus treated as point estimates in the exposure analysis.

Proportion of Dietary Items (P_i)

White (1953) analyzed more than 1,300 pellets and stomach contents from kingfishers in the Maritime Provinces of Canada and found that they prey on fish that are locally abundant and inhabit shallow waters. Alexander (1977) found that kingfishers foraging in trout streams in Michigan had trout as the major dietary component. In other streams, only 17% of the diet included trout and the remainder was composed of other fish (29%), amphibians (27%), insects (19%), crustacea (5%), and birds and mammals (1%). In Michigan trout streams, Salyer and Lagler (1946) found trout comprised 30% of the diet, with other fish (28%) and crustacea (41%) completing the diet. Davis (1982) found that kingfishers in Ohio fed mostly on fish (86%) and crayfish (13%). Cairns (1998) reported that kingfishers in the Canadian Maritime Provinces consumed various types of fish for 91% of their diet, crustaceans for 7.6%, and other prey items accounted for the remainder. Table H.2-3 summarizes the studies reviewed to determine kingfisher diets.

Combining the available data, fish comprised an average of 81.1% (range of 46 to 100%) of the kingfisher diet and crustaceans 10.2% (range of 0 to 41%), with other prey items making up the

difference (i.e., insects 4.2% and amphibians 3.9%). The latter two components were not included in the exposure analyses, however, because of their small contribution to the overall diet. In Reach 5, the mean proportion of fish and crayfish in the diet of kingfishers was assumed to be 88.8% and 11.2%, respectively, based on a revised analysis of the combined literature data after exclusion of insects and amphibians.

The proportions of fish in the diet in Reach 5 and the upstream reference area were assumed to follow a beta distribution in the Monte Carlo analysis and were parameterized to approximate the above proportions (alpha = 55, beta = 9, scale = 1). This parameterization results in a distribution for the fish portion of the diet that has a mean close to 86%, with a range of 70 to 97%, which is representative of the available data. The percent of crayfish in the diet, therefore, was estimated as 100 minus the percent of fish in the diet.

The beta distribution is not an available option in RiskCalc, the software used for conducting the probability bounds analyses. As an alternative, minimum, mean, and maximum values were specified for each dietary item using the means and ranges described above. The minimum, mean, and maximum values were then included as a distribution-free statement in RiskCalc. The result is a set of bounds that include all possible distributions, given the specified minimum, mean, and maximum values specified for the dietary items.

For the Woods Pond and Threemile Pond exposure models, crayfish were not included in the kingfisher diet. The primary reasons for this include:

- The lack of crayfish sightings when conducting other field surveys during the last 3 years at these locations.
- The aquatic vegetation, which acts to conceal crayfish from kingfishers.
- The abundance of cyprinids and centrachids of forage size, which live in the shallow areas and are visually attractive to hunting kingfishers.

Given these factors, it was assumed that fish would replace crayfish in the diet of kingfishers inhabiting the areas around Woods Pond and Threemile Pond. Therefore, in Woods Pond and Threemile Pond, the percent contribution of fish in the diet was assumed to be 100%, and was treated as a point estimate in the Monte Carlo and probability bounds analyses.

Concentration of tPCBs in Fish in Reach 5 (C_i)

A number of fish species have been sampled in Reach 5 including largemouth bass, yellow perch, sunfish, and cyprinids. In Reach 5, 121 samples were collected that were between 2.5 and 17.8 cm in length, the approximate length of fish consumed by kingfishers. The Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 36.4 mg/kg. For the probability bounds analysis, the lower and upper 95% confidence limits determined from the Land H-statistic, 30.5 and 36.4 mg/kg, were used to parameterize a distribution-free statement in RiskCalc.

Concentration of TEQ in Fish in Reach 5 (C_i)

TEQ concentrations were measured in 46 fish samples taken from Reach 5 that were between 2.5 and 17.8 cm in length. Using the decision criteria outlined in Section 6.4, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations. These data were used to calculate the parameters for Monte Carlo and probability bounds analyses. The assumption of how to treat non-detected congeners in the TEQ calculation also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum value was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 712 mg/kg.

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values, assuming that non-detected congeners were equal to half the detection limit, were calculated using the greater of the lower 95% CL and the minimum value, and the lower of the upper 95% CL and the maximum value, respectively. In this

analysis, the 95% LCL and the 95% UCL were selected, with values of 518 and 712 ng/kg, respectively.

Concentrations of COCs in Prey Items From Other Locations (C_i)

The parameterization for the remainder of the prey item COC concentration variables from Reach 5, Reach 6, and the reference areas is presented in Tables H.2-4 and H.2-5. The decision criteria used to parameterize the concentration variables are described in Section 6.4 and follow the logic illustrated in the preceding paragraphs for tPCBs and TEQ.

H.2.1.1.3 Exposure Model Results

The results of the Monte Carlo and probability bounds analyses for exposure of belted kingfishers to tPCBs and TEQ in the PSA and reference areas are presented in Tables H.2-6 and H.2-7, respectively.

Exposure to tPCBs in Reach 5

The Monte Carlo analysis indicated that exposure of kingfishers to tPCBs in Reach 5 could range from a minimum of 11.5 to a maximum of 30.4 mg/kg bw/d. The mean exposure was 18.1 mg/kg bw/d, and the median exposure was 17.9 mg/kg bw/d (Table H.2-6). Of exposure estimates, 80% were between 15.1 and 21.4 mg/kg bw/d. Figure H.2-1 depicts the cumulative distribution of tPCB intake rates for kingfishers in Reach 5.

Sensitivity analysis (Table H.2-8) revealed that the *FMR* power and slope terms were the most important variables (Pearson correlation coefficient (r_p) = 0.66), followed by body weight (r_p = -0.212).

The probability bounds estimated for kingfishers foraging in Reach 5 are depicted in Figure H.2-1. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 10.2 and 17.3 mg/kg bw/d. The 50th percentile ranged between 12.5 and 20.6 mg/kg bw/d, and the 90th percentile ranged between 15.3 and 24.9 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 15.1, the 50th percentile was 17.9, and the 90th percentile was 21.4 mg/kg bw/d.

Other Exposure Scenarios

Exposures of belted kingfishers to tPCBs in Reaches 5 and 6 were similar (Table H.2-6). Exposures in the upstream reference area and Threemile Pond were two to three orders of magnitude lower than in Reaches 5 and 6, having mean total daily intakes of 0.120 and 0.0177 mg/kg bw/d, respectively. As illustrated by the probability bounds distributions, there was a similar degree of uncertainty for all four locations.

Mean exposure of belted kingfishers to TEQ in Reach 5, Reach 6, the upstream reference area, and Threemile Pond was 399, 415, 100, and 12.7 ng/kg bw/d, respectively (Table H.2-7). While exposure of belted kingfishers to tPCBs was highest in Reach 5, TEQ exposure was highest in Reach 6. Figures H.2-1 to H.2-8 depict the cumulative distribution of tPCB and TEQ total daily intake, as well as the corresponding probability bounds, for belted kingfishers at the four areas. For each exposure scenario, sensitivity analysis revealed that the *FMR* power term and *FMR* slope term were the most important variables in the Monte Carlo analyses (Table H.2-8).

H.2.1.2 Osprey

Ospreys were selected as a representative species because of their presence in the Housatonic River watershed, their position at the top of the food web, and their almost exclusive reliance on fish in their diet. As top predators in riverine and lacustrine ecosystems, ospreys are susceptible to the bioaccumulative substances found in these systems. Ospreys have been observed to accumulate tPCBs, PCDDs, and PCDFs from contaminated locations to significant concentrations (Elliott et al. 2001). Ospreys were observed during field surveys in the PSA; however, no nesting has been reported (Appendix A). The habitat is suitable to support ospreys during the breeding season.

H.2.1.2.1 Life History

General Description

Ospreys, also known as fish hawks or fishing eagles, are the only species in the family Pandionidae and therefore, are distinct from the true hawks and eagles, family Accipitridae. The range of these birds covers almost all of North America, except for the extreme north. Although

osprey numbers have declined due to the use of DDT, an increase in osprey numbers has been observed since the ban of the pesticide (Veit and Peterson 1993). Ospreys are large birds with body lengths ranging from 53 to 61 cm, wingspans of up to 180 cm, and average body weights of 1.6 kg for females and 1.4 kg for males (Rattner et al. 2001). Ospreys typically lay clutches of 2 to 3 eggs in early May and have only one brood (Veit and Peterson 1993). Hatchlings are semi-precocial and reach 70% to 80% of their adult body weight within 30 days (Poole 1989).

Habitat

Ospreys use both fresh and saltwater ecosystems, but primarily the latter (Rattner et al. 2001). Breeding habitat for ospreys requires sufficient fish abundance and water that is shallow or clear enough to spot prey (Vana-Miller 1987). Otherwise, the surroundings are of little consequence in nesting site selection; ospreys will choose remote lakes, coastal islands, and even areas close to houses (Bent 1964). Ospreys are tree nesters, but have adapted to man-made structures (Newton 1979).

Ospreys were observed in riverine and shoreline habitats in the PSA, as documented in the 1998-2000 field surveys (Appendix A). Almost all osprey observations in the PSA occurred in late summer and fall, and presumably were of migrating birds or birds wandering from their nesting sites at the end of the breeding period. Although no ospreys are nesting in the PSA, nor are any pairs using it as a frequent or infrequent hunting area during the nesting season, this risk assessment is based on potential use of the study area by nesting pairs, because of the suitability of the habitat, and increasing expansion of ospreys into more territories in both Connecticut and Massachusetts as their numbers increase.

Dietary Habits

Ospreys prefer to forage in shallow waters in lakes and rivers where fish occur near the surface and may be easily seen (DeGraaf and Yamasaki 2001). The birds are almost exclusively piscivorous, preferring medium-sized fish (13 to 40 cm in length). On rare occasions, ospreys will take dead fish or prey on small mammals, reptiles, and crustaceans (Chubbs and Trimper 1998). Ospreys were observed hunting and eating fish in the PSA; koi and goldfish were commonly observed as prey.

H.2.1.2.2 Exposure Model Input Distributions

The input variable parameterizations used in the Monte Carlo and probability bounds analyses to estimate exposure of ospreys to tPCBs and TEQ are summarized in Tables H.2-9 and H.2-10. Because of the large foraging range of ospreys, exposure analyses were conducted for the entire PSA (Reaches 5 and 6 combined). The Threemile Pond reference area and upstream reference areas were included for comparative purposes.

Foraging Time

Ospreys prefer to forage in shallow, calm waters (Poole 1989). The downstream portion of the PSA has an abundance of suitable foraging habitat containing clear water and ample amounts of fish in size classes used by osprey. In contrast, Reach 5A and some areas downstream of the PSA have faster flowing water with riffles, which are not preferred osprey feeding habitat. The foraging radius of these birds is larger when food is less available (Clark 1995; Newton 1979), and has been documented to range from 1.7 km to 15 km (EPA 1993); however, ospreys are opportunistic and will hunt where there is ample available prey that is easily captured close to the nest. The river and backwaters between the WWTP and Woods Pond (Reaches 5B, 5C, and 6) contain shallow, clear water with abundant amounts of fish, primarily sunfish, bass, koi, and carp. The linear distance from the WWTP to Woods Pond Dam is approximately 10 km (6 miles). Given that there is sufficient prey available and suitable foraging habitat within this portion of the PSA, it was assumed that ospreys foraging in this area are able to meet their dietary needs exclusively within this section of the river. The assessment of risk to ospreys inhabiting the PSA of the Housatonic River, therefore, focuses on those birds that would forage exclusively within the PSA during the reproductive cycle.

Body Weight (BW)

Female ospreys are generally larger than males, weighing an average of 1.6 kg and 1.4 kg, respectively (Rattner et al. 2001). Poole (1985) studied ospreys in Massachusetts and found that females ranged from 1.7 kg to 1.9 kg in weight during the breeding season, while males were about 1.4 kg. Brown and Amadon (1968) observed body weights of 1.2 kg to 1.9 kg for females and 1.2 kg to 1.6 kg for males in Nova Scotia.

In the Monte Carlo analysis, mean body weight had an arithmetic mean of 1.67 kg and a standard deviation of 0.214. It was assumed that body weights were distributed normally. The uncertainty in this variable is likely due to variability, rather than lack of knowledge or data. Thus, the same distribution was used in the probability bounds analysis for this input variable.

Food Intake Rate (FIR)

The food intake rate of ospreys has not been well characterized. Food ingestion rate data were available in EPA (1993), however this information was not appropriate for use in this exposure model. These data lacked body weight information, lacked statistical analyses, were estimates themselves, and/or were collected from young or captive birds. The field-based measurements of the daily food intake rate of adult male ospreys (0.21 g/g-day, Poole 1983) were similar to the 25th percentile of the modeled food intake rate described below. Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic rate of free-living birds using the following general equation:

$$FMR\ (kJ/day) = a \times BW(g)^{b} \qquad \text{(Eq. 6)}$$

For both the Monte Carlo and the probability bounds analyses, *FMR* for ospreys was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (body weight [*BW*], *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on error statistics derived from regression analysis of the data reported in Nagy et al. (1999), assuming an underlying normal distribution for each. There were insufficient data to generate an allometric equation for Falconiformes, of which ospreys are members, so the equation for Charadriiformes was used. This Order includes many piscivorous birds and was thought to be a suitable surrogate group. For these birds, the slope term *log a* had a mean of 0.928 and a standard error of 0.197 in log units, and the power term *b* had a mean of 0.768 and a standard error of 0.0874 (Nagy et al. 1999). These values differ slightly from those presented in the paper because two bird species were mistakenly not included in the calculations presented by Nagy et al. (1999) (Nagy 2002). The body weight (*BW*) distribution was described above. The results of the calculation were then converted to kcal/kg bw/d.

Food intake rate is derived from *FMR* using the following equation:

$$FIR(kg/kg\ bw/d) = FMR / \sum_{i=1}^{n} (AE_i \times GE_i) \qquad \text{(Eq. 7)}$$

where AE_i is the assimilation efficiency of the *i*th food item (unitless) by ospreys and GE_i is the gross energy in the *i*th food item (kcal/g). For ospreys, mean assimilation efficiency of fish was 79%. The mean gross energy of fish was 1.2 kcal/g wet weight (EPA 1993). The assimilation efficiency and gross energy variables have small coefficients of variation, and are thus treated as point estimates in the exposure analysis.

Proportion of Dietary Items (P_i)

A number of investigations have identified fish as the sole food source for ospreys (Greene et al. 1983; Hughes 1983; Collopy 1984; Van Daele and Van Daele 1982). Chubbs and Trimper (1998) observed that while fish were the primary prey item taken, small mammals (5.0%) and birds (7.5%) were preyed upon as well. Table H.2-11 summarizes the studies reviewed to determine the diet of ospreys.

For this exposure assessment, because of the relatively small contribution in only some studies of prey other than fish, it was assumed that fish account for 100% of the osprey diet. A point estimate was used in the Monte Carlo and probability bounds analyses.

Concentration of tPCBs in Fish in the PSA

A number of fish species were sampled in the Housatonic River PSA, including largemouth bass, yellow perch, sunfish, cyprinids, goldfish, white sucker, bluegill, and brown bullhead. Of the samples collected, 342 were between 13 and 40 cm in length, the preferred size range for ospreys. The Land H-statistic was used to determine the lower 95% and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 109 mg/kg. For the probability bounds analysis, the lower 95% and upper 95% confidence limits determined from the Land H-statistic, 61.6 and 109 mg/kg, respectively, were used to parameterize a distribution-free statement in RiskCalc.

Concentration of TEQ in Fish in the PSA

TEQ concentrations were determined in 176 fish samples collected from the PSA with lengths between 13 and 40 cm. Using the decision criteria outlined in Section 6.4, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations. This data set was used to calculate the parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat non-detected congeners in the TEQ calculation also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the lower 95% and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum value was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 1,415 ng/kg.

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The range, assuming that non-detected congeners were equal to half the detection limit, was determined using the greater of the lower 95% CL and the minimum value and the lower of the upper 95% CL and the maximum value, respectively. The resulting values ranged from 973 to 1,415 ng/kg.

Concentrations of COCs in Prey Items from Other Locations (C_i)

The parameterizations for the remainder of the prey item COC concentration variables from the reference areas are presented in Tables H.2-12 and H.2-13. The decision criteria used to parameterize the concentration variables are described in Section 6.4 and follow the logic illustrated in the preceding paragraphs for tPCBs and TEQ.

H.2.1.2.3 Exposure Model Results

The results of the Monte Carlo and probability bounds analyses for exposure of ospreys to tPCBs and TEQ in the PSA and reference areas are presented in Tables H.2-14 and H.2-15, respectively.

Exposure to tPCBs in the Primary Study Area

The Monte Carlo analysis indicated that exposure of ospreys to tPCBs in the PSA ranges from a minimum of 2.08 to a maximum of 993 mg/kg bw/d. The mean exposure was 57.2 mg/kg bw/d, and the median exposure was 41.7 mg/kg bw/d (Table H.2-14). Of exposure estimates, 80% were between 14.9 and 115 mg/kg bw/d. Figure H.2-9 depicts the cumulative distribution of tPCB intake rates for ospreys in the PSA.

Sensitivity analysis revealed that the *FMR* power term was the most important variable (Pearson correlation coefficient (r_p) = 0.805), followed by *FMR* slope (r_p = 0.562) and body weight (r_p = -0.0448).

The probability bounds estimated for ospreys foraging in the PSA are depicted in Figure H.2-9. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 8.07 and 16.6 mg/kg bw/d. The 50th percentile ranged between 22.9 and 43.8 mg/kg bw/d, and the 90th percentile ranged between 61.8 and 127 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 14.9, the 50th percentile was 41.7, and the 90th percentile was 115 mg/kg bw/d.

Other Exposure Scenarios

Exposure of ospreys to tPCBs in the upstream reference area and Threemile Pond were considerably lower than in the PSA, with mean total daily intakes of 0.108 and 0.0360 mg/kg bw/d, respectively (Table H.2-14). The uncertainty on these exposure distributions, as illustrated by the probability bounds distributions, indicates a slightly narrower degree of uncertainty for the reference areas.

Mean exposures of ospreys to TEQ in the PSA, upstream reference area, and Threemile Pond were 743, 19.2, and 14.4 ng/kg bw/d, respectively (Table H.2-15). Figures H.2-9 to H.2-14

depict the cumulative distributions of tPCB and TEQ total daily intake, as well as the corresponding probability bounds, for ospreys in the three areas. For each exposure scenario, sensitivity analysis revealed that the *FMR* power term and *FMR* slope term were the most important variables in the Monte Carlo analyses (Table H.2-16).

H.3 EFFECTS ASSESSMENT

Dietary and in ovo exposures of birds to tPCBs and 2,3,7,8-TCDD TEQ are reviewed in this section. The goal of this literature review was to identify the studies to be used to characterize the effects that COCs have on representative bird species for the Housatonic River area. The studies were selected according to their quality and relevance to the selected assessment endpoints, i.e., studies that directly address the effects of tPCBs and TEQ on the reproduction, growth, and survival of birds (WESTON 2000). A summary of bird toxicity studies is presented in Table H.3-1.

Acceptability Criteria for Effects Studies in the Literature—Studies on the effects of COCs to wildlife were considered in the development of effects metrics if sufficient information was provided to permit evaluation of the following:

- Study design.
- Study execution.
- Chemical analysis methods.
- Statistical analyses.

Those studies with major issues (e.g., lack of explanation of statistical analyses) were included in the review of the effects literature and following discussion, but were not considered in the derivation of the effects metrics that were used in the risk characterization. Further detail on the decision criteria for selecting effects metrics are provided in Section 6.6.

These decision criteria were applied to each COC-receptor combination. For tPCBs and dioxins/furans, two measures of concentration were considered: tPCBs and 2,3,7,8-TCDD TEQ (TEQ). For tPCBs, the focus was on studies using mixtures with a higher degree of chlorination (i.e., 54 to 60%) because these mixtures most closely resemble the mixtures occurring in the PSA. For TEQ, the toxicity caused by coplanar tPCBs, dioxins, and furans was considered together using the approach described by Van den Berg et al. (1998) (see Sections 6.4 and Appendix C.10 for more detail).

The effects metric units were consistent with the metrics used in the exposure analysis. To the extent possible, durations, exposure route, and other factors were matched between the exposure and effects metrics.

H.3.1 Contaminants of Concern

H.3.1.1 PCB Mixtures

The majority of toxicity studies for birds, particularly the earlier studies, involved exposure to commercial PCB mixtures (e.g., Aroclor 1260 and 1254). The PCB congener pattern detected in the Housatonic River PSA most closely resembles the profiles of the Aroclor 1254 and 1260 mixtures. A summary of the scientific literature with respect to the avian toxicity of tPCBs is presented in this section, with an emphasis on these commercial mixtures. Figure H.3-1 illustrates the range of effects of Aroclors 1254 and 1260 to various avian species.

H.3.1.1.1 Mortality

Review of the literature clearly shows that avian species have differing sensitivities to oral doses of tPCBs. Of the species studied by Heath et al. (1972a), the most sensitive (after oral dosing for 5 days with Aroclor 1254) was the bobwhite quail, with a median lethality response occurring at a dietary PCB concentration of 604 mg/kg. Other species tested, such as the Japanese quail, mallard duck, and ring-necked pheasant, were less sensitive, with oral LC_{50}s of 2,898, 2,699, and 1,091 mg/kg diet, respectively. In a separate study, pheasants fed 210 mg Aroclor 1254 per day by capsule died within an average of 3.8 days; those fed 20 mg/day died within 46 days on average (Dahlgren et al. 1972a). Tissue analysis of the brains of these animals sampled immediately after their death revealed PCB concentrations in the range of 320 to 770 mg/kg ww. Combining this concentration range with the PCB concentrations in the brains of birds sampled just prior to death led the authors to conclude that PCB concentrations of 300 to 400 mg/kg ww in the brain correlated well with PCB-induced mortality.

Bird et al. (1978) noted a significant increase in chick mortality at an Aroclor 1254 dietary concentration of 20 mg/kg and resulting adipose tissue concentrations of 20 mg/kg ww. Platonow et al. (1973) found dietary doses of 500 mg/kg of Aroclor 1254 fed to day-old cockerels caused death to half the birds within 43 days. Their mortality was associated with brain tissue concentrations of 120 mg/kg ww as well as edema, hemorrhaging, and liver and kidney necrosis. Prestt et al. (1970) estimated the median lethal dietary dose rate of Aroclor 1254 to adult Bengalese finches to be 256 mg/kg/d. These birds had brain tissue concentrations of 290 mg/kg ww and experienced enlarged kidneys and hydropericardium. Stickel et al. (1984)

measured the concentrations of tPCBs in the brain tissue associated with the mortality of several species, including grackles, red-winged blackbirds, brown-headed cowbirds, and starlings, and found that brain tissue concentrations of greater than 310 mg/kg ww were associated with an increased likelihood of death due to PCB poisoning.

Different commercial PCB products vary in their toxicity to avian species. In the studies conducted by Heath et al. (1972a), Aroclor 1248 was consistently the least toxic of the three mixtures (Aroclors 1248, 1254, and 1260) tested. Bobwhite quail and ring-necked pheasants were most sensitive to both the Aroclor 1254 and 1260 formulations over the 5-day exposure at median lethal concentrations of 604 and 1,091 mg/kg, respectively, for Aroclor 1254, and 747 and 1,260 mg/kg for Aroclor 1260. Japanese quail and mallard ducks were the least sensitive to all three Aroclors.

H.3.1.1.2 Reproduction

The reproductive impairment of birds caused by tPCBs has been investigated in several species, in dietary and egg injection studies, as well as field studies examining egg and hatchling concentrations and hatching success. The most commonly noted effects to the reproduction of avian species are reduced egg productivity, egg hatchability, and chick growth rates (CCME 1999). Of the species studied, chickens appear to be the most sensitive, followed by pheasants, turkeys, ducks, and herring gulls (Bosveld and Van den Berg 1994). Total PCBs appear to have no adverse effects on total egg weight, eggshell weight, or eggshell thickness (Lillie et al. 1974; Britton and Huston 1973; Scott 1977).

Lillie et al. (1974) reported that a dietary concentration of 20 mg/kg of Aroclors 1242, 1248, and 1254 given to chickens for 9 weeks significantly decreased egg production. Average egg production over the course of the trials decreased from 79% hen day-egg production in controls to 67.5% in the Aroclors 1242 and 1248 treatments, and 71.3% in the Aroclor 1254 treatment. Scott (1977) reported similar results for hen egg production. In this study, 20 mg/kg of Aroclor 1248 in the diet reduced hen day-egg production to 64.8%, compared to 74.5% for controls.

Platonow and Reinhart (1973), Lillie et al. (1974, 1975), and Ax and Hansen (1975) studied the effects of Aroclor 1254 on the hatchability of eggs from white leghorn hens. In each of these studies, birds were orally exposed to tPCBs in their feed in two or more treatments - Platonow

and Reinhart (1973) dosing at a 5 and 50 mg/kg diet for 14 weeks, Lillie et al. (1974) used dietary treatments of 2 and 20 mg/kg for 9 weeks, and Ax and Hansen (1975) maintained birds on diets of 20 mg/kg for ten weeks. Results after the treatment periods showed a significant reduction in reproductive success in each study. The 20 mg/kg diet in Ax and Hansen (1975) increased embryo mortality to an average of 59% over the course of the study and the 50 mg/kg treatment in Platonow and Reinhart (1973) reduced hatchability to near 0%. The 20 mg/kg treatment in Lillie et al. (1974) reduced hatchability to 69% after 9 weeks of exposure, but a subsequent study (Lillie et al. 1975) detected no adverse effects to hatchability at the same treatment level over 8 weeks of exposure. Tumasonis et al. (1973) reported significant reduction in the hatchability of white leghorn eggs (to 34%) after 2 weeks of exposure to drinking water dosed with 50 mg/L Aroclor 1254, and 0% hatchability after 3 weeks. Platonow and Reinhart (1973) tested the same concentration, but in food (50 mg/kg), and saw a similar response. As effects to chickens were noted in three studies (Lillie et al. 1974; Ax and Hansen 1975; Platonow and Reinhart 1973) at dietary concentrations of 20 to 50 mg/kg, the no effects result from exposure to 20 mg/kg diet in Lillie et al. (1975) was disregarded. This study also had a slightly shorter exposure period than the others.

Pheasants fed 12.5 mg Aroclor 1254 weekly by gavage (Dahlgren et al. 1972b) showed a significantly decreased hatching rate of 50% compared to controls in one trial, but insignificant changes in another trial. Nine-month-old mallard ducks were maintained on a diet containing 25 mg/kg Aroclor 1254 from the start of the study to its conclusion (Custer and Heinz 1980). This included an approximate 4-week period from the start of the study to the first egg laying. This treatment resulted in no significant decreases in clutch size, percent fertile eggs, percent hatching of fertile eggs, or percent of ducklings surviving to 3 weeks of age, compared to controls. Heath et al. (1972b) also investigated the effects of Aroclor 1254 fed to mallards at 25 mg/kg, and bobwhite quail at 50 mg/kg. Mallards were maintained on the diet from about 11 weeks before their first laying season to the conclusion of the study after their second year of laying. No significant differences were observed in any of the measures of reproductive success between control and test birds, including total eggs laid and number of 14-day ducklings per hen-season. Bobwhite were maintained on the diet from 11 weeks pre-laying to the end of three months of laying. No significant effects to reproduction were observed in these birds. Male kestrels fed a diet of day-old cockerels containing 33 mg/kg Aroclor 1254 received an estimated dose of 9-10

mg/kg bw/d. These birds experienced significant decreases in sperm concentration over a collection period of 69 days (Bird et al. 1983). Ring doves fed a diet containing 10 mg/kg Aroclor 1254 experienced near-total reproductive failure 6 months later with only 2 of 20 eggs hatching and fledging and 1 pair laying no eggs (Peakall et al. 1972). Cytogenic studies on the embryos revealed increased chromosome aberration rates, suggesting that tPCBs have a clastogenic, or chromosome-breaking, action.

Effects on hatchability in chickens due to Aroclor 1248 were studied by Scott (1977) and Lillie et al. (1974; 1975). Scott noted that hatchability of hen eggs decreased to 50% after 8-week dietary exposures of 10 mg/kg. The effect was more pronounced at higher doses. As feed concentration increased to 20 mg/kg, egg hatchability decreased to 2.4%. Lillie et al. (1974) observed that egg hatchability was reduced to 1.8% at the end of the 9-week exposure to a diet concentration of 20 mg/kg, whereas average egg hatchability for the control group in the 9-week period was 94%. Lillie et al. (1975) expanded treatment range to include 5 and 10 mg/kg Aroclor 1248 in the diet. The 5 mg/kg concentration did not significantly affect hatchability, but the 10 mg/kg treatment reduced hatchability to 50% at the end of the 8-week exposure, while controls had 90% hatchability.

Aroclor 1242 produced results similar to Aroclor 1254 in hen egg hatchability studies. Britton and Huston (1973) observed that the percent hatchability of fertile hen eggs decreased to 75% following a 6-week maternal dietary dose of 10 mg/kg in feed (0.6 mg/kg bw/d, based on a consumption rate of 6% of body weight daily). Diets containing 20 mg/kg and 40 mg/kg reduced hatchability to 50% and 0%, respectively. Significantly reduced hatchability corresponded to egg yolk concentrations ranging from 2.4 to 5.6 mg/kg ww of Aroclor 1242. Ax and Hansen (1975) maintained birds on diets of 20 mg/kg for ten weeks. Results after the treatment periods showed a significant increased embryo mortality to an average of 55% over the course of the study. Lillie et al. (1974) and Lillie et al. (1975) observed significant reductions in hatchability at Aroclor 1242 concentrations of 20 and 10 mg/kg diet, respectively.

Fernie et al. (2001a, 2001b) examined the effects of tPCBs to first- and second-generation American kestrels. First-generation birds were exposed to an Aroclor mixture (1:1:1, Aroclor 1248:1254:1260) in their feed for 100 days until the eggs hatched. This diet resulted in a PCB dose of 7 mg/kg bw/d and whole egg concentrations of 34 µg/g (mg/kg) ww, compared to 0 µg/g

(mg/kg) in the control group. PCB-dosed birds experienced significantly delayed laying and a reduced number of fledglings per breeding pair. As adults, the birds exposed to the PCB mixture in ovo (second generation) were then paired with non-exposed birds for mating. These birds experienced delayed egg laying; had smaller clutches; and produced fewer fertile eggs, hatchlings, and fledglings per breeding pair. Pairs with a PCB-exposed female experienced complete mortality in 25% of broods, whereas pairs with a PCB-exposed male experienced complete mortality in 63% of broods.

H.3.1.1.3 Other Effects

Sublethal effects observed in avian species exposed to tPCBs include increased EROD and AHH activity, liver necrosis, behavioral changes, decreased weight gain, changes in organ weights, and edema (Hoffman et al. 1996b; Eisler and Belisle 1996; Environment Canada 1998). Growth of bird chicks has been investigated by several groups. Rehfeld et al. (1972a) fed one-day-old New Hampshire X white leghorn chickens a diet containing 40 mg/kg (equivalent to a dose of 3.2 mg/kg bw/d) Aroclor 1248 for 35 days and observed significantly reduced growth rates. Also evident were liver enlargement and spleen atrophy. Rehfeld et al. (1972b) examined the growth of chicks exposed over 42 days and found that dietary concentrations of 30 mg/kg significantly reduced body weight gain. Chicks had reduced body weight gains from tPCBs in the maternal diet (Lillie et al. 1974). Feed concentrations of 20-mg/kg (1.2 mg/kg bw/d) Aroclors 1232 and 1242, and 2-mg/kg (0.12 mg/kg bw/d) Aroclors 1254 and 1260 significantly reduced 3-week body weight gains in newly hatched chicks. Heinz et al. (1980) reported significant weight loss (7%) in adult ring doves at an Aroclor 1254 dietary concentration of 100 mg/kg (7.7 mg/kg bw/d). Drouillard et al. (2001) reported no significant effects to kestrel body weights after dosing with an Aroclor mixture (1248:1254:1260, 1:1:1) for 120 days at an estimated dose of 5 to 7 mg/kg bw/d.

Altered behavior has been observed in birds exposed to tPCBs. Second-generation ring doves taken from a first-generation group exposed to a 10 mg/kg diet of Aroclor 1254 were erratic during the incubation of their eggs, and this led to drastic egg temperature fluctuations (Peakall and Peakall 1973). This may explain the reduced hatching and fledging success of third-generation birds from this investigation. Nest-building behavior may also be adversely affected by PCB exposure (McCarty and Secord 1999a). Tree swallows inhabiting locations along the

PCB-contaminated Hudson River, NY, produced nests of poorer quality compared to reference locations. Nests along the Hudson River had significantly lower weights and significantly fewer feathers lining the nests. Because female swallows build most of the nest cup, and males gather most of the feathers for the lining, both sexes appear to be affected by tPCBs. A high frequency of nest abandonment and burying eggs beneath nest material was observed in Hudson River tree swallow nests (McCarty and Secord 1999b) compared to reference locations.

H.3.1.1.4 Field Studies

A number of studies have attempted to correlate PCB contamination in the environment to reproductive success of wild bird colonies, but it is difficult to obtain definitive results because other contaminants are usually present (de Voogt et al. 2001; Summer et al. 1996). A 10-year review of tPCB concentrations in tissues of fish-eating bird species worldwide (Bosveld and Van den Berg 1994) found that eggs from birds in contaminated areas typically had mean tPCB concentrations of less than 20 µg/g ww egg (mg/kg), but that herring gull egg samples had higher mean tPCB concentrations, with some approaching 100 µg/g ww egg (mg/kg). The belted kingfisher had a reported tPCB concentration in adult tissue in excess of 40 µg/g ww (mg/kg) in contaminated areas.

A belted kingfisher reproduction study was performed in the PSA during the 2002 breeding season (Henning 2002). The objective of the study was to evaluate the relationship between reproductive output of kingfishers and exposure of adult and nestling kingfishers to tPCBs. Nine belted kingfisher burrows were monitored during this study, three of which were depredated before the young could fledge. In the remaining six nests, there was an average of 4.8 nestlings, or 87%, that survived from egg to 26 days. When depredated nests were excluded, fledging rates were consistent with the results of other kingfisher studies reported in the literature (Brooks and Davis 1987). Total daily intake of tPCBs was estimated based on prey concentrations and food ingestion rates. No significant relationships were observed between estimated tPCB dose and reproductive output ($p>0.05$). This comparison of hatchling survival to estimated tPCB dose does not necessarily support a conclusion of no adverse effects to the reproductive success of belted kingfishers. The model used to estimate the total daily intake of tPCBs has limited applicability as it was not possible to attain a sufficiently wide dose gradient with the techniques employed by the investigators in this study (see Section H.4.2).

Tree swallows have been widely studied in the field for reproductive effects from tPCBs, due in part to the ability to implement a robust study design with this species. Tree swallows feed on emerging insects (Blancher and McNicol 1991) and thus, PCB residues in chick tissues and eggs of tree swallows may represent trophic transfer of these contaminants from sediment (Fairchild et al. 1992; Bishop et al. 1999). The highest PCB concentrations ever reported in tree swallows in the literature were found in the Hudson River area in New York (McCarty and Secord 1999b). These concentrations reached as high as 77 mg/kg ww in eggs with mean concentrations ranging from 6 to 30 mg/kg ww (Secord et al. 1999). At this location, the hatchability of eggs and total reproduction (fledglings produced per egg laid) were significantly reduced in the study areas in 1994 compared to a 1991 reference location. Data from 1995, however, showed no significant differences in hatchability or total reproduction between study and reference locations.

Other field studies also have shown no significant differences in reproductive success of tree swallows between study and reference locations. Bishop et al. (1999) studied tree swallow reproduction along the Great Lakes and St. Lawrence River with a reference location in Georgian Bay. Total PCB concentrations in the study locations were 2 to 13 times higher than in the reference location; and although EROD activity, vitamin A levels, and porphyrin concentrations were affected, hatching and fledging success were unaffected at study locations compared to the reference area. Custer et al. (1998) observed tree swallows in the Fox River and lower Green Bay region in Wisconsin. Four locations were included in the study, and mean tPCB concentrations in pippers (1 to 2 days post hatch) and nestlings at these locations ranged from 0.05 to 3.85 mg/kg ww. EROD activity showed a positive correlation with PCB concentration, but differences in hatching success at the locations were unrelated to PCB concentrations in pippers or food. Stapleton et al. (2001) investigated DNA mutation rates of tree swallows inhabiting contaminated and uncontaminated locations and found no differences in the rates despite tPCBs in nestlings reaching concentrations as high as 9.95 mg/kg along the upper Hudson River. Further discussion of tree swallow responses in the Housatonic River is presented in Appendix G and in Custer (2002).

Arena et al. (1999) found significantly decreased fledging success and nest attentiveness in starlings at the Crab Orchard National Wildlife Refuge in Illinois compared to reference locations with no known contamination. Soil samples from the study locations contained Aroclor 1254 at concentrations up to 120,000 mg/kg. They observed a strong relationship

between concentrations of Aroclor 1254 in chick carcasses and animal matter taken from their stomachs. Hoffman et al. (1993) noted reduced hatching success in common terns when eggs accumulated tPCBs of 7.6 mg/kg egg ww compared to a reference location with tPCB concentrations of 4.7 mg/kg egg ww. These eggs were also known to contain DDE and mercury. Cormorant egg hatchability in the Netherlands was negatively correlated to PCB concentrations in eggs (Dirksen et al. 1995) where the mean concentration of the sum of six PCB congeners (PCBs 28, 52, 101, 138, 53, and 180) was 21 mg/kg ww in cormorant eggs at the most contaminated location. However, these effects also correlated with DDE concentrations in cormorant eggs. Egg hatchability and fledging success of cormorants were significantly lower in a colony with higher PCB concentrations in yolk sacs (Van den Berg et al. 1994). Individual PCB congeners were measured in this study, and mean concentrations of 11 common congeners in the yolk sacs of unhatched cormorant embryos ranged from 3.5 to 350 mg/kg lipid weight.

H.3.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

A summary of the literature on the avian toxicity of 2,3,7,8-TCDD TEQ is presented in this section. Figure H.3-2 illustrates the range of effects of these contaminants to various avian species.

H.3.1.2.1 Mortality

In single, oral doses of TCDD, bobwhite quail, mallards, and ringed turtledoves were found to have 37-day LD_{50}s of 15,000, 108,000, and 810,000 ng/kg bw (Hudson et al. 1984). Turtledoves had enlarged livers, and bobwhites experienced severe emaciation and high levels of uric acid salts in connective tissues. Seven days after exposure, signs of intoxication began to appear, including excessive drinking, loss of appetite, hypoactivity, emaciation, tremors, convulsions, and others (Hudson et al. 1984).

Schwetz et al. (1973) exposed 3-day-old white leghorn single-comb cockerels to oral doses of 10, 100, 1,000, and 10,000 ng/kg bw/d TCDD for 21 days and chick mortality and weight were recorded. At the highest treatment level, all chicks died within 15 days; and at the 1,000 ng/kg bw/d treatment level, 8 of 10 chicks died by the 21st day. A no observed adverse effect level (NOAEL) of 100 ng/kg bw/d was established for the mortality of these birds.

Ring-necked pheasants treated with a single intraperitoneal injection of TCDD of 6,250, 25,000, or 100,000 ng/kg bw suffered body weight loss and mortality at the two highest treatment levels (Nosek et al. 1992a). Birds given the single 25,000-ng/kg bw dose experienced body weight loss and 80% mortality at the end of 12 weeks, whereas birds given the 6,250-ng/kg bw dose suffered no mortality and slight, but insignificant, weight loss. A second experiment was conducted by these investigators in which ring-necked pheasants were given smaller intraperitoneal doses weekly of 10, 100, or 1,000 ng/kg bw/week for 10 weeks. This translated to daily doses of 1.4, 14, and 140 ng/kg bw/d. No mortality or body weight effects were observed in birds in the two lowest treatment groups, but the 140-ng/kg bw/d treatment group experienced 60% mortality by the 23rd week (13 weeks after the dosing period). The birds in this group also lost weight over the course of the experiment (to 23 weeks), whereas the other groups were approximately 115% of their initial body weight by the 17th week (Nosek et al. 1992a).

H.3.1.2.2 Reproduction

Nosek et al. (1992a) exposed ring-necked pheasant hens to weekly TCDD intraperitoneal injections of 0.01, 0.1, and 1.0 µg/kg bw/week (1.4, 14, and 140 ng/kg bw/d) for 10 weeks. After the exposure period, hens were mated once per week for 7 weeks, and egg production (cumulative eggs per bird) was monitored. The two lower doses caused no significant impairment to egg production. The highest dose, 140 ng/kg bw/d, caused a decrease in cumulative egg production of approximately 70% over 7 weeks. This treatment level also caused significant reductions in hen body weight as well as significant increases in hen mortality. McKinney et al. (1976) noted that a daily dose of 5 µg/kg ww egg 2,3,7,8-TCDF (5,000 ng/kg ww egg TEQ) for 21 days by gastric intubation resulted in complete mortality of 1-day-old white leghorn chickens within an average of 11.5 days.

Egg injection studies of tPCBs have shown that when similar egg concentrations are attained through injection and through conventional maternal dietary doses, the effects to the chicks are also similar (Hoffman et al. 1996b; Nosek et al. 1993). Embryonic uptake of organochlorines from the yolk is similar to contaminants injected into the yolk and to those accumulated naturally (Peakall and Fox 1987). Bioaccumulative environmental contaminants, such as tPCBs, are concentrated in the egg yolks (Tumasonis et al. 1973; Custer et al. 1997). As a result, studies have been conducted examining the effects of injecting environmentally relevant concentrations

of tPCBs into yolks. Brunstrom et al. (1982) have shown that tPCBs injected into egg yolks are capable of distribution throughout the embryo's body, including fat tissue, liver, kidneys, and bone marrow. Ring-necked pheasant hens fed radiolabeled TCDD were found to eliminate approximately 1% of their body burdens into eggs, and all of the chemical was deposited in the yolk, none in the albumin (Nosek et al. 1992b). Drouillard and Norstrom (2001) summarized the literature on maternal transfer of tPCBs to eggs for several avian species. Egg yolk to maternal adipose tissue PCB concentration ratios ranged from 0.27 in ring doves to 1.2 in chickens and pheasants.

The effects of egg injection of TCDD, TCDF, and PCB congeners expressed as TEQ have been reported by several investigators and include decreased egg production and increased embryonic mortality. Powell et al. (1996a) noted that hatchability of white leghorn chicken eggs significantly decreased at a dose of 160-ng/kg egg TCDD injected into egg yolks, and Henshel et al. (1997) estimated the LD_{50} to be 122 ng/kg egg. Cormorant eggs collected from an isolated colony in Manitoba were injected with 4,000-ng/kg egg TCDD into the yolk, and embryonic mortality was significantly higher than in controls (Powell et al. 1997, 1998). Chickens fed diets containing fish collected from a TCDD- and PCB-contaminated location experienced time- and dose-related decreases in egg hatchability (Summer et al. 1996). Total PCB concentrations in the diet ranged from 0.3 to 6.6 mg/kg, expressed as the sum of Aroclors 1242, 1248, 1254, and 1260 (3.3 to 59 ng/kg TEQ). Nosek et al. (1993) estimated a median lethal dose of TCDD to ring-necked pheasants of 2,180 ng/kg ww egg when administered in egg yolks.

Brunstrom and Andersson (1988) injected chicken eggs that had been incubating for 4 days with 3,3',4,4',5-pentachlorobiphenyl (PCB-126) doses ranging from 0 to 2 µg/kg (0 to 200 ng/kg TEQ) egg. They found significant embryonic mortality in the highest dose group (90%) compared to control groups (vehicle only = 15% mortality). Brunstrom (1990, 1991) investigated the effects of other tPCBs injected into egg yolks of chicks and found that PCB-126 was the most toxic congener. Powell et al. (1996a, 1996b) found similar results for the embryotoxicity of PCB-126 to chickens, then broadened their study to include cormorants (Powell et al. 1997). Chicken embryo LD_{50}s were estimated to be 0.6 and 2.3 µg/kg egg (60 and 230 ng/kg egg TEQ) for the two studies, although the latter was deemed more applicable due to less mortality in the vehicle control dose. Cormorant eggs collected from Lake Winnipegosis, Manitoba, were injected with doses of PCB-126 ranging from 0 to 800 µg/kg ww egg (0 to

80,000 ng/kg ww egg TEQ) and incubated for 21 days. This resulted in significant increases in embryo mortality in the 400,000- and 800,000-ng/kg groups (40,000 and 80,000 ng/kg TEQ), to 87% and 100%, respectively (Powell et al. 1997). A second study involving cormorants resulted in an estimated PCB-126 LD_{50} of 177 µg/kg ww egg (17,700 ng/kg ww egg TEQ) after a single injection into the yolk (Powell et al. 1998). Hoffman et al. (1998) compared the lethality of injecting PCB-126 into the air sac of a variety of birds and found that the white leghorn chicken embryo was the most sensitive, with an LD_{50} of 0.4 µg/kg ww egg (40 ng/kg ww egg TEQ). American kestrel and common tern embryos were less sensitive, with LD_{50}s of 65 and 104 µg/kg ww egg (6,500 and 10,400 ng/kg ww egg TEQ), respectively.

Brunstrom (1988) investigated the embryo toxicity of PCB-77 to a number of avian species. Significantly higher embryonic mortality of chicken embryos was associated with egg injections of 5 or 20 µg/kg egg (250 or 1,000 ng/kg egg TEQ), whereas goose and herring gulls experienced no significant increase in mortality at doses as high as 1,000 µg/kg egg (50,000 ng/kg egg TEQ) and ducks were unaffected at 5,000 µg/kg egg (250,000 ng/kg egg TEQ). Wild turkey embryos were found to be 20 to 100 times less sensitive than chicken embryos to the egg yolk injection of PCB-77. This difference in toxicity may be attributed to the developmental stage of the birds. Ah receptors were found in hepatocytes of 7-day-old chicken embryos, but not in liver cells of 9-day-old turkey embryos (Brunstrom and Lund 1988).

H.3.1.2.3 Other Effects

Laboratory experiments with chickens resulted in beak deformities and edema when subject to in ovo injection of PCB-126 at concentrations of 0.9 and 3.2 µg/kg egg (90 and 320 ng/kg egg TEQ) (Powell et al. 1996a, 1996b). Other effects observed were microthalmia (reduced eye size), brain abnormalities, and leg abnormalities. TCDD has been shown to elicit effects to the developing brain in several avian species (Henshel 1998). In controlled experiments, chickens had brain deformities at doses as low as 10 pg/g TCDD (10 ng/kg egg) administered via egg yolk injection, and wild species such as herons and cormorants exhibited brain asymmetry at accumulated TCDD concentrations of 10 and 19 pg/g (ng/kg) egg.

One-day-old white leghorn cockerels were fed a variety of PCB congeners at 400-mg/kg diet for 21 days, or until death. These chicks experienced a significant reduction in weight gain

compared to controls (McKinney et al. 1976) for three of five PCB congeners. The PCB congeners tested were PCBs -128, -136, -153, 155, and -169. Of these, only PCB-169 has been assigned a TEF. The concentration of this congener in terms of TEQ was 0.4-ppm feed (400,000 ng/kg feed TEQ). Nestling kestrels were dosed with PCB-126 to concentrations of 50, 250, and 1,000 ng/g bw/d (5,000, 25,000, and 100,000 ng/kg bw/d TEQ) for 10 days (Hoffman et al. 1996a). From days 4 to 10, there was a significant correlation between PCB dose and decreased body weight. Bone lengths and spleen weight were more sensitive measures in this study. Humerus, radius-ulna, and tibiotarsus bones were all significantly shorter in the 250 and 1,000 ng/g bw/d (25,000 and 100,000 ng/kg bw/d TEQ) test groups than in the controls. Spleen weight showed a marked dose-response decline. Injection of 0.9 µg/kg egg (90 ng/kg egg TEQ) of PCB-126 prior to incubation produced significantly reduced body weights by the second week of exposure and 3 µg/kg egg (150 ng/kg egg TEQ) of PCB-77 reduced body weights compared to controls at 3 weeks (Powell et al. 1996b).

Chick embryo toxicity has been shown to correlate well with 7-ethoxyresorufin-O-deethylase (EROD) inducing potential (Brunstrom et al. 1990). As with mortality, the strongest EROD-inducing effects are the result of exposure to coplanar PCB congeners. The ED_{50} values for EROD induction in chick embryos by the three PCB congeners (3,3',4,4'-tetrachlorobiphenyl, 3,3',4,4',5-pentachlorobiphenyl, and 3,3',4,4',5,5'-hexachlorobiphenyl) were estimated to be 2, 0.1, and 14 mg/kg egg, respectively (Brunstrom and Andersson 1988). In vitro induction of EROD activity in chicken embryo and hatchling hepatocytes showed that chickens became less sensitive as they aged (Bosveld et al. 1997). The 14-day-old embryo was the most sensitive and the 1-day-old hatchling the least. Chickens have been found to be highly sensitive to tPCBs and may be as much as 10 to 1,000 times as sensitive as wildlife species to MFO induction (Kennedy et al. 1996). Tern hatchlings were found to have NOAELs to CYP1A induction from environmentally relevant dietary PCB concentrations of 0.6 ng/g (600 ng/kg TEQ) fish ww (Bosveld et al. 2000).

H.3.1.2.4 Field Studies

Cormorants in the Great Lakes area exhibited a correlation ($r^2 = 0.703$) between egg mortality and bioassay-derived dioxin equivalence (Tillitt et al. 1992). However, Custer et al. (1999) have suggested that TEQ did not have a significant effect on cormorant reproductive success in Green

Bay, despite significant PCB contamination. They cite insignificant relationships between PCB concentrations in eggs and hatchability and suggest, rather, that DDE was primarily responsible for the observations of in situ decline in cormorant reproductive success at this location. Ospreys in the Fraser and Columbia River systems experienced no significant differences in reproductive success, with 2,930 ng/kg lipid TCDD in egg yolks (Elliott et al. 2001) compared to 33.7 ng/kg lipid in egg yolks at a reference location.

Ospreys exposed to planar halogenated hydrocarbons in the Wisconsin River were investigated from 1992 to 1996 (Woodford et al. 1998). Some of their eggs were collected from nests downstream of two bleached-kraft facilities and from two reference locations upstream to measure contaminant concentrations, while the remaining eggs were left to hatch to measure hatching and fledging rates and weight gain. Results showed that embryonic and chick exposure to PCDDs, PCDFs, and coplanar tPCBs did not affect hatching or fledging rates, but that chick growth may have been reduced at TCDD concentrations ranging from 54 to 67 ng/kg ww egg.

Embryo deformities have been linked to in ovo PCB exposure. Live cormorant and tern eggs collected from locations in the Great Lakes were incubated and opened to reveal deformity rates of 11.9% and 15.1% in eggs with elevated PCB concentrations (Ludwig et al. 1996). Deformity rates in cormorants rose from 4% in Lake Winnipegosis to 16% at North Channel Lake Huron where TEQ reached 400,000 ng/kg ww egg as determined by H4IIE bioassay. Bill defects and edema were the most common deformities. A highly significant correlation between EROD induction and TCDD concentrations in eggs was observed in three great blue heron colonies in British Columbia (Bellward et al. 1990). The reproductive success of great blue herons in British Columbia was also investigated (Elliott et al. 1989). At one study location (Crofton), herons failed to raise any young, and the analyzed egg remains showed TCDD concentrations 3 times higher (210 ng/kg ww egg) than the previous year that had a normal fledging rate. DDE has been cited as a confounding influence in field studies of tPCBs and dioxins (De Voogt et al. 2001). Although other factors, such as predation and human disturbances, could have contributed to the reproductive failure, DDE concentrations in eggs decreased in heron eggs over the 2 years at the Crofton location. Clutch sizes were normal in the other locations (Butler 1992), but fledging success was slightly depressed.

H.3.2 Effects Metrics for Characterizing Risk

The preferred approach to assessing the effects of tPCBs or TEQ to representative species in the Housatonic River PSA was to develop dose-response curves from controlled investigations that treated the representative species (i.e., belted kingfishers, ospreys) with at least five dose levels and measured for a suitably sensitive effects on growth, survival, or reproduction. Controlled toxicity studies on the survival, growth, and reproduction of kingfisher and osprey are not available for tPCBs or TEQ. Similar studies on suitable surrogate species were not found in the literature. These studies are difficult to conduct for birds because there are limited numbers of species amenable to controlled experiments. However, for some of the receptor-COC combinations, NOAELs and lowest observed adverse effect levels (LOAELs) for representative and surrogate species were found in the literature and serve as benchmarks. When toxicity data were lacking for representative or surrogate species, threshold ranges were determined. It was assumed that the toxicity thresholds for the representative species lie within these ranges.

H.3.2.1 Total PCBs

Reproduction has been demonstrated to be a sensitive endpoint for birds exposed to tPCBs for extended durations. Although much work was done to investigate the reproductive effects of tPCBs to various species, no suitable studies were available for the representative avian species in this assessment or for species that could be considered reasonable surrogates. Therefore, a dose-response relationship could not be established between exposure to tPCBs (either Aroclor 1254 or 1260) and adverse reproductive effects to piscivorous birds. It was also not possible to establish a NOAEL or LOAEL for reproductive effects for these species from available studies.

No suitable field data were available to develop field-based benchmarks for piscivorous birds (see Section 6.6) as effects metrics for these groups. The next available option was to establish a range of toxic effects spanning the thresholds for the most sensitive and tolerant avian species. White leghorn chickens are the most sensitive avian species to the reproductive effects of tPCBs. Lillie et al. (1974) exposed hens to 2 mg/kg Aroclor 1254 for 63 days in feed. Assuming hens consume 6% of their body weight in feed per day (CCME 1993; Lillie et al. 1974), these birds received a daily PCB dose of approximately 0.12 mg/kg bw/d. At this treatment level, no significant effects were observed on fertility, egg production, shell thickness, or hatchability, but

the growth rate of chicks was slightly reduced. The 3-week weight gain for the control group was 163 g, whereas the treatment group gained 151 g. Platonow and Reinhart (1973) also exposed white leghorn chickens to Aroclor 1254 via feed. The birds were maintained on a diet containing 5 mg/kg Aroclor 1254 for 98 days and, using the same 6% consumption rate, birds received daily doses of approximately 0.3 mg/kg bw/d. This treatment level caused no adverse effects to egg production, egg fertility, or egg hatchability. When the measures of reproductive success were monitored to 273 days (105 days post-exposure), both egg production and fertility decreased significantly. A dose of 0.12 mg/kg bw/d was assumed to be the threshold for highly sensitive avian species exposed to tPCBs. White leghorn chickens are a domesticated species and the genotype of these birds has been influenced by selective breeding. Because of this, it has been suggested that they are likely to be more sensitive to chemical stressors than wild species. A review of the toxicity of diazinon to birds (WHO 1998) compared published acute oral toxicities to different bird species, including mallard ducks, quail, and domestic chickens. The toxicity of diazinon was similar among these birds, but chickens were less sensitive than ducks or quail, indicating that domestication of a species does not necessarily render it overly sensitive to chemical stressors.

In the available studies, the most reproductively tolerant avian species to tPCBs was the American kestrel. First-generation kestrels fed a mixture of Aroclors 1248, 1254, and 1260 (1:1:1) for 100 days (Fernie et al. 2001b) experienced a significant increase in laying lag (20.8 days compared to 14.5 days for controls) at a reported dose of 7 mg/kg bw/d. Other reproductive measures of effect at this dose, including clutch size and the numbers of fertile eggs, hatchlings, and fledglings per breeding pair decreased, but not significantly. Because the effects observed at this dose were minor, 7 mg/kg bw/d was assumed to be the threshold for tolerant avian species exposed to tPCBs. However, this threshold may underestimate effects because reproductive effects were observed in second-generation kestrels; however, there was insufficient information provided that would refine the threshold further.

The threshold range for the reproductive success of piscivorous birds exposed to tPCBs selected for this assessment is 0.12 to 7.0 mg/kg bw/d based on reproductive studies conducted on white leghorn chickens and American kestrels, respectively.

H.3.2.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

As with tPCBs, reproduction is a sensitive endpoint for birds exposed to TEQ for extended durations. The data for TEQ are also limited, and the options of developing dose-response relationships, NOAELs, LOAELs, or field-based thresholds specific to the representative species for choosing effects metrics (Section 6.6) were not available for TEQ for piscivorous birds. The next available option was to establish a range of toxic effects thresholds for the most sensitive and tolerant avian species known and to assume that the thresholds for the representative species are within these bounds.

The toxicological threshold for the effects of TEQ to sensitive birds is based on a study by Nosek et al. (1992a) where ring-necked pheasants were dosed weekly with TCDD intraperitoneally at doses of 0.01, 0.1, and 1.0 µg/kg bw/week for 10 weeks (1.4, 14, and 140 ng/kg bw/d). In this study, body mass, egg production, and egg hatchability in ring-necked pheasants significantly decreased due to exposure to the highest dose. Cumulative egg production over the 7-week exposure decreased by approximately 70%. Given that the duration of this experiment was 10 weeks and it encompassed a critical life stage, it is considered a chronic exposure. This intraperitoneal exposure is thought to be comparable to oral exposures (EPA 1995) and the 1,000-ng/kg bw/week dose over 10 weeks represents a LOAEL of 140 ng/kg bw/d. The corresponding NOAEL from this study was 14 ng/kg bw/d. A dose of 44 ng/kg bw/d TEQ (geometric mean of LOAEL and NOAEL) was assumed to be the threshold for sensitive avian species exposed to TEQ.

Hoffman et al. (1996a) investigated the effects of PCB-126 on American kestrels. Hatchlings were dosed by oral intubation at concentrations of 50, 250, and 1,000 ng/g bw/d (5,000, 25,000, 100,000 ng/kg bw/d TEQ) for 10 days; and body, organ, and skeletal growth were measured at day 10. Growth of the hatchlings, in terms of mass, decreased over the course of the study for the higher dose level treatments, but not significantly. However, skeletal growth, as measured by humerus length, radius-ulna length, and tibiotarsus length, was significantly reduced at treatment levels of 250 ng/g bw/d (25,000 ng/kg bw/d TEQ). Other effects observed in hatchlings at this dose included significantly decreased spleen weight and increased liver weight. The effects observed at a dose of 25,000 ng/kg bw/d TEQ are significant, but did not translate into

significant effects on hatchling success or growth. Therefore, it was assumed that 25,000 ng/kg bw/d TEQ was the reproductive threshold for tolerant avian species exposed to TEQ.

The threshold range for the reproductive success of piscivorous birds exposed to TEQ is 44 to 25,000 ng/kg bw/d, based on reproductive studies conducted on ring-necked pheasants and American kestrels, respectively.

H.4 RISK CHARACTERIZATION

This section characterizes risk to piscivorous birds exposed to tPCBs and 2,3,7,8-TCDD TEQ in the Housatonic River PSA. The risk characterization includes a WOE methodology consistent with that developed by MDEP (Menzie et al. 1996), which is described in detail in Section 2.9 of the ERA.

The risk characterization for piscivorous birds within the PSA relies on two lines of evidence (probabilistic exposure and effects modeling, and a field study of belted kingfisher productivity) to determine the potential ecological risks for this endpoint. The belted kingfisher and osprey were the two representative species selected for this evaluation.

Section H.4.1 focuses on a comparison of probabilistic exposure estimates calculated for the belted kingfisher and osprey (see Section H.2) to relevant effect doses for tPCBs and TEQ presented in Section H.3. Section H.4.2 reviews the findings of the belted kingfisher field study. Section H.4.3 provides an analysis of the relative significance of the lines of evidence and discusses the overall findings of the risk assessment. Section H.4.4 addresses the sources of uncertainty associated with the risk assessment, H.4.5 discusses the extrapolation of this assessment to other piscivorous birds inhabiting the PSA, and H.4.6 presents the conclusions of this ERA.

The purpose of this risk characterization is to address the following risk questions:

- Are the concentrations of tPCBs and TEQ in the prey of piscivorous birds sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?
- If so, how severe are the risks and what are their potential consequences?

H.4.1 Comparison of Estimated Exposures to Laboratory-Derived Effect Doses

For piscivorous birds (i.e., belted kingfisher and osprey), exposure was assessed separately in Reaches 5 and 6 for kingfishers, and in the PSA (Reaches 5 and 6 combined) for ospreys. The following discussion presents an integration of the exposure distributions and effects threshold ranges developed for each species and exposure area. Each scenario was assigned a category of low, intermediate, or high risk using the following guidance:

- If the probability of exceeding the lower toxicity threshold was less than 20%, the risk was considered to be low.
- If the probability of exceeding the upper toxicity threshold was greater than 20%, the risk was considered to be high.
- All other outcomes were considered to have intermediate risk.

This exercise was conducted separately for the results of the Monte Carlo analysis, and the lower and upper bounds from the probability bounds analyses. The "risk category" refers to the level of risk based on the results of the Monte Carlo analysis. The "risk range" refers to the levels of risk based on the results of the probability bounds analyses. (See Section 6.7.2 for a detailed explanation.) The final section in this risk characterization describes important sources of uncertainty in the evaluation of risks of tPCBs and TEQ to piscivorous bird species.

H.4.1.1 Total PCBs Risk Characterization —Belted Kingfisher and Osprey

The belted kingfisher and osprey risk characterizations relied on the same tPCB effects threshold range (Section H.3). The threshold range for the reproductive success of piscivorous birds exposed to tPCBs is 0.12 to 7.0 mg/kg bw/d based on reproductive studies conducted on white leghorn chickens and American kestrels, respectively.

H.4.1.1.1 Belted Kingfisher

The risks of exposure of belted kingfisher to tPCBs in the PSA and reference areas are summarized in Tables H.4-1 and H.4-2. Kingfishers are estimated to be at high risk from tPCBs in the PSA, with a 100% probability of exceeding the upper toxicity threshold in Reaches 5 and 6, as indicated by the Monte Carlo analyses. The risk category and risk range for kingfishers exposed to tPCBs in the PSA are both high. The risk category for kingfishers is estimated to be intermediate in the upstream reference area and low in the Threemile Pond reference area with risk ranges of low-intermediate and low, respectively.

H.4.1.1.2 Osprey

The Monte Carlo predictions for total daily intake of tPCBs by ospreys indicated that there was a 100% probability of exceeding the lower toxicity threshold and a 99% probability of exceeding

the upper toxicity threshold in the PSA (Table H.4-1). The probability bounds analysis indicated that the probability of exceeding the lower toxicity threshold was 100%, despite uncertainties about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the upper threshold ranged from 92 to 100%, given the same sources of uncertainty. Thus, ospreys feeding and reproducing in the PSA of the Housatonic River during the reproductive period are at a high risk of toxicity from exposure to tPCBs. The risk category and risk range for ospreys exposed to tPCBs in the PSA are both high. The risk category and risk range in the upstream reference area are both intermediate and the risk category and risk range in the Threemile Pond reference area are both low (Table H.4-2).

H.4.1.2 TEQ Risk Characterization — Belted Kingfisher and Osprey

The belted kingfisher and osprey risk characterizations relied on the same TEQ effects threshold range (Section H.3). The threshold range for the reproductive success of piscivorous birds exposed to TEQ is 44 to 25,000 ng/kg bw/d based on toxicity studies conducted on ring-necked pheasants and American kestrels, respectively.

H.4.1.2.1 Belted Kingfisher

The risks of exposure of the belted kingfisher to TEQ in the PSA and reference areas are summarized in Tables H.4-1 and H.4-2. Kingfishers are estimated to be at intermediate risk from exposure to TEQ in the PSA, with a 100% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold in Reaches 5 and 6, as indicated by the Monte Carlo analyses. The risk ranges for kingfishers in Reaches 5 and 6 are both intermediate. The risk category for kingfishers is estimated to be intermediate in the upstream reference area, and low in the Threemile Pond reference area with risk ranges of low-intermediate and low, respectively.

H.4.1.2.2 Osprey

The Monte Carlo predictions for total daily intake of TEQ by ospreys indicated that there was a 100% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold (Table H.4-1). The probability bounds analysis indicated that the

probability of exceeding the lower toxicity threshold was 100%, despite uncertainties about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the upper threshold was 0%, given the same sources of uncertainty. Thus, ospreys feeding and reproducing in the PSA of the Housatonic River are categorized as having an intermediate risk of toxicity from exposure to TEQ based on the results of the Monte Carlo analysis. The corresponding risk range is intermediate based on the results of the probability bounds analysis. The risk categories for the upstream reference area and Threemile Pond reference area are both considered to be low, with risk ranges of low.

H.4.2 Belted Kingfisher Field Study

A study of belted kingfisher reproduction in the PSA was performed by GE during the 2002 breeding season. The study objectives were to determine the relationship between tPCB dose and reproductive output. More specifics on this study can be found in Henning (2002). The study is summarized below.

H.4.2.1 Methods

The belted kingfisher reproduction study was conducted in the spring of 2002, to coincide with the breeding season of these birds. Active kingfisher burrows were sought in the river banks and riparian zone of the PSA. Nests were mapped and monitored every 2-3 weeks using the Peeper Probe (pole-mounted video camera) system. Numbers of eggs and nestlings and parental behavior were some of the endpoints monitored. The probe generally afforded a clear view of the nests. The females, however, tended to block the view of the eggs, and thus data on clutch size and hatching success were not collected.

The report states that total daily intake of tPCBs by kingfishers was estimated based on the concentration of COCs in fish and crayfish samples collected within 1,200 m of each burrow and on prey ingestion rates of adults and nestlings obtained from the literature by Henning (2002). Fish and crayfish in the Housatonic River PSA were sampled by the EPA and GE in the fall between 1994 and 2002. Crayfish samples were associated with specific sampling locations, but fish samples were not. When developing the estimated dose, Henning assumed a more precise location of a fish sample than is possible from the information associated with the sample. River

miles designated in the sample IDs in the database were representative of larger areas of the river, roughly corresponding to a segment of Reach 5A also referred to in sampling records as "Shallow Reach," or Reaches 5B and 5C combined ("Deep Reach").

In addition, the kingfisher diet was assumed to be provided entirely by the section of river within the PSA and to be composed of fish (length 4 to 14 cm) and crayfish in the same proportions as are in the data set. Food ingestion rates were estimated as 0.5 g/g-day for adults (Alexander 1977) and 1.375 g/g-day for fledglings (White 1936). Total daily intake for adults and fledglings was calculated by multiplying tPCB concentrations in fish and crayfish by their respective food intake rates.

However, the total daily intake calculation does not result in a dose gradient necessary to evaluate a dose-response relationship for piscivorous birds consuming Housatonic River fish, for two reasons. First, the fish sampling location is known only at the resolution of a range of river miles (i.e., a river reach level); therefore, an actual river mile cannot be assumed for a sample. Second, fish species sampled may be mobile between life stages and seasons and have integrated contaminant concentrations across these areas. These factors contribute to an averaging of the tPCB concentrations in prey used to evaluate exposure by kingfishers and result in a very narrow exposure gradient, with total daily intake for adult birds ranging from 7.4 to 21 mg/kg bw/d in the GE kingfisher field study (Henning 2002). There is also some concern regarding the diet used in the calculation of kingfisher exposure. Studies are available that describe proportions of prey items typically consumed by kingfishers, yet the investigators represented the kingfisher diet as the proportion of fish and crayfish in the database, which has no relationship to the actual proportions of these items in the kingfisher diet.

Statistical analyses were conducted by first defining dependent and independent variables. Nest outcome (successful or depredated), number of nestlings to survive to 26 days and percent to survive to 26 days were dependent variables. Independent variables were estimated PCB dose and nest density. Student t-tests, linear regressions, and multiple linear regressions were used to examine the effect of independent variables on dependent variables using Power and Sample Size for Windows (PASS) (NCSS, Kaysville, UT).

H.4.2.2 Results

Nine belted kingfisher burrows were monitored during this study, three of which were depredated before the fledging date. Three of the six other nests were located outside the PSA, below Woods Pond; however, the birds that were observed foraging in Woods Pond were believed to be the same individuals. For the successful nests, there was an average of 4.8 nestlings, or 87%, that survived to 26 days. If depredated nests were included in the analysis, the average decreased to 3.9 young per nest, or 58% surviving to 26 days. One additional nest was noted by EPA while conducting oversight of the study, but was not observed by the field investigators, and therefore was not included in the study (Woodlot Alternatives, Inc. 2002).

Estimated tPCB doses for adults and young in the PSA were 13 and 35 mg/kg bw/d, respectively. Reported doses ranged from 7.4 to 21 mg/kg bw/d for adults and 20 to 57 mg/kg bw/d for nestlings. The study reported that there were no significant relationships between estimated tPCB dose and any of the endpoints ($p > 0.05$). The range of estimated total daily intakes in the PSA was narrow, however, and provided an insufficient basis on which to evaluate a dose-response relationship. It is unclear how the doses were derived based upon the known precision with which fish locations are recorded in the project database.

Multivariate models also indicated that combined independent variables (e.g., nest density, tPCB dose) provided no significant relationship between stressors and reproductive effects. The results were similar when depredated nests were included in the analyses. The results of this analysis are similarly confounded as in the previously described analyses, because of the same range of daily tPCB intakes in the PSA. Due to the fact that the tPCB dose gradient was very narrow, and the sample sizes were very small (i.e., $n = 6$), EPA does not believe the study can be used to establish conclusively that tPCB exposure did not adversely affect kingfishers.

H.4.2.3 Discussion

The estimated PCB dose exhibited no significant relationship to any of the reproductive measures, including nest outcome and number and percent of young surviving to 26 days. This comparison of hatchling survival to estimated tPCB dose does not necessarily support a conclusion of no adverse effects to the reproductive success of belted kingfishers. The model

used to estimate the total daily intake of tPCBs has limited applicability as it was not possible to attain a sufficiently wide dose gradient with the techniques employed by the investigators in this study, for the reasons stated above.

The lack of data from reference areas for comparison to the observations in the PSA introduces uncertainty in interpretation of this study. Small sample size may also introduce some uncertainty, as only nine nests, six of which fledged young, were observed in the study.

The kingfisher population in the Housatonic River appears to be breeding successfully, with fledging rates that, when depredated nests are excluded, are similar to other kingfisher observations, and with a population density that is consistent with the literature (Brooks and Davis 1987). There is uncertainty associated with this conclusion, as only one study was available in the literature with which to compare fledging rates.

H.4.3 Weight-of-Evidence Analysis

A WOE analysis procedure was used to assess risks of tPCBs and TEQ to piscivorous birds. The goal of this analysis was to determine whether significant risk is posed to piscivorous birds in the Housatonic River PSA as a result of exposure to tPCBs and TEQ. The analysis follows the methodology proposed by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996; see Section 2.9 for details). The analyses were conducted separately for osprey and belted kingfisher, and for tPCBs and TEQ.

H.4.3.1 Evaluating Measurement Endpoints

Measurement endpoints were assigned relative weights for the ten attributes listed in Menzie et al. (1996) that account for strength of association between the assessment and measurement endpoints, data quality, and study design and execution. For this WOE evaluation, it was assumed that the relative importance of each attribute was equal and therefore, endpoint weighting was based on qualitatively evaluating each attribute, and subsequently determining an overall weight for that endpoint. This approach is consistent with the guidance provided by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996). The measurement

endpoint values are summarized in Table H.4-3 for tPCBs and TEQ. The measurement endpoint values for each attribute are discussed in more detail in the following sections.

H.4.3.1.1 Relationship Between Measurement and Assessment Endpoints

Degree of Association

This attribute quantifies the degree of association between each measurement endpoint and the assessment endpoint. In this analysis, the assessment endpoint was the survival, growth, and reproduction of piscivorous birds. The modeled exposure and effects line of evidence for this attribute was given a moderate value for tPCBs and TEQ and the field study was given a high value.

The modeled exposure and effects line of evidence for this attribute was given a moderate value for tPCBs and TEQ, rather than a high value, because the effects metrics used in the risk characterization were from surrogate species, not the representative bird species. The lower effect thresholds are derived from gallinaceous species, which are not ideally suited as surrogates for piscivorous birds. These studies were, however, applicable in deriving a threshold range (i.e., representing possible sensitive species in the PSA). The upper threshold was based on effects to American kestrels. The exposure model inputs were specific to the representative piscivorous bird species, with the exception of the food intake rate calculation for kingfishers, which relied on an equation derived for all bird species available.

The field study investigating reproductive success of belted kingfishers (Henning 2002) measured the assessment endpoint directly. Kingfisher nests were identified in the PSA and the number of fledging birds per nest was monitored. The field study was given a high value for degree of association.

Stressor/Response Correlation

This attribute measures the ability to correlate the magnitude of response with the degree of exposure. For tPCBs and TEQ, the modeled exposure and effects lines of evidence for this

attribute was given a moderate value for both kingfishers and ospreys. The kingfisher field study was given a moderate value as well.

The modeled exposure and effects line of evidence used toxicity threshold ranges as effects metrics. This made it difficult to determine the relationship between magnitude of response and degree of exposure. The effects metrics, however, were specific to tPCBs and TEQ. Correlations between exposure and magnitude of response have been demonstrated in other species at similar or lower concentration ranges in previous studies (Platonow and Reinhart 1973; Britton and Huston 1973; Lillie et al. 1974). The modeled exposure and effects line of evidence was therefore given a moderate value for stressor-response correlation.

The kingfisher field study did not demonstrate a relationship between tPCB exposure and reduced reproductive output, but the design of the study (e.g., lack of reference site, lack of dose gradient, small sample size) likely precluded the finding of significant relationships. The field study was given a moderate value for stressor-response correlation, as it was specific to the COCs of concern, but the study design was likely inadequate for judging stressor-response correlation.

Utility of Measure

This attribute reflects the quality of the accepted criteria, standards, or performance-based measures used to judge environmental harm. For tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute received a moderate value. The belted kingfisher field study received a moderate value for this attribute also.

The techniques used to model exposure and effects are well established and accepted by the scientific community. The method, as applied here, has limited certainty, as the effects metrics used in the risk characterization were toxicity threshold ranges. The threshold ranges for tPCBs and TEQ are expected to bracket the thresholds of kingfishers and ospreys; however, the broad range limits the utility for judging environmental harm. The modeled exposure and effects line of evidence received a moderate value for this attribute.

Fledging success is a well established measure of avian reproductive success. However, the belted kingfisher field study did not include a reference area on small sample sizes, resulting in low statistical power in the study. Studies of the reproductive success of birds usually include clutch size and hatching success among the measurements, but because kingfishers nest in burrows, gathering this information can be difficult, and was not obtained in this study. Of the nests that were not depredated, kingfishers were reproducing at a rate comparable to another study reported in the literature (Brooks and Davis 1987). This conclusion, however, relies on only one study available to compare fledging rates. The Henning (2002) study also attempted to establish a dose gradient based on the birds' nesting sites in the PSA and the COC concentrations in the prey fish collected in the vicinity of the nest. However, the estimated doses are uncertain because of the lack of ability to associate fish samples with the foraging range specified in the study, and averaging of fish exposure to the COCs because of fish mobility. The field study lines of evidence for this attribute received a moderate value.

H.4.3.1.2 Data Quality

This attribute reflects the degree to which data quality objectives (DQOs), QA/QC procedures, and other recognized characteristics of high quality studies are met for each line of evidence. For tPCBs and TEQ, the measurement endpoints for modeled exposure and effects and the field study lines of evidence were given scores of moderate/high and moderate, respectively.

The DQOs established for the sampling and analysis of tissue samples were met for the tissue residue data used in the exposure analyses for both tPCBs and TEQ. Other model parameters were derived from EPA (1993) and other published journal articles, and the effects metrics were derived from peer reviewed scientific literature. A value of moderate/high was given for the modeled exposure and effects line of evidence.

Although the field study appeared to follow accepted protocols cited in the study (EPA was not provided with an opportunity to review standard operating procedures or protocols prior to receiving the study report), there were a few concerns with the data generated in study. Few samples were included in the study as only nine nests were found, of which only six fledged young. A tenth nest was noted during EPA oversight, but was not included in the study. Clutch size data were not available as adult birds blocked the view of the clutches in the burrows. The

study did not include a reference site and few other studies were available in the literature with which to compare reproductive success. The calculation of dose for birds in each nest is of questionable value, as limitations to the approach produced a narrow dose gradient. The field study received a moderate value for this attribute.

H.4.3.1.3 Study Design

Site Specificity

This attribute reflects the site specificity of data, media, species, environmental conditions, and habitat for each measurement endpoint. For tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute received a low/moderate value. The field study received a moderate/high value for this attribute.

The low/moderate value for the modeled exposure and effects line of evidence was chosen because prey tissues were collected from the site, but other model inputs (e.g., body weight, free metabolic rate) did not reflect the site-specific environmental conditions of the PSA. Sensitivity analyses of the exposure models identified the free metabolic rate power and slope terms as the most important variables to the exposure probability distributions. These variables were derived from birds other than the representative species, and in different locations. The effects studies used to develop the threshold ranges were conducted in laboratories at various sites.

The belted kingfisher field study was conducted for the most part within the PSA, however, some nests were downstream of Woods Pond, outside of the PSA. While the birds inhabiting these nests were observed foraging in Woods Pond, they also may have foraged downstream of the Woods Pond Dam, where lower concentrations of contaminants were measured, and concentrations of COCs in prey are likely lower. No reference areas were included in the study. This study received a moderate/high value for this attribute.

Sensitivity

This attribute refers to the ability of the measurement endpoint to detect changes due to a stressor against a background of natural variability and other sources of uncertainty. For both tPCBs and

TEQ, the modeled exposure and effects line of evidence for this attribute was given a low/moderate value. The field study was given a low/moderate value.

The modeled exposure and effects line of evidence directly assessed the exposure that ospreys and kingfishers would receive in the PSA, but suitable effects studies for these species were not available, so a threshold range spanning sensitive to tolerant birds was used. The only difference the model is able to detect with some degree of certainty is the probability of surpassing the low or high effect threshold. Given the breadth of the probability bounds and the separation between the thresholds, a very large difference in total daily intake is necessary to detect this difference. This line of evidence was therefore given a low/moderate value for this attribute.

Field studies are rarely able to detect differences that are twofold or less. This particular study had limited ability to detect differences in reproductive success as there was no reference area with which to compare the measure of success and the estimated dose gradient was insufficiently broad to detect effects within the PSA. For these reasons, a low/moderate value was assigned to the field study.

Spatial Representativeness

This attribute reflects the degree of spatial overlap of study area, measurement locations, locations of stressors, locations of representative species, and points of potential exposure to those stressors. For both tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute was given a moderate value. The belted kingfisher field study received a moderate/high value for this attribute.

The modeled exposures relied on COC concentrations in prey tissues collected from the Housatonic River area. However, none of the measures of spatial representativeness applied to the other model inputs. The toxicity studies used to derive the effects metrics all had overlap of four of the five criteria (i.e., not the study area). This line of evidence was given a moderate value for this attribute.

The belted kingfisher field study was given a moderate/high value for this attribute, as sampling locations were included throughout the study area, and included relevant stressors, representative

species and points of exposure. Some of the sampling locations were located outside of the PSA, with kingfisher nests south of Woods Pond Dam included in the study.

Temporal Representativeness

This attribute describes the degree of coincidence in time between data collection and exposure. For both tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute was given a moderate value. The belted kingfisher field study received a moderate value for this attribute also.

The tissue samples used to parameterize the model were collected in multiple sampling events, but these did not completely overlap with the reproductive cycle of piscivorous birds in the Housatonic River area. Data for several other model parameters, such as body weight, may or may not have been collected during the birds' breeding seasons. The effects studies selected for the modeled exposure and effects line of evidence spanned the reproductive cycles of the bird species studied. This line of evidence was therefore given a moderate value.

The kingfisher field study was conducted during the species' reproductive cycle, but as the study measured only one reproductive cycle, it was considered a single sampling event. Moderate variability would be expected in the reproductive success of kingfishers over time. The field study was thus given a moderate value for this attribute.

Quantitative Measure

This attribute indicates the degree to which the response can be quantified by a given measurement endpoint. For both tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute was given a moderate/high value. The belted kingfisher field study received a moderate value for this attribute.

The results from the modeled exposure were probabilistic and highly quantitative and propagated uncertainty associated with the modeling procedures. However, the use of effects threshold ranges as the effects metrics impaired the ability to quantify a response to the stressors. The results of the comparison of modeled exposure with effects thresholds are quantitative, in that the probabilities of exceeding the thresholds were determined. The use of thresholds, however,

means that magnitude of effect could not be assessed, as would be the case with dose-response curves. The biological significance of exceeding a threshold is unknown. This line of evidence was given a moderate/high value for this attribute.

The field study results are quantitative, however, the dose gradient was too narrow to detect a dose-response relationship. In addition, the sample size was very small (i.e., n=6) for the statistics used (i.e., t-test and regression). Student's t-tests were apparently performed even though there were no reference sites for comparison. The field study was therefore given a moderate value.

Standard Method

This attribute quantified the extent to which measurement endpoint studies adhered to recognized scientific protocols. For both tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute was given a moderate value. The belted kingfisher field study received a moderate/high value for this attribute.

For the modeled exposure and effects line of evidence, generally accepted methods and guidance were used and were applicable for the endpoint and site. Probability bounds analysis, however, is a relatively new technique for propagating uncertainty, and the use of threshold ranges for the effects metrics is not ideal. This line of evidence was given a moderate value for this attribute.

The field study protocol was not available for review by EPA prior to the study, and the methods provided after the completion of the study revealed some issues, such as:

- Lack of reference areas for the kingfisher study.
- Lack of clutch size data.
- Lack of nest-specific fish concentrations for estimating TDI in the kingfisher study.

Despite these limitations, the study appeared to follow standard field protocols. This attribute was given a moderate/high value.

Total Value

The total value assigned to each measurement endpoint is an average of the values from the attributes (Table H.4-3). For tPCBs and TEQ, the modeled exposure and effects line of evidence for ospreys and kingfishers was given a moderate value and the belted kingfisher field study line of evidence was given a moderate/high value.

H.4.3.2 Magnitude of Response

The measurement endpoint value was used together with the magnitude of response in the measurement endpoint to judge the WOE for adverse effects on representative species. The magnitude of response can be presented as qualitative or quantitative measures. We use qualitative measures here.

The total value, evidence of harm, and magnitude of responses are presented in Table H.4-4 for tPCBs and Table H.4-5 for TEQ.

The modeled exposure and effects lines of evidence indicated a significant potential that exposure to tPCBs and TEQ would cause thresholds for sensitive piscivorous bird species to be exceeded in the PSA. The analyses further indicated that thresholds for even tolerant species could be exceeded in the PSA. The risk estimates for TEQ, however, were more uncertain than for tPCBs. Risks to piscivorous birds in reference areas from exposure to tPCBs and TEQ were low.

The results of the belted kingfisher field study, conversely, provided no evidence of adverse effects to the reproductive success of these birds in the PSA. The conclusions drawn from this study are not strongly supported by the statistical analyses of the results because of small sample sizes and lack of a suitable reference site, but the nests that were monitored showed that these birds are reproducing in the PSA.

H.4.3.3 Concurrence Among Measurement Endpoints

The final component in the WOE approach examines the agreement, logical connections, interdependencies, and correlations among the measurement endpoints as they relate to each

assessment endpoint. The methodology for representing concurrence involves the use of a graphical method where measurement endpoints are plotted on a matrix that also includes the weight of each endpoint and degree of response.

Table H.4-6 depicts the outcome for belted kingfishers for tPCBs and Table H.4-7 for TEQ. The results from the modeled exposure and effects lines of evidence suggests that tPCBs and TEQ pose a risk to belted kingfishers inhabiting the PSA of the Housatonic River. The belted kingfisher field study, however, suggests that the reproductive success of these birds may not be impaired by tPCBs and TEQ in the PSA. The uncertainty in the modeled exposure and effects line of evidence, outlined below, may mean that this approach overestimated the risks of tPCBs and TEQ to piscivorous birds in the PSA. The WOE assessment therefore supports a conclusion of low risk to belted kingfishers exposed to tPCBs and TEQ in the PSA. This conclusion, however, is highly uncertain because of the conflicting results in the WOE assessment.

The weight-of-evidence analysis for ospreys supports a conclusion of high risk for these birds from exposure to tPCBs and TEQ in the PSA (Tables H.4-6 and H.4-7). There has been no reproduction by ospreys in the PSA in recent years. The habitat is suitable to support ospreys, there is sufficient prey available, and they have been observed foraging in the area, so the lack of breeding in the PSA in recent years may be of concern. The physiological differences between kingfishers and ospreys preclude extrapolation of the results of the kingfisher field study to ospreys. For these reasons, the WOE assessment supports a conclusion of high risk for ospreys exposed to tPCBs and TEQ in the PSA. This conclusion, however, is based on a single line of evidence (modeled exposure and effects) and is therefore uncertain.

H.4.4 Sources of Uncertainty

The assessment of risk to piscivorous birds contains uncertainties. Each source of uncertainty can influence the estimates of risk, therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to piscivorous birds are described below.

- In this assessment, it was assumed that dietary exposure represented the most important pathway for exposure of piscivorous birds to COCs. Although unlikely to provide a major contribution to the risk, other pathways could increase the exposure

and perhaps increase risk slightly (Moore et al. 1999). Deterministic calculations were conducted in which estimates of exposure to COCs via drinking water and inhalation were included in the exposure model. Inclusion of these routes did not substantially increase overall exposure of piscivorous birds to the COCs.

- The Monte Carlo sensitivity analyses suggested that the free metabolic rate (FMR) slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. However, no suitable direct measurements of free metabolic rate are available for the representative wildlife species. Similarly, suitable measured food intake rates are not available for the free-living belted kingfisher and osprey. Therefore, free metabolic rates were estimated using allometric equations. The use of allometric equations introduces some degree of uncertainty into the exposure estimates because they are subject to model-fitting error, and are based on species different from the representative species used in this assessment. For example, there were insufficient data to generate an allometric equation for coraciiformes, of which belted kingfishers are members, so the equation for all birds was used. Given the lack of empirical data on species specific to this assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as input for the allometric slope and power terms.

- The free metabolic rate equations require body weight as an input variable. Data on the body weights of individuals in the PSA were not available. As a result, body weights of the representative species were estimated from the literature. Body weights of ospreys and kingfishers are generally well documented and typically have little variation among mature adults. The potential magnitude and direction of the uncertainty associated with lack of site-specific information on body weight are unknown, but are likely small.

- Because no stomach contents or other dietary analyses are available for the representative species in the PSA, dietary compositions were derived from those reported in the literature for birds collected from other geographical locations. Some potential dietary items were not included in the exposure analyses because it was anticipated that these items would be consumed on a limited basis, if at all, in the PSA. Ospreys and kingfishers, for example, have been reported to occasionally prey on reptiles and amphibians. These items have not been included in the diets of these birds for this assessment because of the abundance of fish available in the PSA. The potential magnitude and direction of the uncertainty associated with lack of information on diet are unknown. The uncertainty due to lack of knowledge of the diet of these birds in the PSA was partially addressed by using distributions to represent variability in diet.

- Sample sizes were limited for the analyses of COC concentrations in some prey items, specifically, crayfish. To address this uncertainty in the Monte Carlo analysis, the UCL or data set maximum (see Section 6.4 and Appendix C.5) was used as an estimate of COC concentrations in prey items. The potential magnitude of the uncertainty associated with small sample sizes for COC concentrations is unknown,

but this approach would likely overestimate exposure. The probability bounds analysis used an unbiased approach (e.g., distribution free range from LCL to UCL) to deal with sample size uncertainty.

- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). As a result, decision criteria were developed (see Section 6.4 of the ERA) for co-eluted congeners to determine TEQ concentrations used as distribution parameters in the Monte Carlo and probability bounds analyses. These criteria were designed to explicitly incorporate this source of uncertainty in the probabilistic analyses. Thus, this source of uncertainty has been incorporated in this risk assessment.

- The foraging time of belted kingfishers and ospreys in the PSA was assumed to be 100%. This assumption is valid for many kingfishers because they have small foraging ranges. However, the maximum foraging radius of ospreys is roughly the length of the PSA. The availability of prey fish and ample suitable foraging area within the PSA suggest that ospreys in the area could forage exclusively within the PSA. These birds could, however, also forage beyond the PSA, thus reducing exposure to COCs.

- The greatest source of uncertainty of the effects assessment was associated with the lack of toxicity studies involving the representative species. There were no toxicity studies available for belted kingfishers or ospreys exposed to tPCBs or TEQ. As a result, laboratory studies involving other species were used to estimate effects to piscivorous birds. This extrapolation introduced uncertainty in the effects assessment because of the variations in physiological and biochemical factors such as uptake, metabolism, and disposition that can alter the potential toxicity of a contaminant. The sensitivity of birds to an environmental contaminant may differ from that of a laboratory or domestic species due to behavioral and ecological parameters including stress factors (e.g., competition, seasonal changes in temperature or food availability), disease, and exposure to other contaminants. Inbred laboratory animal strains may also have an unusual sensitivity or resistance to a tested substance. To address uncertainty in the effects assessment, a threshold range was used in which effects to tolerant and sensitive species were considered. It is assumed that the toxicity thresholds for the representative species lie within these ranges.

- The effects metrics used to estimate risk to representative species considered Aroclor 1254 and 1260 mixtures. Some uncertainty is inherent in extrapolating from those mixtures to the specific congener patterns observed in the PSA of the Housatonic River. The use of the TEQ approach takes the specific congener profile of the PSA into account because individual PCB congeners are included in the exposure model rather than tPCB concentrations.

- The belted kingfisher field study methods generally followed accepted protocols, but there were several shortcomings. For example, there was no reference site, no information was provided regarding nest search intensity, the researchers were unable to determine clutch size, and there were too few visits to the nest during the

reproductive cycle. These shortcomings limit the ability to draw rigorous conclusions.

- The statistics used in the belted kingfisher field study were not clearly stated. Student t-tests were apparently performed even though there were no reference sites with which comparisons could be made. A power analysis of the results would have been useful. The sample sizes were very small (i.e., n=6) for the statistics used (i.e., t-test and regression).

- The approach used to estimate dose in the belted kingfisher field study had a number of shortcomings. The investigators assumed a foraging radius of 1,200 m and attempted to identify prey samples within this radius of each burrow. The fish samples had a "river mile" location associated with each sample, but this is a coarse measure that does not allow location of the sample with sufficient precision to assign specific fish to a specific 1,200-m foraging radius. Fish are also mobile within the PSA, meaning that they receive their total exposure from many areas of the river and that concentrations in fish do not vary substantially within the PSA. As a result, the dose gradient achieved by this approach is likely too narrow to detect a significant dose-response relationship.

- The belted kingfisher field study results do not definitively support the conclusions of low risk because the data are limited. There are several conclusions drawn by the authors that are not strongly supported by the information presented in the report. The conclusion that the kingfisher population is consistent with the quality of habitat present is speculative. Survival to 26 days and densities were compared with the results from only one study (Brooks and Davis 1987). It is inappropriate to conclude that the Housatonic River kingfishers fall within the range reported for other kingfisher populations when only one study is referenced. While the GE study provides no evidence of impaired reproduction or population density attributable to PCBs, it fails to acknowledge the limitations associated with the use of only one metric to evaluate reproduction.

H.4.5 Extrapolation to Other Species

The belted kingfisher and osprey were chosen to represent piscivorous birds inhabiting the Housatonic River area. The belted kingfisher and osprey are the only birds in this group that are common to the area. Other piscivorous birds (e.g., pied-billed grebe, great blue heron) are addressed in Appendix K.

H.4.6 Conclusions

The WOE analysis indicated that exposure of piscivorous birds, such as the belted kingfisher and osprey, to tPCBs and TEQ in the PSA could lead to adverse reproductive effects in some species.

The lines of evidence used to support this conclusion were the comparison of modeled exposure with effects to piscivorous birds and the field study of the reproductive success of belted kingfishers in the PSA.

For the modeled exposure and effects line of evidence, the effects characterization employed a toxicity threshold range to describe the potential effects of tPCBs and TEQ to piscivorous birds. The most sensitive and most tolerant bird species were used to develop the toxicity threshold ranges, with the assumption that representative piscivorous bird species would begin to experience adverse effects in this range.

The most tolerant bird species to tPCBs found in the literature was the American kestrel, which experienced only minor effects after being dosed with 7.0 mg/kg bw/d for 100 days (Fernie et al. 2001b). The modeled exposure results indicated that each of the representative piscivorous bird species are likely to have a daily intake greater than 7.0 mg/kg bw/d. This means that piscivorous birds in the PSA are likely to receive a tPCB dose that is greater than what the least sensitive species known can tolerate.

For TEQ, the risk picture is less clear because the threshold range for this COC is very wide (44 to 25,000 ng/kg bw/d). Exposure estimates for representative piscivorous bird species in the PSA fell within this range. Thus, without effects data specific to kingfishers and ospreys, it is difficult to make definitive conclusions about the risks of TEQ to these species.

A belted kingfisher reproduction study was undertaken in the PSA during the 2002 breeding season Henning (2002). The study evaluated the relationship between tPCB dose and reproductive output. Nine belted kingfisher burrows were monitored during this study, three of which were depredated before the young could fledge. For the six successful nests, there was an average of 4.8 nestlings, or 87%, that survived to 26 days. When depredated nests were excluded, fledging rates are consistent with the only other kingfisher study reported in the literature (Brooks and Davis 1987). Total daily intake of tPCBs was estimated based on prey concentrations and food ingestion rates. No significant relationships were observed between estimated tPCB dose and reproductive output ($p>0.05$), although this analysis did not truly provide the dose gradient necessary to evaluate a dose-response relationship for piscivorous birds

consuming Housatonic River fish. No reference areas were included in the study to compare kingfisher productivity in contaminated and uncontaminated sites.

For the assessment of risks to kingfishers, both lines of evidence were integrated. The modeled exposure and effects line of evidence indicated that kingfishers in the PSA are likely to receive a tPCB dose greater than what the most tolerant species known can endure. For TEQ, the risk picture is less clear because the threshold range for this COC is very wide and the exposure estimates for kingfishers fell within this range. Thus, without effects data specific to kingfishers, it is difficult to make definitive conclusions about the risks of TEQ to this species. The field study of kingfisher productivity, however, indicated that these birds are able to reproduce in the PSA. This line of evidence was given a higher weighting than the exposure and effects modeling, despite concerns about the study. Therefore, kingfishers are considered to be at low risk in the PSA as a result of exposure to tPCBs and TEQ. The conclusion of low risk to kingfishers is uncertain because the two lines of evidence did not give concordant results.

For ospreys, only the modeled exposure and effects line of evidence was available to assess risk to these birds. As with kingfishers, this line of evidence indicated that ospreys in the PSA are likely to receive a tPCB dose that is greater than what the most tolerant species known can bear. The risks due to exposure to TEQ are unclear, as the estimates for exposure also fell within toxicity threshold range. Ospreys, however, lack a site-specific study that investigates the effects of COCs in the PSA. The PSA contains suitable habitat for ospreys with abundant prey, raising the possibility that they are not resident in the area because of contaminants. Ospreys are therefore considered to be at risk in the PSA as a result of exposure to tPCBs and TEQ.

ERA Summary

The weight-of-evidence analysis suggests that belted kingfishers may be at low risk as a result of exposure to tPCBs and TEQ in the Housatonic River PSA. Although modeled exposure and effects indicated high risk for tPCBs and intermediate risk for TEQ, a field study of kingfisher productivity indicated that the birds were reproducing in the PSA. The conclusion of low risk to kingfishers is highly uncertain because the two lines of evidence did not give concordant results.

Ospreys may be at high risk from exposure to tPCBs and intermediate risk from exposure to TEQ in the Housatonic River PSA. In the PSA, exposure of piscivorous birds to tPCBs is greater than concentrations that caused adverse effects in the most tolerant species studied. The conclusion of high risk to ospreys is uncertain because only one line of evidence was available.

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APPENDIX H
ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS BIRDS

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Table H.2-1

Input Variables Used in the Monte Carlo Exposure Analysis for Total Daily Intake of tPCBs and TEQ by Belted Kingfishers Inhabiting the Housatonic River Area

Variable	Distribution	Parameters
Body weight (*BW*; g)	Normal	0 = 150; s = 14.7
Free metabolic rate (*FMR*; kcal/kg bw/d) *a* = FMR slope *b* = FMR power	$FMR = a\ BW^b$ Normal Normal	 $log_{10}\ a$: 0 = 1.02; s = 0.0393 0 = 0.681; s = 0.0182
Gross energy (*GE*; kcal/kg)		
Fish	Point Estimate	1,200
Crayfish	Point Estimate	1,100
Assimilation efficiency (*AE*; unitless)		
Fish	Point Estimate	0.79
Crayfish	Point Estimate	0.77
Proportion fish in diet R5 and UR (P_f; unitless)	Beta	α = 55; β = 9; scale = 1
Proportion fish in diet R6 and 3M (P_f; unitless)	Point Estimate	1.0
Proportion crayfish in diet R5 and UR (P_c; unitless)	Beta	$1\text{-}P_f$
Proportion forage time in PSA (*PT*; unitless)	Point Estimate	1.0

R5 = Reach 5; R6 = Reach 6; UR = Upstream Reference; 3M = Threemile Pond

Table H.2-2

Input Variables Used in the RiskCalc Probability Bounds Analysis for Total Daily Intake of tPCBs and TEQ by Belted Kingfishers Inhabiting the Housatonic River Area

Variable	Distribution	Parameters
Body weight (*BW*; g)	Normal	0 = 150; s = 14.7
Free metabolic rate (FMR; kcal/kg bw/d) *a* = FMR slope *b* = FMR power	$FMR = a\ BW^b$ Normal Normal	 $log_{10}\ a$: 0 = 1.02; s = 0.0393 0 = 0.681; s = 0.0182
Gross energy (*GE*; kcal/kg)		
Fish	Point Estimate	1,200
Crayfish	Point Estimate	1,100
Assimilation efficiency (*AE*; unitless)		
Fish	Point Estimate	0.79
Crayfish	Point Estimate	0.77
Proportion fish in diet R5 and UR (P_f; unitless)	Minmaxmean	0.697, 0.967, 0.859
Proportion fish in diet R6 and 3M (P_f; unitless)	Point Estimate	1.0
Proportion crayfish in diet R5 and UR (P_cH3; unitless)	Minmaxmean	1-P_f
Proportion forage time in PSA (*PT*; unitless)	Point Estimate	1.0

R5 = Reach 5; R6 = Reach 6; UR = Upstream Reference; 3M = Threemile Pond

Table H.2-3

Diet of the Belted Kingfisher

	Summer	% Fish	% Crust	% Insects	% Amph	Location	Reference
Salmonids	25					Maritime Provinces, Canada	Cairns 1998
Freshwater fish	30.8						
Estuarine fish	35.4						
Crustaceans	7.6						
Insects	1						
Percent Composition		91.2	7.6	1	0		
Trout	17					Lower Michigan/lake, % wet weight; stomach contents	Alexander 1977
Non-trout fish	29						
Crustaceans	5						
Insects	19						
Amphibians	27						
Birds and mammals	1						
Unidentified	2						
Percent Composition		46	5	19	27		
Trout	80					Lower Michigan/stream, % wet weight; stomach contents	Alexander 1977
Non-trout fish	6						
Crustaceans	2						
Insects	3						
Amphibians	9						
Percent Composition		86	2	3	9		

Table H.2-3

Diet of the Belted Kingfisher
(Continued)

	Summer	% Fish	% Crust	% Insects	% Amph	Location	Reference
Trout	29					Lower Michigan/river, % wet weight; stomach contents	Alexander 1977
Non-trout fish	32						
Unidentified fish	2						
Crustacea	17						
Insects	3						
Amphibians	13						
Vegetation	1						
Unidentified	3						
Percent Composition		63	17	3	13		
Trout	30					Michigan/trout streams, % wet volume/stomach contents	Salyer and Lagler 1946 in EPA 1993
Other game and pan fish (perch, centrachids)	13						
Forage fish (e.g., minnows, sticklebacks, sculpins)	15						
Unidentified fish	1						
Crayfish	41						
Percent Composition		59	41	0	0		

Table H.2-3

Diet of the Belted Kingfisher
(Continued)

	Summer	% Fish	% Crust	% Insects	% Amph	Location	Reference
Game and pan fish (mostly perch)	17.5					Michigan/lakes, streams, % wet volume/stomach contents	Salyer and Lagler 1946 in EPA 1993
Forage fish (minnows, sticklebacks, sculpins, etc.)	49.1						
Other fish	2						
Fish remains	0.9						
Frogs	2.3						
Crayfish	7.4						
Insects	21						
Percent Composition		69	7	21	2		
Game and pan fish (perch, centrachids)	10.15					Michigan/non-trout streams, % wet volume/stomach contents	Salyer and Lagler 1946 in EPA 1993
Forage fish (minnows, sticklebacks, etc.)	31.3						
Other fish	16.2						
Fish remains	0.1						
Crayfish	39.6						
Insects	2.2						
Percent Composition		58	40	2	0		
Salmon	20.1					Maritime Provinces, Canada, small salmon streams	White 1953 in EPA 1993
Trout	6						
Suckers	9.7						
Minnows	40.4						
Sticklebacks	12.7						
Other fish	9.7						
Insects	1.3						
Percent Composition		98.7	0	1.3	0		

Table H.2-3

Diet of the Belted Kingfisher
(Continued)

	Summer	% Fish	% Crust	% Insects	% Amph	Location	Reference
Salmon	24					Maritime Provinces, Canada, large salmon rivers	White 1953 in EPA 1993
Trout	7						
Suckers	20						
Minnows	24						
Sticklebacks	8						
Insects	4						
Percent Composition		96	0	4	0		
Salmon fry	7					Nova Scotia/river; % of total number of prey; stomach contents	White 1936 in EPA 1993
Salmon-1 yr old	58						
Trout	4						
Sticklebacks	27						
Suckers	4						
Percent Composition		100	0	0	0		
Salmon fry	11					Nova Scotia/riparian-streams, % of total number of prey; fecal pellets	White 1936 in EPA 1993
Salmon-1 yr old	42						
Salmon -2 yr old	1						
Trout	15						
Sticklebacks	30						
Killifish	1						
Percent Composition		100	0	0	0		

Table H.2-3

Diet of the Belted Kingfisher
(Continued)

	Summer	% Fish	% Crust	% Insects	% Amph	Location	Reference
Crayfish	13					Southwest Ohio/creek, % of total number of prey brought to nestlings	Davis 1982 in EPA 1993
Cyprinids	77						
Minnows	13						
Stonerollers	38						
Unidentified	26						
Other fish	10						
Percent Composition		87	13	0	0		
Fish: yellow perch, common shiner, short-nosed dace, horny-head chub, and mud minnow	100					Minnesota	Cornwell 1963
Percent Composition		100	0	0	0		

Table H.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Belted Kingfishers

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Fish	Reach 5	121	2.59	25.6	29.0	36.0	99.3	UCL > Max: No	36.4	Distribution-Free Range Min=30.5 Max=36.4
Fish	Reach 6	95	8.80	22.0	27.0	32.0	99.8	UCL > Max: No	30.4	Distribution-Free Range Min=26.0 Max=30.4
Fish	Upstream Reference Area	42	0.0762	0.148	0.161	0.203	2.36	UCL > Max: No	0.249	Distribution-Free Range Min=0.183 Max=0.249
Fish	Threemile Pond	22	0.0161	0.0210	0.0244	0.0303	0.0628	UCL > Max: No	0.0330	Distribution-Free Range Min=0.0250 Max=0.0330
Crayfish	Reach 5	40	2.59	6.63	8.15	14.8	52.1	UCL > Max: No	14.8	Distribution-Free Range Min=10.1 Max=14.8
Crayfish	Upstream Reference Area	10	0.0246	0.0274	0.0391	0.519	0.0664	UCL > Max: No	0.0530	Distribution-Free Range Min=0.0339 Max=0.0530

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

Table H.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Belted Kingfishers

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Fish	Reach 5	46	78.6	342	502	648	2,663	Co-elution issue: No Detection limit issue: No UCL > Max: No	712	Distribution-Free Range Min=518 Max=712
Fish	Reach 6	29	315	369	590	732	2,330	Co-elution issue: No Detection limit issue: No UCL > Max: No	772	Distribution-Free Range Min=558 Max=772
Fish	Upstream Reference Area	40	9.47	20.9	28.3	32.4	107	Co-elution issue: No Detection limit issue: Yes UCL > Max: No	Triangular Distribution Min=29.6 (ND=0) Best=35.7 (ND=DL/2) Max=42.5 (ND=DL)	Distribution-Free Range Min=21.0 (ND=0) Max=42.5 (ND=DL)
Fish	Threemile Pond	21	11.4	13.2	17.3	24.9	44.7	Co-elution issue: No Detection limit issue: Yes UCL > Max: No	Triangular Distribution Min=15.1 (ND=0) Best=23.5 (ND=DL/2) Max=32.4 (ND=DL)	Distribution-Free Range Min=9.13 (ND=0) Max=32.4 (ND=DL)
Crayfish	Reach 5	12	61.2	128	188	621	1,024	Co-elution issue: No Detection limit issue: No UCL > Max: No	825	Distribution-Free Range Min=224 Max=825
Crayfish	Upstream Reference Area	3	221	225	229	609	988	Co-elution issue: No Detection limit issue: No UCL > Max: N/A[c]	988	Distribution-Free Range Min=221 Max=988

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

Table H.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Belted Kingfishers (Continued)

[b] If assumptions about concentrations for samples below the detection limit or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL and UCL were used in the probability bounds analysis. When the number of samples was below 5, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis.

[c] Confidence limits on the mean could not be calculated because of sample size.

Table H.2-6

Exposure Model Results for Belted Kingfishers Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile
Reach 5	11.5	30.4	18.1	15.1	17.9	21.4	10.2	12.5	15.3	17.3	20.6	24.9
Reach 6	10.1	27.6	16.3	13.7	16.2	19.2	11.6	13.8	16.2	13.9	16.4	19.5
Upstream Reference Area	0.0753	0.201	0.120	0.0993	0.118	0.142	0.0607	0.0742	0.0903	0.113	0.134	0.162
Threemile Pond – Reference Area	0.0110	0.0300	0.0177	0.0148	0.0176	0.0209	0.0111	0.0133	0.0156	0.0151	0.0178	0.0212

Table H.2-7

Exposure Model Results for Belted Kingfishers Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile
Reach 5	243	676	399	333	395	469	175	214	261	397	480	589
Reach 6	256	702	415	347	411	489	249	296	349	353	415	496
Upstream Reference Area	29.4	296	100	66.2	96.9	137	11.0	14.1	24.3	111	169	226
Threemile Pond – Reference Area	5.73	26.5	12.7	9.57	12.6	16.1	4.07	4.84	5.71	14.8	17.4	20.8

Table H.2-8

Sensitivity Results from Monte Carlo Analysis of Belted Kingfishers Exposed to tPCBs and 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variables	Correlation Coefficient
Total PCBs	Reach 5	FMRpower	0.665
		FMRslope	0.661
		Proportion of fish in diet	0.187
		Body weight	-0.212
	Reach 6	FMRpower	0.677
		FMRslope	0.674
		Body weight	-0.216
		Proportion of fish in diet	0.00973
	Upstream Reference Areas	FMRpower	0.653
		FMRslope	0.648
		Proportion of fish in diet	0.261
		Body weight	-0.208
	Threemile Pond Reference Area	FMRpower	0.677
		FMRslope	0.674
		Body weight	-0.216
		Proportion of fish in diet	0.00973
2,3,7,8-TCDD TEQ	Reach 5	FMRpower	0.673
		FMRslope	0.671
		Proportion of fish in diet	-0.0769
		Body weight	-0.216
	Reach 6	FMRpower	0.677
		FMRslope	0.674
		Body weight	-0.216
		Proportion of fish in diet	0.00973
	Upstream Reference Area	Proportion of fish in diet	-0.873
		FMRslope	0.304
		FMRpower	0.301
		Body weight	-0.103
		TEQ in fish	0.0273
	Threemile Pond Reference Area	TEQ in fish	0.746
		FMRslope	0.440
		FMRpower	0.435
		Body weight	-0.136
		Proportion of fish in diet	0.00943

Table H.2-9

Input Variables Used in the Monte Carlo Exposure Analysis for Total Daily Intake of tPCBs and TEQ by Ospreys Inhabiting the Housatonic River Area

Variable	Distribution	Parameters
Body weight (*BW*; g)	Normal	0 = 1,696; s = 214
Free metabolic rate (*FMR*; kcal/kg bw/d) *a* = FMR slope *b* = FMR power	$FMR = a\ BW^b$ Normal Normal	 $log_{10}\ a$: 0 = 0.928; s = 0.197 0 = 0.768; s = 0.0874
Gross energy (*GE*; kcal/kg)	Point Estimate	1,200
Assimilation efficiency (*AE*; unitless)	Point Estimate	0.79
Proportion fish in diet (P_f; unitless)	Point Estimate	1.0
Proportion forage time in PSA (*PT*; unitless)	Point Estimate	1.0

Table H.2-10

Input Variables Used in the RiskCalc Probability Bounds Analysis for Total Daily Intake of PCBs and TEQ by Ospreys Inhabiting the Housatonic River Area

Variable	Distribution	Parameters
Body weight (*BW*; g)	Normal	0 = 1,696; s = 214
Free metabolic rate (*FMR*; kcal/kg bw/d) *a* = FMR slope *b* = FMR power	$FMR = a\ BW^b$ Normal Normal	 $log_{10}\ a$: 0 = 0.928; s = 0.197 0 = 0.768; s = 0.0874
Gross energy (*GE*; kcal/kg)	Point Estimate	1,200
Assimilation efficiency (*AE*; unitless)	Point Estimate	0.79
Proportion fish in diet (P_f; unitless)	Point Estimate	1.0
Proportion forage time in PSA (*PT*; unitless)	Point Estimate	1.0

Table H.2-11

Diet of Ospreys

	Spring	Summer	Fall	Winter	% Fish	% Birds	% Mammals	Location	Reference
Alewife		32						Nova Scotia, Canada/harbor, bay; % wet weight; observed captures	Greene et al. 1983 in EPA 1993
Smelt		5							
Pollock		53							
Winter flounder		10							
Percent Composition					100	0	0		
Starry flounder		95						SE Alaska/NS; % wet weight; observed captures, noting fish length	Hughes 1983 in EPA 1993
Cutthroat trout		5							
Percent Composition					100	0	0		
Carp		67						W Oregon/NS; % wet weight; observed captures, noting fish length	Hughes 1983 in EPA 1993
Crappie		33							
Percent Composition					100	0	0		
Gizzard shad	63							Florida/lake; % of prey caught; identified at nests	Collopy 1984 in EPA 1993
Sunfish	29								
Largemouth bass	5								
Golden shiner	3								
Percent Composition					100	0	0		
Brown bullhead	37.7							Idaho/reservoir; % of fish caught; observed captures	Van Daele and Van Daele 1982 in EPA 1993
Salmonids	20.8								
Northern squawfish	19.3								
Yellow perch	11.6								
Largescale sucker	10.6								
Percent Composition					100	0	0		

Table H.2-11

Diet of Ospreys
(Continued)

	Spring	Summer	Fall	Winter	% Fish	% Birds	% Mammals	Location	Reference
Brown bullhead	36.4							Prey captures brought to the nest in Cascade Reservoir, Idaho (%)	Van Daele and Van Daele 1982 in EPA 1993
Salmonids	23.6								
Northern squawfish	10.9								
Largescale sucker	10.9								
Yellow perch	5.5								
Unknown	12.7								
Percent Composition					100	0	0		
Fish		87.5						Labrador summer of 1995	Chubbs and Trimper 1998
Small mammals (mice, voles)		7.5							
Birds (sparrow like passerines)		5							
Percent Composition					87.5	5	7.5		
Fish		100						Labrador summer of 1996	
Percent Composition					100	0	0		

Table H.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Ospreys

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Fish	PSA	342	3.03	37.9	59.3	99.2	447	UCL > Max: No	109	Distribution-Free Range Min=61.6 Max=109
Fish	Upstream Reference Area	52	0.0816	0.442	0.178	0.230	0.338	UCL > Max: No	0.206	Distribution-Free Range Min=0.174 Max=0.206
Fish	Threemile Pond	53	0.0112	0.0328	0.0487	0.0751	0.193	UCL > Max: No	0.0686	Distribution-Free Range Min=0.0507 Max=0.0686

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

Table H.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Ospreys

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Fish	PSA	176	78.6	511	890	1,540	5,372	Co-elution issue: No Detection limit issue: No UCL > Max: No	1,415	Distribution-Free Range Min=973 Max=1,415
Fish	Upstream Reference Area	45	12.1	21.9	28.5	38.1	107	Co-elution issue: No Detection limit issue: Yes UCL > Max: No	Triangular Distribution Min=31.1 (ND=0) Best=36.3 (ND=DL/2) Max=42.1 (ND=DL)	Distribution-Free Range Min=23.1 Max=42.1
Fish	Threemile Pond	42	13.4	17.8	24.5	31.6	39.5	Co-elution issue: No Detection limit issue: Yes UCL > Max: No	Triangular Distribution Min=21.9 (ND=0) Best=27.3 (ND=DL/2) Max=33.3 (ND=DL)	Distribution-Free Range Min=16.7 Max=33.3

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] If assumptions about concentrations for samples below the detection limit or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL and UCL were used in the probability bounds analysis.

Table H.2-14

Exposure Model Results for Ospreys Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile
Primary Study Area	2.08	993	57.2	14.9	41.7	115	8.07	22.9	61.8	16.6	43.8	127
Upstream Reference Area	0.00394	1.88	0.108	0.0281	0.0787	0.217	0.0228	0.0646	0.175	0.0314	0.0827	0.240
Threemile Pond – Reference Area	0.00131	0.625	0.0360	0.00936	0.0262	0.0721	0.00664	0.0188	0.0509	0.0105	0.0276	0.0799

Table H.2-15

Exposure Model Results for Ospreys Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile	10th %-ile	50th %-ile	90th %-ile
Primary Study Area	27.1	12,897	743	193	541	1,488	127	361	977	216	568	1,649
Upstream Reference Area	0.613	308	19.2	4.93	13.9	38.2	3.03	8.58	23.2	6.43	16.9	49.1
Threemile Pond – Reference Area	0.489	231	14.4	3.71	10.5	29.2	2.19	6.20	16.8	5.08	13.4	38.8

Table H.2-16

Sensitivity Results from Monte Carlo Analysis of Ospreys Exposed to tPCBs and 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variables	Correlation Coefficient
Total PCBs	Primary Study Area	FMRpower	0.805
		FMRslope	0.562
		Body weight	-0.0448
	Upstream Reference Area	FMRpower	0.805
		FMRslope	0.562
		Body weight	-0.0448
	Threemile Pond Reference Area	FMRpower	0.805
		FMRslope	0.562
		Body weight	-0.0448
2,3,7,8-TCDD TEQ	Primary Study Area	FMRpower	0.805
		FMRslope	0.562
		Body weight	-0.0448
	Upstream Reference Area	FMRpower	0.802
		FMRslope	0.560
		TEQ in fish	0.0680
		Body weight	-0.0434
	Threemile Pond Reference Area	FMRpower	0.800
		FMRslope	0.558
		TEQ in fish	0.117
		Body weight	-0.0452

Table H.3-1

Summary of PCB and TCDD Effects on Birds

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Aroclor 1242						
White Leghorn Chicken	41 wk/F	1.2[a]	63	75.6[a]	Decreased egg hatchability	Lillie et al. 1974
White Leghorn Chicken	41 wk/F	1.2[a]	63	75.6[a]	Decreased egg production	Lillie et al. 1974
White Leghorn Chicken	10 m/F	0.6[a] (diet)	42	25.2[a]	Decreased egg hatchability	Britton and Huston 1973
Japanese Quail	9-14 d	>6,000 (ppm diet)	5		LC_{50}	Heath et al. 1972
Bobwhite Quail	9-14 d	2,098 (ppm diet)	5		LC_{50}	Heath et al. 1972
Mallard Duck	9-14 d	3,182 (ppm diet)	5		LC_{50}	Heath et al. 1972
Ring-necked Pheasant	9-14 d	2,078 (ppm diet)	5		LC_{50}	Heath et al. 1972
Aroclor 1248						
White Leghorn Chicken	Adult/F	0.6[a]	28	33.6[a]	Decreased egg hatchability	Scott 1977
White Leghorn Chicken	Adult/F	1.2[a]	56	67.2[a]	Decreased egg production, hatchability	Scott 1977
White Leghorn Chicken	41 wk/F	1.2[a]	63	75.6[a]	Decreased egg production, hatchability	Lillie et al. 1974
White Leghorn X New Hampshire	1 day	4.0[a]	35	140[a]	Decreased body weight gain	Rehfeld et al. 1971
Japanese Quail	9-14 d	4,844 (ppm diet)	5		LC_{50}	Heath et al. 1972
Bobwhite Quail	9-14 d	1,175 (ppm diet)	5		LC_{50}	Heath et al. 1972
Mallard Duck	9-14 d	2,798 (ppm diet)	5		LC_{50}	Heath et al. 1972

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Ring-necked Pheasant	9-14 d	1,312 (ppm diet)	5		LC_{50}	Heath et al. 1972
Screech Owl	Adult	3 (ppm diet)	360		No significant effects on number of eggs laid, number of eggs hatched, or number of young fledged	McLane and Hughes 1980
Aroclor 1254						
Japanese Quail	9-14 d	2,898 (ppm diet)	5		LC_{50}	Heath et al. 1972
Bobwhite Quail	9-14 d	604 (ppm diet)	5		LC_{50}	Heath et al. 1972
Mallard Duck	9-14 d	2,699 (ppm diet)	5		LC_{50}	Heath et al. 1972
Ring-necked Pheasant	9-14 d	1,091 (ppm diet)	5		LC_{50}	Heath et al. 1972
Pheasant	11wk/F	20	Until death	792-1,082	Mortality (100%); average days to death= 46; reduced heart, liver, kidney and spleen wt.	Dahlgren et al. 1972a
Pheasant	6-9 m	28.5	35	997.5	32% mortality	Dahlgren et al. 1972a
Ring-necked Pheasant	1 year/F	1.8	112	202	Significantly reduced egg production, egg hatchability, chick survival to 6 wks, chick wt at 6 wks	Dahlgren et al. 1972b
Ring Dove	Adult	0.74	56	41.44	Significant increases to dopamine and norepinephrine concentrations	Heinz et al. 1980
Ring Dove	Adult	7.7	56	431.2	% wt change (-7.1); increased liver weight dopamine and norepinephrine concentrations	Heinz et al. 1980

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Ring Dove	M-F	10[b] (ppm diet)			Eggs hatched (33%); hatched chicks fledged (58%); increase in variation of egg temperature	Peakall and Peakall 1973
Ring Dove (2nd gen)	F	10[b] (ppm diet)			Eggs hatched (42%); hatched chicks fledged (67%)	Peakall and Peakall 1973
White Leghorn Chicken	41w/F	1.2 [a]	63	75.6 [a]	Significant decrease in hatchability of fertile eggs (81%)	Lillie et al. 1974
White Leghorn Chicken	29w/F	1.2 [a]	56	67.2 [a]	No effects	Lillie et al. 1975
White Leghorn Chicken	F	1.2 [a]	70	84 [a]	Significant increase in embryo mortality (59%)	Ax and Hansen 1975
White Leghorn Chicken	1 d	30 [a]	43	1,290 [a]	50% mortality within 43 days, severe edema and lesions	Platonow et al. 1973
White Leghorn Chicken	6 m/M-F	3 [a]	98	294 [a]	Significant decrease in hen day egg production (40%) and egg hatchability (~100%)	Platonow and Reinhart 1973
White Leghorn Chicken	5 m/F	50 (ppm water)	14		Significantly reduced hatchability (34.4%)	Tumasonis et al. 1973
Bobwhite Quail	1 yr/F	50 (ppm diet)	1 laying season		No effects	Heath et al. 1972b
Mallard Ducks	1yr/F	25 (ppm diet)	2 laying seasons		No effects	Heath et al. 1972b
Mallard Ducks	9 mo/F	25 (ppm diet)	77		No effects	Custer and Heinz 1980

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Bengalese Finch	Adult/M-F	253.6	56	14,202	Estimated LD_{50}; average brain conc. – 290 mg/kg; average liver conc. – 345 mg/kg	Prestt et al. 1970
Starling	Adult/M-F	1,500 (ppm diet)	4		4 d to 50% mortality; average brain tissue conc. 439 mg/kg in dead birds: 179 mg/kg in sacrificed	Stickel et al. 1984
Red-winged Blackbird, Cow-bird, Grackle	Adult/M-F	1,500 (ppm diet)	6, 7, 8		6, 7, and 8 d to 50% mortality respectively. Average brain tissue conc. 579 mg/kg in dead birds: <156 mg/kg in sacrificed	Stickel et al. 1984
Wild Birds	Adult/M-F				Estimated PCB concentration in brain tissue associated with avian mortality: 310 mg/kg	Stickel et al. 1984
Aroclor 1260						
Japanese Quail	9-14 d	2,186 (ppm diet)	5[a]		LC_{50}	Heath et al. 1972
Bobwhite Quail	9-14 d	747 (ppm diet)	5[a]		LC_{50}	Heath et al. 1972
Mallard Duck	9-14 d	1,975 (ppm diet)	5[a]		LC_{50}	Heath et al. 1972
Ring-necked Pheasant	9-14 d	1,260 (ppm diet)	5[a]		LC_{50}	Heath et al. 1972
Hubbart Chicken	1 day/M	24[a]	60	1,440[a]	Body weight decrease (703 g); liver weight increase (29.7 g); spleen weight increase (0.9 g); 15% mortality	Vos and Koeman 1970
PCB-77						
White Leghorn Chicken	Embryo		Single inj	0.009 (egg)	Calculated LD_{50}	Powell et al. 1996a

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
White Leghorn Chicken	Embryo		Single inj	0.003 (egg)	Reduced 3 wk body weight (-7.5%)	Powell et al. 1996a
White Leghorn Chicken	Embryo		Single inj	0.009 (egg)	Calculated LD_{50}	Brunstrom and Andersson 1988
White Leghorn Chicken	Embryo		Single inj	0.005 (egg)	Increased mortality	Brunstrom 1988
Brown Leghorn Chicken	Embryo		Single inj	0.005 (egg)	Increased mortality	Brunstrom 1988
New Hampshire Chicken	Embryo		Single inj	0.005 (egg)	Increased mortality	Brunstrom 1988
Rhode Island Red Chicken	Embryo		Single inj	0.2 (egg)	Increased mortality	Brunstrom 1988
Duck	Embryo		Single inj	5 (egg)	No change in mortality	Brunstrom 1988
Goose	Embryo		Single inj	1 (egg)	No change in mortality	Brunstrom 1988
Herring Gull	Embryo		Single inj	1 (egg)	No change in mortality	Brunstrom 1988
White Leghorn Chicken	Embryo		Single inj	0.002 (egg)	ED_{50} EROD induction	Brunstrom and Andersson 1988
White Leghorn Chicken	Embryo		Single inj	0.0146 (egg)	ED_{50} lymphoid development inhibition	Brunstrom et al. 1990
Turkey	Embryo		Single inj	1 (egg)	Increased mortality (60%)	Brunstrom and Lund 1988
American Kestrel	Embryo		Single inj	0.688 (egg)	Calculated LD_{50}	Hoffman et al. 1996a
American Kestrel	Embryo		Single inj	1 (egg)	Increased mortality (23%), decreased hatching success (38%)	Hoffman et al. 1998
Ring-necked Pheasant	Embryo		Single inj	1 (egg)	100% mortality	Brunstrom and Reutergardh 1986

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
PCB-105						
White Leghorn Chicken	Embryo		Single inj	5.59 (egg)	Calculated LD_{50}	Powell et al. 1996a
White Leghorn Chicken	Embryo		Single inj	2.20 (egg)	Calculated LD_{50}	Brunstrom 1990
PCB-126						
American Kestrel	1 d/M-F	0.25 mg/kg bw/d	10	25	Significantly decreased bone length, decreased spleen wt. Decrease in bw, but not significant.	Hoffman et al. 1996a
American Kestrel	Embryo		Single inj	0.065	Calculated LD_{50}	Hoffman et al. 1996a
Common Tern	Embryo		Single inj	0.104	Calculated LD_{50}	Hoffman et al. 1998
Common Tern	Embryo		Single inj	0.044	Reduced hatching success (60%)	Hoffman et al. 1998
White Leghorn Chicken	Embryo		Single inj	0.0006	Calculated LD_{50}	Powell et al. 1996a
White Leghorn Chicken	Embryo		Single inj	0.0009	Reduced 3 wk body weight	Powell et al. 1996a
White Leghorn Chicken	Embryo		Single inj	0.002	Calculated LD_{50}	Powell et al. 1996b
White Leghorn Chicken	Embryo		Single inj	0.003	Calculated LD_{50}	Brunstrom and Andersson 1988
Double-crested Cormorant	Embryo		Single inj	0.158	Calculated LD_{50}	Powell et al. 1997
Double-crested Cormorant	Embryo		Single inj	0.177	Calculated LD_{50}	Powell et al. 1998
Turkey	Embryo		Single inj	0.02	Increased mortality	Brunstrom 1989

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
White Leghorn Chicken	Embryo		Single inj	0.0001	ED_{50} EROD induction	Brunstrom and Andersson 1988
White Leghorn Chicken	Embryo		Single inj	0.0013	ED_{50} lymphoid development inhibition	Brunstrom et al. 1990
White Leghorn Chicken	Embryo		Single inj	0.001	Calculated LD_{50}	Fox and Grasman 1999
White Leghorn Chicken	Embryo		Single inj	0.001	Increased mortality	Zhao et al. 1997
PCB-156						
White Leghorn Chicken	Embryo		Single inj	1.5	Calculated LD_{50}	Brunstrom 1990
PCB-157						
White Leghorn Chicken	Embryo		Single inj	2.5	Calculated LD_{50}	Brunstrom 1990
PCB-169						
White Leghorn Chicken	Embryo		Single inj	0.17	Calculated LD_{50}	Brunstrom and Andersson 1988
White Leghorn Chicken	Embryo		Single inj	0.144	ED_{50} EROD induction	Brunstrom and Andersson 1988
White Leghorn Chicken	Embryo		Single inj	0.108	ED_{50} lymphoid development inhibition	Brunstrom et al. 1990
2,3,7,8–TCDD						
Ring-necked Pheasant	F		Single inj	0.025	80% mortality	Nosek et al. 1992

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Ring-necked Pheasant	F		Weekly ip inj	0.01	100% embryonic mortality, decreased egg production, decreased weight gain	Nosek et al. 1992
Ring-necked Pheasant	Embryo		Single inj	0.0022	LD_{50} hatchability	Nosek et al. 1993
Chicken	Embryo		Single inj	0.00015	LD_{50} hatchability	Henshel et al. 1997
Chicken	Embryo		Single inj	0.0001	Reduced 21-d embryonic weight	Henshel et al. 1997
Chicken	Embryo		Single inj	0.0003	Reduced hatching weight	Hoffman et al. 1998
Heron	Embryo		Single inj	0.000021	ED_{50} brain deformities	Henshel 1998
Chicken	Embryo		Single inj	0.000037	ED_{50} brain deformities	Henshel 1998
Cormorant	Embryo		Single inj	0.000034	ED_{50} brain deformities	Henshel 1998
Cormorant	Embryo		Single inj	0.004	Increased mortality (50%)	Powell et al. 1997
White Leghorn Chicken	Embryo		Single inj	0.00016	LD_{50} hatchability	Powell et al. 1996b
Total PCBs						
White Leghorn Chicken	Adult/F	6.6	56		Decreased hatchability	Summer et al. 1996
American Kestrel	Adult/M-F	7	100	34 (egg)	2nd generation increased brood mortality; decreased female clutch size	Fernie et al. 2001a
American Kestrel	Adult/M-F	7	100	34 (egg)	Decreased number of fertile eggs, hatchlings, and fledglings per breeding pair	Fernie et al. 2001b
Common Tern	21 d/M-F	1,250 ng/kg ww TEQ in diet			EROD induction 10x controls	Bosveld et al. 2000

Table H.3-1

Summary of PCB and TCDD Effects on Birds (Continued)

Species	Age/Sex	Dose (mg/kg bw/d)	Duration (day)	Total Dose (mg/kg bw)	Endpoint	Reference
Complex Mixtures (Field-Based Studies)						
Cormorant	Adult/F	21 (egg)	Trophic		Decreased egg hatchability compared to 10 mg/kg egg site	Dirksen et al. 1995
Tree Swallow	Adult/ Nestlings	0.155	Trophic		No effects on growth or reproduction	Custer et al. 1998
Tree Swallow	Adult/F	9.3-29.5 (egg); 3.7-62.2 (nestling)	Trophic		Decreased hatching rates, increased hatchling mortality in 1994, but not 1995	McCarty and Secord 1999b
Tree Swallow	Adult/M-F		Trophic		Smaller nests, fewer feathers lining nest at test site compared to reference	McCarty and Secord 1999a
Tree Swallow	Adult/M-F		Trophic		No reproductive effects at whole body PCB concentrations of 11,100 ng/kg egg	Bishop et al. 1999
Barn Swallow	Adult/M-F	0.52 (estimated)	Trophic		No reproductive effects	Henning et al. 1997
Robin	Adult/M-F	5.7 (estimated)			No reproductive effects	Henning et al. 1997
Heron	Adult/M-F	210 ng/kg TCDD egg	Trophic		Complete colony hatching failure; 9-66 ng/kg had no effects	Elliott et al. 1989

[a] Estimated using food consumption rate: body weight ratio of 6% (CCME 1999).

[b] Second-generation birds which had been continuously maintained on a diet containing 10 mg Aroclor 1254/kg diet.

Table H.4-1

Summary of Exceedance Probabilities for Piscivorous Birds from the Housatonic River PSA

Bird/Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LB		*FOMC*		*UB*		*LB*		*FOMC*		*UB*	
	LT	UT	LT	UT	LT	UT	LT	UT	LT	UT	LT	UT
Belted Kingfisher												
Reach 5	100	100	100	100	100	100	100	0	100	0	100	0
Reach 6	100	100	100	100	100	100	100	0	100	0	100	0
Upstream Reference Area	0	0	46	0	79	0	0	0	100	0	100	0
Threemile Pond	0	0	0	0	0	0	0	0	0	0	0	0
Osprey												
Reaches 5 and 6	100	92	100	99	100	100	100	0	100	0	100	0
Upstream Reference Area	21	0	30	0	32	0	1	0	7	0	12	0
Threemile Pond	0	0	3	0	4	0	0	0	4	0	7	0

LB = Lower Probability Bound.
FOMC = First Order Monte Carlo.
UB = Upper Probability Bound.
LT = Lower Toxicity Threshold.
UT = Upper Toxicity Threshold.

Table H.4-2

Summary of Qualitative Risk Statements for Piscivorous Birds from the Housatonic River PSA

Bird/Location	Qualitative Risk Statements				
	tPCBs			TEQ	
	Risk Category	Risk Range		Risk Category	Risk Range
Belted Kingfisher					
Reach 5	High	High		Intermediate	Intermediate
Reach 6	High	High		Intermediate	Intermediate
Upstream Reference Area	Intermediate	Low-intermediate		Intermediate	Low-intermediate
Threemile Pond	Low	Low		Low	Low
Osprey					
Reaches 5 and 6	High	High		Intermediate	Intermediate
Upstream Reference Area	Intermediate	Intermediate		Low	Low
Threemile Pond	Low	Low		Low	Low

Table H.4-3

Weighting of Measurement Endpoints for Piscivorous Birds
Weight-of-Evidence Evaluation

Attributes	Modeled Exposure and Effects for tPCBs and TEQ	GE Kingfisher Field Study (Henning 2002)	Rationale
I. Relationship Between Measurement and Assessment Endpoints			
1. Degree of Association	M	H	Assessment endpoint linked to measurement endpoint for MEE, but not directly measured, and surrogate species are used in the effects metrics. Field study directly measured the assessment endpoint.
2. Stressor/Response	M	M	Correlation has been demonstrated in previous studies, but the modeled exposure and effects was unable to correlate exposure and response because of the wide effects threshold range. Exposure/response relationship could not be measured in the field study, given the small sample sizes and narrow dose range.
3. Utility of Measure	M	M	Modeled exposure and effects procedures used are standardized and widely accepted, the primary limitation was lack of species-specific effects data and the large toxicity threshold range, which reduce certainty. Statistical power of study was low, and therefore the statistical analyses have limited applicability. There was also no reference area for comparison to the PSA.

Table H.4-3

Weighting of Measurement Endpoints for Piscivorous Birds Weight-of-Evidence Evaluation

(Continued)

Attributes	Modeled Exposure and Effects for tPCBs and TEQ	GE Kingfisher Field Study (Henning 2002)	Rationale
II. Data Quality			
4. Data Quality	M/H	M	The tissue data in the exposure analysis were subject to a rigorous QA/QC check, other model parameters were derived from EPA (1993) and other published journal articles, and the effects metrics were derived from published journal articles. The field study appeared to follow acceptable procedures, but there were a few concerns, namely: few samples, no clutch size data, no reference site data, narrow dose gradient, one or more missed nests, and few other suitable papers in the literature.
III. Study Design			
5. Site Specificity	L/M	M/H	Biological tissue data used in exposure models were site specific. Other exposure model parameters were not necessarily representative of site conditions, and effects measures were laboratory based, not site specific. A field study was conducted in the PSA, although some kingfishers included in the study likely foraged downstream of the PSA. No reference sites were included in the study.
6. Sensitivity	L/M	L/M	Modeled exposure and effects directly assessed exposure, but effects studies for the representative species were not available, so a threshold range spanning sensitive to tolerant birds was employed. This range was too broad to be very sensitive. Sample sizes were generally small, estimated dose gradient was small, and no reference area was available for comparison. Field studies are generally not able to detect potential subtle effects associated with COCs.

Table H.4-3

Weighting of Measurement Endpoints for Piscivorous Birds Weight-of-Evidence Evaluation

(Continued)

Attributes	Modeled Exposure and Effects for tPCBs and TEQ	GE Kingfisher Field Study (Henning 2002)	Rationale
7. Spatial Representativeness	M	M/H	Modeled exposures relied on tissue data collected throughout the study area. Effects assessment used toxicity studies conducted in laboratories. Four of five criteria overlapped with the field study, as some kingfisher nests were located outside of the PSA and likely foraged outside as well.
8. Temporal Representativeness	M	M	Tissue data for the exposure model were not collected at times coinciding with the breeding season. Effects studies did span the reproductive cycles of the birds. Field study was conducted during the breeding season of belted kingfishers, but the study was a single sampling event.
9. Quantitative Measure	M/H	M	Probabilistic exposure and effects modeling was highly quantitative and propagated uncertainty associated with modeling procedures. Given the broad range of the effects thresholds, results do not clearly reflect biological significance. Field study results were quantitative, however small sample sizes limited the statistical power.
10. Standard Method	M	M/H	Generally accepted exposure and effects modeling procedures were followed, but probability bounds analysis is a relatively new technique for propagating uncertainty, and the use of a threshold range for the effects portion is not common. Field study protocol was not available for review prior to study and the methods that were presented after the completion of the studies introduced some concerns.
Overall Endpoint Value	M	M/H	– – –

L = low
M = moderate
H = high

Table H.4-4

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	M	Yes (Kingfisher) Yes (Osprey)	High (Kingfisher) High (Osprey)
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)

L = low
M = moderate
H = high

Table H.4-5

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	M	Yes (Kingfisher) Yes (Osprey)	Intermediate (Kingfisher) Intermediate (Osprey)
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)

L = low
M = moderate
H = high

Table H.4-6

Risk Analysis Summary for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint:	Survival, growth, and reproduction of piscivorous birds.

	Weighting Factors (increasing confidence of weight)				
Harm/Magnitude	**Low**	**Low/ Moderate**	**Modera te**	**Moderate/High**	**High**
Yes/High			MEE-KF MEE-O		
Yes/Intermediate					
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low				FS-KF	
No/Intermediate					
No/High					

MEE = Modeled exposure and effects
FS = Field study
KF = Kingfisher
O = Osprey

Table H.4-7

Risk Analysis Summary for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint:	Survival, growth, and reproduction of piscivorous birds.

	Weighting Factors (increasing confidence of weight)				
Harm/Magnitude	**Low**	**Low/ Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High					
Yes/Intermediate			MEE-KF MEE-O		
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low				FS-KF	
No/Intermediate					
No/High					

MEE = Modeled exposure and effects
FS = Field study
KF = Kingfisher
O = Osprey

APPENDIX H
ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS BIRDS

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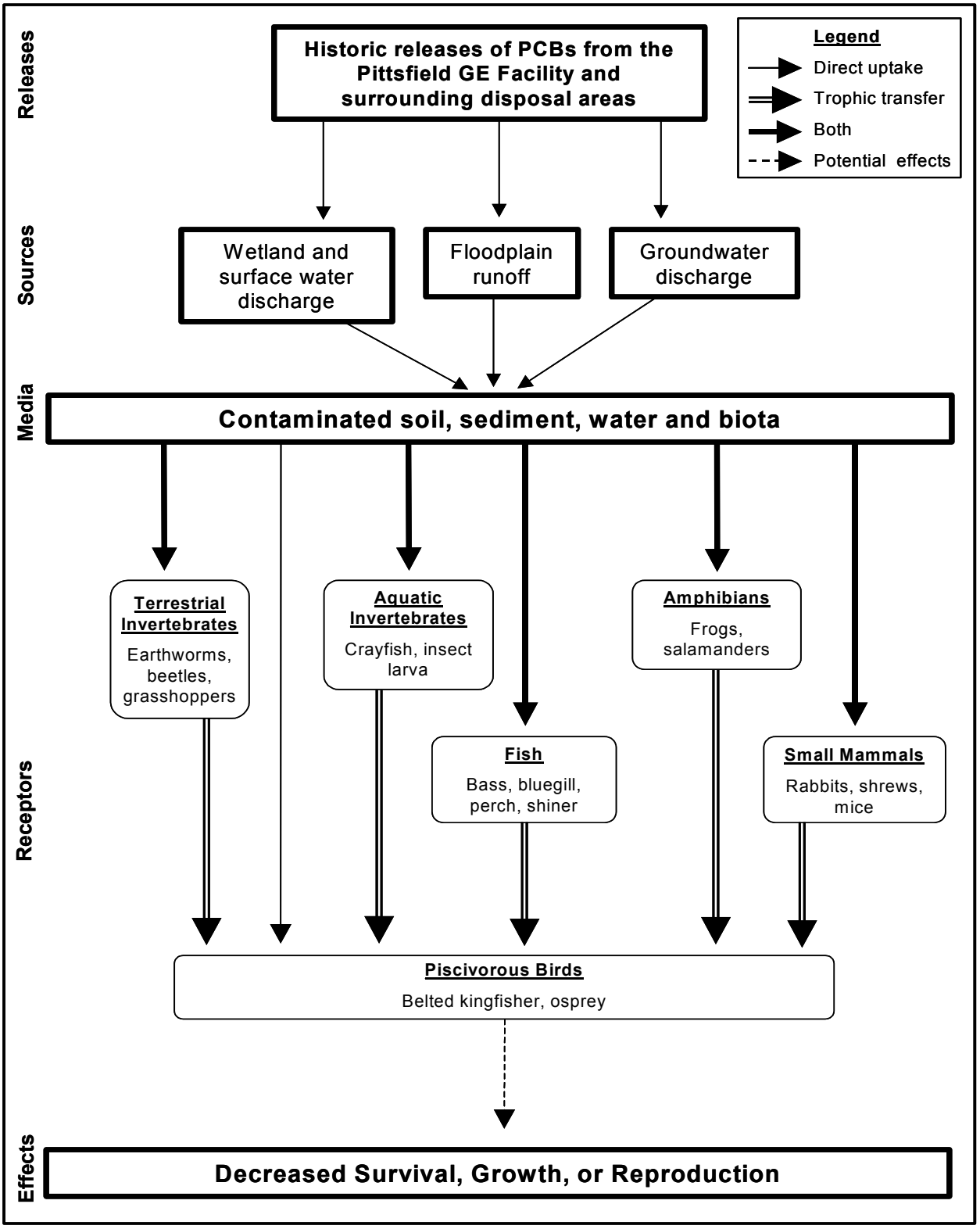


Figure H.1-1 Conceptual Model for the Exposure of Piscivorous Birds to tPCBs and TEQ in the Housatonic River PSA

EXPOSURE

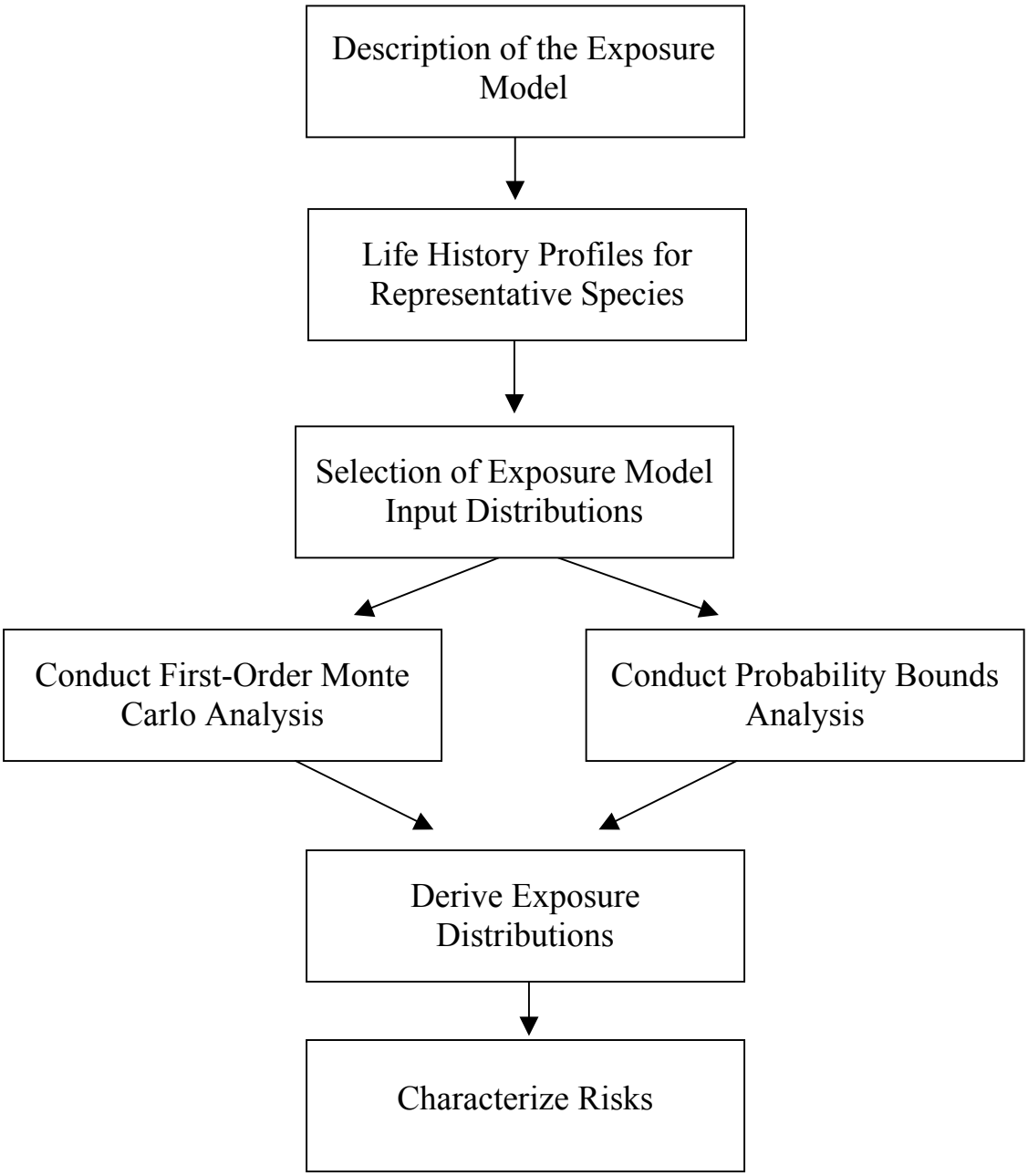


Figure H.1-2 Overview of Approach Used to Assess Modeled Exposure of Piscivorous Birds to Contaminants of Concern (COCs) in the Housatonic River PSA

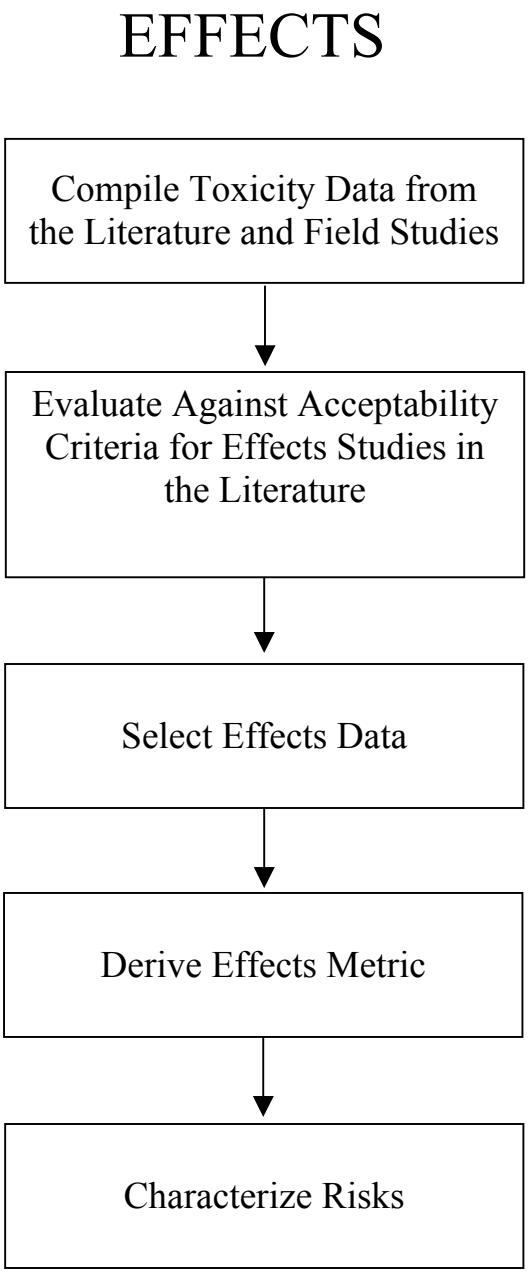


Figure H.1-3 Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Piscivorous Birds in the Housatonic River PSA

RISK CHARACTERIZATION

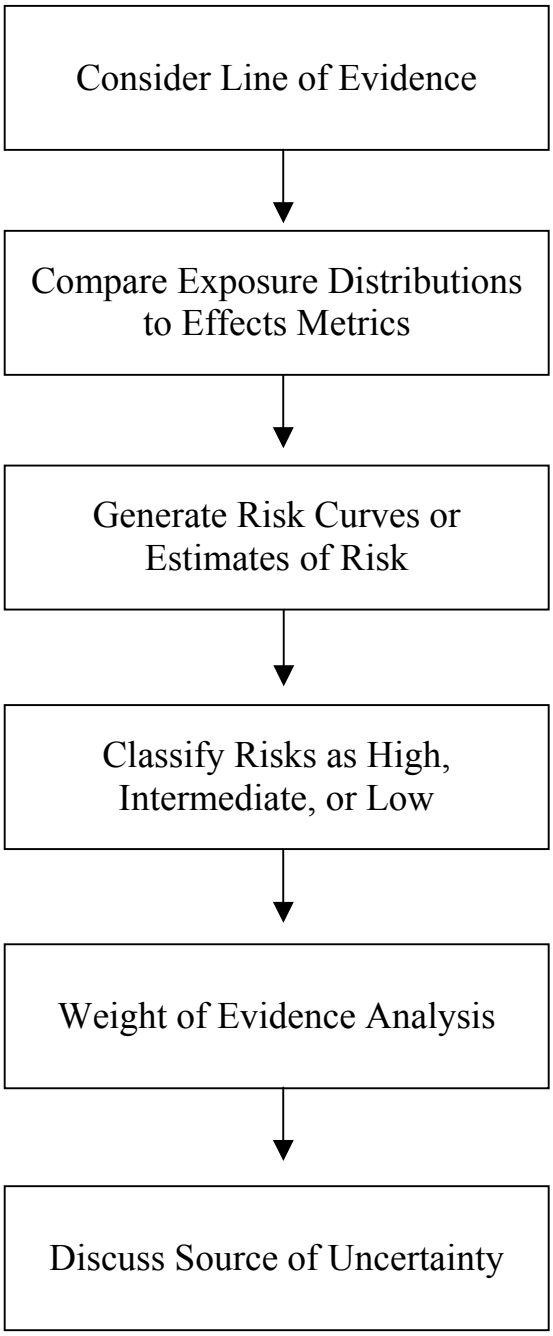
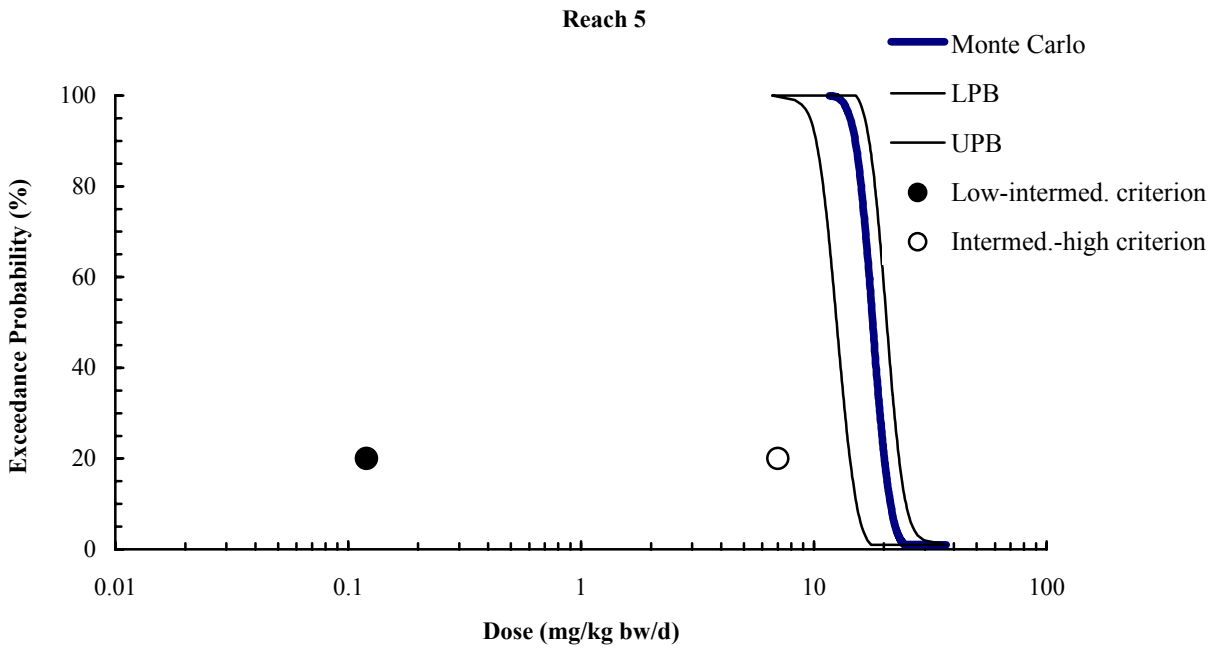
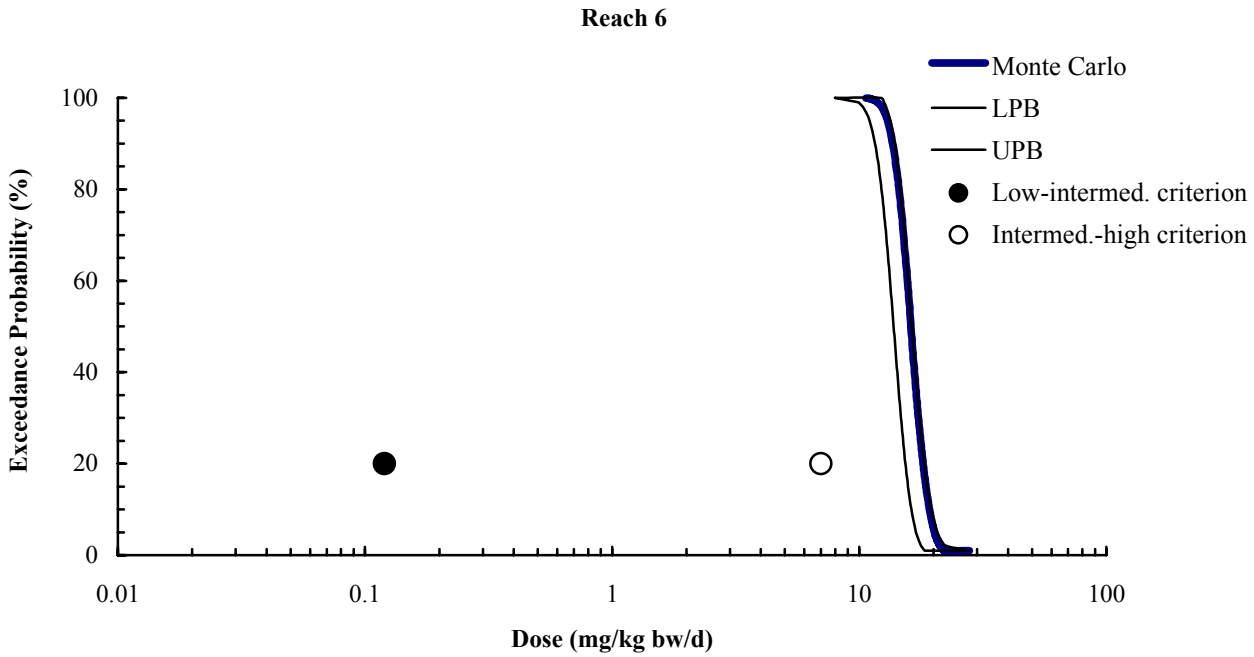


Figure H.1-4 Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Birds in the Housatonic River PSA



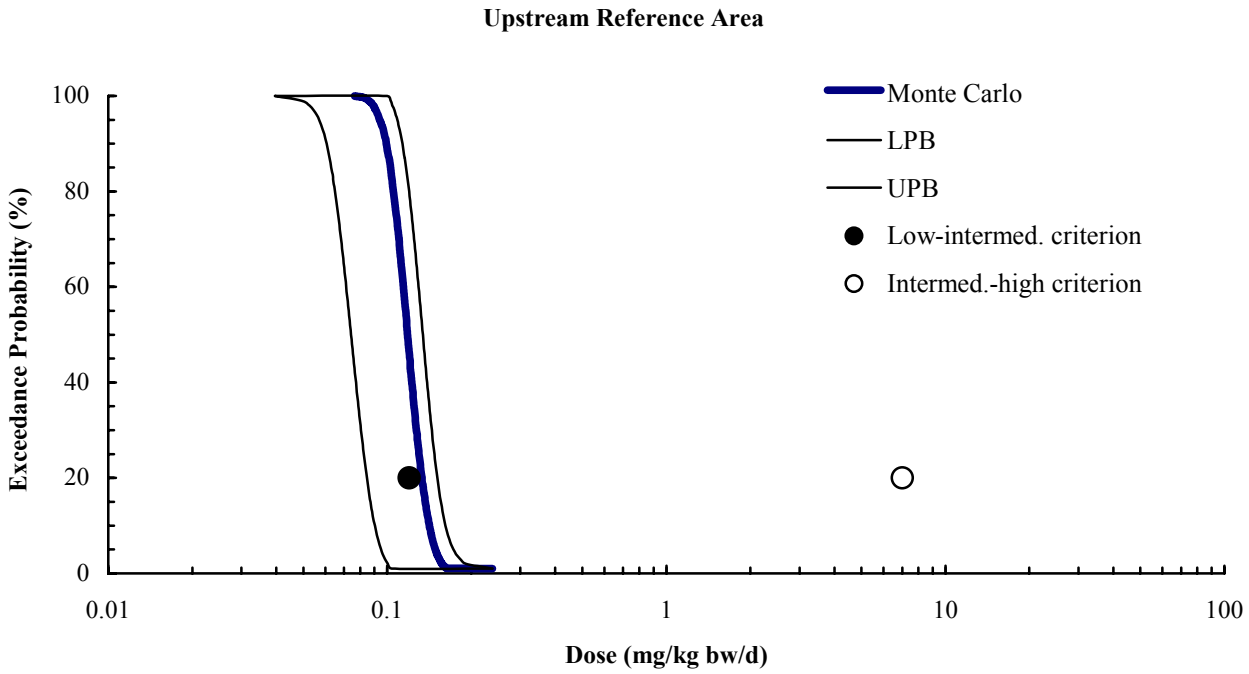
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-1 Exposure of Belted Kingfishers to tPCBs in Reach 5 of the Housatonic River PSA



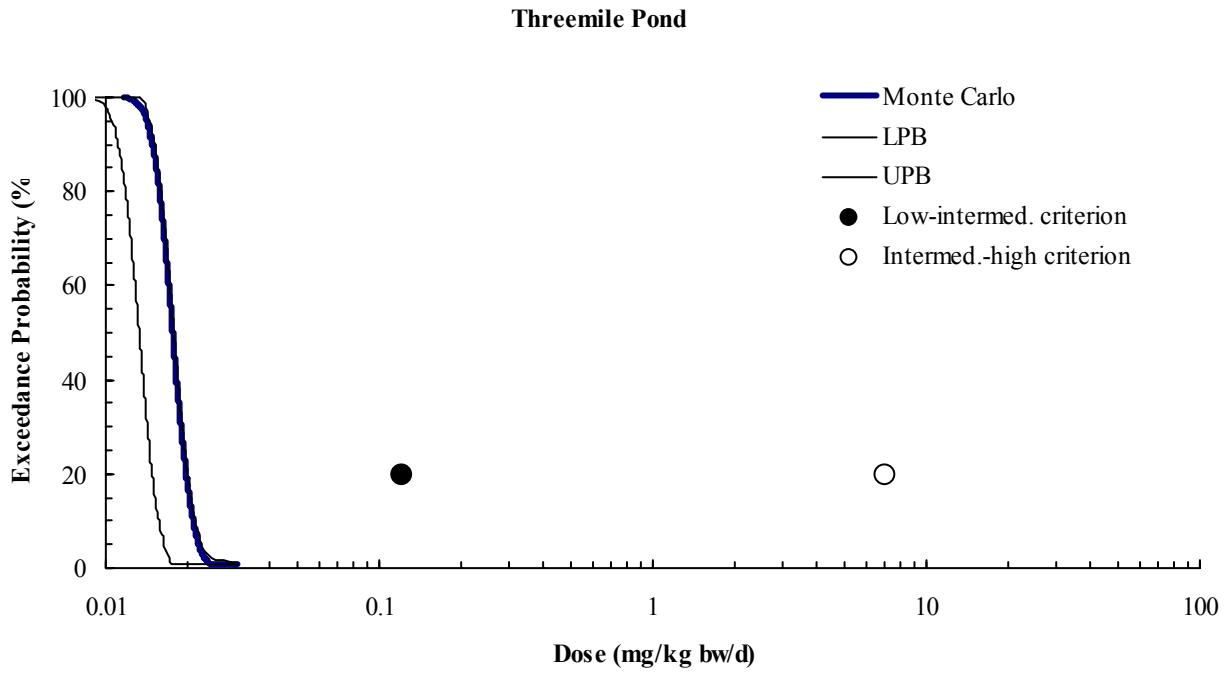
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-2 Exposure of Belted Kingfishers to tPCBs in Reach 6 of the Housatonic River PSA



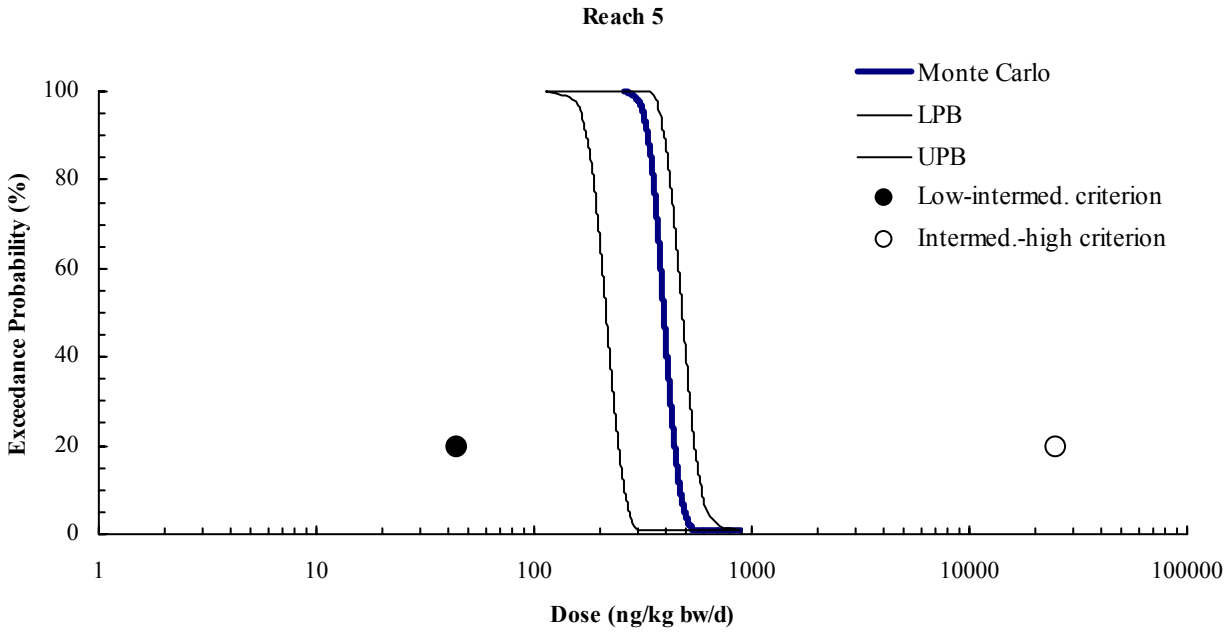
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-3 Exposure of Belted Kingfishers to tPCBs in the Housatonic River Upstream Reference Area



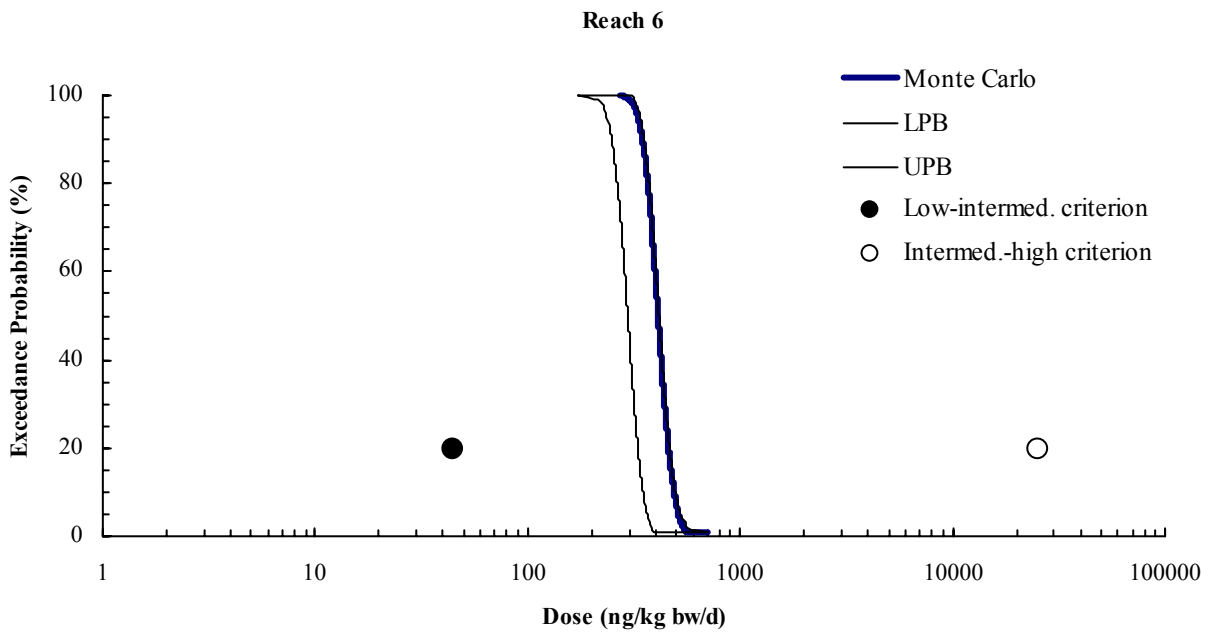
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-4 Exposure of Belted Kingfishers to tPCBs in the Threemile Pond Reference Area



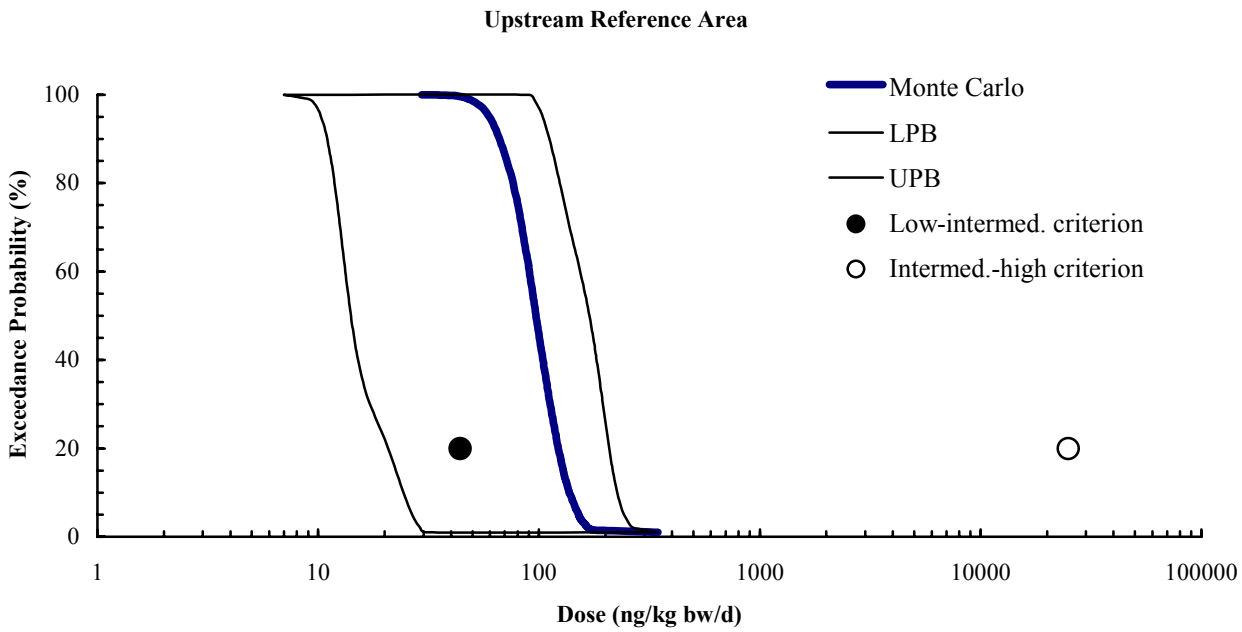
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-5 Exposure of Belted Kingfishers to TEQ in Reach 5 of the Housatonic River PSA



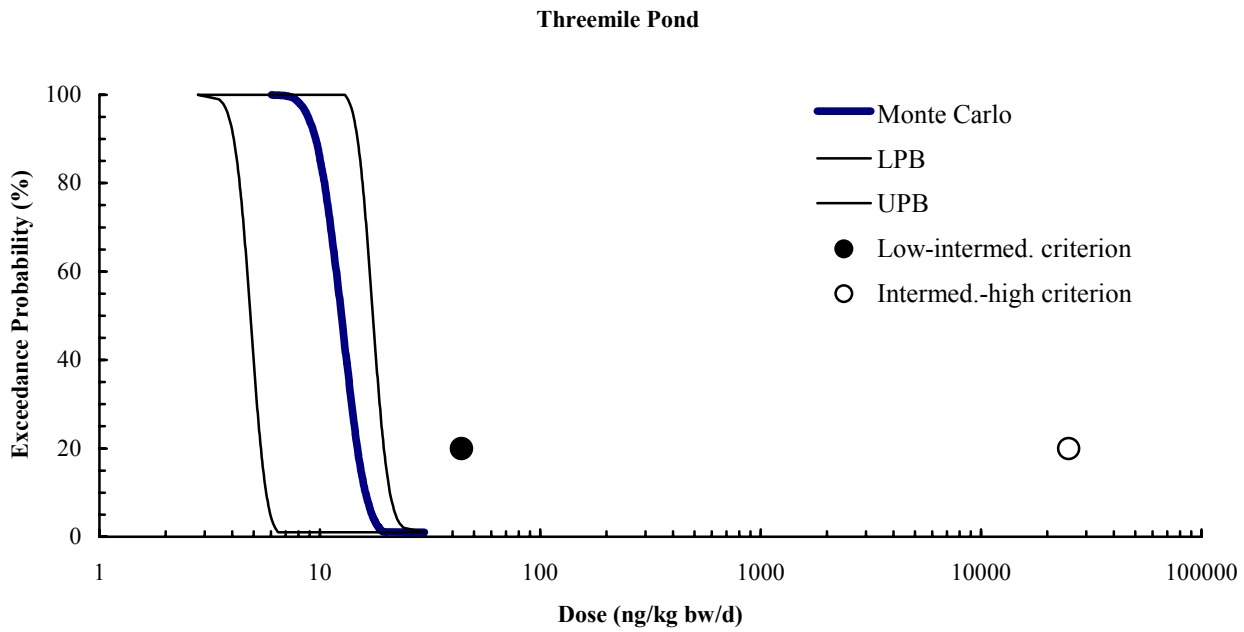
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-6 Exposure of Belted Kingfishers to TEQ in Reach 6 of the Housatonic River PSA



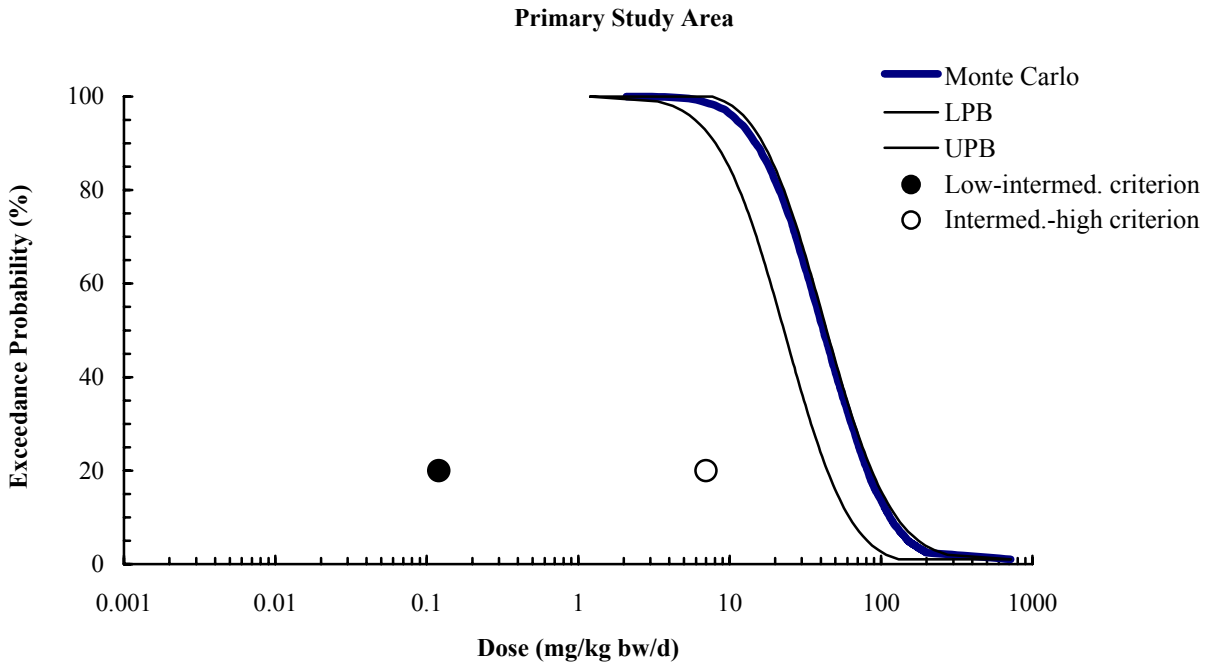
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-7 Exposure of Belted Kingfishers to TEQ in the Housatonic River Upstream Reference Area



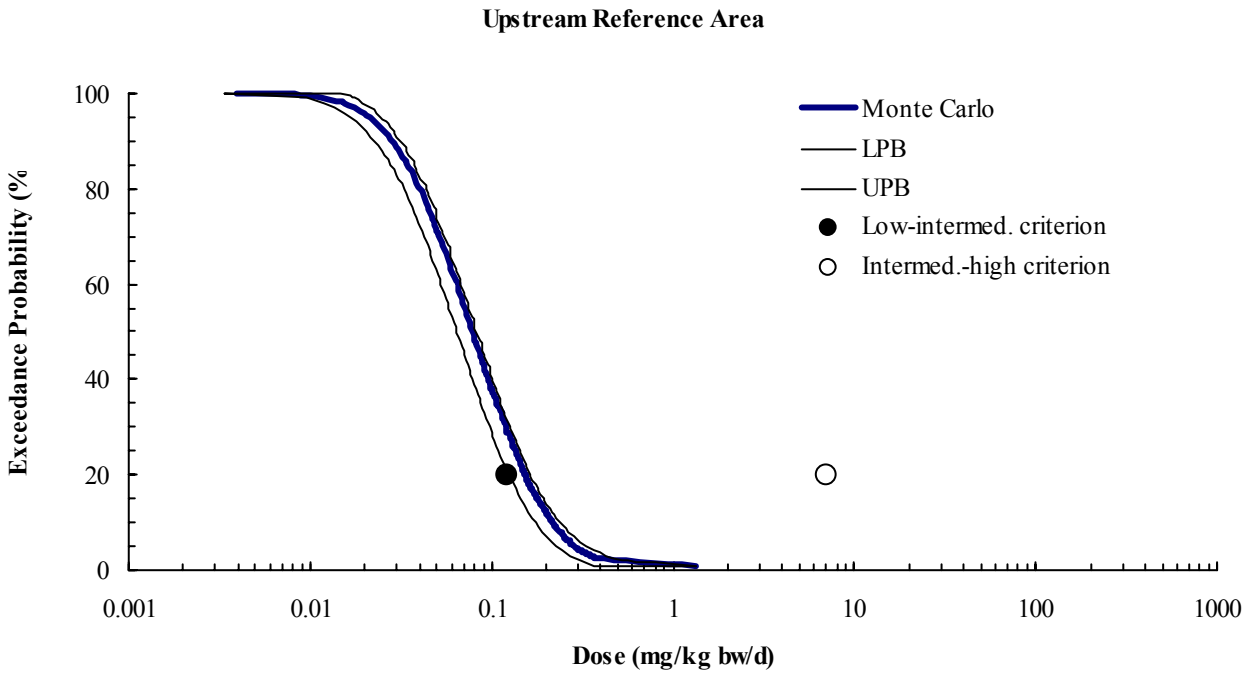
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-8 Exposure of Belted Kingfishers to TEQ in the Threemile Pond Reference Area



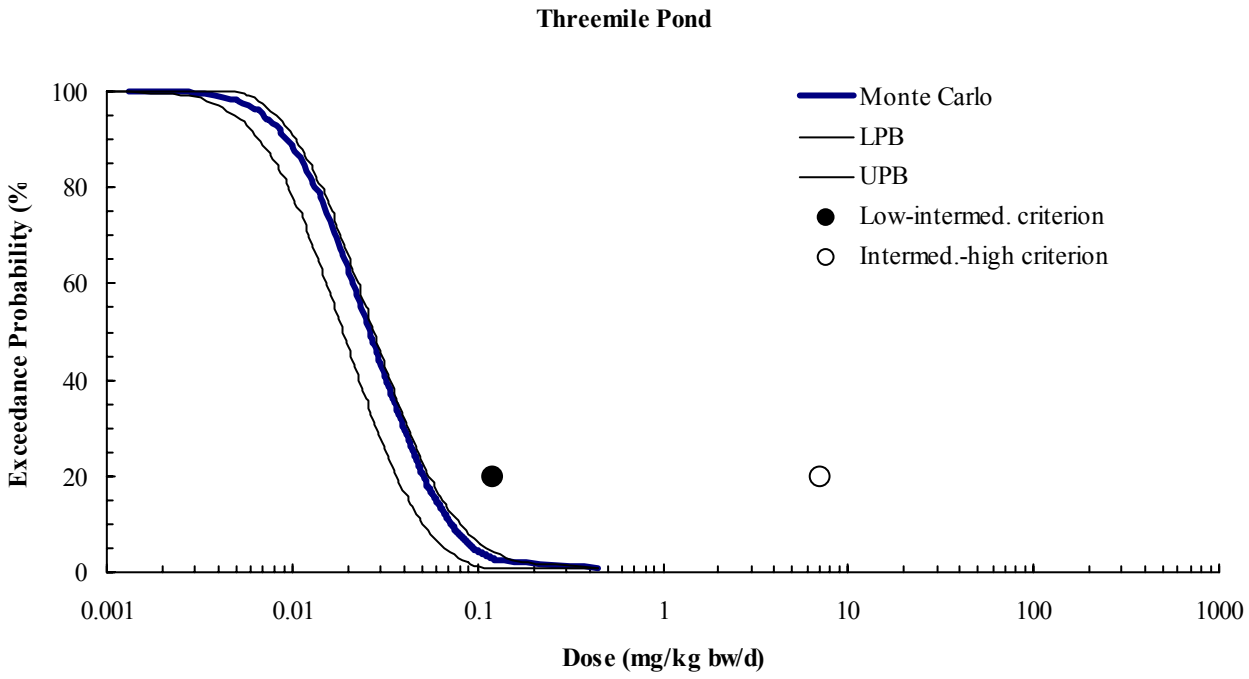
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-9 Exposure of Ospreys to tPCBs in Reaches 5 and 6 of the Housatonic River PSA



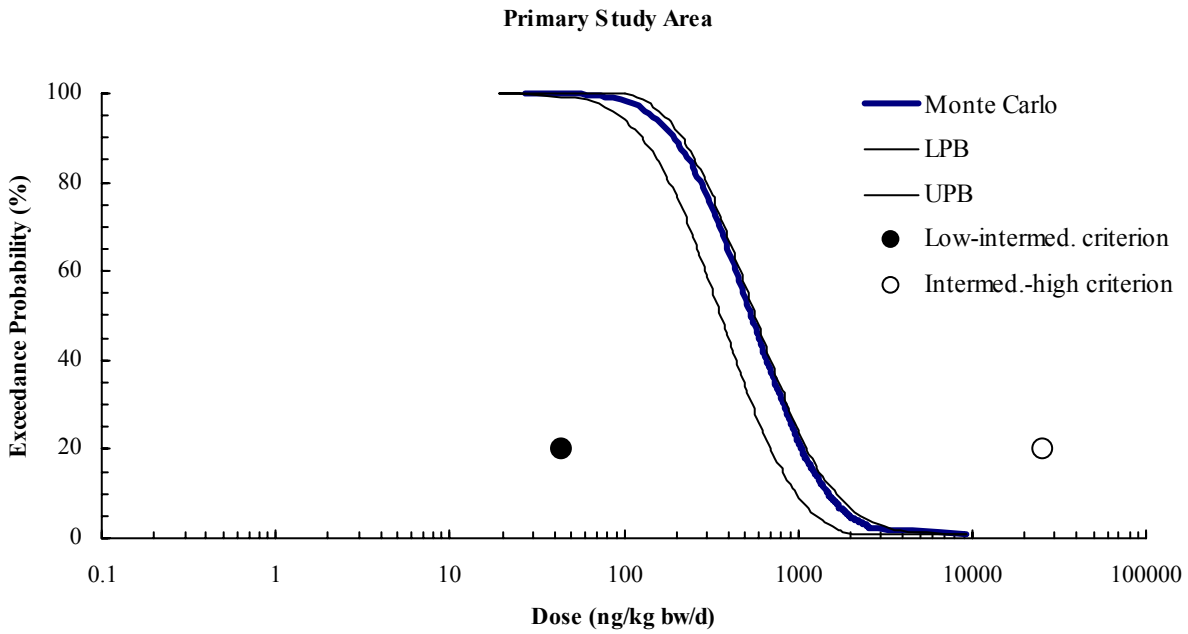
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-10 Exposure of Ospreys to tPCBs in the Housatonic River Upstream Reference Area



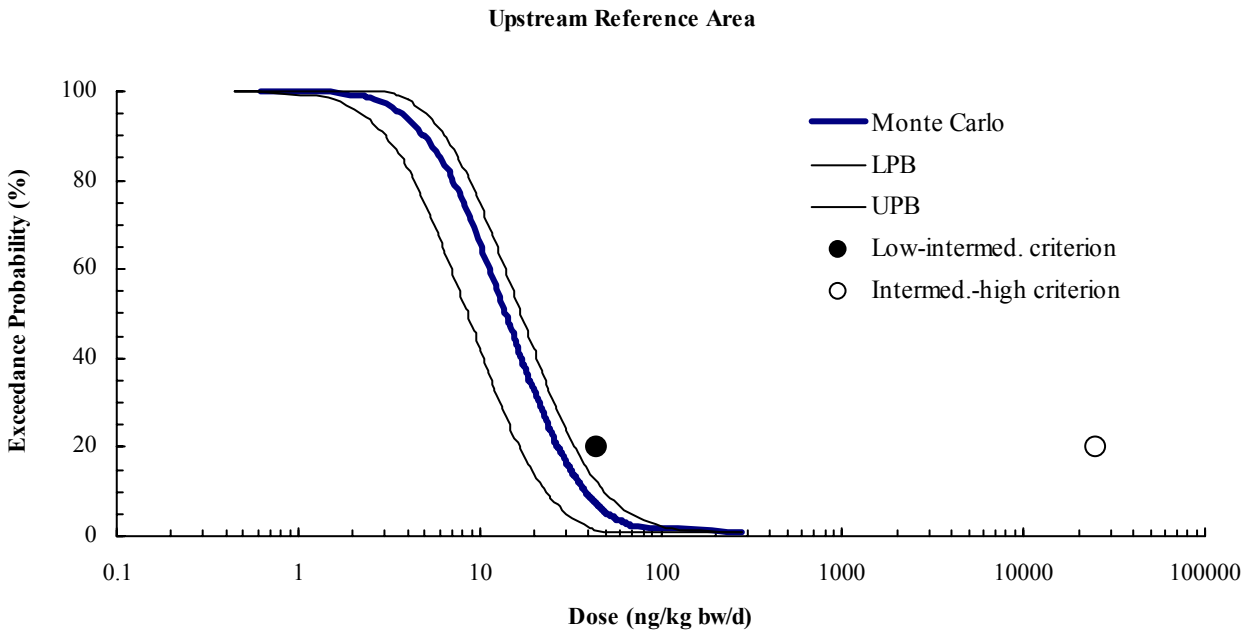
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-11 Exposure of Ospreys to tPCBs in the Threemile Pond Reference Area



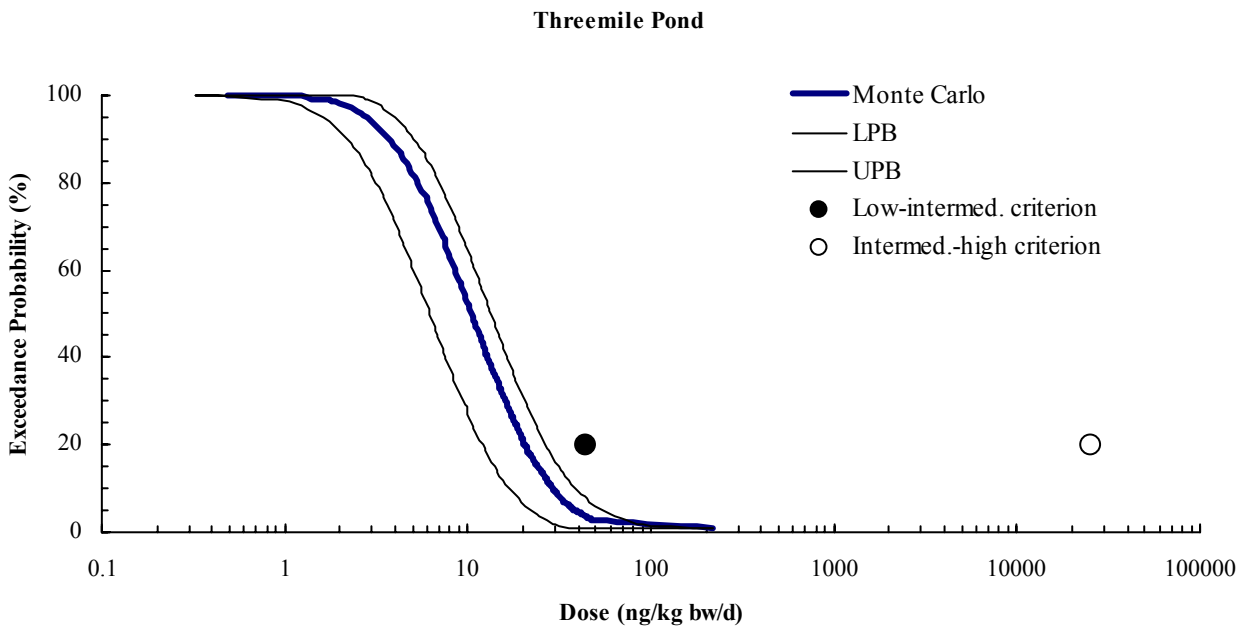
LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-12 Exposure of Ospreys to TEQ in Reaches 5 and 6 of the Housatonic River PSA



LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-13 Exposure of Ospreys to TEQ in the Upstream Reference Area



LPB = Lower probability bound
UPB = Upper probability bound

Figure H.2-14 Exposure of Ospreys to TEQ in the Threemile Pond Reference Area

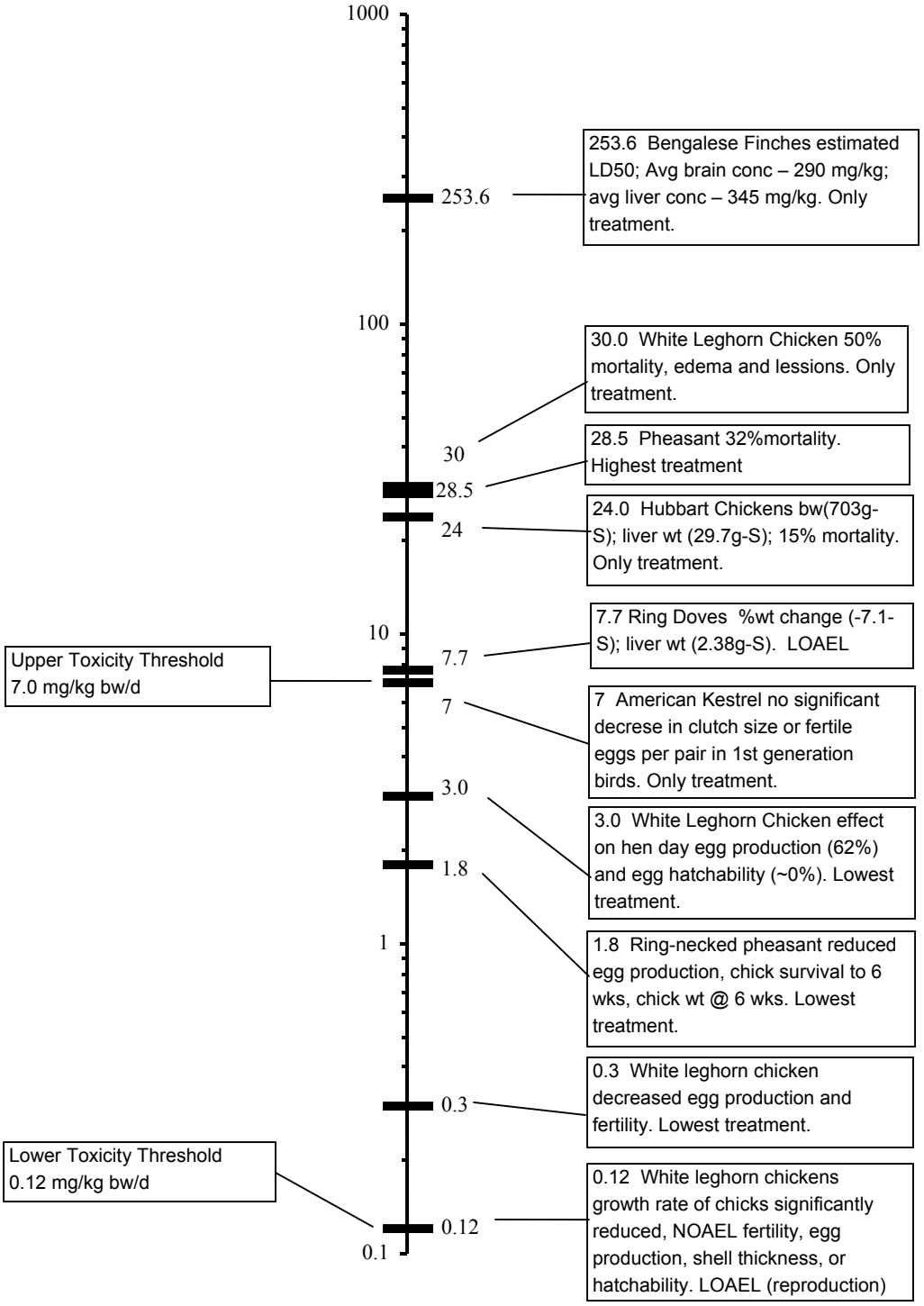


Figure H.3-1 Effects of Aroclor 1254/1260 on Avian Species (mg/kg bw/d)

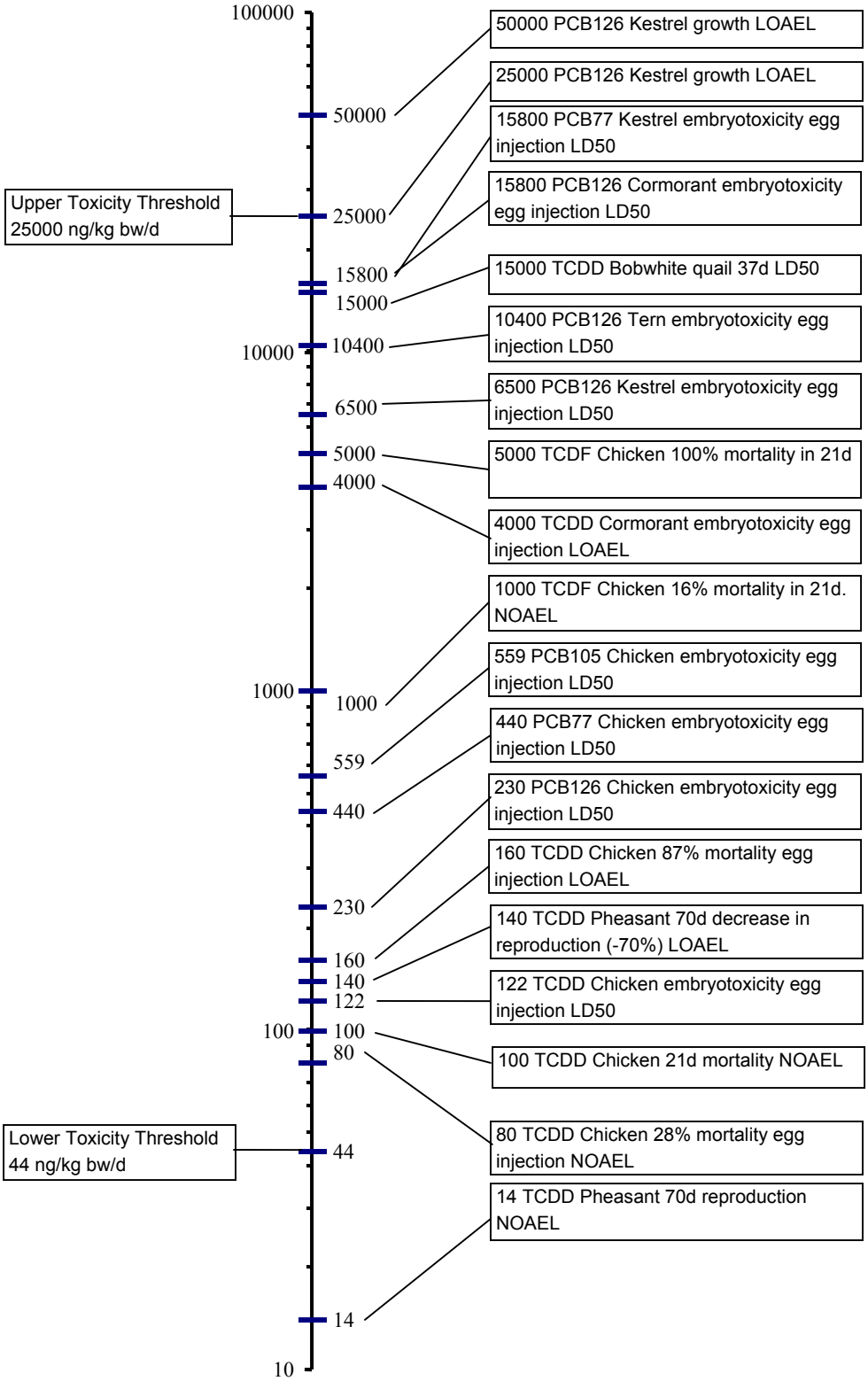


Figure H.3-2 Effects of 2,3,7,8-TCDD TEQ on Avian Species (ng TEQ/kg bw/d)

APPENDIX I

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS MAMMALS

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LIST OF ACRONYMS

BBL	Blasland, Bouck, & Lee
BROD	benzyloxyresorufin-O-deethylase
BW	body weight
CERC	Columbia Environmental Research Center
CLs	confidence limits
CO_2	carbon dioxide
COCs	contaminants of concern
COPCs	contaminants of potential concern
CYP	cytochrome P450
DL	detection limit
ECOD	ethoxycoumarin-O-deethylase
ERA	ecological risk assessment
EROD	ethoxyresorufin-O-deethylase
FIR	food intake rate
FMR	free metabolic rate
GE	General Electric Company
kcal	kilocalories
MATC	maximum acceptable threshold concentration
MDFW	Massachusetts Division of Fisheries and Wildlife
MSU	Michigan State University
NOAEL	no observed adverse effect level
PROD	pentoxyresorufin-O-deethylase
SOP	standard operating procedure
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
TDI	total daily intake
TEF	toxic equivalency factor
TEQ	toxic equivalence
tPCBs	total PCBs
WOE	weight-of-evidence

APPENDIX I

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS MAMMALS

I.1 INTRODUCTION

The purpose of this appendix is to characterize and quantify the current and potential risks posed to piscivorous mammals exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain, focusing on total PCBs (tPCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The river is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters. The Primary Study Area (PSA) includes the river and the 10-year floodplain beginning at the confluence of the East and West Branches of the Housatonic River downstream of the GE facility, to Woods Pond Dam (Figure 1.1-2).

A Pre-ERA was conducted to narrow the scope of the ecological risk assessment (ERA) by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A three-tiered deterministic approach was used to screen COPCs. The deterministic assessments compared potential conservative estimates of exposure with conservative adverse effects benchmarks to identify which contaminants are of potential concern to omnivorous and carnivorous mammals in the Housatonic River. A risk quotient (total daily intake/effect benchmark) for piscivorous mammals greater than one in the Housatonic River area resulted in the COPCs being screened through to the next tier assessment, and to the probabilistic ERA, if necessary. In the COPC screening specific to this endpoint, several other COPCs (primarily organochlorine pesticides) were screened out because their actual concentrations in the PSA were likely much lower than the measured values due to laboratory interference (see Section 2.4). In summary, the COPCs that screened through to the probabilistic risk assessment for omnivorous and carnivorous mammals were the contaminants of concern (COCs) tPCBs and 2,3,7,8-TCDD toxic equivalence (TEQ).

Total PCBs detected in Housatonic River media samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is

calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4).

I.1.1 Overview of Approach

A step-wise approach was used to assess the risks of tPCBs and TEQ to piscivorous mammals in the Housatonic River watershed. The four main steps in this process include:

1. Derivation of a conceptual model (Figure I.1-1).
2. Assessment of exposure of piscivorous mammals to COCs (Figure I.1-2).
3. Assessment of the effects of COCs on piscivorous mammals (Figure I.1-3).
4. Characterization of risks to the piscivorous mammal community (Figure I.1-4).

I.1.2 Conceptual Model

The conceptual model presented in Figure I.1-1 illustrates the exposure pathways for piscivorous mammals exposed to tPCBs and TEQ in the PSA.

Total PCBs and TEQ are persistent, hydrophobic, and lipophilic. Therefore, they are bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001). Piscivorous mammals that reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally through diet (mainly fish in addition to aquatic invertebrates, small mammals, and waterfowl) and trophic transfer. Other routes of exposure, considered to be less important to overall exposure, include inhalation, water consumption, and sediment ingestion (Moore et al. 1999).

The problem formulation (see Section 2) identified mink (*Mustela vison*) and river otter (*Lutra canadensis*) as representative species for piscivorous mammals potentially exposed to tPCBs and TEQ from consumption of contaminated prey. Life history profiles for these receptors are found in the text boxes. Additional life history information on these species is presented in Sections I.2.1.5.1 and I.2.1.6.1.

The assessment endpoint that is the subject of this appendix is the survival, growth, and reproduction of piscivorous mammals in the Housatonic River PSA. The measurement endpoints used to evaluate the assessment endpoint were: (1) the comparisons of modeled

exposure to doses reported in the literature to cause adverse effects from tPCBs and other COPCs ingested in the diet to the survival, reproduction, or growth of piscivorous mammals; (2) quantitative field surveys to determine the abundance of piscivorous mammals in the Housatonic River PSA relative to uncontaminated reference areas within the watershed; and (3) a feeding study using fish collected from the PSA to determine whether a diet of site-specific fish has adverse effects on the survival and reproduction of farm-raised mink. The feeding and field studies are discussed in the risk characterization section, and were not used to develop effect levels for comparison to modeled results to maintain the independence of these lines of evidence.

I.1.3 Exposure Assessment

The exposure assessment estimates the exposure of piscivorous mammals to tPCBs and TEQ in the Housatonic River PSA (Figure I.1-2), beginning with a description of the exposure model. The exposure model was parameterized using life history information on the representative species and concentrations of tPCBs and TEQ in prey collected in the PSA and reference areas. Monte Carlo and probability bounds analyses were then conducted to estimate exposure for each COC.

I.1.4 Effects Assessment

The effects assessment provides an overview of the literature on the effects of tPCBs and TEQ to survival, growth, and reproduction of mink and river otter, selected as the representative wildlife species (Figure I.1-3). An extensive literature search was conducted to locate studies with data and information on the survival, growth, and reproduction of the representative species exposed to the COCs. Each of the studies was evaluated using defined acceptability criteria. Studies were selected and used to derive appropriate effects metric.

I.1.5 Risk Characterization

The risk characterization evaluates the likelihood that adverse effects may occur as a result of wildlife exposure to tPCBs and TEQ (Figure I.1-4). Three independent lines of evidence were available to characterize risks to piscivorous mammals from exposure to tPCBs and TEQ:

- **Modeled Exposure and Effects.** The purpose of this line of evidence was to determine the extent to which the concentrations of tPCBs and TEQ ingested in the

diet might cause adverse effects to the survival, reproduction, or growth of piscivorous mammals. A probabilistic food web model was used to estimate exposure of tPCBs and TEQ to mink and river otter. The total daily intakes for mink and otter in the PSA and reference areas were calculated using equations from the *Wildlife Exposure Factors Handbook* (EPA 1993) and related publications. These estimated exposures were compared to the results of toxicological studies reported in the literature to determine if the piscivorous mammalian species are being exposed to tPCBs and TEQ at levels likely to induce adverse effects.

- **Field Surveys.** The purpose of the field surveys was to determine the abundance of piscivorous mammals in the Housatonic River floodplain relative to uncontaminated reference areas within the watershed. Both EPA and GE performed field surveys.

- **Mink Feeding Study.** The purpose of the feeding study was to determine whether a diet containing varying amounts of Housatonic River fish causes adverse effects in farm-raised mink. The effects metrics included survival, growth, and reproduction of females and their offspring.

A weight-of-evidence (WOE) assessment was conducted to combine the results from each line of evidence. The section concludes with a discussion of sources of uncertainty in the assessment of risks of COCs to piscivorous mammals, and the conclusions of the risk characterization.

I.1.6 Organization

This appendix is organized as follows:

- Section I.2 describes the exposure model, input parameters, and techniques to propagate uncertainty. Also presented in this section are the input data and exposure results for mink and otter.

- Section I.3 describes the effects to piscivorous mammals exposed to tPCBs and TEQ.

- Section I.4, Risk Characterization, presents the modeled exposure and effects, field surveys, and feeding studies of mink. This section also includes a discussion of the WOE assessment, the sources of uncertainty regarding risk estimates, and the conclusions regarding risk for piscivorous mammals exposed to tPCBs and TEQ in the Housatonic River.

I.2 EXPOSURE ASSESSMENT

This section evaluates exposure of representative species of piscivorous mammals to tPCBs and TEQ in Reaches 5 (confluence to Woods Pond) and 6 (Woods Pond), also referred to as the Primary Study Area (PSA) of the Housatonic River. Exposure assessments were also conducted for two reference areas for comparative purposes. One of the reference areas is located upstream of the GE facility on the East Branch of the Housatonic River in Dalton, MA. The other reference area is Threemile Pond located in Sheffield, MA, which is in the Housatonic River drainage, but at a higher elevation draining to the river.

The representative species for the piscivorous mammal endpoint include two indigenous species: mink and river otter. Exposure of these species to tPCBs and TEQ was estimated using a total daily intake (TDI) model adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993).

The exposure period was chosen to account for the seasonal variability in composition of the diet of mink (which can be very opportunistic), and, to a lesser extent, river otter. The exposure period also encompasses the reproductive cycle of these species. Total PCBs and TEQ are known to cause reproductive effects in piscivorous mammals at low concentrations after chronic exposures (see Section I.3). Exposure was also controlled in the mink feeding study, but is not addressed in this section.

This section begins with a description of the general model used for each of the exposure assessments. Subsequent sections describe the inputs used in the exposure model. The section concludes with a description of the Monte Carlo and probability bounds analyses conducted to estimate total daily intake of tPCBs and TEQ for each of the representative piscivorous mammal species in the Housatonic River PSA and reference areas.

I.2.1 Exposure Model for Piscivorous Mammals

The model used to estimate exposure of piscivorous mammals to tPCBs and TEQ focused on ingestion of these contaminants through the diet. Other exposure routes (e.g., water), while considered, were determined to be of much less importance for estimating risk from tPCBs and TEQ. The model used in the exposure analysis was:

$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i \qquad \text{(Eq. 1)}$$

where

TDI	=	Total daily intake (mg/kg bw/d tPCB, ng/kg bw/d TEQ)
P_i	=	Proportion of the *i*th food item in the diet (unitless)
FIR	=	Normalized food intake rate (kg/kg bw/d)
C_i	=	Concentration in *i*th food item (mg/kg tPCB, ng/kg TEQ)
FT	=	Foraging time in the study area (unitless)

The dietary component of the model reflects the diet of mink and river otter in terms of the proportion of fish, birds, invertebrates, and mammals consumed. The model does not include intake terms for drinking water and preening exposure routes because the magnitude of intake from those sources is likely to be very small. The effects of omitting those components on the overall risk assessment are discussed in the uncertainty section. Exposure was calculated for Reaches 5 and 6 as well as for the two reference locations.

I.2.1.1 Choosing Exposure Distributions

Input distributions were generally assigned as follows:

- Lognormal distributions for variables that were right-skewed with a lower bound of zero and no upper bound (e.g., amount of contaminant of concern in fish);
- Beta distributions for variables bounded by zero and one (e.g., proportion of prey item in the diet);
- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight); and
- Point estimates for minor variables or variables with low coefficients of variation.

In certain situations (e.g., poor fit to the data), other distributions were selected or other approaches were used. These latter situations are described in detail where they occurred in the following sections.

I.2.1.2 Incorporation of Spatial Averaging in Derivation of Estimated Prey Concentrations

Concentrations of tPCBs and TEQ vary spatially in prey. Many wildlife species forage over distances ranging from tens of meters to greater than 10 km. An individual integrates the spatial variation in tissue concentrations of their prey. Therefore, estimates of central tendency were used in the exposure model as an expression of the spatial and temporal averaging of COC concentrations in prey tissue. In the probabilistic exposure analyses, it was assumed that the spatially and temporally averaged exposure estimates did not vary between individuals foraging in the same area (EPA, 1999).

To account for uncertainty due to sample size, the measure of centrality in the Monte Carlo analysis was the minimum of: (1) the 95% upper confidence limit calculated using the Land H-statistic, or (2) the maximum concentration measured In the probability bounds analyses, however, the uncertainty regarding the arithmetic mean was accounted for with a different procedure. The procedure involved using the Land H-statistic to estimate the lower 95% and upper 95% confidence limits (CLs) on the mean (Gilbert 1987), and then using these values to derive bounds on all possible distributions that exist within this range. This approach results in an expression of the uncertainty about the true value of the arithmetic mean that arises due to limited sample sizes. In cases where the 95% upper confidence limit could not be estimated or exceeded the maximum, other techniques were used to derive bounds on the mean. These techniques and criteria for their use are described in Section 6.5.

I.2.1.3 Techniques for Propagating Uncertainty

In this assessment, two types of probabilistic analysis were undertaken: Monte Carlo analysis and probability bounds analysis. The former is the most common probabilistic method employed in risk assessments, and guidance for its use in Superfund assessments is available (EPA 1997). Although Monte Carlo methods are appropriate for the determination of exposure risks when input distributions are known precisely, they do not adequately represent the effects of uncertainty around the input distributions (Ferson 1996). In many ERAs, the available data are limited and consequently the input distributions used to calculate risks are uncertain.

Probability bounds analysis is a tool for separating variability and uncertainty to obtain bounds on the result that explicitly account for the uncertainty about the input distributions. As in Monte Carlo analysis, the overall slopes of the bounds indicate how much variability exists in the system. The distance between the bounds is an indication of the uncertainty that exists due to lack of knowledge. More detailed descriptions of Monte Carlo analysis and probability bounds analysis are provided in Section 6.5 and Appendix C.4.

For each Monte Carlo analysis, a sensitivity analysis was performed to determine the relative influence that each input variable had on the output exposure distribution. This was done by determining the correlation between randomly chosen input values for a particular variable, and the corresponding output exposure estimates derived from the simulation. Input variables that have a strong influence on the output exposure estimate tend to have high correlation coefficients, although this interpretation becomes problematic when there are dependencies between input variables. The results of the sensitivity analyses are included in this exposure assessment.

The exposure models for the ERA contain multiple variables, some of which may be correlated. The assumption of independence can be inappropriate because even subtle dependencies can affect the estimated exposure. If correlations are not accounted for, the variance and the tails of the exposure distribution may be poorly estimated. This risk assessment uses several approaches to quantify correlations between variables. These approaches include simulation of observed correlations, assumption of perfect covariance (e.g., when the diet consists of two prey items, the proportion of one item in the diet is equal to one minus the other item), or no assumptions at all about dependencies (all possible relationships between two variables can occur). The specific approach used depended on the type of data and the application. In cases where independence of variables seemed intuitively obvious (e.g., COC concentration in the prey item and proportion of that item in the diet), independence was assumed.

I.2.1.4 Treatment of Non-Detects

The approach for generating summary statistics or distributions when a data set includes samples with COC concentrations below the detection limit is described in detail in Appendix C.2. In summary, for data sets with some non-detect concentrations, summary statistics were generated

assuming that the contaminant concentration was zero (ND = 0), and assuming that contaminant concentrations were equal to the detection limit (ND = DL). If the ratio between the summary statistic of interest (e.g., mean) calculated assuming ND = DL and the summary statistic assuming ND = 0 was less than or equal to 1.3, the analysis was performed assuming that all non-detected chemical concentrations were equal to half the detection limit (ND = ½ DL).[1] If the ratio was greater than 1.3 and the sample size and detection frequency were sufficient (i.e., n >5, detection frequency >25%), the probability plot method described in Appendix C.2 was used to generate estimated concentrations for samples with COC concentrations below the detection limit. If the sample size and detection frequency were not adequate, then the exposure calculations were done assuming ND = 0, ND = ½ DL, and ND = DL for the Monte Carlo analysis, and ND = 0 and ND = DL for the probability bounds analysis. For a more detailed discussion of the treatment of non-detects, see Appendix C.2, Figure 4.

I.2.1.5 Mink

Mink (Figure I.2-1) were selected as a representative species for piscivorous mammals because they are known to occur in the Housatonic River watershed, the area contains suitable mink habitat (Appendix A), and their prey are directly and indirectly (through their diet) exposed to COCs. Furthermore, piscivorous mammals such as mink are exposed to high levels of tPCBs and TEQ because of their consumption of aquatic prey and their position at the top of the food chain. The diet of mink, consisting of fish, crayfish, amphibians, insects, and waterfowl (Linscombe et al. 1982) makes them susceptible to exposure to bioaccumulative substances (Moore et al. 1997, 1999). Moreover, mink have demonstrated a sensitivity to tPCBs and TEQ (Bleavins et al. 1980). The literature indicates that there is a positive correlation between tPCB concentrations in fish and concentrations of PCBs in mustelid species (Foley et al. 1988). Total PCBs have been shown to accumulate readily in wild mink and reach harmful concentrations (Aulerich et al. 1971; Bleavins et al. 1980; Foley et al. 1988; Heaton et al. 1995; Wren et al. 1987a, 1987b).

---

[1]This decision criterion supplements the procedures described in Appendix C.2.

I.2.1.5.1 Life History

Life History of Mink (Mustela vison)

Mink are small, fur-bearing animals with characteristic elongated bodies, short legs, and long tails. Mink are one of the most widespread mammalian carnivores and their range spans much of the continental United States and Canada.

Habitat – Require access to open water such as streams, tidal flats, marshes, shallow rivers, lakes, and swamps, also suitable cover in the form of overhanging vegetation, rock crevices, exposed roots, log jams, and undercut banks.

Home Range – Adult males occupy home ranges exclusive of other adult males, and may include the ranges of one or more females. Male home ranges vary from 309 to 776 ha and female home ranges range from 7.8 to 20.4 ha. Riverine home ranges are linear (between 1.0 and 6.0 km of shoreline), while those in marsh habitats tend to be more circular.

Dietary Habits – Primary food items include fish, small mammals, benthic invertebrates, birds, and amphibians. Opportunistic with a diet that varies depending on the availability of prey items. Mean percentage of prey items in diet: fish 23%; mammals, 15%; birds, 11.0%; invertebrates, 36%; and amphibians and reptiles, 15%.

General Description

Mink are small fur-bearing animals belonging to the family Mustelidae. Mustelid species differ in size, but in general have long bodies, short legs, non-retractile claws, short heads, medium to long tails, and well-developed anal musk glands (Whitaker and Hamilton 1998). Mink are one of the most widespread mammalian carnivores in North America. Their range includes much of the continental United States and Canada, except for areas in the far north, Mexico, and arid areas of the United States.

Habitat

Mink are semi-aquatic, thus, the presence of a water body is an important factor in habitat selection. Types of water bodies used by mink include small streams, tidal flats, cattail marshes, rivers, lakes, bogs, swamps, and bottomland woods (Gerell 1970, as cited in Allen 1986). Surface water must be present for at least 9 months out of the year to provide optimal foraging habitat (Allen 1986). Mink typically forage within sight of open water (Kurta 1995) and do not stray more than 200 m from the shoreline (Allen 1986). The size of the water body and water depth can play a role in habitat selection. For example, large open water areas are not suitable for mink, unless the water is shallow (Dunstone and O'Connor 1979).

Cover availability also affects habitat use by mink. Cover can be provided by overhanging or emergent vegetation, rocks or rock crevices, exposed roots, debris, log jams, undercut banks, or boulders (Allen 1986). In smaller wetlands, the presence of woody vegetation within 100 m of the edge of the shoreline can further enhance habitat use by mink (Allen 1986). The optimum cover conditions for mink in emergent wetlands range from 50 to 75% cover. The availability of suitable den sites may also affect habitat use by mink. Typically, several dens are located close to preferred foraging sites within an individual's home range (Allen 1986). Dens are established in burrows excavated by other animals, tree root cavities, rock piles, log jams, and old beaver and muskrat lodges. Several dens may be established and used at the same time (Eagle and Sargeant 1985; Allen 1986; Lariviere 1999).

Dietary Habits

To determine the dietary preferences of mink, a literature search was performed to identify field studies that had examined mink dietary composition through scat and/or stomach content analysis. Based on the studies listed in Table I.2-1, the primary food items of mink likely include small mammals, fish, benthic invertebrates, birds, amphibians, and insects.

Mink are opportunistic and their diet varies widely depending on the availability of prey items. For example, Gilbert and Nancekivell (1982) examined mink scats in a drainage system dominated by lakes and a second system dominated by streams. They found that mink diet between the two systems varied in proportion to availability in each habitat. However, fish were an important component of the diet in both systems. Proulx et al. (1987) reported mink diet based on scat analysis in Ontario marsh habitat and found that small mammals and birds were the primary components.

To estimate the diet for mink living in the Housatonic PSA, studies in Table I.2-1 were evaluated for similarity to the habitat in the PSA. For example, Birks and Dunstone (1985) surveyed mink diet along the Scottish coastline but that study was excluded from the analysis because the prey species and habitat along the coast of Scotland are substantially different from that found in a freshwater riverine system in Massachusetts. Studies that reported mink diets from the northeastern United States and southeastern Canada were preferred over studies from other locations. This resulted in five studies selected to estimate the mink diet in the PSA, including

Hamilton (1940), Hamilton (1959), Alexander (1977), Burgess and Bider (1980), and Sealander (1943). Based on the diets reported in these five studies, the mean percentage of each prey item estimated in the mink diet was: fish, 23% (range 0 to 67.4%); mammals, 15% (range 0 to 25%); birds, 11.0% (range 0 to 39%); invertebrates, 36% (range 0 to 54%); and amphibians and reptiles, 15.0% (0 to 30%) (Table I.2-2). The dietary component represented by birds was generally composed of waterfowl. Similarly, crayfish dominated the invertebrate component of the diet. Distributions were used to represent each dietary component to account for the variation observed in the mink diet studies reported in the literature.

Fish Size Range

Melquist et al. (1981) found that fish taken by mink were mostly cyprinids between 7 and 12 cm long. Similarly, Hamilton (1940) recorded that the average length of fish taken by mink ranged from 7.6 to 10.2 cm. According to Alexander (1977), mink in rivers and streams in lower Michigan and New York consume fish ranging from 15 to 18 cm. Based on this information, fish used in the exposure analyses were limited to a minimum length of 7 cm and a maximum length of 20 cm.

Mink Home Range

Adult male mink occupy home ranges that are exclusive of other adult males, but may include the home ranges of one or more females (Mitchell 1961; Birks and Dunstone 1985; Whitaker and Hamilton 1998). Male home ranges are larger (average of 85.4% larger) than those of females and have been reported to range from 766 to 1,920 acres (310 to 777 hectares) (Arnold and Fritzell 1987, as cited in Lariviere 1999; Arnold and Fritzell 1990; Whitaker and Hamilton 1998). Female home ranges in Montana were from 19.3 to 50.4 acres in size (7.81 to 20.4 hectares) (Mitchell 1961). Riverine home ranges are basically linear, whereas those in marsh habitats tend to be more circular (Birks and Linn 1982; Eagle and Whitman 1987, as described in the EPA *Wildlife Exposure Factors Handbook*, 1993). The foraging range of mink ranges between 1.0 and 6.0 km of shoreline (Gerell 1970; Linn and Birks 1981). Thus, one to several mink could have their entire foraging ranges located within the area of the river from the confluence to Woods Pond, or more if tributary habitat is included in the estimate. In this assessment, exposure for mink is estimated for the individuals that spend 100% of their time foraging in the PSA. However, estimates of exposures for mink that spend 50, 25, and 10% of

their time foraging in the PSA are also provided to demonstrate a range of COC exposure for mink that only partially use the PSA for foraging. This was accomplished by multiplying the calculated exposures for 100% foraging time in the PSA by area use factors of 0.50, 0.25, and 0.10.

I.2.1.5.2 Exposure Model Input Distributions

The input parameters used in the Monte Carlo and probability bounds analyses are summarized in Table I.2-3. The following sections provide additional information for each variable in the exposure models.

Body Weight (BW)

Body weight is not used in the model directly, but is a required variable in allometric models (e.g., Nagy 1987) to estimate food intake or free metabolic rates. Average body weights (wet weight of wild animals) of female mink range from 550 g (Mitchell 1961) to 970 g (Hornshaw et al. 1983) and males range from 630 to 1,000 g (Whitaker and Hamilton 1998). For the Monte Carlo analysis, the mean weight of females was estimated to be 685 g with a standard deviation of 122. The exposure modeling considers females because the effects metric used in the risk assessment is female reproductive success. Body weights were assumed to be distributed normally. There is low uncertainty associated with this variable. The uncertainty in this variable is due to variability, rather than lack of knowledge or data (i.e., the variable is easily measured and many studies have been conducted that measured this variable). Accordingly, the same distribution was used in the probability bounds analysis.

Food Intake Rate (FIR)

The daily energy requirements of mink vary depending on environmental conditions and the stage of the reproductive cycle. However, the long-term average daily consumption of dry matter is about 0.040 kg/kg of body mass for males and 0.0530 kg/kg of body mass for female captive mink (Bleavins and Aulerich 1981; Lariviere 1999). A 1.0-kg mink living in a laboratory requires about 150 kilocalories (kcal) of digestible energy every day for maintenance. A nursing female can require three times that amount at 3 weeks post-partum (Lariviere 1999). However, a nursing female food intake rate was not considered in this assessment because nursing is a short-term event relative to the time scale of this assessment (annual exposure). The

time scale of this exposure assessment was chosen to be approximately 1 year based on the extended reproductive cycle of mink (mating starts in early March) and the duration of the mink feeding study, which evaluated the effects on young mink until they were 6 months old.

For the purpose of the ERA for piscivorous mammals, *FIR* was estimated using an allometric equation rather than using literature-reported values for captive mink. An allometric model-derived *FIR* better approximates the increased energy demand of wild mink resulting from higher activity levels incurred while foraging, defending and inspecting territory, and avoiding predators (Lamprey 1964; Buechner and Golley 1967; Koplin et al. 1980).

Food intake rate (FIR) is derived using the following equation:

$$FIR = \frac{FMR}{\sum_{n}^{i=1} AE_i \cdot GE_i} \qquad \text{(Eq. 2)}$$

where:

FMR	=	Normalized free metabolic rate (kcal/kg bw/d)
AE_i	=	Assimilation efficiency of *i*th food item (unitless)
GE_i	=	Gross energy of *i*th food item (kcal/kg)
P_i	=	Proportion of the *i*th food item in the diet (unitless).

The exposure model used the allometric relationship of Nagy et al. (1999) developed for carnivorous mammals to estimate the free metabolic rate for female mink. The general form of the model is:

$$FMR\ (kJ/d) = a \cdot BW(g)^b \qquad \text{(Eq. 3)}$$

For the Monte Carlo and probability bounds analyses, *FMR* was estimated using a probabilistic approach where distributions of the input variables (body weight [*BW*], *a*, *b*) were used rather than point estimates. The slope (*a*) and power (*b*) distributions were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999), assuming an underlying normal distribution for each. For carnivorous mammals, log (*a*) had a reported mean

of 0.367 and a standard error of 0.223, and (*b*) had a reported mean of 0.850 and a standard error of 0.055 (Nagy et al. 1999).

Assimilation Efficiency (AE_i)

Average assimilation efficiency for mammals consuming fish and amphibians is 0.91, invertebrates 0.87, and birds and mammals 0.84 (EPA 1993; Grodzinski and Wunder 1975; Barrett and Stueck 1976). No data were available for assimilation efficiency of mammals consuming amphibians, but it is likely to be similar to that for mammals consuming fish. These variables were treated as point estimates in Monte Carlo simulations and probability bounds analyses because of their relatively small coefficients of variation.

Gross Energy (GE_i)

The mean gross energies reported in the literature were as follows: fish and amphibians (assumed for amphibians) 1.20 kcal/g (Thayer et al. 1973; EPA 1993), invertebrates 1.10 kcal/g (Jorgensen et al. 1991; Minnich 1982; Thayer et al. 1973), and birds and mammals 1.8 kcal/g (EPA 1993). These variables were treated as point estimates in the analysis because of their relatively small coefficient of variation. Gross energy is easily measured and thus, measurement error is likely to be low.

Proportions of Dietary Items (P_i)

The primary food items in the mink diet include small mammals, fish, benthic invertebrates (crayfish), birds (waterfowl), amphibians (Alexander 1977; Burgess and Bider 1980; Cowan and Reilly 1973; Gilbert and Nanckivell 1982; Hamilton 1959, 1940; Melquist et al. 1981; Proulx et al. 1987) (Table I.2-2). Combining the available data, an average of 23% (range of 0 to 64.7%) of the mink diet consists of fish. Mammals comprise 15% of the diet, reptiles and amphibians also constitute an average of 15% (range of 0 to 30%) of the diet, birds (i.e., waterfowl) 11% (range of 0 to 39%) of the diet, and invertebrates constitute 36% of the diet. The proportion of prey type in the diet was assumed to follow a beta distribution in the Monte Carlo analysis and was parameterized to approximate the above averages and ranges (Table I.2-3).

The beta distribution is not an available option in RiskCalc, the software used for conducting the probability bounds analyses. As an alternative, minimum, mean, and maximum values were specified for each dietary item using the means and ranges described above. The minimum,

mean, and maximum values were then included as a distribution-free statement in RiskCalc. The results bound all possible distributions, given the specified minimum, mean, and maximum values specified for the dietary items.

I.2.1.5.3 Exposure Concentrations

This section discusses the concentrations of tPCBs and TEQ in prey of mink in Reaches 5 and 6 and in prey of river otter in Reaches 5 and 6 combined. For the sake of brevity, only one exposure scenario is discussed in detail. The remainder of prey concentration data for other exposure scenarios (e.g., reference areas) is summarized in the text and presented in Tables I.2-4, I.2-5, I.2-12, and I.2-13).

EPA (1992) states that because of the uncertainty associated with estimating the true average concentration for a site, the 95% upper confidence limit (UCL) of the arithmetic mean should be used for this variable. For lognormal data, EPA (1992) recommends the Land method using the H-statistic. Several authors (e.g., Ott 1995; Seiler and Alvarez 1996; Hattis and Burmaster 1994) have argued that concentrations of contaminants in environmental media tend to be lognormally distributed and that this may be expected because of mechanistic reasons. Current EPA guidance (EPA 1997; also see Haimes et al. 1994) state that distributions should be chosen for input variables on the basis of mechanistic or theoretical reasons, if possible, because such distributions have the highest degree of confidence. As a result, concentrations of contaminants in prey were assumed to be lognormally distributed in this ERA, and hence the Land H-statistic was used to estimate the 95% UCL. To determine the reasonableness of this assumption, the Shapiro-Wilk test was used to test for lognormality. Over two thirds of the data sets used in the wildlife assessments passed the test for lognormality (i.e., p 0.05), which supports the assumption of lognormality for concentrations of contaminants. That said, it is recognized that the Land method can produce high values for the UCL, particularly when data are not lognormally distributed, samples size is small, or variation is high (Singh et al. 1997; Schultz and Griffin 1999). EPA's (1992) guidance recognized this problem and recommended that the maximum detected concentration be used when the calculated UCL exceeds this value. This guidance was followed in this assessment.

Concentration of tPCBs in Fish in Reach 5 (C_i)

A number of fish species (e.g., largemouth bass, yellow perch, and pumpkinseed) were sampled in Reach 5. Of the samples collected, 99 were fish between 7 and 20 cm in length, the approximate length of fish consumed by mink (Melquist et al. 1981; Alexander 1977; Hamilton 1940). The Land H-statistic was used to determine the lower and upper 95% confidence limits (CLs) on the mean. The lower value of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 38.3 mg/kg. For the probability bounds analysis, the lower 95% and upper 95% confidence limits determined from the Land H-statistic were used to parameterize a distribution-free statement in RiskCalc. The lower and upper CLs were 31.7 mg/kg and 38.3 mg/kg, respectively (Table I.2-4). Table I.2-4 summarizes concentration parameters for fish and invertebrates.

Estimation of Whole Body Waterfowl Concentrations

Waterfowl (wood ducks and mallards) were collected from the PSA and the Threemile Pond reference area. The birds collected from the PSA resided in or had been raised within the PSA, based on observations for field monitoring activities. However, birds collected at Threemile Pond were believed to have been migrating because they were all collected in a single trap day after no birds had been observed during previous trapping activity. Differences in Aroclor/congener compositions between the reference tissue samples and the site-related samples support this observation. Breast and liver tissue from the waterfowl were analyzed for tPCBs and TEQ congeners. However, offal concentrations of tPCBs and TEQ were not determined. The following method was used to estimate offal concentrations and whole body concentrations of tPCBs and TEQ.

Offal concentrations were estimated for high, moderate, and low concentration scenarios. The low concentration scenario assumed that the offal had a COC concentration of zero. The moderate concentration scenario assumed that the offal had a COC concentration equal to the breast concentration. The high concentration scenario assumed that the offal had a COC concentration equal to the liver concentration (where tPCBs and TEQ tend to accumulate at high concentrations relative to other organs or tissues).

For each concentration scenario, the whole body concentration (*wbconc*)was estimated using the formula:

$$wbconc = \frac{(bwt \cdot bconc) + (lwt \cdot lconc) + (owt \cdot oconc)}{(bwt + lwt + owt)} \qquad \text{(Eq. 4)}$$

where:

bwt = Tissue weight for the breast (kg)
bconc = Concentration for the breast (kg)
lwt = Tissue weight for the liver (kg)
lconc = Concentration for the liver
owt = Tissue weight for the offal (mg/kg tPCB; ng/kg TEQ)
oconc = Estimated concentration for the offal (mg/kg tPCB; ng/kg TEQ).

Concentration of tPCBs in Birds for Reach 5 (C_i)

A total of 18 duck tissue samples were available from Reach 5. The Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The reconstitution of the bird tissues (see preceding section) was considered in applying the Land H-statistic. The approach outlined in Section 6.4 was adapted to account for the uncertainty about whole body concentration of tPCBs following reconstitution. For tPCBs in birds, the assumption of how to treat the low (offal = zero concentration) and high (offal = higher of breast and liver concentration) estimates of whole body concentrations had an important influence on the estimated tPCB concentrations. Therefore, a triangular distribution was used in the Monte Carlo analysis to capture the uncertainty about the estimate of the measure of centrality. The Land H-statistic was used to determine the upper 95% confidence limits on the mean concentrations for the low, moderate, and high concentration estimates. The minimum value of the triangular distribution was calculated using the lower of the upper 95% CL and the maximum value in the low (offal = zero) estimate. The best estimate of the triangular distribution was calculated using the lower of the upper 95% CL and the maximum value in the moderate (offal = breast) estimate. The triangular distribution maximum was calculated using the lower of the upper 95% CL and the maximum value for the high (offal = liver) estimate. In this case, the minimum, best estimate, and maximum values were all the upper 95% CLs. The resulting triangular distribution

parameters were: minimum = 2.38 mg/kg, best estimate = 11.4 mg/kg, and maximum = 16.4 mg/kg.

In the probability bounds analysis, the uncertainty regarding the tPCB concentrations was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values were calculated using the higher of the lower 95% CL and the minimum value from the offal = zero estimate and the lower of the upper 95% CL and the maximum value from the offal = liver estimate, respectively. In this case, the lower and upper 95% CLs were chosen, and the resulting values ranged from 1.25 to 16.4 mg/kg. Table I.2-4 provides the additional concentration parameters for birds, mammals, and amphibians.

I.2.1.5.4 Concentrations of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Prey

Concentration of TEQ in Fish in Reach 5 (C_i)

TEQ were determined for 54 fish samples that ranged from 7 to 20 cm in length collected in Reach 5. Using the approach outlined in Section 3.4, the assumption of how to treat non-detected congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean, assuming that non-detected congener concentrations were equal to half the detection limit (ND = ½ DL). The lower of the upper 95% CL and the maximum value was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 443 ng/kg. In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values, assuming that non-detected congeners were equal to half the method detection limit (DL), were calculated using the greater of the lower 95% CL and the minimum value and the lower of the upper 95% CL and the maximum value, respectively. The resulting values ranged from 346 to 443 ng/kg (Table I.2-5).

Table I.2-5 includes the other concentration parameters for fish, invertebrates, and mammals.

Concentration of TEQ in Birds in Reach 5 (C_i)

TEQ was determined in 18 duck tissue samples collected from Reach 5. The Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The reconstitution of the bird tissues (see preceding section) was considered in applying the Land H-statistic. The approach outlined in Section 6.4 was modified to account for the uncertainty about whole body concentration of TEQ following reconstitution. For TEQ in birds, the assumption of how to treat the low (offal = zero concentration) and high (offal = higher of breast and liver concentration) estimates of whole body concentrations did have an important influence on the estimated TEQ concentration. Therefore, a triangular distribution was used in the Monte Carlo analysis to capture the uncertainty about the estimate of the measure of centrality.

Using the approach outlined in Section 6.4, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations. The assumption of how to treat non-detected congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that ND = ½ DL. The Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean concentrations for the low, moderate, and the high estimates. The minimum value of the triangular distribution was calculated using the lower of the upper 95% CL and the maximum value in the low (offal = zero) estimate. The best estimate of the triangular distribution was calculated using the lower of the upper 95% CL and the maximum value of the moderate (offal = breast) estimate. The triangular distribution maximum was calculated using the lower of the upper 95% CL and the maximum value for the high (offal = liver) estimate. In this case, the minimum, best estimate, and maximum values were all the upper 95% CLs. The resulting triangular distribution parameters were: minimum = 564, best estimate = 2,459, and maximum = 3,538 ng/kg.

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values were calculated using the higher of the lower 95% CL and

the minimum value with offal = zero and the lower of the upper 95% CL and the maximum value with offal = liver, respectively. In this case, the lower and upper 95% CLs were chosen, and the resulting values ranged from 230 to 3,538 ng/kg. Table I.2-5 includes the exposure parameters used for birds and amphibians.

I.2.1.5.5 Exposure Model Results

The results of the Monte Carlo and probability bounds analyses for exposure of mink to tPCBs and 2,3,7,8-TCDD TEQ in the PSA are presented in Tables I.2-6 and I.2-7, respectively. Figures I.2-2 to I.2-9 depict the cumulative distributions of total daily intake of tPCBs and TEQ, as well as the corresponding probability bounds for mink at the four areas.

Exposure to tPCBs in Reach 5

PSA Foraging Time: 100%

The Monte Carlo analysis indicated that exposure of mink to tPCBs in Reach 5 ranged from a minimum of 0.308 to a maximum of 82.5 mg/kg bw/d. The mean exposure was 5.29 mg/kg bw/d, and the median exposure was 3.97 mg/kg bw/d (Table I.2-6). Of the exposure estimates, 90% were between 1.15 and 13.6 mg/kg bw/d. Figure I.2-2 depicts the cumulative distribution of total daily intake rates of tPCBs for mink at this location.

The probability bounds estimated for mink foraging in Reach 5 are shown in Figure I.2-2. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 0.0244 and 4.11 mg/kg bw/d. The 50th percentile ranged between 0.292 and 8.47 mg/kg bw/d, and the 90th percentile ranged between 1.72 and 22.5 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 1.52, the 50th percentile was 3.97, and the 90th percentile was 10.4 mg/kg bw/d.

Mink foraging in Reach 5 less than 100% of time would have their exposures reduced in proportion to the amount of time they spend outside the PSA (e.g., 90% foraging outside Reach 5 would reduce exposure by a factor of 10).

Sensitivity analysis revealed that the *FMR* slope term (Pearson correlation coefficient $r_p = 0.840$) was the most important variable, followed by the *FMR* power term ($r_p = 0.459$), proportion of fish in diet ($r_p = 0.140$), and proportion of birds in the diet ($r_p = -0.140$) (Table I.2-8).

Exposure to TEQ in Reach 5

PSA Foraging Time: 100%

The Monte Carlo analysis indicated that exposure of mink to TEQ ranged from a minimum of 7.90 to a maximum of 3,614 ng/kg bw/d in Reach 5. The mean exposure was 198 ng/kg bw/d and the median exposure was 147 ng/kg bw/d (Table I.2-7). Of the exposure estimates, 90% were between 42.5 and 509 ng/kg bw/d. Figure I.2-6 shows the cumulative distribution of intake rates of TEQ for mink in Reach 5.

The probability bounds estimated for exposure to TEQ for mink foraging in Reach 5 are depicted in Figure I.2-6. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 0.329 and 177 ng/kg bw/d. The 50th percentile ranged between 3.97 and 367 ng/kg bw/d, and the 90th percentile ranged between 17.0 and 982 ng/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 55.8, the 50th percentile was 147, and the 90th percentile was 392 ng/kg bw/d (Table I.2-7).

Mink foraging in Reach 5 less than 100% of time would have their exposures reduced in proportion to the amount of time they spend outside the PSA (e.g., 90% foraging outside Reach 5 would reduce exposure by a factor of 10).

Sensitivity analysis revealed that the *FMR* slope term (Pearson correlation coefficient $r_p = 0.849$) was the most important variable, followed by *FMR* power term ($r_p = 0.478$), proportion of mammals in diet ($r_p = 0.138$), proportion of fish in diet ($r_p = -0.0757$), proportion of invertebrates in diet ($r_p = -0.0751$), concentration of TEQ in birds ($r_p = 0.00586$), and body weight ($r_p = -0.0504$). Table I.2-9 lists the remaining parameters.

Other Exposure Scenarios

The mean exposure to tPCBs of mink foraging in Reach 6 100% of time was 3.87 mg/kg bw/d (Table I.2-6), which is slightly lower than that found in Reach 5. Exposure of mink to tPCBs at the upstream and Threemile Pond reference areas was about 200 times lower than in Reaches 5 and 6. The mean exposures at the upstream and Threemile Pond were 0.0177 mg/kg bw/d and 0.152 mg/kg bw/d, respectively.

For comparison purposes, exposures were calculated for scenarios involving mink that forage only partially in the PSA. The mean exposure to PCBs was 0.387 mg/kg bw/d for mink obtaining 10% of their diet in the PSA. The mean daily exposures for mink feeding 50 and 25% of the time in the PSA were 1.94 and 0.968 mg/kg, respectively.

Mean exposures to TEQ of mink feeding 100% of the time in Reach 6, the upstream reference area, and the Threemile Pond reference area were 153, 6.48, and 42.6 ng/kg bw/d, respectively. Exposure in Reach 6 is about 30% lower than in Reach 5, but is about 23 times higher than in the upstream reference area, and nearly 4 times higher than in the Threemile Pond reference area. In comparison, mean TEQ exposure of mink feeding 10% of the time in Reach 6 was 15.3 ng/kg bw/d. The mean daily exposures for mink feeding 50 and 25% of the time in the PSA were 76.5 and 38.3 ng/kg.

For each exposure scenario, sensitivity analysis revealed that the *FMR* power term and the *FMR* slope term were the most sensitive variables in the Monte Carlo analyses. The exposure model results for exposure of mink to tPCBs and TEQ in the PSA and reference areas are presented in Tables I.2-6 and I.2-7, and the sensitivity analyses are presented in Table I.2-8 and Table I.2-9.

I.2.1.6 River Otter

The river otter (Figure I.2-10) was selected as a representative species for piscivorous mammals because they are known to occur in the Housatonic River watershed, the PSA contains suitable otter habitat (Appendix A), and the fish and crayfish they consume are directly and indirectly (through their diet) exposed to tPCBs and TEQ. Because otter feed mainly on fish, they are exposed to high concentrations of bioaccumulative contaminants.

I.2.1.6.1 Life History

Life History of River Otter (Lutra canadensis)

River otter are long-bodied, short-legged, semi-aquatic mustelids that occur throughout most of Canada and the continental United States. Male otter in the eastern United States are quite large and range in weight from 8 to 11 kg. Females range from 7.5 to 8 kg.

Habitat - Remain close to aquatic habitats such as lakes, marshes, streams, seashores, rivers, creeks, and bayous. In New England, preferentially select riverine and lacustrine systems. Have numerous denning and nesting sites within home range, used over the course of the year. Denning and resting sites may be located in log jams, riparian vegetation, snow or ice cavities, riprap, talus rock, boulders, brush and log piles, undercut banks, and dens constructed by other animals.

Home Range - Average size of the home range for adult otter is about 30 km of shoreline. Lactating females have the smallest home ranges. Other than family groups, are typically solitary. Will form temporary associations that may consist of related or unrelated individuals. Home ranges shown to overlap extensively, with some sharing essentially the same home range.

Dietary Habits - Diet somewhat variable, primarily consisting of aquatic animals, particularly fish; other prey includes crayfish, amphibians, turtles, birds, small mammals, and insects. Prefer to forage in shallow water and eat primarily slow-moving, shallow-dwelling fish, such as chubs, suckers, catfish, daces, darters, and schooling fish such as bluegill and other sunfish.

General Description

River otter are long-bodied, short-legged, semi-aquatic mustelids. They occur throughout most of Canada and the continental United States, except for the southwestern United States (Lowery 1974; Choate et al. 1994). For otter from the eastern United States, males range in weight from 8 to 11 kg and females from 7.5 to 8 kg (Whitaker and Hamilton 1998). Body length ranges from 66 to 76 cm, with a 30 to 43 cm tail.

Otter Home Range

River otter use areas associated with aquatic habitats such as lakes, marshes, streams, seashores, rivers, creeks, and bayous, especially those bordered by trees. In New England, river otter will preferentially select riverine and lacustrine systems. Habitat use by river otter in Maine is positively correlated with the length of the stream and average shoreline diversity (e.g., the amount of shallow habitat available for foraging) (Dubuc et al. 1990). In Massachusetts, river

otter use a variety of palustrine, riverine, and lacustrine wetland systems, with no particular preference for any single habitat type (Newman and Griffin 1994).

River otter have numerous denning and nesting sites within their home range, which they use over the course of the year. Denning and resting sites may be located in log jams, riparian vegetation, snow or ice cavities, riprap, talus rock, boulders, brush and log piles, undercut banks, and dens constructed by other animals (Melquist and Hornocker 1983).

The average size of the home range for adult otter is about 30 km (18 miles) of shoreline (Melquist and Hornocker 1983). Males tend to range more than females. Lactating females range the least (EPA 1993). Home ranges include activity centers, where a river otter spends at least 10% of its time during a given season. Activity centers are located in areas with both an abundant prey base and sufficient shelter (Melquist and Hornocker 1983). Activity centers vary during the course of the year with changing prey availability.

Other than family groups, river otter are typically solitary. They will, however, form temporary associations that may consist of related or unrelated individuals. Home ranges have been shown to overlap extensively, with some river otter sharing essentially the same home range. Separation appears to occur at the activity centers, with individuals or family groups using different activity centers within the home range or the same activity centers, but at different times throughout the day (Melquist and Hornocker 1983).

Given the size of the home range, exposure analyses were conducted for Reaches 5 and 6 combined. The analyses were also conducted for the upstream and Threemile Pond reference areas for comparative purposes.

In this assessment, exposure for river otter that spend 100% of their time foraging in the PSA was estimated. However, exposures for 50, 25, and 10% foraging in the PSA were also estimated to represent river otter that might only partially feed in the PSA. This was done by multiplying the calculated exposures for 100% foraging time in the PSA by the area use factors of 0.50, 0.25, and 0.10.

Dietary Habits

The diet of river otter is somewhat variable, but consists primarily of aquatic animals, particularly fish (Andersen and Woolf 1987; Greer 1955; Melquist and Hornocker 1983; Palmer and Fowler 1975). Other prey, including crayfish, amphibians, turtles, birds, small mammals, and insects, also may be consumed (Whitaker and Hamilton 1998; Burt and Grossenheider 1976; Liers 1951). River otter prefer to forage in shallow water and eat slow-moving, shallow-dwelling fish, such as chubs, suckers, catfish, daces, darters, and schooling fish such as bluegill and other sunfish (Whitaker and Hamilton 1998; Sheldon and Toll 1964). Fish were found in 70% of the otter stomachs surveyed from water bodies in the Adirondacks (Whitaker and Hamilton 1998).

To determine the dietary preferences of river otter, a literature search was performed to isolate field studies that examined otter dietary composition through scat and/or stomach analysis. The studies were screened to identify those that considered otter diet in habitats similar to the Housatonic River study area. Table I.2-10 summarizes the percentage of each prey item in the diets of river otter as reported in the studies reviewed.

Fish Prey Size

Fish prey of river otter can range from 2 to 50 cm in length (Melquist and Hornocker 1983). In some areas, fish captured were typically less than 15 cm (Hamilton 1961; Lagler and Ostenson 1942; Alexander 1977). Greer (1956), however, indicated that fish captured by otter ranged from 15 to 25 cm. Based on these observations, the exposure analysis for otter included tissue samples for fish ranging in length from 2 to 50 cm.

I.2.1.6.2 Exposure Model Input Distributions

The input parameters used in the Monte Carlo and probability bounds analyses are summarized in Table I.2-11 and described in detail below.

Body Weight (BW)

Body weight is not used in the model directly, but is a required variable in allometric models (e.g., Nagy 1987) to estimate food intake or free metabolic rates. Whitaker and Hamilton (1998)

reported that body weights ranged from 8 to 11 kg (average of 9.2 kg) for males and from 7.5 to 8.0 kg (average of 7.9 kg) for females in eastern United States populations.

In the Monte Carlo analysis, body weight was assumed to be normally distributed with a mean of 8.36 kg and a standard deviation of 1.6 kg. The same distribution was used in the probability bounds analysis for this input variable. The uncertainty in this variable is small and is likely due to variability, rather than lack of knowledge or data gaps.

Food Intake Rate (FIR)

For the purpose of the ERA for piscivorous mammals, *FIR* was estimated using an allometric equation rather than using literature-reported values for captive mink because it better approximates the increased energy demand of wild mink resulting from higher activity levels incurred while foraging, defending and inspecting territory, and avoiding predators (Lamprey 1964; Buechner and Golley 1967; Koplin et al. 1980). Food intake rate (*FIR*) is derived using the following equation:

$$FIR = \frac{FMR}{\sum_{n}^{i=1} AE_i \cdot GE_i} \qquad \text{(Eq. 5)}$$

where:

FMR = Normalized free metabolic rate (kcal/kg bw/d)

AE_i = Assimilation efficiency of *i*th food item (unitless)

GE_i = Gross energy of *i*th food item (kcal/kg)

P_i = Proportion of the *i*th food item in the diet (unitless)

FMR is estimated from the allometric relationship of Nagy et al. (1999) developed for carnivorous mammals. The general form of the model is:

$$FMR\ (kJ/d) = a \cdot BW(g)^b \qquad \text{(Eq. 6)}$$

For the Monte Carlo and probability bounds analyses, *FMR* was estimated using a probabilistic approach where distributions of the input variables (body weight [*BW*], *a*, *b*) were used rather than point estimates. The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy et al. (1999), assuming an underlying normal distribution for each. For mammals, log *a* had a reported mean of 0.367 and a standard error of 0.223, and *b* had a reported mean of 0.850 and a standard error of 0.055 (Nagy et al. 1999).

Assimilation Efficiency (AE_i)

Average assimilation efficiency for fish consumed by mammals is 0.91 and for crustaceans is 0.87 (EPA 1993). These variables were treated as point estimates in the Monte Carlo simulations and probability bounds analyses because of their low coefficients of variation.

Gross Energy (GE_i)

Average gross energies of fish and crayfish reported in the literature were 1.20 kcal/g (Thayer et al. 1973; EPA 1993) and 1.10 kcal/g (Jorgensen et al. 1991; Minnich 1982; Thayer et al. 1973), respectively. These variables were treated as point estimates in the Monte Carlo simulations and probability bounds analyses because of their low coefficients of variation.

Proportion of Dietary Items (P_i)

According to several sources (Melquist and Hornocker 1983; Greer 1955; Anderson and Woolf 1987) the diet of otter is nearly 100% fish in some locations and times of the year. Sheldon and Toll (1964) and Melquist and Hornocker (1983) found fish remains in 92 to 100% of the analyzed scats. However, considering the preponderance of crayfish exoskeletons often seen in otter scat, crayfish are a common prey item during some times of the year. The *Wildlife Exposure Factors Handbook* (EPA 1993) summarized several studies where crayfish were found to be an important seasonal food source. Information from scat analyses from the Housatonic River watershed indicated that crayfish constituted about 20% of the otter diet during the winter when the scat was collected (Appendix A).

A summary of the information provided in these studies indicates that approximately 80% of the river otter diet consists of fish. Invertebrates constitute 8 to 20% of the diet. Other prey may occasionally be consumed by river otter.

The proportion of prey type in the diet was assumed to follow a beta distribution and was parameterized to approximate the above proportions. Beta distribution parameters for crayfish were alpha = 7.67, beta = 1.67, and scale = 0.24. Beta distribution parameters for fish were one minus the proportion for crayfish (Table I.2-11).

The beta distribution is not an available option in RiskCalc, the software used for conducting the probability bounds analyses. As an alternative, minimum, mean, and maximum values were specified for each dietary item using the means and ranges described above. The minimum, mean, and maximum values were then included as a distribution-free statement in RiskCalc. The result is probability bounds that include all possible distributions, given the specified minimum, mean, and maximum values specified for the dietary items.

I.2.1.6.3 Concentrations of tPCBs in Prey

Concentration of tPCBs in Fish from Reaches 5 and 6 (C_i)

In Reaches 5 and 6, 418 fish samples ranging between 2 and 50 cm in length were included in the analysis. The Land H-statistic was used to determine the lower 95% and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 66.8 mg/kg. For the probability bounds analysis, the lower 95% and upper 95% confidence limits determined from the Land H-statistic were used to parameterize a distribution-free statement in RiskCalc (Table I.2-12). Table I.2-12 summarizes the concentration parameters for fish and crayfish.

I.2.1.6.4 Concentrations of TEQ in Prey

Concentration of TEQ in Fish from Reaches 5 and 6 (C_i)

TEQ was determined in 236 samples of fish ranging between 2 and 50 cm in length, in Reaches 5 and 6. Using the decision criteria outlined in Section 6.4, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-

201/PCB-157/PCB-173) concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses.

The assumption of how to treat non-detected congeners in the TEQ calculation also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this case, the upper 95% CL was the lower of the two, with a value of 727 ng/kg.

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values, assuming that ND = ½ DL, were calculated using the greater of the lower 95% CL and the minimum value and the lower of the upper 95% CL and the maximum value, respectively. The resulting values ranged from 542 to 727 ng/kg (Table I.2-13). Table I.2-13 summarizes the concentration data for fish and crayfish.

I.2.1.6.5 Exposure Model Results

The results of the Monte Carlo and probability bounds analyses for exposure of river otter to tPCBs and TEQ in the PSA are presented in Tables I.2-14 and I.2-15, respectively. The results of the sensitivity analysis are presented in Table I.2-16. Figures I.2-11 to I.2-16 depict the cumulative distributions of total daily intake of tPCBs and TEQ, as well as the corresponding probability bounds.

Exposure to tPCBs in Reaches 5 and 6

PSA Foraging Time: 100%

The Monte Carlo analysis indicated that exposure to tPCBs of otter foraging in the PSA 100% of time could range from a minimum of 0.251 to a maximum of 111 mg/kg bw/d. The mean exposure was 8.42 mg/kg bw/d and the median exposure was 6.02 mg/kg bw/d (Table I.2-14), with 90% of exposure estimates were between 1.60 and 22.8 mg/kg bw/d. Figure I.2-11 shows the cumulative distribution of total daily intake rates of tPCBs for river otter in the PSA.

The probability bounds estimated for river otter foraging in the PSA 100% of the time are shown in Figure I.2-11. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 1.59 and 8.12 mg/kg bw/d. The 50th percentile ranged between 3.27 and 14.2 mg/kg bw/d, and the 90th percentile ranged between 5.78 and 53.0 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 2.15, the 50th percentile was 6.03, and the 90th percentile was 17.1 mg/kg bw/d (Table I.2-14).

Otter foraging in Reaches 5 and 6 less than 100% of time would have their exposures reduced in proportion to the amount of time they spend outside the PSA (e.g., 90% foraging outside Reach 5 would reduce exposure by a factor of 10).

Sensitivity analysis revealed that the *FMR* slope term (r_p = 0.770) was the most important variable, followed by the *FMR* power term (r_p = 0.592), and proportion of crayfish in the diet (r_p = -0.0279) (Table I.2-16).

Exposure to TEQ in Reaches 5 and 6

PSA Feeding Time: 100%

The Monte Carlo analysis indicated that exposure to TEQ of river otter in the PSA 100% of the time could range from a minimum of 2.20 ng/kg bw/d to a maximum of 1,714 ng/kg bw/d. The mean exposure was 104 ng/kg bw/d and the median exposure was 74.2 ng/kg bw/d (Table I.2-15). Of the exposure estimates, 90% were between 19.3 and 287 ng/kg bw/d. Figure I.2-14 shows the cumulative distribution of TEQ intake rates for river otter in the PSA.

The probability bounds estimated for otter foraging in the PSA 100% of time are shown in Figure I.2-14. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 23.1 and 47.8 ng/kg bw/d. The 50th percentile ranged between 52.1 and 102 ng/kg bw/d, and the 90th percentile ranged between 128 and 288 ng/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 263, the 50th percentile was 74.2, and the 90th percentile was 212 ng/kg bw/d.

Otter foraging in Reaches 5 and 6 less than 100% of time would have their exposures reduced in proportion to the amount of time they spend outside the PSA (e.g., 90% foraging outside Reach 5 would reduce exposure by a factor of 10) (Table I.2-15).

Sensitivity analysis revealed that the *FMR* slope term (r_p = 0.779) was the most important variable, followed by *FMR* power term (r_p = 0.597), body weight (r_p = -0.0361), proportion of crayfish (r_p - 0.0275), and concentration of TEQ in crayfish (r_p = 0.00930) (Table I.2-16).

Other Exposure Scenarios

The mean total daily intakes of tPCBs of otter foraging 100% of time in the upstream and Threemile Pond reference areas were 0.0287 mg/kg bw/d and 0.00713 mg/kg bw/d, respectively (Table I.2-14). This is about two to three orders of magnitude lower than that found in Reaches 5 and 6 (8.42 mg/kg bw/d). In comparison, the mean exposure to tPCBs was as low as 0.000713 mg/kg bw/d for otter feeding in Threemile Pond 10% of the time. The mean daily exposure for mink feeding 50 and 25% of the time in the Threemile Pond area were 0.00357 and 0.00178 mg/kg, respectively.

The mean exposures to TEQ of otter in Reaches 5 and 6, the upstream reference area, and the Threemile Pond reference area were 104, 17.6, and 1.49 ng/kg bw/d, respectively. Mean total daily intakes could be as low as 0.149 mg/kg bw/d for otter that feed in Threemile Pond only 10% of the time (Table I.2-15).

For each exposure scenario, sensitivity analysis revealed that the *FMR* power term and the *FMR* slope term were the most important variables in the Monte Carlo analyses. The exposure model results for exposure of otter to tPCBs and TEQ in the PSA are presented in Tables I.2-14 and I.2-15, and the sensitivity analyses are presented in Table I.2-16.

I.3 EFFECTS ASSESSMENT

Studies on the effects of COCs to wildlife were considered in the development of appropriate effects metrics if sufficient information was provided to permit evaluation of study design, study execution, chemical analysis methods, statistical analyses, and other key aspects of the study. Those studies with major issues (e.g., lack of documentation of statistical methods) were included in the review of the effects literature that follows, but were not considered in the derivation of the effects metrics that will be used in the risk characterization.

I.3.1 Contaminants of Concern

I.3.1.1 tPCBs

The following sections focus on the effects of Aroclor 1254 and 1260 on mink and otter. The congener profiles in these mixtures most closely represent the congener profiles observed in fish and other media in the Housatonic River.

I.3.1.1.1 Overview of Toxicity

Information on the toxicity of tPCBs to piscivorous mammals was obtained from a literature search on the effects of PCBs to mink and otter, as well as other small mammals. Piscivorous mammals are known to be sensitive to the effects of tPCBs (Safe 1994; Eisler and Belisle 1996; Tillitt et al. 1996).

A synopsis of the studies selected from the literature is presented in Figures I.3-1 and I.3-2. Table I.3-1 contains a more comprehensive review of available data. The studies in Table I.3-1 met the following criteria:

- Mink or otter were tested.
- The study used a dietary route of exposure.
- Ecologically relevant toxicity metrics were used.
- Acceptable test and analytical methods were used.

All toxicity estimates presented in this assessment are reported as daily dietary doses, normalized to a body weight of 1.0 kg (mg/kg bw/d tPCBs or ng/kg bw/d TEQ) to be consistent with the exposure model.

Exposure of mink and otter to tPCBs and TEQ can produce a variety of physiological responses, ranging from enzyme induction (Aulerich et al. 1985) to death (Safe 1994; Eisler and Belisle 1996; Tillitt et al. 1996). This risk assessment focused on effects that have a direct and immediate influence on the long-term maintenance of mink and otter local subpopulations. Accordingly, preference was given to studies that reported effect concentrations associated with mortality, reproductive impairment, and growth rate. Studies that reported serious histopathological effects, such as damage to the liver, kidney, or brain, were also considered. Less consideration was given to studies that reported effect concentrations associated with changes in biochemical functions of organs.

In addition to reduced survival, growth, and fecundity, tPCBs and TEQ have been observed to cause anorexia (Aulerich et al. 1971, 1973, 1987; Platonow and Karstad 1973; Hornshaw et al. 1986; Heaton et al. 1995), abnormal molting and nail growth (Aulerich et al. 1987), gastric ulcers (Aulerich et al. 1971, 1973, 1987; Platonow and Karstad 1973), bloody stools (Aulerich et al. 1971, 1973, 1987), and listlessness or nervousness (Platonow and Karstad 1973; Heaton et al. 1995).

I.3.1.1.2 Mortality

Dietary Exposure

In a study where the diet was prepared from cattle that had consumed feed contaminated with Aroclor 1254 (Figure I.3-1; Table I.3-1; Platonow and Karstad 1973), a dose of 0.0890 mg/kg bw/d consumed by female mink over 160 days of exposure caused 100% mortality in the offspring. The treatment also caused 17% mortality in adult females, but not in males. Hornshaw et al. (1983) fed female mink with contaminated carp containing a dose of 0.210 mg/kg bw/d of PCBs identified as Aroclor 1254. After 7 months of this feeding regime, the mink were allowed to reproduce. None of the young were born alive. A slightly higher dose of 0.280 mg/kg bw/d caused 100% kit mortality 4 weeks after birth. Adult female mink experienced 12% mortality after 10 months of continuous exposure to this treatment (Aulerich and Ringer 1977). Total mortality in adults was observed at a dose of 0.500 mg/kg bw/d (Platonow and Karstad 1973). Only 105 days of dietary exposure at this concentration were required to kill all the adults.

In another study, female mink were exposed to a dose of 0.700 mg/kg bw/d. Of these individuals, 30% died after 9 months of exposure (Aulerich and Ringer 1977). Mortality increased to 71% in response to a dose of 1.40 mg/kg bw/d. Ranch-raised mink exposed to 0.140 mg/kg bw/d reported as Aroclor 1254 from field-collected carp experienced lower survival in lactating offspring (Wren et al. 1987b). However, the carp contained other contaminants that could have contributed to the toxic response.

Dietary LC_{50} tests with mink performed by Hornshaw et al. (1986) using Aroclor 1254 revealed average LC_{50}s from 6.58 mg/kg bw/d to 8.12 mg/kg bw/d. One of the highest estimates of acute doses was reported by Aulerich et al. (1973), who found a 48-hour LD_{50} of 140 mg/kg bw/d. Dietary exposure of female mink to a dose of 0.004 mg/kg bw/d tPCBs (42 to 60 % chlorine) in carp for 3 to 6 weeks resulted in 15% mortality in kits (Heaton et al. 1995). Mortality increased to 69% at a dose of 0.1 mg/kg bw/d after 3 weeks of exposure and 71% after 6 weeks of exposure. At the dose of 0.210 mg/kg bw/d, kit mortality was 71% after 3 weeks of exposure and 89% after 6 weeks of exposure. Total kit mortality was observed at a dose of 0.360 mg/kg bw/d, with death being observed in as little as 24 hours after receiving the dose. Jensen et al. (1977) exposed female mink to a dose of 1.54 mg/kg bw/d of Aroclor 1254 for 66 days. After the treatment, no live kits were born to exposed females. Ringer et al. (1972) exposed mink to a diet spiked with 4.20 mg/kg bw/d PCBs (equal amounts of Aroclors 1242, 1248, and 1254). All adult mink died prior to whelping.

A dose of 0.350 mg/kg bw/d of 2,3,6,2',3',6'-HxCB (PCB-136) resulted in decreased survival time of newborn kits (Aulerich et al. 1985). Exposure of female mink to 0.00140 mg/kg bw/d of the congener 3,4,5,3',4',5'-HxCB (PCB-169) for 120 days had no effect on mink mortality (Aulerich et al. 1987). Mortality was observed at 0.007 mg/kg bw/d. Exposure for 135 days induced 50% mortality (Aulerich et al. 1987). A dose of 0.0140 mg/kg bw/d caused 50% mortality by day 88.

Tissue Residue Effects Data

The available tissue concentration data indicate that adverse effect concentrations begin at approximately 1.0 mg/kg ww (whole body). Heaton et al. (1995) reported a no observed adverse effect level (NOAEL) of 0.09 mg/kg liver tissue. The lowest observed adverse effect level

(LOAEL) was reported to be 2.20 mg/kg liver. In another study, Leonards et al. (1995) reported an EC_{50} of 1.20 mg/kg mink tissue for relative litter size. An EC_{50} for kit survival was reported to be 2.40 mg/kg mink tissue (whole body). The study by Leonards et al. (1995) was based on a compilation of mink toxicity data for PCB technical mixtures containing 16 to 60% chlorine.

I.3.1.1.3 Reproduction

Farm-raised mink exposed to 0.140 mg/kg bw/d Aroclor 1254 experienced reduced survival of lactating offspring. However, no declines in fertility, whelping, or fecundity were observed (Wren et al. 1987b).

Kihlstrom et al. (1992) exposed female mink to 1.64 mg of Aroclor 1254 per individual (1.28 mg/kg bw/d) in food for 105 days. The exposure caused all kits to be stillborn. The dose also increased the number of interrupted pregnancies.

Aulerich and Ringer (1977) reported that exposure of mink to 0.280 mg/kg bw/d of Aroclor 1254 did not affect birth rate, birth weight, or survival. However, a dose of 2.80 mg/kg bw/d caused reduced whelping and growth rate of kits. At 0.7 mg/kg bw/d, no whelping was observed, although survival was unaffected (Bleavins et al. 1980). Decreased mink fecundity has been observed following exposure to 0.08 mg/kg bw/d (0.7 mg/kg diet) (Brunstrom et al. 1991). In another study, Aulerich et al. (1985) exposed mink to dietary concentrations of Aroclor 1254 over extended exposure periods (several weeks). A concentration of 2.5 mg/kg bw/d was associated with reduced fecundity. Only one female whelped and the kit that was born died after birth. This dietary exposure is equivalent to a dose of 0.288 mg/kg bw/d given the food intake rate of 115 g/d.

Male and female mink fed PCB-contaminated diets (Saginaw Bay carp) had decreased breeding performance. Kit body weight and survival were reduced at birth following exposure to 0.140 mg/kg bw/d of tPCBs in diet (Restum et al. 1998).

I.3.1.1.4 Other Effects

Prolonged consumption of diet contaminated with 2.5 mg/kg 2,3,6,2',3',6'-HxCB (0.350 mg/kg bw/d) did not cause adverse effects on the growth rate of juvenile mink (Aulerich et al. 1985). A dose of 0.7 mg/kg bw/d led to increased norepinephrine and dopamine activity.

Exposure of female mink to 0.0014 mg/kg bw/d of 3,4,5,3',4',5-HxCB (PCB-169) for 120 days resulted in increased liver weight (Aulerich et al. 1987). This treatment had no effect on growth rate. Such effects were observed at 0.007 mg/kg bw/d. The growth rate dropped just after 90 days of exposure (Aulerich et al. 1987).

I.3.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

This section describes the toxicity of the most potent dioxin, 2,3,7,8-TCDD, as well as dioxin-like compounds that have the same mode of action and act on the same receptor, including the dioxin-like PCB congeners.

I.3.1.2.1 Mortality

Mature female mink fed diets with 0.600, 16.0, 53.0, 180, and 1,400 ng/kg of 2,3,7,8-TCDD (equivalent to a dose of 0.0840, 2.24, 7.42, 25.2, and 196 ng/kg bw/d) for a maximum of 132 days exhibited 17% mortality, as well as lethargy and bloody stools at the highest dose concentration (Hochstein et al. 2001). Final body weights were inversely related to dietary TCDD concentration and there was a dose-dependent drop in kit weight from birth to week 3 of exposure. At the highest dose concentration of 196 ng/kg bw/d various physiological functions were depressed.

Hochstein et al. (1998) exposed female mink to 1, 10, 100, 1,000, 10,000, and 100,000 ng/kg of TCDD in the diet (daily dose equivalent of 0.14, 1.4, 14, 140, 1,400, and 14,000 ng/kg bw/d) for 125 days. A dose-dependent wasting syndrome (decrease in body weight) was observed. Mortality reached 12.5%, 62.5%, and 100% after 28 days of exposure to 140, 1,400, and 14,000 ng/kg bw/d, respectively. After 125 days of exposure, mortality reached 100% in the 1,400- and 14,000 ng/kg exposure groups.

Newborn mink given doses (intraperitoneal injection) of 100 and 1,000 ng TCDD/kg bw experienced 100% mortality at the higher dose after 12 days. The lower dose caused depressed body weight and 62% mortality (Aulerich et al. 1988). Adult mink administered a single oral dose of 2,500 ng/kg bw TCDD had significantly reduced body weights after 3 weeks (Hochstein et al. 1988).

At 0.250 ng/kg bw/d, Heaton et al. (1995) observed 15% mortality to mink kits after exposure for 3 weeks. A 69% mortality in kits was reported at a dose of 3.6 ng/kg bw/d. Mortality increased to 100% at a dose of 10.7 ng/kg bw/d.

A dose of 0.350 mg/kg bw/d of 2,3,6,2',3',6'-HxCB resulted in decreased survival of newborn kits (Aulerich et al. 1985). Exposure of female mink to 0.00140 mg/kg bw/d of the isomer 3,4,5,3',4',5-HxCB for 120 days had no effect on mink mortality (Aulerich et al. 1987). Mortality was observed at 0.007 mg/kg bw/d. An exposure duration of 135 days was required to induce 50% mortality (Aulerich et al. 1987). A dose of 0.0140 mg/kg bw/d caused 50% mortality by day 88.

I.3.1.2.2 Reproduction

Adult mink exposed to 0.6, 16, 53, 180, and 1,400 ng/kg TCDD in a diet of field-collected fish (daily dose equivalent of 0.084, 2.24, 7.42, 25.2, and 196 ng/kg bw/d) for up to 132 days produced offspring that had reduced survival from birth to week 3 of exposure (Hochstein et al. 2001).

There is some evidence that TCDD interferes with ovulation. Ushinohama et al. (2001) administered a dose of 32,000 ng/kg by gavage to female rats. The treatment led to reduced body weight gains as well as to reduced ovarian weights. Infertility and fetal loss have been observed at a dose of 0.01 μg/kg/day (10 ng/kg bw/d TEQ) administered to rats (Murray et al. 1979). A LOAEL of 1 ng/kg/day was estimated by Nisbet and Paxton (1982) using Murray et al. (1979) data. Ovulation was delayed and fewer ova produced.

A dose of 0.350 mg/kg bw/d of 2,3,6,2',3',6'-HxCB caused reduced litter size (Aulerich et al. 1985). Female mink exposed to 0.00140 mg/kg bw/d of the isomer 3,4,5,3',4',5-HxCB for 120

days did not experience adverse effects on reproduction (Aulerich et al. 1987). A dose of 0.0140 mg/kg bw/d was associated with a total absence of whelping.

I.3.1.2.3 Other Effects

Hochstein et al. (2001) reported that body weights of adult female mink were inversely proportional to dietary TCDD concentration. Doses ranged from 0.084 to 196 ng/kg bw/d.

In a study that examined the effects of dietary TCDD on mink, Render et al. (2001) reported that 2,400 ng/kg diet (336 ng/kg bw/d) fed to mink juveniles for 36 days resulted in reduced weight gain in the exposed animals. Also, the treated animals had loose and displaced incisor teeth. In an earlier study, Render et al. (2000) reported that adult female mink exposed to 5,000 ng/kg TCDD in the diet (700 ng/kg bw/d) for 6 months had nests of squamous epithelium in periodontal ligaments and osteolysis in the alveolar bone. Aulerich et al. (2001) reported that weanling male rats exposed to 10,000 ng/kg TCDD (1,400 ng/kg bw/d) for 101 days in the diet experienced a dose-dependent decrease in body weight gain. However, no jaw lesions were observed.

Newborn mink injected with 100 ng/kg bw TCDD for 12 days experienced depressed body weight (Aulerich et al. 1988). Adult mink administered a single oral dose of 2,500 ng/kg bw had significantly reduced body weight after 3 weeks (Hochstein et al. 1988). Ushinohama et al. (2001) administered a dose of 32,000 ng/kg bw by gavage to female rats, and reported reduced body weight gain.

I.3.2 Mink Feeding Study

It was hypothesized when developing the conceptual model for the ERA that contaminants in the prey of piscivorous mammals foraging in the PSA may have caused adverse effects on the survival, reproduction, and/or growth of exposed individuals, based on lack of observations of mink or otter (or their sign) during EPA field investigations. To test this hypothesis, a feeding study was performed by researchers at Michigan State University (MSU) (Bursian et al. 2002), the results of which are described below.

In this study, fish were collected from the PSA, frozen, and sent to MSU. These fish were mixed with ocean herring in varying proportions to derive a control diet formulated to meet the

nutritional requirements of farm-raised mink (all diets were 30% fish, 70% formulated mink diet) and five treatment diets containing target concentrations ranging from 0.25 to 4 mg/kg tPCBs. These doses were established during the design of the study to span the range of known effect thresholds from previous studies on the effects of PCB exposure in mink. The diets were fed to captive adult female mink for approximately 160 days. The exposure period for adult females began approximately 2 months prior to mating, and continued through mating and whelping of the kits. Some kits were exposed for an additional 6 months following whelping. A number of endpoints were measured during the study including: feed consumption rate, mating success, gestation length, number of kits born, adult and kit survival, body weights, organ weights, and tissue histology. Biochemical parameters and the histopathology of the jaws of mink kits were also measured. The following sections describe the methodology used in the study and the study results. A more detailed description of the study can be found in Bursian et al. (2002).

I.3.2.1 Methodology

I.3.2.1.1 Fish Preparation

Fish were collected from the PSA (Woods Pond area) of the Housatonic River and shipped to the MSU Experimental Fur Farm (East Lansing, MI). Atlantic herring were purchased from Boston Feed Company (Natick, MA) to serve as an uncontaminated source of fish for use in mixing the necessary range of dietary PCB concentrations while maintaining 30% total fish content in each diet. Samples were analyzed for tPCB concentrations. The results indicated that the herring contained an average of 0.0506 ±0.0005 mg/kg tPCBs, and the PSA fish contained an average of 113 ±5.4 mg/kg tPCBs.

I.3.2.1.2 Dietary Treatments

The treatment diets were based on the standard MSU Experimental Fur Farm ranch diet formulated to meet the nutritional requirements of mink. Each diet contained 30% fish, which was assumed to be the average quantity of fish consumed by mink in the wild (Heaton et al. 1995). The control diet contained 30% ocean herring. The remaining five treatment diets contained a mixture of ocean herring and PSA fish to obtain targeted concentrations of tPCBs of 0.25, 0.50, 1.0, 2.0, and 4.0 mg/kg based on the dilution of PSA fish containing 113 mg/kg tPCBs with ocean herring. The percentages of PSA fish added to each treatment diet were 0%

for the control treatment, 0.22% for the 0.25 mg/kg treatment, 0.44% for the 0.5 mg/kg treatment, 0.88% for the 1.0 mg/kg treatment, 1.75% for the 2.0 mg/kg treatment, and 3.51% for the 4 mg/kg treatment. Six grab samples consisting of six pooled subsamples per grab sample were collected for subsequent contaminant analysis. The measured concentrations achieved in the treatments were 0.055 mg/kg tPCBs in the control, 0.34 mg/kg in the 0.25 target treatment, 0.61 mg/kg in the 0.5 target treatment, 0.96mg/kg in the 1.0 target treatment, 1.6 mg/kg in the 2.0 target treatment, and 3.7 mg/kg in the 4.0 target treatment.

I.3.2.1.3 Study Design

A total of 72 female mink were randomly assigned to six treatment groups (12 mink/group). Mink were housed individually in wire cages containing a wooden nest box. The mink were placed on the control diet (30% ocean herring) 7 days prior to the experiment. Because some species of fish contain thiaminase, the mink were provided supplemental thiamine to prevent Chastek's paralysis (Gnaedinger 1963; Green et al. 1942; Long and Shaw 1943). Body weight was recorded on the first and last days of the acclimation period. Feed consumption was measured for two consecutive days during the acclimation period. After the treatments began, any animals that died during the study were examined by a veterinary pathologist. Each treated female was given an opportunity to mate every fourth day with an untreated male until a successful mating occurred. Nest boxes were checked on a daily basis for the presence of mink kits. The gender of the kits was determined at birth and numbers of live and stillborn young counted. All surviving adults and six kits (three males and three females) from each treatment group were euthanized and necropsied when the kits were 6 weeks old. The liver was analyzed for microsomal enzyme activities, contaminant concentrations, and histology. Histological examination also included brain, kidneys, spleen, heart, and adrenal glands.

I.3.2.2 Results

The results of the mink feeding study are described in detail in Bursian et al. (2002). In this section, only major findings are presented. The presence of tPCBs, TEQ, and other COCs in the diet did not have a significant effect on food intake rate of adult female mink. Consumption of diets containing tPCBs, TEQ, and other COCs derived from Housatonic River fish had no significant effect on breeding success (number of females bred/total number of females) or

whelping success (number of females whelping/number of females bred) of female mink. Gestation length was not significantly altered by exposure treatments. Average litter size and kit survival at birth and 3 weeks of age were also not affected by the exposure treatments. The decrease in percent survival of kits in the 3.7 mg/kg tPCB treatment group at 6 weeks of age was statistically significant (i.e., 46% lower compared to controls) compared to kits in the control and 1.6 mg/kg tPCB treatment groups (Table I.3-2).

There were no significant differences between treatments for adult female body weights at the beginning of the study, during the pre-breeding period, and at 3 and 6 weeks post whelping. However, there was a significant treatment by date interaction for kit body weights from birth to 6 weeks of age. At 3 weeks of age, kits in the 0.56 mg/kg tPCB treatment group had significantly higher body weights when compared to kits in the other five groups. Kits in the 3.7 mg/kg tPCB treatment group had significantly lower body weights when compared to kits in the other groups. At 6 weeks of age, however, mean kit body weight in the 3.7 mg/kg tPCB treatment group was only slightly lower than mean kit body weights observed in the control treatment (251 ±16.2 g versus 293 ±11.3 g, respectively). From 10 to 30 weeks of age, differences in kit body weights between treatments were minor and did not have a dose-dependent relationship with either tPCBs or TEQ.

Absolute and relative (expressed as a percentage of body weight) brain, heart, spleen, liver, kidney, and adrenal gland weights of adult females were not significantly different between treatment groups at necropsy. Absolute brain, spleen, kidney, and adrenal gland weights in kits necropsied at 6 weeks of age were not significantly different between treatment groups. Differences in absolute heart and liver weights of kits between treatments were minor and did not have a dose-dependent relationship with either tPCBs or TEQ. Relative spleen and adrenal gland weights were not significantly different between treatment groups. There were no significant differences in the relative weights of the brain, liver, kidneys, and heart in males and of the heart in females. Females in the 3.7 mg/kg tPCB group had significantly higher relative brain, liver and, kidney weights compared to females in the other five treatment groups at 6 weeks of age.

Absolute brain, kidney, heart, and adrenal gland weights in kits necropsied at the end of the 30-week study were not significantly different between treatment groups. Minor differences in absolute spleen and liver weights between treatments were observed, but the effects did not have a dose-dependent relationship with either tPCBs or TEQ. Relative brain, heart, and adrenal gland weights were not significantly different between treatment groups. The relative spleen weights of kits in the 3.7 mg/kg tPCB treatment group were significantly higher compared to the control and 0.34 mg/kg tPCB treatment groups. Other organ effects were minor and not dose-dependent. The results of the histological examination of the tissues of major internal organs of the adult female mink and their kits did not show remarkable changes attributable to the treatment diets. These results are not discussed here, but are presented in detail in Bursian et al. (2002).

I.3.2.3 Mink Jaw Lesion Study

The purpose of this study was to examine the histopathology of jaws of mink from the feeding study by Bursian et al. (2002). The objective was to determine whether the dietary treatments induced lesions as had been observed previously in other studies of mink fed PCB-126 and TCDD. The evaluation was conducted on 6-month kits necropsied at the end of the mink feeding study.

Methodology

The skulls of 6-month-old mink kits were fixed in a 10% formalin-saline solution at necropsy, decalcified in 5% nitric acid, rinsed, trimmed, processed using a routine histotechnologic method, and embedded in paraffin. Tissues were sectioned at 6 microns and stained with hematoxylin and eosin. Jaws from 36 kits were examined for pathologies: 6 jaw samples from each of the control and 0.34, 0.61, 0.96, 1.6, and 3.7 mg tPCBs/kg treatments. The observed lesions were graded as mild, moderate, or severe based on the number and size of foci of squamous cell proliferation in maxilla and mandibles.

Results and Discussion

While none of the mink kits had gross abnormalities of the maxilla and mandible, in some treatments histological evidence, in the form of proliferation of periodontal squamous epithelial

cells, was present. Nests of squamous epithelium were found adjacent to the teeth and some had cystic centers. The proliferation resulted in focal loss of alveolar bone. Squamous cell proliferation was apparent in 17%, 33%, and 100% of kits in the 0.96, 1.6, and 3.7 mg tPCBs/kg treatments, respectively. No lesions were observed in the controls and the 0.34 and 0.61 mg tPCBs/kg treatments. The lesions appeared to start from the caudal molar region of the jaw and advanced to the pre-molar, canine, and incisor regions. The initial lesions in the molar region usually consisted of large cysts lined with thick layers of stratified squamous epithelium and filled with floating, sloughed squamous cells. The subsequent lesions in the pre-molar, canine, and incisor regions of the jaw were characterized as multiple nodules of compact stratified squamous epithelium.

These results indicate that dietary concentrations of PCBs, specifically PCB-126, as low as 54 ng/kg in the diet (0.96 mg tPCBs/kg diet) can induce maxillary and mandibular squamous cell proliferation. Exposure of mink to higher concentrations of PCB-126 for longer periods of time, as would be expected in the Housatonic River ecosystem, would undoubtedly cause increased severity of the lesions leading to erosion of the mandible and maxilla with resultant loss of teeth. Such an effect could ultimately cause the animal to die of starvation (Bursian et al. 2003; Bursian et al. 2002).

I.3.2.4 Mink Enzyme Study

Tillitt et al. (2003) supplemented the MSU studies by measuring hepatic O-dealkylase activities associated with cytochrome P450 (CYP) isozymes induced in mink fed diets containing fish collected from the PSA in the MSU study. Specific activities were measured against four separate substrates to measure the induction of CYP enzymes in maternal and F1 generations of the exposed mink. The induction of CYP enzymes is a good indicator of exposure to coplanar PCBs, dioxins, and furans and indicates a first level of toxicological response to dioxins and dioxin-like contaminants (Aulerich et al. 2003). Hepatic activities were measured because the majority of detoxification of xenobiotics occurs in the liver.

I.3.2.4.1 Methodology

In the feeding study by Bursian et al. (2002), 36 offspring (kits) along with the adults were sacrificed at 6 weeks after whelping. Another 36 kits were sacrificed at 6 months post whelping. The livers were removed and placed in 1.2-ml cryovials and frozen in liquid nitrogen. Frozen liver samples from the parental generation, 6-week old offspring, and 6-month-old offspring were transmitted to the Columbia Environmental Research Center (CERC) for analysis. The analyses consisted of microsomal preparation and various O-dealkylase assays. All procedures were executed according to CERC standard operating procedures (SOPs) and QA/QC procedures.

I.3.2.4.2 Results

BROD and PROD Activity

The (benzyloxyresorufin-O-deethylase or BROD and pentoxyresorufin-O-deethylase or PROD) responses of mink to various dietary treatments of Housatonic River fish are presented (Tables I.3-3 through to I.3-5). There was no significant induction of BROD by any of the dietary treatments in the maternal mink (Table I.3-3). The BROD activity in the 6-week old kits was not significantly different from the control mink in any of the treatments except at the greatest dose (3.7 mg tPCB/kg diet, or 3.54% Housatonic River fish) where it was significantly reduced as compared to the control ($p < 0.05$, Table I.3-4). No dose-response relationship was apparent in either the maternal or 6-week old kit responses.

Hepatic BROD activity was significantly elevated at all of the treatment doses of the 6-month old kits (Table I.3-5). However, the increased hepatic BROD activity of these kits may have been due to the reduced activity in the control mink in this age group rather than to the treatment response. (Bursian, pers. comm.).

The induction of PROD in maternal mink and their offspring was minimal (Tables I.3-3 to I.3-5) and significant in only the smallest dose group (0.34 mg tPCB/kg diet, or 0.22% Housatonic River fish) in the 6-week-old and 6-month-old kits. No dose-response relationships were evident in any of the treatment groups. Further, no significant induction of hepatic PROD was observed in any of the treatment groups of the 6-month-old kits. A general decrease in induction of both BROD and PROD was observed in both ages of the kits with respect to increasing dose.

ECOD and EROD Activity

The ethoxycoumarin-O-deethylase (ECOD) activity was elevated in every dose and age group relative to controls (Tables I.3-3 to I.3-5). The dose-response relationship of Housatonic River fish dietary exposure and hepatic ECOD responses in mink of all age groups was monotonic and consistent with an increased exposure to Ah-receptor ligands and dioxin-like contaminants. However, significant increases ($p < 0.05$) in the maternal mink ECOD activities were associated with the two highest dose treatments (1.75 and 3.7 mg tPCB/kg diet of Housatonic River fish). The ECOD rates in the 6-month-old kits were significantly elevated over controls at all treatment doses (0.56, 0.90, 1.75, and 3.7 mg tPCB/kg diet). There were no statistically significant increases in the ECOD rates of any of the 6-week old kits (Figure I.3-3).

The hepatic ethoxyresorufin-O-deethylase (EROD) activities were elevated in every treatment group that received dietary exposure to Housatonic River fish. Maternal mink and 6-week old kit EROD inductions were significantly greater ($p < 0.05$) than controls in all but the lowest treatment group. The largest inductions of EROD were observed in the 6-month-old kits. All treatments yielded significant increases in EROD activity (Figure I.3-4).

I.3.2.4.3 Discussion of ECOD and EROD Activity

Induction of CYP2B-related activity in mink (BROD and PROD) was not substantial at any of the doses of fish from the Housatonic River. Only a few dose-age treatment combinations had significant inductions toward BROD or PROD activities. Further, none of the increases in BROD or PROD activities occurred in a dose-dependent fashion. Thus, the amounts of di- to tetra-ortho-chloro-substituted PCBs (PCB congeners thought to be responsible for CYP2B-related enzyme inductions) were either below a threshold of activation of these enzymes in the dietary treatments or the enzyme induction pathways were saturated. Further analysis (protein content or message) would be required to discern which of these occurred in these studies.

Induction of CYP1A1-related hepatic enzyme activities (ECOD and EROD) was observed to occur in a dose-dependent manner in all ages of mink examined. Significant increases in these Ah-receptor-regulated enzymes were observed even in treatments with only a small amount of fish from the Housatonic River (0.44%) in their diets. These results confirm the known sensitivity of mink to the effects of tPCBs and other related dioxin-like compounds. The results

also confirm that only a small amount of fish (< 0.5%) from the Housatonic River would be required in the diets of mink to activate Ah-receptor pathways and processes in mink (Tillitt et al. 2003).

I.3.2.5 Discussion

Consumption of diets containing tPCBs and TEQ derived from fish collected from the Housatonic River did not have an adverse effect on adult mink reproduction as assessed by breeding success, whelping success, and gestation length. Kit survival at 6 weeks of age, however, was significantly decreased in the 3.7 mg/kg tPCB (68.5 ng/kg TEQ) treatment group.

The results of the Bursian et al. (2002) study indicated that effects on the number of live kits whelped and kit survival to 6 weeks of age did not occur at dietary concentrations of tPCBs and TEQ that caused adverse effects in other feeding studies (e.g., Saginaw Bay study), but at higher concentrations. There are two possible explanations for this apparent difference in response, including:

- Other COCs present in the Saginaw Bay fish, but not in the Housatonic River fish, may have contributed to the effects observed in the mink fed Saginaw Bay fish.
- The congener mixture in the Housatonic River fish may have a lower toxicity to mink than the congener mixture in the Saginaw Bay fish.

The total daily intake (TDI) of tPCBs by mink in the 1.6 mg/kg treatment group was 0.169 mg/kg bw/d. In the 3.7 mg/kg tPCB treatment group, the TDI was 0.414 mg/kg bw/d. These treatments represent the NOAEL and LOAEL for kit survival at 6 weeks of age in this study. The corresponding TDIs for TEQ were 1.69 ng/kg bw/d (NOAEL) and 7.67 ng/kg bw/d (LOAEL), respectively. At a tPCB dose lower than the NOAEL from the Housatonic River study (0.134 mg/kg bw/d), Heaton et al. (1995) observed a 64% reduction in kit survival to 6 weeks of age. At a tPCB dose lower than the LOAEL from the Housatonic River study (0.32 mg/kg bw/d), Heaton et al. (1995) observed no kit survival to 6 weeks of age. Therefore, differences in food intake rate do not explain the difference in results between the Saginaw Bay mink feeding study and the Housatonic River mink feeding study.

To determine whether differences in the toxicity of the congener mixtures in Housatonic River and Saginaw Bay fish might explain the difference in results between the two feeding studies, TEQ was calculated for each of the Saginaw Bay dietary treatments. This was done using the congener concentrations reported in Tillitt et al. (1996) and the TEFs developed by Van den Berg et al. (1998). At a dose slightly below the LOAEL for the Housatonic River study (6.83 ng/kg bw/d TEQ), an 87% reduction in kit survival to 6 weeks of age was observed by Tillitt et al. Kit survival was significantly reduced in the Housatonic River study at 7.67 ng/kg bw/d, but to a lesser extent (46%). The NOAEL from the Housatonic River study expressed as TEQ (1.69 ng/kg bw/d) is more than two times lower than the lowest dietary dose treatment in the Saginaw Bay study (3.57 ng/kg bw/d) despite the fact that the tPCB doses in the two treatments were quite similar. It appears that most of the difference in results between the Saginaw Bay and Housatonic River mink feeding studies is due to the reduced toxicity of the congener mixture in Housatonic River fish. When the results are expressed as dioxin-like TEQ, the results of the two studies are more comparable. Nevertheless, there exists the possibility that other COCs contributed more strongly to the effects observed in the Saginaw Bay study than was the case in the Housatonic River study.

In conclusion, it should be noted that the dietary doses encountered by mink in the PSA (described below) are much greater than those tested in the feeding study (the highest dose in the feeding study contained only 3.51% of fish from the PSA).

I.3.2.6 Housatonic River Fish and Experimental Diet Congener Comparison

The composition of PCB congeners in the fish blend used in the Michigan State University (MSU) study (Bursian et al. 2002) was compared to the congener composition measured in Housatonic River fish species and sizes likely to be consumed by mink, that were used to determine modeled exposure concentrations. This comparison was performed to determine whether there were potential differences in toxicity between the diet used in the feeding experiment and fish consumed by wild mink in the PSA. See Appendix C.7 for more details.

The experimental diet contained two species of fish from the PSA, carp and goldfish, because of the logistics associated with obtaining the amount of fish required to perform the study. However, there are at least six species of fish in the 7- to 20-cm range that mink may consume in

the PSA. These species include fallfish, yellow perch, golden shiner, largemouth bass, pumpkinseed, and brown bullhead.

The fish component of the mink diet from the feeding study was analyzed by the USGS laboratory for 136 individual congeners, plus 2 co-eluting pairs of congeners. These congeners collectively total over 95% of Aroclor 1260 as measured by Frame et al. (2001). The Housatonic River fish (i.e., those used in the exposure analyses for mink in Section I.2) were analyzed by the GERG laboratory for 71 individual congeners, 22 co-eluting pairs, and 2 co-eluting triplets. These congeners also collectively total over 95% of Aroclor 1260 as measured by Frame et al. (2001). The individual congeners or congener groups common to the two analyses was used for this evaluation. The congeners used in the assessment included 61 individual congeners, 20 pairs, and 2 triplicate congener groups for a total of 83 congeners/congener groups.

I.3.2.6.1 Congener Analysis

For the purpose of this analysis, the concentrations of non-detected congeners were assumed to be equal to zero (ND = 0). This was reasonable because as the sum of the concentrations of non-detects assumed to be equal to the detection limit (ND = DL) was less than 1% of the total congener concentration for any sample (with the exception of the ocean herring). The congener concentration for each sample was normalized to the concentration of total congeners for that sample. The results for each congener represent the relative percent contribution of that particular congener to the sum of all congener concentrations for that sample.

The average normalized concentration for each congener within each of the six fish species used in the exposure analyses, and all species combined, were calculated. The standard error for each of the congeners was calculated as a measure of variability. These statistics were calculated for the five treatment diet blends from the feeding study. There was very little variability in normalized congener concentrations among the feeding study treatments. The congener pattern in the control (ocean herring) could not be evaluated due to the high number of non-detects and the low tPCB concentrations (55 µg/kg as a sum of the congeners). There was a substantial amount of variability in the congener composition in the Housatonic River fish tissue concentrations used for the modeling (Figures I.3-5 and I.3-6).

A separate analysis was performed for the coplanar congeners PCB-77, 81, 126, and 169. The non-detect congener results were treated by both setting the non-detects at zero (ND = 0) and setting the non-detects at the respective detection limit (ND = DL). The congener results for each of the four congeners were normalized to the total concentration of the four coplanar congeners (not the total of all of the congeners). Average normalized fish concentrations were calculated for exposure modeling and dietary treatment estimation. The standard error for each of the congeners was calculated as a measure of variability. The normalized percentages were multiplied by the mammalian TEQ from Van den Berg et al. (1998) to yield a relative measure of the toxicity as TEQ in the diet blend from the feeding study and the Housatonic River fish.

The mean plus two standard errors for each normalized congener were plotted for each fish species and treatment. A comparison was performed between the fish species used in the exposure analyses and the dietary treatment. Similar graphs were developed for all fish species combined, as well as for coplanar congeners.

I.3.2.6.2 Results and Discussion

The congener patterns in the feeding study diet were comparable to those in the fish used in the exposure analyses (Figure I.3-5). There were some exceptions, however. The following congeners were higher (difference between means greater than two standard errors) in the feeding study diet relative to the fish samples used in the exposure analyses:

- PCB-149/123.
- PCB-170/190.
- PCB-174.
- PCB-136.
- PCB-42/37/59.
- PCB-130.
- PCB-22/51.
- PCB-209.

The following congeners were lower in the feeding study diet relative to the fish samples used in the exposure analyses:

- PCB-82.
- PCB-56.
- PCB-67.

When the analysis was repeated for all fish combined, there was an increase in the number of congener concentrations that differed by more than two standard errors between the feeding study diet and exposure analysis fish. That increase was attributed to a large drop in the standard error due to the increased number of samples (N = 92). There were 15 congeners that were higher and 27 congeners that were lower in the feeding study diet than in the exposure analysis fish.

The percent contribution of several coplanar congeners to the tPCB mixture differs slightly in the fish used in the feeding study versus the fish used in the exposure analyses. The mean percent contribution of the most toxic congener, PCB-126, in the fish used in the exposure analyses was about 0.022% compared to approximately 0.005% in the fish used in the feeding study (Figure I.3-6). However, because the error bars ($\pm$ 2 standard errors) for the two means overlap, it is unlikely that these differences are statistically meaningful. Therefore, the PCB composition in fish from the feeding study can be treated as similar to that in fish used in the exposure analyses and the results from both studies are directly comparable.

I.3.3 Effects Metrics for Modeling Risk

I.3.3.1 Selection of Effects Metrics

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species, to concentration- or dose-response curves for the functional group of interest (e.g., benthic invertebrates, mammalian piscivores). While site-specific studies can be used to derive effects metrics, for this independent line of evidence effects characterization preferentially relied on concentration- or dose-response curves, but defaulted to benchmarks or other estimates of effect (e.g., NOAEL, LOAEL) when insufficient data were available to derive dose-response curves. Effects associated with growth, survival, and reproduction are generally the preferred measures of effect. Further details on the decision criteria used in selecting effects metrics is provided in Section 6.6 of the ERA.

The decision criteria were applied to each COC-receptor combination. For PCBs and dioxins/furans, two measures of concentration were considered: tPCBs and 2,3,7,8-TCDD TEQ. For tPCBs, the focus was on studies using mixtures with a higher degree of chlorination (i.e., 54

to 60%) because these mixtures most closely resemble the mixtures occurring in the PSA. For TEQ, the toxicity caused by coplanar PCBs, dioxins, and furans was considered together using the approach described by Van den Berg et al. (1998) (see Section 6.4 for more detail). In all cases, the effects metrics had the same units as did the metrics used in the exposure analysis. Dose-response relationships were combined with the corresponding exposure distribution in risk characterization to derive risk curves that characterize the relationship between probability and magnitude of effect.

It was considered preferable to assess the effects of tPCBs and TEQ to the representative species in the Housatonic River using the results of controlled investigations that treated the species with at least five dose levels and measured for effects on growth, survival, or reproduction. However, controlled toxicity studies were available only for mink exposed to tPCBs. As a result, data for mink were used to assess toxicity to the river otter, with the assumption that the two species share similar sensitivity to tPCBs.

I.3.3.2 tPCBs for Mink and River Otter

Data were available from several long-term feeding studies to develop a dose-response curve for mink exposed to Aroclor 1254; however, there were few data available on the effects of tPCBs specifically to otter. Mink and otter both belong to the *Mustelidae* family. Because mink and otter have similar aquatic-based diets, belong to the same family, and have similar physical characteristics, it was assumed that the dose-response curve developed for mink is appropriate for the river otter. It is recognized that mustelids may vary in sensitivity to tPCBs (Leonards et al. 1997). For example, Bleavins et al. (1980) found that ferrets are less sensitive to tPCBs than mink. However, numerous researchers have used mink effects data as a representation of the effects of tPCBs to otter (e.g., Traas et al. 2001; Leonards et al. 1997).

Derivation of a dose-response curve requires long-term feeding studies that singly or combined have at least five dose treatments for sensitive endpoints such as mortality or reproductive success. There were four studies that met these criteria and had five or more doses. These studies included Hornshaw et al. (1983; 6 treatments), Hornshaw et al. (1986; 11 treatments), Aulerich and Ringer (1977; 6 treatments), and Bleavins et al. (1980; 5 treatments). Four other studies reported dose-response data for mortality and fecundity, but had fewer than five dose

concentrations. The studies were those performed by Platonow and Karstad (1973; 2 treatments), Aulerich et al. (1985; 2 treatments), and Heaton et al. (1995; 4 treatments). The derivation of a dose-response relationship for fecundity was further refined to include only those studies that had PCB mixtures with at least 54% chlorine content, used a well-characterized diet, and used similar, state-of-the-art exposure protocols and laboratory facilities such as those at Michigan State University.

The acceptable studies that met these criteria were the Bleavins et al. (1980) and Aulerich et al. (1985) studies. Because both studies used similar protocols, exposure duration, and species (a similar strain of farm-raised mink) they were combined to yield a data set with nine treatments for fecundity.

The curve fitting of dose-response data to a generalized linear model (GLiM) was performed using SAS® (SAS Institute, Cary, NC) (Bailer and Oris 1997). Fecundity data were subjected to a logarithmic link function and assumed to have a Poisson error distribution. Figure I.3-7 depicts the data set and fitted model for female mink fecundity. The dose-fecundity model (Figure I.3-7) fitted by the GLiM method using the log link function and a Poisson error distribution was highly significant ($P < 0.0001$; F value = 31.2; degrees of freedom 15; $\beta_0 = -2.3$; $\beta_1 = -3.0$; seβ_0 = 0.90; seβ_1 = 0.72; corr $\beta_0\beta_1$= 0.979).

I.3.3.3 2,3,7,8-TCDD Toxic Equivalence for Mink and River Otter

There were four studies that estimated effects of TEQ to survival, reproduction, and growth of mink following chronic exposures. The studies were by Heaton et al. (1995), Hochstein et al. (1998, 2001), and Aulerich et al. (1988). However, these studies employed field-collected fish that had contaminants other than dioxins or dioxin-like compounds. Therefore, the observed responses could not be conclusively linked to TCDD and equivalents, which precluded the development of either a dose-response curve, or a NOAEL or LOAEL specific to TEQ from these studies. The third option was to use field-based information on mink and otter to develop a field-based threshold. The studies by Heaton et al. (1995), Hockstein et al. (1998, 2001), and Aulerich et al. (1988) involved exposing mink to fish contaminated with TEQ and other contaminants. Based on a review of these studies, it appears that adverse effects on growth in kits begin to occur at concentrations of 3.6 ng/kg bw/d (lower toxicity threshold; Heaton et al.

1995). The highest dose that did not cause adverse effects was 36 ng/kg bw/d (upper toxicity threshold; Hochstein et al. 2001). Thus, the threshold range, based on studies that used field-collected fish, is 3.6 to 36 ng/kg bw/d for piscivorous mammals.

I.4 RISK CHARACTERIZATION

I.4.1 Introduction

This section characterizes the risks to piscivorous mammals exposed to PCBs and TEQ in the PSA of the Housatonic River. The risk characterization approach includes an application of the WOE methodology developed by MDEP (Menzie et al. 1996).

The risk characterization for piscivorous mammals in the PSA used three lines of evidence to determine risks for this endpoint. The three lines of evidence include: (1) field survey information from the PSA and nearby reference areas; (2) a study in which female mink were fed fish collected from the PSA to determine effects on reproduction, kit survival, and growth and development; and (3) comparison of estimated exposures to laboratory-derived effects endpoints. These lines of evidence are considered to be independent and are combined at the end of this section to develop the WOE assessment.

The risk questions addressed in this section include:

- Are the concentrations of tPCBs and TEQ in the prey of piscivorous mammals sufficient to cause adverse effects to individuals inhabiting the Housatonic River PSA?
- If so, how severe are the risks and what are their potential consequences?

I.4.2 Mink/Otter Field Surveys

Field surveys of mammals in the PSA, including some specific to mink and otter, were conducted by EPA in 1998, 1999, and 2000 (Appendix A). A subsequent survey was performed by GE (BBL 2002). The methods and results of the EPA survey are summarized in the next section and the GE survey is described in the following section. A more detailed description of the EPA survey can be found in Appendix A, and the GE survey in BBL (2002).

I.4.2.1 Introduction and Methods (of EPA Study)

Mink and Otter Surveys

Surveys for mink and otter in the PSA and reference areas were performed over multiple years to characterize the extent and seasonality of use, and to compare differences in use patterns between the PSA and reference areas.

Snow Tracking

Mammal snow track counts (Halpin 1984, Halpin and Bissonette 1988) were conducted during the winters of 1998-1999 (hereafter 1999) and 1999-2000 (hereafter 2000) in the riparian habitats of the PSA, as well as in four reference areas (Threemile Pond, Washington Mountain Lake in October Mountain State Forest, Muddy Pond in Hinsdale Flats SWMA, and Ashley Lake). During the 1999 survey, six 500-m (1,650-ft) transects were established in the PSA so that many of the habitat types used by mink and otter (e.g., low-gradient stream, black ash–red maple–tamarack calcareous seepage swamp, shrub swamp, deep emergent marsh, shallow emergent marsh, and wet meadow) were sampled (Appendix A, Map 6-1). Transects were surveyed after a fresh snowfall for a minimum of two or three snow events each winter. Mammal tracks were recorded according to species (or lowest identifiable taxonomic level), and photographs were taken of tracks of representative species (e.g., mink, otter). Transect locations were recorded and plotted on a map of the PSA. Ashley Lake and Washington Mountain Lake in October Mountain State Forest were surveyed as reference areas during the 1999 winter. Because the lakes were small, entire shorelines were surveyed rather than establishing transects. During the 2000 surveys, three transects were established in the PSA and were sampled using methods similar to the 1999 transects (Map 6-1). During that winter, however, mink and otter scents were placed every 60 m (200 feet) along transects in an effort to attract these animals to the transects and to determine whether or not these species were present in the PSA (Appendix A). In addition, three transects were established at each of four reference areas (Appendix A, Maps 6-2, 6-3, 6-4, and 6-5). Identification of tracks followed methods in Murie (1974), Halfpenny and Biesiot (1986), Forrest (1988), and Rezendes (1999).

Scent Post Surveys

Scent post station surveys (Conner et al. 1983) were used during the fall of 1998 and 1999 and concurrent with the winter 2000 snow tracking surveys to determine the presence of mink and river otter in the PSA (WESTON 2000). Three transects were established during the fall surveys, one each in the upper, middle, and lower portions of the PSA (Appendix A, Map 6-6). Each transect ran parallel to the shoreline for 500 m (1,650 feet) and consisted of 10 scent post stations placed at 60-m (200-foot) intervals and 0.3 to 1.5 m (1 to 5 feet) from the shoreline. Each scent station consisted of a 1-m (3.3-foot) diameter circle of moist sand sifted into place, with a wooden dowel placed in the center and smeared with a commercial lure (Leon Lures® Mink #1 Super All Call and Otter Super All Call) (Linhart and Knowlton 1975, Phillips 1982). Lures were alternated between stations so that half of the stations on each transect were baited with mink lure and half with otter lure (Humphrey and Zinn 1982).

During the winter 2000 surveys, scent posts were placed along the snow tracking transects in the PSA (Map 6-1) and in each of the four reference areas (Maps 6-2, 6-3, 6-4, and 6.5). The methods of applying, spacing, and alternating the scents were the same as the fall surveys, except that, rather than using sand as a track medium, the snow surrounding each scent post was examined.

Otter Scat Analysis

River otter scat discovered during the scent post and snow tracking surveys was collected to analyze prey species. After collection, scats were measured, photographed, and analyzed for prey composition. Fish scales were removed and placed in envelopes to dry. The scales were shipped to the Laconia, New Hampshire Office of Fishery Assistance, USFWS, where they were identified to lowest possible taxonomic group and aged. Identifications were based upon voucher scales taken during previous fish sampling events from the Housatonic River, or from other areas. That information was summarized by the USFWS (Smithwood 2002).

I.4.2.2 Results (EPA Study)

I.4.2.2.1 Mink

Mink tracks (Appendix A, Figure 6-6) and scat were observed at several locations in the PSA during snow tracking surveys. Tracks were observed at each end of the PSA, occurring near the confluence of the East and West Branch of the Housatonic River and near Willow Creek; no observations were made in the middle portion of the PSA. One set of tracks near the confluence exhibited a continuous pattern before blood-stained snow was observed, suggesting that the mink had killed a cottontail or other prey item. It appeared that the rabbit left tracks in the snow immediately in front of the mink that was apparently chasing it. The tracks near Willow Creek were associated with a hole in the ice in the impounded portion of the creek behind the railroad tracks, an area of little or no detected PCBs.

During the fall 1999 scent post survey, an additional set of mustelid tracks and scat were observed just south of the confluence of the East and West Branch Housatonic River. Due to sexual dimorphism among mustelid species, male long-tailed weasel and female mink overlap in size. Therefore, tracks alone cannot always distinguish between these species. The size of the observed tracks fell within the overlap of these two species and could not be positively identified. Scat found at this location was composed of fragments of bones and fur from a small mammal (Appendix A, Figure 6-7).

In conclusion, mink were observed twice as much in reference areas as in the PSA floodplain (0.037 sightings/hour vs. 0.019 sightings/hour, respectively) (Table I.4-1).

I.4.2.2.2 River Otter

Historical trapping data from the MDFW show that river otter have been present in the Housatonic River watershed (1977–1999) (S. Langlois, MDFW, personal communication 2000). However, it is not known if these individuals were captured from the Housatonic River or other bodies of water within the extensive watershed.

River otter were not observed in the PSA during 1998 or 1999 snow tracking or scent post surveys. However, river otter tracks, slides, and scats were observed in the PSA during February 2000 during the winter track and scent post surveys. One set of slides, tracks, and scat occurred

approximately 485 m south of the confluence of the East and West Branches of the Housatonic River in the PSA. Scats were observed one month later and appeared old, having likely been buried under the snow during earlier visits. They consisted primarily of fish scales and bones, with some containing small amounts of crayfish exoskeletons. Another set of slides, tracks, and scat was observed further downstream, just north of New Lenox Road during the winter 2000 snow tracking and scent post surveys (Appendix A, Figure 6-3 and Figure 6-4). The individual appeared to have been attracted by the lure and deposited its scat and scent markings. An additional river otter scat was collected adjacent to Woodland Road, approximately 800 m (0.5 mile) downstream of the Yokun Brook confluence. This scat was fresh at the time of collection on 22 March 2000, and consisted of fish scales and crayfish fragments. This was an incidental observation, not the direct result of snow tracking or scent post surveys. Nevertheless, it appears that river otter from outside areas move into the PSA to find food.

Ten otter scat samples were collected, seven from the PSA and three from Muddy Pond at the Hinsdale Flats SWMA reference area. Scats were composed of primarily fish scales and bones (Appendix A, Figure 6-5). Five of these scats contained small amounts of crayfish exoskeleton as well; total crayfish composition never exceeded 20 percent of the scat volume. Scat analysis by the U.S. Fish and Wildlife Service (Smithwood 2002) showed that all families of fish from the PSA were present except Salmonidae and Percicthyidae, although Centrachids were the only family represented in all scats. Otter foraged on both juvenile and adult fish, and the majority of the scales were from fish under 3 years of age. These results fall within those reported in the literature.

River otter were commonly observed at all four of the reference areas during the 1998 through 2000 field surveys. Ashley Lake had 0.049 sightings per hour, Washington Mountain Lake had 0.167 sightings per hour, Threemile Pond had 0.167 sightings per hour, and Muddy Pond had 0.231 sightings per hour. River otter or indications of their presence were observed on four occasions during 260.5 hours of survey (0.015 sightings per hour) in the PSA, while they were encountered 8.7 times more frequently (14 times during 108 hours) in reference areas (Table I.4-1).

I.4.2.2.3 Discussion

River otter and mink habitat is often associated with beaver activity; beaver ponds provide an abundant supply of prey, stable water levels, den sites, and escape cover (Newman and Griffin 1994). Along with beaver activity, vertical banks, rock formations, and backwater sloughs have been shown to be associated with denning sites for river otter, whereas mink frequently use muskrat dens and lodges. Points of land, tributary streams, fallen logs, log jams, conifer trees, and pools have all been correlated with river otter latrines (Sheldon and Toll 1964, Dubuc et al. 1990, Newman 1990, Swimley et al. 1998). The Housatonic River in the PSA offers an abundance of habitats that meet these requirements. However, otter and mink sign were very infrequent, much lower in occurrence than what would be expected considering the available habitats and food resources. For example, despite hundreds of hours conducting track and scent post surveys for otter and mink, and thousands of hours of field time spent characterizing the ecological communities in the PSA, very few otter or signs of otter were observed. Conversely, despite substantially fewer hours spent at reference areas, otter were frequently observed.

I.4.2.3 Mink Presence, Distribution, and Abundance in the PSA (GE Survey)

Introduction

GE retained BBL to determine the presence or absence and possible distribution of wild mink in the Housatonic River area between the spring of 2001 and the spring of 2003 (Bernstein et al. 2003). The study during the winter of 2002/2003 was expanded to include river otter. The methods used were similar to those in the EPA surveys, and consisted of looking for tracks in soft sand at scent post stations in the spring, summer, and fall (mink only), and snow tracking (mink and otter) during the winter months. Surveys were performed in suitable mink habitat, including irregular shorelines and backwaters with dense, wooded cover near the water. Additional efforts included setting traps, scented burrows, and deploying motion sensitive cameras. The observations were conducted along the Housatonic River between New Lenox Road and Woods Pond (the midpoint of Reach 5B, and Reach 5C and 6; no work was performed in the upstream half of the PSA).

Materials and Methods

According to Bernstein (2003), in June of 2001, 70 scent post stations were placed along the main stem of the Housatonic River in the PSA. An additional 30 scent post stations were placed on tributaries. The scent post stations consisted of an attractant placed in the center of 1-m circle cleared of vegetation and covered with fine sand. The attractants included pieces of wool scented with mink urine, amino acid disks, and mink scat from a commercial fur farm. The stations were monitored on 3 days every month until December of 2001.

During the winter (between December and March 2001/2002 [mink] and 2002/2003 [mink and river otter], field staff looked for snow tracks in areas that were likely to support mink and river otter, such as open springs and natural dens. Any observed tracks were photographed for subsequent identification by experts.

Results and Discussion

The study reported that 35 sets of mink tracks were observed between April 2001 and March 2002. A total of 33 sets of mink and 41 sets of river otter tracks were observed in 2003. However, only 4 out of the 35 sets of mink tracks were observed in the snow-free months. In 2003, all mink and river otter tracks were observed in winter. This suggests that either the observation methods used in the spring, summer, and fall were ineffective or mink and river otter were not present in those seasons.

If the lack of sightings during the spring, summer, and fall was due to the fact that no mink were present in the PSA (a hypothesis supported by very few tracks at post stations, no photographs recorded by the motion-sensitive camera in the snow-free months, and no tracks in scented burrows), then the tracks detected in winter likely belonged to transient mink rather than to local residents. Local residents would leave many tracks during the snow-free months of the year. Also, the tracks may have been mistaken for muskrat or other tracks. Misidentification of some mink tracks was an issue in the field survey, based on EPA's review of photographs provided by Bernstein et al. (2003) and observations recorded during EPA oversight of the study (Woodlot Alternatives, Inc. 2002). The 2003 survey discovered one confirmed and one suspected river otter den site. However, the presence of one den site in winter is not evidence of a river otter population living and reproducing in the PSA.

Conclusions

The Bernstein et al. (2003) report did not discuss the implications of the disproportionate number of sightings in winter versus other seasons, apparent ineffectiveness of scent posts, lack of inclusion of reference areas outside the PSA, uncertainties associated with the lack of tracking expertise and experience, results of motion-sensitive camera trials (i.e., no mink or river otter observed in the snow-free months), uncertainty in determining species or sex of mink from tracks, and uncertainty in attributing sets of tracks from areas located close together to separate individuals. Therefore, EPA does not believe that the conclusions of the report, that 4 to 10 individuals have established home ranges within the PSA, can be substantiated based on the data.

Instead, the data collected in the study suggest that there are no permanent and resident mink or river otter in the PSA, and the mink or river otter that do enter the PSA are transient individuals. Mink and river otter often extend their foraging range in winter, when food supplies are scarce (EPA 1993). The PSA contains good mink habitat and food. Thus, mink are likely attracted to it in winter. The data collected in this study are in agreement with the data collected in the EPA study.

In summary, this report cited incidences of mink and river otter signs in the PSA. However, the study had several limitations, which led to conclusions that are not supported by the data. These limitations include (Woodlot Alternatives, Inc. 2002):

- Virtually all mink and river otter track observations were made in winter.
- Errors made during the construction of scent posts may have rendered the posts ineffective.
- Lack of experience in wildlife tracking in some cases resulted in errors in proper identification of tracks as identified by EPA oversight personnel while in the field.
- The poor quality of track photos precludes the ability of an expert not present in the field to identify species, age, or sex. No supporting evidence was provided for the determination of sex of mink in photographed snow tracks (e.g., photos did not include a scale as a reference, yet mink cannot be sexed without track length and lope distance measurements).
- Relatively few photographs of tracks were included in the report compared to the number of observations reported, thus precluding the ability to verify most of the observations.

- No confirmed tracks were observed in the presence of EPA oversight personnel.
- No mink/otter sightings using motion-sensitive cameras in snow-free months or lures placed in burrows were made.
- The study reported few mink sign during the spring, summer, and fall, such as tracks in mud and sand, scat, kill leftovers, and active burrows belonging to resident mink. The lack of mink sign occurred despite the deployment of 100 scent stations, motion-sensitive cameras, scented burrows, and many hours of direct observations.
- No efforts were made to determine whether the discovered river otter den was permanent or temporary, and no determination of sex of river otter was made.

I.4.3 Comparison of Estimated Exposures to Laboratory-Derived Effects Doses

Exposure was assessed for mink and river otter in the PSA. Because Reaches 5 and 6 combined roughly correspond to the size of the home range for otter, these reaches were combined for the otter analyses. For mink, the assessment was conducted separately for each reach because of the smaller foraging range. For comparative purposes, exposure was also estimated for mink and otter in two reference areas: the upstream reference area and Threemile Pond. Moreover, exposure was also estimated for mink and river otter foraging 50, 25, and 10% of time in the PSA. The probabilistic exposure analysis for piscivorous mammals used a dietary modeling approach for calculating exposure to tPCBs and TEQ. A review of the possible exposure pathways and their relative importance to the overall exposure confirmed that ingestion of food items (fish, invertebrates, waterfowl, amphibians, and small mammals for mink, and fish and crayfish for river otter) was the only pathway of concern for these species.

The effects characterization provides an overview of the current literature on the potential effects of tPCBs and TEQ to piscivorous mammals. Numerous studies tested toxicity to mink directly and, thus, were highly relevant. However, no such studies were identified for river otter. There were sufficient data for mink exposed to tPCBs to derive a dose-response relationship. This relationship was used with the results of the exposure analyses to derive risk curves for mink and river otter. Dose-response curves for mink and river otter could not be developed for TEQ. Therefore, a threshold range was developed instead.

The following integrates the results of the exposure and effects assessments developed for each species, COC, and exposure area. The categories of low, intermediate, and high risk were derived using the following approach:

- If risk curves were available:
  - If the probability of 10% or greater effect was less than 20%, then the risk to piscivorous mammals was low.
  - If the probability of 20% or greater effect was greater than 50%, then the risk to piscivorous mammals was high.
  - All other outcomes were considered to have intermediate risk.
- If no risk curves were available:
  - If the probability of exceeding the lower toxicity threshold was less than 20%, the risk to piscivorous mammals was low.
  - If the probability of exceeding the upper toxicity threshold was greater than 20%, the risk to piscivorous mammals was high.
  - All other outcomes for the lower and upper thresholds were considered to have intermediate risk.

The results of the Monte Carlo analysis were used to determine the "risk category." A similar exercise was conducted using the results of the probability bounds analysis. The lower and upper-bound risk categories are expressed as the "risk range."

I.4.3.1 tPCB Risk Characterization – Mink and River Otter

The dose-response relationship for tPCBs was based on data from the studies by Bleavins et al. (1980) and Aulerich et al. (1985), which examined the effects of dietary exposure to the fecundity of female mink. The dose-response relationship indicated that 10 and 20% declines in fecundity would be expected at doses of 0.0128 and 0.0272 mg/kg bw/d tPCBs, respectively.

I.4.3.1.1 Mink – Reach 5

The Monte Carlo estimates of total daily intake of tPCBs by mink foraging in Reach 5 100% of the time indicated that there was a 100% probability of exceeding the 10 and 20% effect doses (Figure I.4-1; Table I.4-2). Further, there was also a 100% probability of a 50% or greater

reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 98 to 100%, given uncertainties that existed about parameterization of the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effect dose ranges from 86 to 100%, given the same sources of uncertainty. Thus, mink exposed to tPCBs in Reach 5 of the Housatonic River were considered to be in the high-risk category with a risk range of high (Table I.4-3).

The potential for exposure to tPCBs at this location was so high that even individuals foraging in Reach 5 10% of the time had a 100% probability of exceeding the 20% effect dose (Table I.4-2) according to the results of the Monte Carlo analyses (Figure I.4-2).

I.4.3.1.2 Mink – Reach 6

The Monte Carlo estimates of total daily intake of tPCBs by mink foraging in Reach 6 100% of the time indicated that there was a 100% probability of exceeding the 10 and 20% effect doses (Figure I.4-3; Table I.4-2). Further, there was also a 100% probability of a 50% or greater reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 59 to 100%, given uncertainties that existed about parameterization of the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effect dose ranges from 40 to 100%, given the same sources of uncertainty. Thus, mink exposed to tPCBs in Reach 6 of the Housatonic River were considered to be in the high risk category with a risk range of intermediate to high (Table I.4-3). Similar to Reach 5, the potential for exposure at Reach 6 was also very high, individuals who forage in Reach 6 only 10% of the time have a 100% probability of exceeding the 20% effect dose (Table I.4-2), according to the results of the Monte Carlo analyses (Figure I.4-4).

I.4.3.1.3 Mink – Upstream Reference Area

The Monte Carlo estimates of total daily intake of tPCBs by mink foraging in the upstream reference area 100% of the time indicated that there was a 50% probability of exceeding the 10% effect dose, and a 17% probability of exceeding the 20% effect dose (Figure I.4-5; Table I.4-2). There was 0% probability of a 50% or greater reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 2 to 95%, given uncertainties that existed about how to parameterize the Monte Carlo exposure model.

The corresponding probability of exceeding the 20% effect dose ranges from 0 to 65%, given the same sources of uncertainty. Thus, mink exposed to tPCBs in the upstream reference area were considered to be in the intermediate-risk category with a risk range of low to high (Table I.4-3). However, those individuals that forage 50% of the time or less at this location have a very low probability of daily intake rates exceeding the 10% and 20% effect doses. Figure I.4-6 depicts risks for mink foraging 10% of the time in the upstream reference areas.

I.4.3.1.4 Mink – Threemile Pond Reference Area

The Monte Carlo predictions for total daily intake of tPCBs by mink foraging in the Threemile Pond reference area 100% of the time indicated that there was a 91% probability of exceeding the 10% effect dose, and a 78% probability of exceeding the 20% effect dose (Figure I.4-7; Table I.4-2). There was a 37% probability of a 50% or greater reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose could range from 0 to 100%, given uncertainties that existed about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effect dose could range from 0 to 98%, given the same sources of uncertainty. Thus, mink exposed to tPCBs in the Threemile Pond reference area were considered to be in the high-risk category with a risk range of low to high (Table I.4-3). Individuals that forage 10% of the time at this location have a 50% and 13% probability of exceeding the 10 and 20% effect doses, respectively (Figure I.4-8).

I.4.3.1.5 River Otter – Reaches 5 and 6

The Monte Carlo predictions for total daily intake of tPCBs by river otter foraging in Reaches 5 and 6 100% of the time indicated that there was a 100% probability of exceeding the 10 and 20% effect doses (Figure I.4-9; Table I.4-2). Further, there was also a 100% probability of a 50% or greater reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10 and 20% effect doses was 100%, despite uncertainties that exist about parameterization of the Monte Carlo exposure model. Thus, river otter exposed to tPCBs in Reaches 5 and 6 were considered to be in the high-risk category (Table I.4-3). The lowering of the foraging time from 100% to 10% at this location did not decrease the risk rating, suggesting that the potential for exposure and risk in Reaches 5 and 6 is very high (Figure I.4-10).

I.4.3.1.6 River Otter – Upstream Reference Area

The Monte Carlo predictions for total daily intake of tPCBs by river otter foraging in the upstream reference area 100% of the time indicated that there was a 72% probability of exceeding the 10% effect dose, and a 37% probability of exceeding the 20% effect dose (Figure I.4-11; Table I.4-2). There was a 6% probability of a 50% or greater reduction in fecundity. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 69 to 89%, given the sources of uncertainty that existed about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effect dose ranges from 26 to 49%, given the same sources of uncertainty. Thus, river otter exposed to tPCBs in the upstream reference area are considered to be in the intermediate-risk category with a risk range of low to intermediate (Table I.4-3). River otter that forage 10% of the time at this location have 6% and 39% probabilities of exceeding the 10% and 20% effects thresholds. Those probabilities drop to zero for individuals that forage only 10% of the time in the upstream reference area (Figure I.4-12).

I.4.3.1.7 River Otter – Threemile Pond Reference Area

The Monte Carlo predictions for total daily intake of tPCBs by river otter foraging in the Threemile Pond reference area 100% of the time indicated that there was a 13% probability of exceeding the 10% effect dose (Figure I.4-13; Table I.4-2). The probability of exceeding the 20% effect dose was 0%. The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 6 to 19%, given uncertainties that existed about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effect dose ranges from 1% to 5%, given the same sources of uncertainty. Thus, river otter exposed to tPCBs in the Threemile Pond reference area are considered to be in the low-risk category with a risk range of low to intermediate (Table I.4-3). The probability of exceeding the 10% effect dose, however, decreased to 0% for otter that forage at this location only 10% of time (Figure I.4-14).

I.4.3.2 TEQ Risk Characterization – Mink and River Otter

Dose-response curves, NOAELs, or LOAELs could not be developed for mink and river otter exposed to TEQ. The next option was to use field-based information on mink and otter to

develop a field-based threshold range. The studies by Heaton et al. (1995), Hochstein et al. (1998, 2001), and Aulerich et al. (1988) involved exposing mink to fish contaminated with TEQ and other contaminants. Based on a review of these studies, a lower toxicity threshold of 3.6 ng/kg bw/d and an upper toxicity threshold of 36 ng/kg bw/d were selected as the field-based threshold range for mink and river otter.

I.4.3.2.1 Mink – Reach 5

The Monte Carlo predictions for total daily intake of TEQ by mink foraging in Reach 5 100% of the time indicated that there was a 100% probability of exceeding the lower effect threshold and a 97% probability of exceeding the upper effect threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold ranges from 52% to 100%, given the uncertainty in the Monte Carlo model. The corresponding probability of exceeding the upper threshold ranges from 3% to 100%, given the same sources of uncertainty in the Monte Carlo model. Thus, mink exposed to TEQ in Reach 5 are considered to be in the high-risk category with a risk range of intermediate to high (Table I.4-3). Reduced foraging time decreases the risk of adverse effects, but the decrease is modest. At 10% foraging time, there is still a 97% probability of exceeding the 10% effects threshold and a 12% probability of exceeding the 20% effects threshold (Table I.4-2).

I.4.3.2.2 Mink – Reach 6

The Monte Carlo predictions for total daily intake of TEQ by mink foraging in Reach 6 100% of the time indicated that there was a 100% probability of exceeding the lower effect threshold and an 83% probability of exceeding the upper effect threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold ranges from 28% to 100%. The corresponding probability of exceeding the upper threshold ranges from 3% to 100%, given the uncertainty in the Monte Carlo model. Thus, mink exposed to TEQ in Reach 6 of the Housatonic River were considered to be in the high-risk category with a risk range of intermediate to high (Table I.4-3). Reduction of foraging time to 10% does not reduce risk to the low category.

I.4.3.2.3 Mink – Upstream Reference Area

The Monte Carlo predictions for total daily intake of TEQ by mink foraging in the upstream reference area 100% of the time indicated that there was a 64% probability of exceeding the lower effect threshold and a 0% probability of exceeding the upper effect threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold ranges from 16% to 100%. The corresponding probability of exceeding the upper threshold ranges from 0 to 82%, given the uncertainty in the Monte Carlo model. Thus, mink exposed to TEQ in the upstream reference area of the Housatonic River were considered to be in the intermediate-risk category with a risk range of low to high (Table I.4-3). That risk decreased to the low category for individuals that foraged less than 25% of the time at this location.

I.4.3.2.4 Mink – Threemile Pond Reference Area

The Monte Carlo predictions for total daily intake of TEQ by mink foraging in the Threemile Pond reference area 100% of the time indicated that there was a 96% probability of exceeding the lower effect threshold and a 32% probability of exceeding the upper effect threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold ranges from 0 to 100%. The corresponding probability of exceeding the upper threshold ranges from 0 to 90%, given the uncertainty in the Monte Carlo model. Thus, mink exposed to TEQ in Threemile Pond were considered to be in the low-risk category with a risk range of low to high (Table I.4-3). Reducing foraging time from 100% to 10% had the effect of decreasing the probabilities of exceeding the 10% and 20% effects thresholds to 32 and 0%, respectively.

I.4.3.2.5 River Otter – Reaches 5 and 6

The Monte Carlo predictions for total daily intake of TEQ by river otter foraging in Reaches 5 and 6 100% of the time indicated that there was 100% probability of exceeding the lower effect threshold (Table I.4-2) and an 82% probability of exceeding the upper effect threshold. The probability bounds analysis indicated that the probability of exceeding the lower threshold was 100%. The probability of exceeding the upper threshold ranges from 72% to 97%, given the uncertainties as to how to parameterize the Monte Carlo exposure model. Thus, river otter exposed to TEQ in Reaches 5 and 6 of the Housatonic River area were considered to be in the high-risk category (Table I.4-3). Lowering of foraging time to 10% decreased risk to the

intermediate category. Thus, the level of risk remained above the low category despite a 10-fold drop in exposure at this location.

I.4.3.2.6 River Otter – Upstream Reference Area

The Monte Carlo predictions for total daily intake of TEQ by river otter foraging in the upstream reference area 100% of the time indicated that there was a 94% probability of exceeding the lower effects threshold and a 10% probability of exceeding the upper effects threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold could range from 15% to 100%. The corresponding probability of exceeding the upper threshold ranges from 0 to 19%. Thus, river otter exposed to TEQ in the upstream reference area of the Housatonic River were considered to be in the intermediate risk category with a risk range of low to intermediate (Table I.4-3). The risk remained in the intermediate category even for otter that foraged only 10% of the time at this location.

I.4.3.2.7 River Otter – Threemile Pond Reference Area

The Monte Carlo predictions for total daily intake of TEQ by river otter foraging in the Threemile Pond reference area 100% of the time indicated that there was a 7% probability of exceeding the lower toxicity threshold, and a 0% probability of exceeding the upper threshold (Table I.4-2). The probability bounds analysis indicated that the probability of exceeding the lower threshold ranges from 0 to 21%, given uncertainties that existed about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the upper threshold was 0%. Thus, river otter exposed to TEQ in the Threemile Pond reference area of the Housatonic River were in the low-risk category with a risk range of low to intermediate (Table I.4-3).

I.4.4 Weight-of-Evidence Analysis

A WOE analysis is used to combine the three major lines of evidence described in the preceding sections for mink and river otter. The goal of this analysis is to determine whether a significant risk of harm is posed to piscivorous mammals in the Housatonic River PSA as a result of exposure to tPCBs and TEQ. The analysis follows the methodology proposed by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996; see Section 2.8 for details). The analyses are conducted separately for tPCBs and TEQ.

I.4.4.1 Evaluating Measurement Endpoints

Measurement endpoints are assigned a relative weight for each of the attributes that account for strength of association between the assessment and measurement endpoints, data quality, and study design and execution. The relative importance of each attribute is indicated by an attribute weighting factor following the guidance provided by Menzie et al. (1996). The attribute weighting factors and measurement endpoint values are presented in Table I.4-4. The discussion of the attribute values for each measurement endpoint is provided in the following section.

I.4.4.1.1 Relationship Between Measurement and Assessment Endpoints

Degree of Association

This attribute quantifies the degree of association between each measurement and the assessment endpoint. In the analysis the assessment endpoint was the survival, growth, and reproduction of piscivorous mammals. For both tPCBs and TEQ, the field surveys by EPA and GE, the MSU feeding study, and modeled exposure and effects lines of evidence were given values of low/ moderate (EPA and GE), high, and high, respectively. The field surveys were designed to record presence, relative abundance, and habitat usage of mink and otter in and around the PSA. Although the surveys were directed toward piscivorous mammals, they did not measure survival, growth, or reproduction directly. Thus, the values assigned to the surveys for this attribute were discounted accordingly.

The MSU feeding study used one of the representative species to determine the direct effects on survival, growth, and reproduction resulting from the feeding of contaminated fish from the PSA. Therefore, there was a high degree of association between the measurement and the assessment endpoint.

The modeled exposure and effects line of evidence directly measured effects on survival, growth, and reproduction of one or both of the two piscivorous mammals considered in this assessment. Thus, the degree of association between the measurement and the assessment endpoints was high.

Stressor/Response Correlation

This attribute measures the strength of correlation between assessment and measurement endpoints in terms of the ability to correlate the magnitude of response with the degree of exposure. For tPCBs, the measurement endpoints for the field surveys (EPA and GE), feeding study, and modeled exposure and effects lines of evidence (tPCBs and TEQ) were given values of moderate, low/moderate, moderate/high, high, and moderate, respectively.

The two field surveys were designed to determine the relative abundance of mink and river otter. In the EPA study, both within and outside the PSA, and in the GE study, solely within the PSA, no reference areas were evaluated. The surveys were not designed to explicitly determine the strength of the relationship between magnitude of effect and degree of exposure. However the inclusion of reference areas provided a measure of relative response in the EPA study, resulting in the values of low/moderate for the GE study and moderate for the EPA study.

The MSU feeding study and PCB modeled exposure and effects lines of evidence both explicitly quantified the relationship for tPCBs and response, and were given a high value. However. for TEQ, the modeled exposure and effects line of evidence was given a moderate value because the effects metric used in the risk characterization was a threshold range, and not a dose-response relationship. The effects metrics, however, were specific to TEQ.

Utility of Measure

This attribute reflects the quality of the accepted criteria, standards, or performance-based measures used to judge environmental harm. For PCBs, the measurement endpoints for the field surveys (EPA and GE), feeding study, and modeled exposure and effects lines of evidence (tPCBs and TEQ) were given values of moderate, low/moderate, high, moderate/high, and moderate respectively.

The EPA field survey was based on techniques that are accepted and have been applied in the field. However, the survey by GE had several issues with study methods and interpretation of results (see Section I.4.2.3), which led to a lower weighting.

The MSU feeding study methods and the techniques used to model exposure and effects are well established, published, and accepted by the scientific community. These measurement endpoints

are sensitive and capable of detecting effects to survival, growth, and reproduction of piscivorous mammals. Thus, the MSU study was given a high value.

The modeled exposure and effects line of evidence for tPCBs was given a moderate/high value because the procedures used were standardized and widely accepted, and the dose/response relationship was derived for mink. The modeled exposure line of evidence for TEQ was given a moderate value because the effects metric used in the risk characterization was a threshold range. While thresholds are considered by regulators and risk assessors to be useful tools in an ERA, they may be of more limited use for making inferences about magnitude of risk.

I.4.4.1.2 Data Quality

Quality of Data

This attribute reflects the degree to which data quality objectives and other recognized characteristics of high quality studies are met. The factors affecting the quality of the study is the appropriateness and implementation of the experimental design and the minimization of the confounding factors. The field survey conducted by EPA was given a moderate/high value. The feeding study was given a high value. For these lines of evidence, data were collected and analyzed using experimental designs and methodologies developed to achieve the DQOs of the ERA.

The field survey performed by GE was given a value of low because of the problems with identifying mink and river otter tracks and other methodological issues (Section I.4.2.3). In addition, prior to implementation of the study, EPA was not provided the opportunity to comment on the study design and to better refine the DQOs and determine the suitability of the data for use in the ERA.

The DQOs established for the sampling and analysis of tissue samples collected by EPA were met for the tissue residue data used in the exposure analyses for both tPCBs and TEQ. Other model parameters were derived from EPA (1993) and other published journal articles using standard methods, and the effects metrics were derived from the peer-reviewed literature, many of which could be evaluated for achievement of DQOs. A value of moderate/high was given for this endpoint.

I.4.4.1.3 Study Design

Site Specificity

The value for this attribute reflects the site specificity of data, media, species, environmental conditions, and habitat for each measurement endpoint. For this attribute, the values for tPCBs and TEQ were the same.

The field survey line of evidence for the EPA study was assigned a high value because the surveys were conducted in the Housatonic River PSA and nearby reference areas, and focused on the habitats likely to be frequented by piscivorous mammals. The GE field study was given a moderate/high value because it failed to include reference areas.

The MSU feeding study was assigned a moderate value because although tissue from the site was used for the exposure to the species of interest, in the feeding study, the site-specific environmental conditions and habitat types found in the Housatonic River area were not replicated.

The modeling of exposure and effects was assigned a value of low/moderate. The prey tissue analyses used in the exposure models were site specific, and other exposure parameters were representative of site conditions. The effects data, however, were laboratory based and did not reflect the site-specific environmental conditions found in the Housatonic River area.

Sensitivity

This attribute refers to the ability of the measurement endpoint to detect changes due to a stressor against a background of natural variability and other sources of uncertainty. For both tPCBs and TEQ, the field surveys (EPA and GE), MSU feeding study, and modeled exposure and effects lines of evidence were given values of moderate, low/moderate, high, and high, respectively.

Given the level of effort and techniques employed, field surveys for piscivorous mammals were expected to detect differences beyond 100-fold or more between the PSA and reference areas. Field surveys, however, are rarely able to detect differences that are two-fold or less. For these reasons, a moderate value was assigned to this measurement endpoint for the EPA study because reference areas were included. The GE field survey was given a low/moderate value because of the limitations with survey methodologies and lack of a reference areas (Section I.4.2.3). In the

MSU mink feeding study, all treatment differences two-fold or higher were detected as significant; therefore, a high value for this attribute was assigned.

For both tPCBs and TEQ, the modeled exposure and effects line of evidence for this attribute was given a high value. The modeled exposure and effects line of evidence directly assessed the exposure-response relationship for the representative species, and mink are particularly sensitive to effects from exposure to PCBs. In addition, laboratory studies from which effects data were derived yielded a dose-response curve, hence the high value.

Spatial Representativeness

This attribute reflects the degree of spatial overlap of study area, measurement locations, locations of stressors, locations of receptors, and points of potential exposure to those receptors. The field surveys (EPA and GE) were given values of high and moderate/high, respectively. The mink feeding study and modeled exposure and effects lines of evidence for both tPCBs and TEQ were all given the moderate/high values.

The field survey performed by EPA evaluated the entire PSA and reference areas, thus, the high value. The biological survey perfumed by GE did not include Reaches 5A and half of 5B in the PSA or any reference areas, hence the reduced value. The feeding study relied on tissues collected from limited locations in the PSA and, thus, the value was reduced.

Some of the exposure parameters in the exposure model were not measured on-site, but were taken from the published literature (e.g., body weight, gross energy, assimilation efficiency). In addition, the effects assessment used toxicity studies conducted in laboratories. Thus, the modeling and effects was assigned a value of moderate/high.

Temporal Representativeness

This attribute describes the degree of coincidence in time between data collection and exposure. For both tPCBs and TEQ, the field surveys (EPA and GE), the MSU feeding study, and modeled exposure and effects lines of evidence were given values of high and moderate/high, moderate/high, and moderate/high, respectively.

The field survey by EPA (Appendix A) occurred over a period of 3 years. In comparison, the survey by GE spanned two seasons. Thus, the EPA field survey was given a value of high, and

the GE field survey was given a moderate/high value. Thus, the feeding study measurement endpoint was given a moderate/high value.

The feeding study was a study that included pre-breeding of adults and the reproductive cycle of mink. While the fish collected for this study were from a short-term sampling event, fish concentrations in the PSA have not exhibited temporal trends in recent years. Thus, the feeding study measurement endpoint was given a moderate/high value.

The exposure model was designed to span the reproductive cycle of mink. The tissue samples used to parameterize the model were collected in multiple sampling events over a period of several years. Thus, these samples are expected to adequately represent the variability in exposure that mink would encounter at the site in the absence of temporal trends. The effects studies selected for tPCBs and TEQ spanned the reproductive cycle of mink and river otter. However, some of the exposure parameters used in the modeling were not site-specific, but were obtained from the literature (e.g., body weight, gross energy, assimilation efficiency). In addition, the effects assessment used toxicity studies conducted in laboratories (i.e., the effect metrics were not site specific). The weighting given for this attribute was moderate/high.

Quantitative Measure

This attribute indicates the degree to which the response can be quantified by a given measurement endpoint. For both tPCBs and TEQ, the field surveys (EPA and GE), feeding study, and modeled exposure and effects lines of evidence were given values of low, high, and high, respectively. The field surveys yielded results that could not be directly attributed to the COCs; therefore, a lower value for this attribute was assigned.

The results from the MSU feeding study yielded quantitative response data for survival, growth, and reproduction of mink that were tested for statistical significance. Thus, the feeding study measurement endpoint was given a high value.

The results from the modeled exposure and effects line of evidence are probabilistic and highly quantitative, although they are not designed for tests of statistical significance (Menzie et al. 1996). In addition, given the use of dose-response curves, results reflect biological significance. Using professional judgment, a high value for this line of evidence was assigned.

Standard Method

This attribute quantifies the extent to which measurement endpoint studies adhered to recognized scientific protocols. For both tPCBs and TEQ, the field surveys by EPA and the feeding study were given a high value. For these lines of evidence, generally accepted methods were used and were directly applicable for the endpoint and site. The field survey by GE was given a value of moderate/high because of limitations in the study methodology (Section I.4.2.3).

The modeled exposure and effects line of evidence included methods, that while generally accepted or discussed in guidance, were slightly modified to be applicable to the goals of the assessment. The probability bounds analysis method is also a relatively new technique for propagating uncertainty. The weighting given for this attribute was moderate/high.

I.4.4.1.4 Conclusions

The values of each measurement endpoint are listed in Table I.4-5 and Table I.4-6. The EPA field survey was determined to have a moderate/high value, the GE survey had a moderate value, the MSU feeding study had a high value, and the modeled exposure and effects for tPCBs and TEQ had a moderate/high value.

I.4.4.2 Magnitude of Response in Measurement Endpoints

The measurement endpoint values are used, together with the magnitude of response in the measurement endpoints, to judge the WOE for adverse effects for the assessment endpoint. The magnitude of response can be presented as qualitative or quantitative measures. Qualitative measures were used here.

The total attribute weighting values, evidence of harm, and magnitude of responses for each measurement endpoint are presented in Table I.4-5 for tPCBs and in Table I.4-6 for TEQ. All three major lines of evidence (except the field survey results as reported by GE) indicated that the elevated concentrations of tPCBs and TEQ in the PSA of the Housatonic River are causing adverse effects of high magnitude to mink and river otter.

The EPA field survey indicated that mink and river otter are rarely present in the PSA and have not established home territories close to the main channel in the PSA. Mink, and to a greater

extent, river otter, were observed much more often in nearby reference areas despite a less intensive sampling effort. The GE report (BBL 2002) presented both contrary and supportive information. Their study corroborated EPA's observations that mink and river otter occur in the PSA primarily during the winter season when individuals expand their territories. The GE report, however, concluded that mink are present in the PSA year-round at expected densities, despite a lack of supporting data (i.e., only 5% of observations of mink were during the growing season). The GE study also had several methodological shortcomings (Section I.4.2.3) that discount the proposed conclusions.

The MSU feeding study indicated that feeding adult female mink with a diet containing 3.51% fish collected from the PSA caused a statistically significant reduction (46% compared to controls) in kit survival to 6 weeks of age. Mink are opportunistic predators that feed on a variety of prey items in addition to fish. Stomach content analyses conducted in a variety of habitats have consistently shown, however, that the proportion of fish in the mink diet is much higher than 3.5%, typically being around 30% and as high as 85% (see EPA 1993 for a summary of relevant studies). Further, other prey items in the mink diet (e.g., small invertebrates and mammals) are also contaminated with tPCBs and TEQ in the PSA, which have been measured at high concentrations. These considerations indicate that mink resident in the PSA would likely experience catastrophic effects on early kit survival. This conclusion is not altered by the potential reduction in toxicity based on fish species and sizes more typically consumed by mink than those collected and used in the feeding study.

The results from the jaw lesion study indicate that exposure to relatively low levels of PCB-126 (0.96 mg tPCBs/kg diet) can induce maxillary and mandibular squamous cell proliferation. Exposure of mink to higher PCB concentrations and for longer periods, as expected in the Housatonic River ecosystem, would undoubtedly cause increased severity of the lesions leading to loss of teeth and possibly death of starvation The occurrence of jaw lesions coincides with the induction of Ah-receptor-regulated enzymes (ECOD and EROD) also in a dose-response manner.

The results from the modeled exposure and effects indicated that there was a high probability of adverse effects to mink and otter exposed to tPCBs and TEQ in the PSA. Risks are also above

the low category for mink and river otter in the reference areas, particularly for tPCBs. Exposure of piscivorous mammals to tPCBs and TEQ is one to three orders of magnitude higher in the PSA than would occur in the reference areas. As a result, mink and otter are at risk in the PSA, even if they only forage in the area for short periods of time (less than or equal to 10% of foraging time). This would not be the case in the reference areas, where mink and otter could likely forage for months without experiencing adverse effects.

I.4.4.3 Concurrence Among Measurement Endpoints

The final component in the WOE approach addresses the examination of agreement, logical connections, interdependencies, and correlations among the measurement endpoints as they relate to each assessment endpoint. The methodology for detecting concurrence involves the use of a graphical method where measurement endpoints are plotted on a matrix that also includes the weight of each endpoint and degree of response. Tables I.4-7 and I.4-8 depict the outcomes for mink and river otter exposed to tPCBs and TEQ, respectively. Clearly, the three major lines of evidence all suggest that tPCBs and TEQ pose a very serious risk to mink and river otter inhabiting the PSA of the Housatonic River. The data presented in the GE study support this conclusion as their surveys did not confirm that mink and otter were residing in the study area during the growing season.

I.4.5 Sources of Uncertainty

The assessment of risk to piscivorous mammals contains uncertainties. Each source of uncertainty can influence the estimates of risk, therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to piscivorous mammals are described below.

- In this assessment, it was assumed that dietary exposure represents the most important pathway of exposure for piscivorous mammals exposed to COCs. Although unlikely to provide a major contribution to the risk, other pathways could increase the exposure and perhaps increase risk slightly (Moore et al. 1999). Other pathways include drinking water intake, incidental ingestion of sediment, inhalation, transdermal uptake, and preening activity. When drinking water was included in a screening level analysis for piscivorous mammals, the results showed negligible increases in exposure due to drinking water and their likely low importance. The

remaining pathways were not assessed due to the difficulty in quantifying intake via those routes.

- The Monte Carlo sensitivity analyses indicated that the free metabolic rate (*FMR*) slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. However, no measurements of free metabolic rate were available for the representative wildlife species. Similarly, measured food intake rates were not available for mink and river otter or reasonable surrogate species. Therefore, free metabolic rates were estimated using allometric equations. The use of allometric equations introduces some uncertainty into the exposure estimates because they have model-fitting error and are based on species different from the representative species used in this assessment. For mink and river otter, the carnivora model of Nagy et al. (1999) was selected as the most appropriate allometric model to estimate free metabolic rate. Examples of other species used in the model included cat, fox, dog, and wolf. Given the lack of data on representative species used in the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as inputs for the allometric slope and power terms.

- The free-living metabolic rate equations require body weight as an input variable. Data on the body weight of mink and river otter living in the PSA are not available. As a result, body weights for those receptors were estimated from the literature. The potential magnitude and direction of the uncertainty associated with lack of site-specific information on body weights of mink and otter are unknown, but are likely small.

- Because no stomach contents or other dietary analyses were available for mink in the PSA, dietary compositions were derived from those reported in the literature from other similar geographical locations. The potential magnitude and direction of the uncertainty associated with lack of information on diet are unknown. The uncertainty due to lack of knowledge on the diet of mink in the PSA was partially addressed by using distributions to represent variability in diets at other similar sites. Small mammals were the most contaminated prey, thus, any increases in the proportion of this type of prey consumed would lead to increases in exposure. Conversely, amphibians were the least contaminated prey, thus, increases in the intake of this prey item would lead to decreases in exposure to tPCBs and TEQ.

- Sample sizes were limited for the analyses of COC concentrations in some prey items. For example, there were only four amphibian samples for tPCBs from the upstream reference area, only one amphibian sample for TEQ from the upstream reference area and the Threemile Pond reference area, and only three invertebrate samples for TEQ from the upstream reference area Uncertainty due to sample size was explicitly addressed in the uncertainty analyses. In the Monte Carlo analysis, sample size uncertainty was addressed by use of the 95% upper confidence limit (UCL) on the mean. The use of the UCL addressed uncertainty, but it was biased toward overestimating exposure. In the probability bounds analysis, uncertainty was

addressed by specifying concentration variables as a range from the 5% lower confidence limit (LCL) to the UCL. This treatment of uncertainty was unbiased.

- Data on concentrations of tPCBs and TEQ in crayfish and mammals were not available for Reach 6. In those cases, the concentrations of tPCBs and TEQ in prey were estimated using ratios between prey items at other locations for which full data sets existed. This type of extrapolation introduces some uncertainty regarding the concentration of COCs in prey tissue, although the magnitude and direction of this uncertainty is difficult to judge.

- In some instances, data on concentrations of tPCBs and TEQ in crayfish, birds, and mammals from reference areas were not available. In those cases, an assumption was made that these prey items contained no detectable residues of the two contaminants. The magnitude of uncertainty introduced by this assumption is likely to be small because sediment data indicate that detectable tPCB and TEQ residues are rare at those sites.

- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). This source of uncertainty was addressed in the uncertainty analyses by estimating prey concentrations assuming concentrations of PCB-123 and PCB-157 were equal to zero, and assuming that concentrations of PCB-123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. The resulting TEQ estimates were then compared. If the ratio of the upper to lower bound TEQ estimates was less than 1.3, this source of uncertainty was deemed unimportant and disregarded. If the ratio exceeded 1.3, the uncertainty due to the co-elution was propagated through the uncertainty analyses.

- The base exposure scenario for mink and river otter assumed that these animals would forage 100% of their time in the PSA. This assumption is reasonable given the similarity between the size of the PSA and the foraging ranges of these species. However, some individuals might forage part of the time outside the PSA on less contaminated prey. The exposure and risk analyses indicated that individual mink and river otter that forage even a small fraction of time in the PSA (≤ 10%) are at high risk, particularly for tPCBs.

- There was some uncertainty in the effects assessment associated with the lack of toxicity studies involving river otter. As a result, existing toxicity information for mink was used to estimate effects on river otter. This approximation, however, likely introduces relatively little uncertainty because both species belong to the same taxonomic group and are likely to have similar sensitivities to tPCBs and TEQ.

- To derive dose-response relationships for tPCBs, data from several studies were combined into a single data set. Although, all studies used the same experimental protocol, laboratory facilities, analytical laboratories, and even the same strain of mink, some differences in methodology between the studies are likely. It is not possible to quantify the magnitude of that uncertainty, although it is expected to be small.

- The effects metrics used to estimate risk to piscivorous mammals via exposure models were derived for Aroclor 1254 mixtures. Some uncertainty is inherent in extrapolating from studies using the Aroclor 1254 mixture to the specific congener patterns observed in weathered mixtures in the PSA of the Housatonic River. The feeding study with mink using fish from the PSA suggested that the PCB mixture in fish (most closely resembling Aroclor 1260) was less toxic than the PCB mixture reported in literature (Aroclor 1254). Thus, the risk to mink and river otter estimated by the exposure model was slightly overestimated. This overestimate, however, does not affect the final risk conclusion due to the very high exposure rates for mink and river otter.

- The comparison of PCB congeners in the diet-fish blend used in the MSU study to the congener composition measured in Housatonic River fish revealed (with few exceptions) that the congener patterns (and potency) in the feeding study diet were comparable to those in the fish used in the exposure analyses. However, there was some uncertainty as to the influence of PCB-126 on the toxicity of the treatment diet (PCB-126 content of 0.005%) vs exposure analysis fish (PCB-126 content of 0.022%). Although the percentages were within two standard errors of each other (criteria for similarity), the difference might have contributed to the lower than expected toxicity of tPCBs observed in the feeding study.

- There was some uncertainty whether other COCs present in Saginaw Bay fish contributed to the increased toxicity of those fish compared to fish from the Housatonic River.

- It was uncertain whether the food intake rates of the mink fed fish from the Housatonic River were comparable to the corresponding rates observed for mink in the Saginaw Bay study.

- There was uncertainty whether the congener mixture in the Housatonic River fish has the same potency as the mixture in the Saginaw Bay fish.

- The GE mink and otter study lacks critical information needed to confirm track identification (i.e., multiple measurements with a scale) and sex determination (i.e., photographs of tracks, most without a scale, were sent out of state to a scientist in Louisiana). Data on spacing between paired tracks and on lope distance were not presented to segregate male long-tailed weasels from female mink. Without this supporting information the results and interpretation remain questionable.

- In the GE study, the use of a study area that represents only a portion of Housatonic River and adjacent floodplain known to be affected by PCB contamination, and the lack of reference areas creates uncertainty, and limits the ability to draw inferences about whether the number of individual mink and river otter observed in the PSA is normal.

- There is some uncertainty in the assessment of risks downstream of Woods Pond. This is due to the potential errors in measuring tPCB tissue residues in fish as well as

in the derivation of the MATC. However, the magnitude of this uncertainty is likely small.

I.4.6 Comparison to Other Piscivorous Mammals

There are no other piscivorous mammals other than mink and river otter in the PSA.

I.4.7 Risk Downstream of PSA

The risk for mink (and river otter) associated with exposure to tPCBs downstream of Woods Pond was assessed by comparing concentrations of tPCBs in prey fish (5 to 20 cm) in Reaches 7 to 16 to a maximum acceptable threshold concentration (MATC) developed specifically for mink (also used for river otter). For the downstream assessment for mink, it was assumed that the fish constituted 23% of the diet and invertebrates (mostly crayfish) 36% of the diet. The remaining 41% of the diet consisted of other uncontaminated dietary items. No crayfish data were available for the downstream reaches. However, crayfish residues in the PSA were similar to the fish residues. Thus, it was assumed that 59% of the diet of mink foraging downstream of Woods Pond was composed of fish. For the downstream assessment of river otter, it was assumed that 100% of its diet was composed of fish. On average, however, river otter consume about 80% of fish and 20% of crayfish. Crayfish data were not available for the downstream reaches and levels in crayfish were approximated using fish residues (which were similar in the PSA). Thus, the assumption of 100% fish diet for downstream otter was reasonable.

The MATC of 2.65 mg/kg tPCBs in fish (whole body, wet weight) was developed as the geometric mean of the NOAEL and LOAEL developed by Bursian et al. (2002) in a site-specific study of the toxicity of Housatonic River fish to mink. The LOAEL was based on the observation of significantly reduced mink kit survivability at 6 weeks of age. The value of this LOAEL was estimated at 3.7 mg/kg feed supplied to reproducing dams. The NOAEL was based on the same endpoint and its value was 1.6 mg/kg feed.

To determine the extent and types of habitats available for mink and river otter downstream, U.S. Fish and Wildlife Service National Wetland Inventory, U.S. Geological Survey Topographical Quadrangle maps, and some aerial photos of the river were examined in detail. The species-habitat matrix in Appendix A.2 (Ecological Characterization of the Housatonic River

Downstream of Woods Pond) identified potential habitat for mink and otter. According to this analysis, potential mink habitat is ubiquitous and includes all areas except high gradient stream, calcareous rock cliff, cultural grassland, agricultural cropland, and residential/industrial development. Potential river otter habitat is less abundant and centers more on larger wetland systems, with slower flowing water, or with impounded water. Any places where the river is impounded, or near a lake or pond, there is potential river otter habitat.

Fish tissue data were obtained from sampling efforts from 1998 to 2002. The results of the analysis are presented in Figures I.4-15 and I.4-16. Potential risk to mink and river otter exists in river sections from Woods Pond to the end of Reach 10 (mink) and 12 (river otter).

I.4.8 Conclusions

For piscivorous mammals, three lines of evidence were available, including field surveys, a site-specific mink feeding study, and exposure and effects modeling. The weight-of-evidence analysis indicates an intermediate to high risk for mink and river otter to tPCBs and TEQ in the PSA.

Field surveys by EPA and GE were conducted to determine the presence of mink and river otter in the PSA. The surveys were not designed to provide a quantitative evaluation of the relationship between exposure to COCs and the survival, growth, and reproduction of piscivorous mammals in the PSA. Instead, the surveys determined the presence and relative abundance of piscivorous mammals. Signs of mink and river otter were observed in the PSA, but mostly in winter, suggesting that mink and river otter that are present in the PSA are there on transient basis. In the EPA study, these results could be compared to those obtained in reference areas within the watershed.

The mink feeding study was designed to determine the effects on survival, growth and reproduction of mink fed a diet containing fish from the PSA. The results from this study indicated that feeding adult female mink with a diet containing as little as 3.51% fish from the PSA caused a statistically significant reduction (46% compared to controls) in kit survival to 6 weeks of age. Because mink in the wild typically consume between 0 and 65% fish in their diet (mean 23%), the associated risk expected to the population in the PSA is correspondingly higher.

Further, the jaw lesion study indicates that erosion of the jaw occurs at even lower doses and exhibits a dose-response. Such effects could eventually lead to starvation. The occurrence of jaw lesions coincides with the induction of Ah-receptor-regulated enzymes (ECOD and EROD) also in a dose-responsive manner.

The modeling of exposure and effects line of evidence was used independently to determine the level of risk to the representative mammal species, mink and river otter. The effects characterization developed a dose-response curve to describe the potential effects of tPCBs to piscivorous mammals. Toxicity benchmarks based on mink studies were developed for TEQ. The dose-response curve for effects of tPCBs to piscivorous mammals indicated that 10% and 20% declines in fecundity would be expected at doses of 0.0128 and 0.0272 mg/kg bw/d, respectively. For TEQ benchmarks (reduction in kit growth), the lower threshold was set at 3.6 ng/kg bw/d and the upper threshold was set at 36 ng/kg bw/d. The modeled exposure results indicated that the daily intake rates of tPCBs by mink and river otter were far greater than the toxicity thresholds. This means that mink and river otter feeding in the PSA receive tPCB doses that cause adverse reproductive effects. A similar conclusion was reached for TEQ.

ERA Summary

The weight-of-evidence analysis indicates an intermediate to high risk for mink and river otter exposed to tPCBs and TEQ in the PSA.

The risk continues to be elevated for individuals that forage only a small fraction of their time in the PSA.

Downstream of Woods Pond (Reach 6), the mink and river otter may be at risk from exposure to tPCBs and TEQ as far as Reach 10 and 12, respectively.

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APPENDIX I
ASSESSMENT ENDPOINT — SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS MAMMALS

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Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink

Authors	Gilbert and Nancekiveil 1982		Proulx *et al.* 1987										Korschgen 1958		Hamilton 1936	Hamilton 1940		Alexander 1977	
Location	Northeast Alberta		Ontario										Missouri		New York	New York		North Central Lower Michigan	
Habitat	Lake	Stream	Marsh										Various		Various	Marsh		Streams/rivers	
Season	Year round		Summer (low water)		Fall		Summer (low water)		Winter		Summer (high water)		December		Winter (Nov-Mar)	Summer		Spring	Summer
Sex	Both		Both										Both		Both	Both		Both	
Number	140	61	93		61		8		8		7		372		70	300		41	41
A = % occurrence, B = % volume	A	A	A	B	A	B	A	B	A	B	A	B	A	B	A	A	B	A	A
Fish																			
Centrarchids	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Catastomids	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cottids	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Minnow, shiner, etc.	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Bass, sunfish, etc.	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Killifish	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Perch	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Pickerel	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mudminnows	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mottled sculpin	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cyprinide (unidentified)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Northern pike	2.1	1.6	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Stickleback	27.9	3.3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
White sucker	2.1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Bullhead	–	–	–	–	–	–	–	–	–	–	–	–	0.8	0.2	–	–	–	–	–
Artic grayling	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Eel	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Salmonid	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	54.0	54.0
Blenny	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Rockling	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Butterfish	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Sea scorpion	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Gobiidae	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Flatfish	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Unclassified fish	–	–	–	–	–	–	–	–	–	–	–	–	30.1	19.7	–	–	–	19.0	19.0
Total fish	31.4	6.6	0.0	0.0	1.6	0.0	0.0	0.0	12.5	3.5	0.0	0.0	30.9	19.9	18.8	41.0	27.3	73	73.0
Mammals																			
Shrews (Soricidae)	11.4	13.1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Sorex sp.	–	–	1.0	0.1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Short-tailed shrew (*Blarina brevicauda*)	–	–	5.4	3.5	3.3	2.8	–	–	–	–	–	–	–	–	–	–	–	–	–
Least shrew (*Crypototis parva*)	–	–	–	–	–	–	–	–	–	–	–	–	0.8	0.2	–	–	–	–	–
Cricetidae (mice, rats, voles)	1.4	1.6	–	–	–	–	–	–	–	–	–	–	–	–	32.9	3.0	3.0	–	–
Prairie Mole (*Scalopus a. machrinus*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Rat (*Rattus norvegicus*)	–	–	–	–	–	–	–	–	–	–	–	–	0.3	0.1	–	–	–	–	–
Cotton rat (*Sigmodon hispidus*)	–	–	–	–	–	–	–	–	–	–	–	–	1.6	4.5	–	–	–	–	–
Mice	–	–	–	–	–	–	–	–	–	–	–	–	1.3	0.2	–	–	–	–	–
Mice (*Peromyscus spp.*)	–	–	1.0	0.1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Deer mouse	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Pine Mouse (*Pitymys p. scalopsoides*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
House mouse (*Mus musculus*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Harvest mouse (*Reithrodontomys sp.*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Jumping mouse (*Zapus* spp.)	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Northern bog lemming (*Synaptomys borealis*)	2.9	1.6	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Lemming (*Synaptomys cooperi*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Vole (*Clethrionomys spp.*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink (Continued)

Authors	Gilbert and Nancekiveil 1982		Proulx *et al.* 1987										Korschgen 1958		Hamilton 1936	Hamilton 1940		Alexander 1977	
Location	Northeast Alberta		Ontario										Missouri		New York	New York		North Central Lower Michigan	
Habitat	Lake	Stream	Marsh										Various		Various	Marsh		Streams/rivers	
Season	Year round		Summer (low water)		Fall		Summer (low water)		Winter		Summer (high water)		December		Winter (Nov-Mar)	Summer		Spring	Summer
Sex	Both		Both										Both		Both	Both		Both	
Number	140	61	93		61		8		8		7		372		70	300		41	41
A = % occurrence, B = % volume	A	A	A	B	A	B	A	B	A	B	A	B	A	B	A	A	B	A	A
Redback vole (*Clethrionomys grapperi*)	3.6	3.3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Phenacomys (*Phenacomys sp.*)	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Meadow vole (*Mictotus pennsylvanicus*)	–	–	20.4	15.5	13.1	10.8	–	–	25.0	5.3	42.8	9.0	–	–	–	–	–	–	–
Meadow mouse (*Microtus ochrogaster*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Microtus spp.	4.3	6.6	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Microtinae	5.0	31.2	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mole (*Condylura*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2.4	0.3	0.3	–	–
Yellow pine chipmunk	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Squirrel	–	–	–	–	–	–	–	–	–	–	–	–	1.3	2.2	–	–	–	–	–
Total small mammals	170.0	118.4	120.8	19.2	77.4	13.6	8.0	0.0	33.0	5.3	49.8	9.0	377.3	7.2	105.3	303.3	3.3	41.0	41.0
Woodchuk (*Marmota monax*)	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Muskrat (*Ondatra zibethicus*)	21.4	8.2	32.3	32.7	41.0	39.0	12.5	17.1	37.5	24.0	–	–	1.1	1.3	14.1	49.3	38.0	–	–
Lagomorph (rabbits/pikas)	–	–	–	–	–	–	–	–	–	–	–	–	5.9	10.2	4.7	1.0	1.0	–	–
Snowshoe hare (*Lepus americanus*)	19.3	42.6	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cottontail (*Sylvilagus f. mearnsii*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Short-tailed weasel (*Mustela erminea*)	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mink (*Mustela vison*)	8.6	3.3	1.0	0.1	8.2	2.8	–	–	–	–	–	–	–	–	–	–	–	–	–
Domestic cat	–	–	–	–	–	–	–	–	–	–	–	–	0.3	0.3	–	–	–	–	–
Unknown mammal	–	–	1.0	0.1	1.6	0.1	–	–	–	–	–	–	23.4	24.1	–	0.3	0.3	5.75	5.75
Total mammals	63.6	83.6	62.1	52.1	67.2	55.5	12.5	17.1	62.5	29.3	42.8	9.0	785.3	50.3	229.4	657.3	45.9	5.75	5.75
Birds																			
Gaviforms (loons)	1.4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Anseriforms (ducks and geese)	3.6	6.6	24.7	17.4	8.2	10.8	25.0	4.0	25.0	7.6	14.3	2.7	–	–	–	–	–	–	–
Gaviforms or Anseriforms	12.9	3.3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Ciconiiforms (herons, egrets, bitterns)	–	1.6	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Gruiforms (rails, moorhen, coot)	7.1	4.9	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Passiformes (songbirds)	1.4	–	5.4	2.3	–	–	37.5	10.2	–	–	–	–	3.8	3.7	–	–	–	–	–
Charadiforms (gulls, sandpipers, terns)	0.7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Charadiforms and Gruiforms	–	–	6.5	6.9	–	–	12.5	2.8	–	–	–	–	–	–	–	–	–	–	–
Columbiformes (doves)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Galliformes (pheasent, grouse, quail)	–	–	–	–	–	–	–	–	–	–	–	–	0.3	0.2	–	–	–	–	–
Gallus domesticus (Domestic Chicken)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Grebes	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Unknown birds	–	–	5.4	0.8	6.6	0.6	12.5	11.4	–	–	–	–	1.9	1.7	–	–	–	5.75	5.75
Total birds	32.9	16.4	42.0	27.4	14.8	11.4	87.5	28.4	25.0	7.6	14.3	2.7	5.9	5.6	0.0	9.7	9.1	5.75	5.75
Reptiles and Amphibians																			
Snake	–	–	2.2	1.6	–	–	12.5	0.3	12.5	4.7	–	–	0.3	0.1	–	1.0	2.7	–	–
Turtle	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0.7	1.1	–	–
Frogs (unclassified)	–	–	5.4	1.3	27.9	16.1	–	–	–	–	–	–	25.0	23.6	2.4	5.7	3.4	4	4.0
Green frog (*Rana clamitans*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Ball frog (*Rana catesbeiana*)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Leopard frog	–	–	–	–	–	–	–	–	–	–	–	–	0.3	0.9	–	–	–	–	–
Spring peeper	–	–	–	–	–	–	–	–	–	–	–	–	0.3	0.4	–	–	–	–	–
Salamanders	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Total reptile and amphibian	0.0	0.0	7.6	2.9	27.9	16.1	12.5	0.3	12.5	4.7	0.0	0.0	25.9	25.0	2.4	7.3	7.2	4.0	4.0
Invertebrates																			
Insects	11.4	3.3	25.8	3.7	41.0	6.3	–	–	–	–	14.3	0.3	–	–	7.1	–	–	1	1

Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink (Continued)

Authors	Gilbert and Nancekiveil 1982		Proulx *et al.* 1987										Korschgen 1958		Hamilton 1936	Hamilton 1940		Alexander 1977	
Location	Northeast Alberta		Ontario										Missouri		New York	New York		North Central Lower Michigan	
Habitat	Lake	Stream	Marsh										Various		Various	Marsh		Streams/rivers	
Season	Year round		Summer (low water)		Fall		Summer (low water)		Winter		Summer (high water)		December		Winter (Nov-Mar)	Summer		Spring	Summer
Sex	Both		Both										Both		Both	Both		Both	
Number	140	61	93		61		8		8		7		372		70	300		41	41
A = % occurrence, B = % volume	A	A	A	B	A	B	A	B	A	B	A	B	A	B	A	A	B	A	A
Aquatic beetles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	39.3	13.9	–	–
Mollusk	0.7	–	11.8	0.8	6.6	0.5	–	–	–	–	–	–	4.0	0.4	–	0.3	tr	–	–
Cestoda (B)	1.4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Crustacea (B)	0.4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Isopoda (T)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Crayfish (B)	–	–	25.8	12.1	14.8	4.5	–	–	–	–	71.4	35.2	19.9	9.3	16.5	–	–	7.5	7.5
Crab (B)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Other Decopoda (B)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Earthworms (T)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Total invertebrates	35.0	8.2	63.4	16.6	62.4	11.3	0.0	0.0	0.0	0.0	85.7	35.5	395.9	9.7	93.5	339.7	13.9	8.5	8.5
Other																			
Eggshell	–	–	4.3	0.4	1.6	1.1	–	–	12.5	3.5	–	–	–	–	–	–	–	–	–
Vegetation	–	–	2.2	0.6	8.2	4.5	12.5	0.6	–	–	14.3	0.3	1.9	0.7	1.2	–	–	0.5	0.5
Unidentified	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2.5	2.5
Total Other	0.0	0.0	6.5	1.0	9.8	5.6	12.5	0.6	12.5	3.5	14.3	0.3	1.9	0.7	1.2	0.0	0.0	3.0	3.0

Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink (Continued)

Authors	Melquist et al. 1981	Cowan & Reilly 1973		Hamilton 1959		Burgess 1980		Sealander 1943	
Location	Idaho	North Dakota		New York State		Lac Carre, QC		Southern Michigan	
Habitat	River drainage	Riverine		Various		Stream		Various	
Season	1976-1979	Summer	Fall	Fall / winter	Summer	Summer / fall		Winter	
Sex		Both		Both	Both	Both		Both	
Number	659	281	281	630 stomach contents	561 scats	40 scats		201	201
A = % occurrence, B = % volume	A	B	B	A	A	A	B	A	B
Fish									
Centrarchids	–	–	–	–	–	–	–	6	3
Catasttomids	–	–	–	–	–	–	–	2	1
Cottids	–	–	–	–	–	–	–	–	–
Minnow, shiner, etc.	–	–	–	14.0	–	–	–	–	–
Bass, sunfish, etc.	–	–	–	1.7	–	–	–	–	–
Killifish	–	–	–	1.3	–	–	–	–	–
Perch	–	–	–	1.1	–	–	–	–	–
Pickerel	–	–	–	0.6	–	–	–	–	–
Mudminnows	–	–	–	0.2	–	–	–	–	–
Mottled sculpin	7.0	–	–	–	–	–	–	–	–
Cyprinide (unidentified)	29.0	–	–	–	–	–	–	1	1
Northern pike	–	–	–	–	–	–	–	–	–
Stickleback	–	–	–	–	–	–	–	–	–
White sucker	–	–	–	1.1	–	–	–	–	–
Bullhead	–	–	–	0.6	–	–	–	–	–
Artic grayling	–	–	–	–	–	–	–	–	–
Eel	–	–	–	–	–	–	–	–	–
Salmonid	13.0	–	–	2.5	–	–	–	–	–
Blenny	–	–	–	–	–	–	–	–	–
Rockling	–	–	–	–	–	–	–	–	–
Butterfish	–	–	–	–	–	–	–	–	–
Sea scorpion	–	–	–	–	–	–	–	–	–
Gobiidae	–	–	–	–	–	–	–	–	–
Flatfish	–	–	–	–	–	–	–	2	1
Unclassified fish	12.0	–	–	11.4	–	–	–	2	1
Total fish	61	0.0	0	34.5	0.0	0.0	0.0	11	6
Mammals									
Shrews (Soricidae)	2	–	–	–	–	–	–	–	–
Sorex sp.	–	–	–	0.5	–	–	–	–	–
Short-tailed shrew (*Blarina brevicauda*)	–	–	–	4.8	32.4	10.0	7.6	2	trace
Least shrew (*Crypototis parva*)	–	–	–	–	–	–	–	–	–
Cricetidae (mice, rats, voles)	–	–	–	–	–	–	–	–	–
Prairie Mole (*Scalopus a. machrinus*)	–	–	–	–	–	–	–	1	trace
Rat (*Rattus norvegicus*)	–	–	–	0.6	–	–	–	1	trace
Cotton rat (*Sigmodon hispidus*)	–	–	–	–	–	–	–	–	–
Mice	–	–	–	–	–	–	–	1	1
Mice (*Peromyscus spp.*)	–	–	–	1.7	–	–	–	–	–
Deer mouse	6	–	–	–	–	–	–	3	4
Pine Mouse (*Pitymys p. scalopsoides*)	–	–	–	–	–	–	–	3	1
House mouse (*Mus musculus*)	–	–	–	–	–	–	–	–	–
Harvest mouse (*Reithrodontomys sp.*)	–	–	–	–	–	–	–	–	–
Jumping mouse (*Zapus* spp.)	1	–	–	–	–	–	–	–	–
Northern bog lemming (*Synaptomys borealis*)	–	–	–	–	–	–	–	–	–
Lemming (*Synaptomys cooperi*)	–	–	–	0.3	–	–	–	–	–
Vole (*Clethrionomys spp.*)	–	–	–	–	–	–	–	–	–

Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink (Continued)

Authors	Melquist et al. 1981	Cowan & Reilly 1973		Hamilton 1959		Burgess 1980		Sealander 1943	
Location	Idaho	North Dakota		New York State		Lac Carre, QC		Southern Michigan	
Habitat	River drainage	Riverine		Various		Stream		Various	
Season	1976-1979	Summer	Fall	Fall / winter	Summer	Summer / fall		Winter	
Sex		Both		Both	Both	Both		Both	
Number	659	281	281	630 stomach contents	561 scats	40 scats		201	201
A = % occurrence, B = % volume	A	B	B	A	A	A	B	A	B
Redback vole (*Clethrionomys grapperi*)	–	–	–	–	–	–	–	–	–
Phenacomys (*Phenacomys sp.*)	–	–	–	–	–	–	–	–	–
Meadow vole (*Mictotus pennsylvanicus*)	–	36.1	36.1	–	–	–	–	8	5
Meadow mouse (*Microtus ochrogaster*)	24	–	–	12.4	–	–	–	–	–
Microtus spp.	–	–	–	–	–	–	–	–	–
Microtinae	–	–	–	1.1	–	–	–	–	–
Mole (*Condylura*)	–	–	–	–	–	–	–	3	4
Yellow pine chipmunk	1	–	–	–	–	–	–	–	–
Squirrel	–	–	–	0.6	–	–	–	–	–
Total small mammals	693.0	317.1	317.1	22.0	32.4	10.0	7.6	19	11
Woodchuk (*Marmota monax*)	–	–	–	–	–	–	–	–	–
Muskrat (*Ondatra zibethicus*)	5.0	18.3	18.3	5.4	–	–	–	36	31
Lagomorph (rabbits/pikas)	–	–	–	2.9	–	32.5	29.6	–	–
Snowshoe hare (*Lepus americanus*)	–	–	–	–	–	10.0	10.0	–	–
Cottontail (*Sylvilagus f. mearnsii*)	–	–	–	–	–	–	–	15	14
Short-tailed weasel (*Mustela erminea*)	–	–	–	0.2	–	–	–	–	–
Mink (*Mustela vison*)	–	–	–	–	–	–	–	–	–
Domestic cat	–	–	–	–	–	–	–	–	–
Unknown mammal	2	9.2	9.2	4.1	–	–	–	–	–
Total mammals	43	64	64	56.6	64.8	42.5	39.6	70	56
Birds									
Gaviforms (loons)	–	–	–	–	–	–	–	–	–
Anseriforms (ducks and geese)	9	–	–	0.2	–	7.5	9.3	–	–
Gaviforms or Anseriforms	–	15	15	–	44.0	–	–	–	–
Ciconiiforms (herons, egrets, bitterns)	–	–	–	–	–	–	–	–	–
Gruiforms (rails, moorhen, coot)	–	–	–	–	–	–	–	1	4
Passiformes (songbirds)	–	12	12	1.3	–	–	–	–	–
Charadiforms (gulls, sandpipers, terns)	–	–	–	–	–	–	–	–	–
Charadiforms and Gruiforms	–	–	–	–	–	–	–	–	–
Columbiformes (doves)	–	–	–	–	–	–	–	5	7
Galliformes (pheasent, grouse, quail)	–	–	–	0.8	–	–	–	2	3
Gallus domesticus (Domestic Chicken)	–	–	–	–	–	–	–	–	–
Grebes	–	–	–	–	–	–	–	–	–
Unknown birds	10	2	2	–	–	–	–	5	1
Total birds	19	27	27	2.3	44.0	7.5	9.3	13	15
Reptiles and Amphibians									
Snake	2	2	2	1.3	–	–	–	–	–
Turtle	–	–	–	0.2	–	–	–	8	2
Frogs (unclassified)	–	1.0	1.0	18.9	9.3	5.0	5.0	7	6
Green frog (*Rana clamitans*)	–	–	–	–	–	–	–	3	3
Ball frog (*Rana catesbeiana*)	–	–	–	–	–	–	–	1	trace
Leopard frog	–	–	–	–	–	–	–	4	9
Spring peeper	–	–	–	–	–	–	–	25	22
Salamanders	–	–	–	3.2	–	–	–	–	–
Total reptile and amphibian	2.0	2.0	2	23.6	9.3	12.5	12.0	48.0	42.0
Invertebrates									
Insects	15	6	6	6.8	29.2	–	–	–	–

Table I.2-1

Dietary Study Data Used to Estimate Dietary Preferences of Mink (Continued)

Authors	Melquist et al. 1981	Cowan & Reilly 1973		Hamilton 1959		Burgess 1980		Sealander 1943	
Location	Idaho	North Dakota		New York State		Lac Carre, QC		Southern Michigan	
Habitat	River drainage	Riverine		Various		Stream		Various	
Season	1976-1979	Summer	Fall	Fall / winter	Summer	Summer / fall		Winter	
Sex		Both		Both	Both	Both		Both	
Number	659	281	281	630 stomach contents	561 scats	40 scats		201	201
A = % occurrence, B = % volume	A	B	B	A	A	A	B	A	B
Aquatic beetles	7	–	–	–	–	–	–	–	–
Mollusk	–	–	–	1.6	23.0	–	–	–	–
Cestoda (B)	–	–	–	–	–	–	–	–	–
Crustacea (B)	–	–	–	–		15.0	6.3	–	–
Isopoda (T)	–	–	–	–	–	–	–	6	2
Crayfish (B)	–	2	2	14.1	12.7	–	–	–	–
Crab (B)	–	–	–	0.3	–	–	–	–	–
Other Decapoda (B)	–	–	–	–	–	–	–	6	2
Earthworms (T)	–	–	–	2.4	–	–	–	–	–
Total invertebrates	24	7.0	7.0	25.2	42.6	20.0	20.0	12.0	4.0
Other									
Eggshell	–	–	–	0.3	–	–	–	–	–
Vegetation	–	2	2	–	–	–	–	–	–
Unidentified	–	–	–	–	–	–	–	–	–
Total Other	0.0	2.0	2.0	0.3	0.0	0.0	0.0	0.0	0.0

Table I.2-2

Mean Proportion of Prey in the Diet of Mink Used in Exposure Modeling

Prey Item	Adjusted Mean (%)
Fish	23.0
Mammals	15
Birds	11
Invertebrates	36
Amphibians and reptiles	15
Total Percent	**100**

Table I.2-3

Input Variables Used in the Monte Carlo Exposure and Probability Bounds Analysis for Total Daily Intake of tPCBs and 2,3,7,8-TCDD TEQ by Mink Inhabiting the Housatonic River Area

Variable	Distribution	Parameters		
Body weight (BW; g)	Normal	0 = 685; s = 122		
Free metabolic rate (FMR; kcal/kg bw/d)	$Y=aBW^b$	Log a=0.367, s=log 0.223; b=0.850, s=0.055		
Gross energy (GE; kcal/g ww)				
Fish	Point Estimate	1.2		
Crayfish	Point Estimate	1.1		
Birds	Point Estimate	1.8		
Mammals	Point Estimate	1.8		
Amphibians	Point Estimate	1.2		
Assimilation efficiency (AE; unitless)				
Fish	Point Estimate	0.91		
Crayfish	Point Estimate	0.87		
Birds	Point Estimate	0.84		
Mammals	Point Estimate	0.84		
Amphibians	Point Estimate	0.91		
Proportion of Prey Items in Diet	Monte Carlo			
		Alpha	Beta	Scale
Fish	Beta	2.07	4.50	0.730
Invertebrates	Beta	2.93	1.50	0.541
Birds	Beta	1.31	4.00	0.447
Mammals	Beta	2.20	1.40	0.240
Amphibians	Beta	2.00	2.50	0.341
	Probability Bounds			
Fish	Range	Min=0	Max=0.647	
Invertebrates	Range	Min=0	Max=0.541	
Birds	Range	Min=0	Max=0.392	
Mammals	Range	Min=0	Max=0.247	
Amphibians	Range	Min=0	Max=0.341	

Table I.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Mink

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Amphibians	Reach 5	29	0.028	1.13	2.45	5.37	9.22	UCL > Max: Yes	Point Estimate Max = 9.22	Distribution Free Range Min = 0.028 Max = 9.22
	Reach 6	9	1.48	3.04	4.35	5.45	6.01	UCL > Max: Yes	Point Estimate Max = 6.01	Distribution Free Range Min = 1.48 Max = 6.01
	Upstream Reference Area	4	0.029	0.03	0.034	0.043	0.061	UCL > Max: Yes	Point Estimate Max = 0.061	Distribution Free Range Min = 0.029 Max = 0.061
	Threemile Pond Reference Area	5	0.005	0.013	0.015	0.017	0.023	UCL > Max: Yes	Point Estimate Max = 0.023	Distribution Free Range Min = 0.005 Max = 0.023
Mammals	Reach 5	76	0.151	1.78	4.98	29.3	148	UCL > Max: No	Point Estimate UCL = 80.7	Distribution Free Range LCL = 23.2 UCL = 80.7
	Reach 6	-								
	Upstream Reference Area	-								
	Threemile Pond Reference Area	-								

Table I.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Mink (Continued)

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Invertebrates	Reach 5	40	2.59	6.63	8.15	14.8	52.1	UCL > Max: No	Point Estimate UCL = 14.8	Distribution Free Range LCL = 10.1 UCL = 14.8
	Reach 6	-								
	Upstream Reference Area	10	0.025	0.027	0.039	0.052	0.066	UCL > Max: No	Point Estimate UCL = 0.053	Distribution Free Range LCL = 0.034 UCL = 0.053
	Threemile Pond Reference Area	-	-	-	-	-	-			
Fish	Reach 5	99	2.59	24.6	29.9	39.2	99.3	UCL > Max: No	Point Estimate UCL = 38.3	Distribution Free Range LCL = 31.7 UCL = 38.3
	Reach 6	86	8.80	22.0	27.9	34.0	99.8	UCL > Max: No	Point Estimate UCL = 32.1	Distribution Free Range LCL = 27.3 UCL = 32.1
	Upstream Reference Area	45	0.08	0.148	0.161	0.204	2.36	UCL > Max: No	Point Estimate UCL = 0.242	Distribution Free Range LCL = 0.181 UCL = 0.242
	Threemile Pond Reference	31	0.016	0.022	0.028	0.051	0.102	UCL > Max: No	Point Estimate UCL = 0.045	Distribution Free Range LCL = 0.032 UCL = 0.045

Table I.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Mink (Continued)

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Birds	Reach 5	18	1.04	4.81	6.45	9.45	18.9	Reconstitution Issue[c]: Yes UCL > Max: NA	Triangular Distribution Min = 2.38 Best = 11.4 Max = 16.4	Distribution Free Range Min = 1.25 Max = 16.4
	Reach 6	7	1.65	4.58	5.87	8.00	11.4	Reconstitution Issue[c]: Yes UCL > Max: NA	Triangular Distribution Min = 2.61 Best = 11.4 Max = 22.3	Distribution Free Range Min = 0.848 Max = 22.3
	Upstream Reference Area	-								
	Threemile Pond Reference Area	20	0.006	0.012	0.214	0.626	3.04	Reconstitution Issue[c]: Yes UCL > Max: NA	Triangular Distribution Min = 0.689 Best = 0.691 Max = 3.04	Distribution Free Range Min = 0.088 Max = 3.04

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations. For birds, statistics are calculated as for other prey species and the reported values are for the moderate (offal = breast) estimate.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

[c] For Monte Carlo inputs the minimum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for the low (offal = 0) estimate. The best estimate of the triangular distribution was the lower of maximum value and the upper 95% CL for the moderate (offal = breast) estimate. The maximum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for high (offal = liver) estimate. For the probability bounds, the minimum value was the higher of the minimum value and the lower 95% CL from the low (offal = 0) estimate. The maximum value was the lower of the maximum values and the upper 95% CL from the high (offal = liver) estimate.

Table I.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of 2,3,7,8-TCDD TEQ in Prey of Mink

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Amphibians	Reach 5	10	29.2	58.8	91.6	123	223	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: Yes	Triangular Distribution Min = 175 (ND=0) Best = 187 (ND=DL/2) Max = 197 (ND=100)	Distribution Free Range Min = 158 (ND=0) Max = 197 (ND=100)
	Reach 6	5	91	96.9	234	267	274	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: Yes	Triangular Distribution Min = 238 (ND=0) Best = 274 (ND=DL/2) Max = 318 (ND=100)	Distribution Free Range Min = 96.4 (ND=0) Max = 318 (ND=100)
	Upstream Reference Area	1	63.9	-	-	-	63.9	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: Yes	Triangular Distribution Min = 28 (ND=0) Best = 63.9 (ND=DL/2) Max = 99.7 (ND=100)	Distribution Free Range Min = 28 (ND=0) Max = 99.7 (ND=100)
	Threemile Pond Reference Area	1	94.4	-	-	-	94.4	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: Yes	Triangular Distribution Min = 44.8 (ND=0) Best = 94.4 (ND=DL/2) Max = 144 (ND=100)	Distribution Free Range Min = 44.8 (ND=0) Max = 144 (ND=100)

Table I.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of 2,3,7,8-TCDD TEQ in Prey of Mink (Continued)

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Mammals	Reach 5	12	14.7	179	290	1107	4,038	Detection Limit Issue: No Co-elution Issue: No UCL > Max: Yes	Point Estimate Max = 4,038	Distribution Free Range Min = 14.7 Max = 4,038
	Reach 6	-								
	Upstream Reference Area	-								
	Threemile Pond Reference Area	-								
Invertebrates	Reach 5	12	73.4	141	164	426	905	Detection Limit Issue: No Co-elution Issue: Yes UCL > Max: No	Triangular Distribution LCL = 438 (Co-elute=0) Best = 515 (Mean LCL/UCL) UCL = 592 (Co-elute = 100)	Distribution Free Range LCL = 141 UCL = 592
	Reach 6	-								
	Upstream Reference Area	3	47.7	65.8	83.9	279	473	Detection Limit Issue: No Co-elution Issue: No UCL > Max: No	Point Estimate UCL = 473	Distribution Free Range LCL = 47.7 UCL = 473
	Threemile Pond Reference Area	-								

Table I.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of 2,3,7,8-TCDD TEQ in Prey of Mink (Continued)

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Fish	Reach 5	54	56.5	253	331	471	1,565	Detection Limit Issue: No Co-elution Issue: No UCL > Max: No	Point Estimate UCL = 443	Distribution Free Range LCL = 346 UCL = 443
	Reach 6	38	163	296	362	511	1,207	Detection Limit Issue: No Co-elution Issue: No UCL > Max: No	Point Estimate UCL = 482	Distribution Free Range LCL = 376 UCL = 482
	Upstream Reference Area	43	7.26	11.7	12.3	15.6	70.1	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: No	Triangular Distribution LCL = 10.7 (ND=0) Best = 16.6 (Mean LCL/UCL) UCL = 22.7 (ND=DL)	Distribution Free Range LCL = 8.19 UCL = 22.7
	Threemile Pond Reference Area	29	7.40	8.48	9.34	14.2	18.8	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: No	Triangular Distribution LCL = 7.17 (ND=0) Best = 12.3 (Mean LCL/UCL) UCL = 18.0 (ND = DL)	Distribution Free Range LCL = 4.58 UCL = 18.0

Table I.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of 2,3,7,8-TCDD TEQ in Prey of Mink (Continued)

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Birds	Reach 5	18	128	532	858	1596	6,689	Co-elution issue: No Detection limit issue: No Reconstitution issue[c]: Yes UCL > Max: No	Triangular Distribution LCL = 564 Best = 2,459 UCL = 3,538	Distribution Free Range LCL = 230 UCL = 3,538
	Reach 6	7	145	401	423	934	4,753	Co-elution issue: No Detection limit issue: No Reconstitution issue[c]: Yes UCL > Max: No	Triangular Distribution LCL = 1,072 Best = 4,752 UCL = 4,889	Distribution Free Range LCL = 147 UCL = 4,889
	Upstream Reference Area	-								
	Threemile Pond Reference Area	20	7.93	12.4	38.9	126	643	Co-elution issue: No Detection limit issue: No Reconstitution issue[c]: Yes UCL > Max: No	Triangular Distribution LCL = 121 Best = 180 UCL = 312	Distribution Free Range LCL = 15.1 UCL = 312

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations. For birds, statistics are calculated as for other prey species and the reported values are for the moderate (offal = breast) estimate.

[b] Co-elution was not an issue when estimating concentrations of congeners in prey tissues. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

[c] For Monte Carlo inputs the minimum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for the low (offal = 0) estimate. The best estimate of the triangular distribution was the lower of maximum value and the upper 95% CL for the moderate (offal = breast) estimate. The maximum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for high (offal = liver) estimate. For the probability bounds, the minimum value was the higher of the minimum value and the lower 95% CL from the low (offal = 0) estimate. The maximum value was the lower of the maximum values and the upper 95% CL from the high (offal = liver) estimate.

Table I.2-6

Exposure Model Results for Mink Exposed to tPCBs (mg/kg bw/d) in the PSA

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
100% Foraging Time													
Reach 5	0.308	82.5	5.29	3.97	1.52	3.97	10.4	0.0244	0.292	1.72	4.11	8.47	22.5
Reach 6	0.0571	96.1	3.87	2.34	0.634	2.34	8.59	0.00624	0.0175	1.63	3.24	7.69	22.1
Upstream Reference Area	0.000797	0.285	0.0177	0.0128	0.00451	0.0128	0.0358	0.0000871	0.000991	0.00707	0.0140	0.0305	0.0847
Threemile Pond	0.00101	10.8	0.152	0.0696	0.0153	0.0696	0.328	0.0000388	0.000109	0.00352	0.149	0.379	1.15

Table I.2-7

Exposure Model Results for Mink Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d) in the PSA

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
100% Foraging Time													
Reach 5	7.90	3,614	198	147	55.8	147	392	0.329	3.97	17.0	177	367	982
Reach 6	1.38	5,817	153	91.2	25.1	91.2	335	0.0967	0.306	17.8	139	343	1,026
Upstream Reference Area	0.195	110	6.48	4.78	1.76	4.78	13.1	0.0890	1.30	4.58	30.7	58.0	149
Threemile Pond	0.386	1,678	42.6	22.0	5.97	22.0	90.4	0.0287	0.0975	5.19	36.6	85.4	245

Table I.2-8

Sensitivity Analysis Results from Monte Carlo Analysis of Mink Exposed to tPCBs

Chemical	Location	Exposure Model Input Variable	Correlation Coefficient
tPCBs	Reach 5	FMR slope term	0.840
		FMR power term	0.459
		Proportion of fish in diet	0.140
		Proportion of birds in diet	-0.140
		Body weight	-0.0366
		Proportion of amphibians in diet	0.020
		Proportion of invertebrates in diet	-0.03851
		Concentration in birds	0.00770
		Proportion of mammals in diet	-0.0775
	Reach 6	Proportion of fish in diet	0.648
		FMR slope term	0.612
		Proportion of invertebrates in diet	-0.309
		FMR power term	0.325
		Proportion of mammals in diet	-0.305
		Proportion of birds in diet	-0.0261
		Concentration in birds	0.0514
		Body weight	-0.0203
		Proportion of amphibians in diet	0.0621
	Upstream Reference Area	FMR slope term	0.778
		Proportion of amphibians in diet	0.0514
		FMR power term	0.414
		Proportion of fish in diet	0.394
		Proportion of birds in diet	-0.181
		Proportion of mammals in diet	-0.149
		Proportion of invertebrates in diet	-0.0501
		Body weight	-0.0244

Table I.2-8

Sensitivity Analysis Results from Monte Carlo Analysis of Mink Exposed to tPCBs (Continued)

Chemical	Location	Exposure Model Input Variable	Correlation Coefficient
tPCBs (continued)	Threemile Pond Reference Area	Proportion of birds in diet	0.619
		FMR slope term	0.518
		Proportion of inverts in diet	-0.632
		FMR power term	0.279
		Concentration in birds	0.256
		Proportion of mammals in diet	-0.0518
		Proportion of fish in diet	0.0782
		Body weight	-0.0215
		Proportion of amphibians in diet	0.0254

Table I.2-9

Sensitivity Analysis Results from Monte Carlo Analysis of Mink Exposed to 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variable	Correlation Coefficient
2,3,7,8-TCDD TEQ	Reach 5	FMR slope term	0.849
		FMR power term	0.478
		Proportion of mammals in diet	0.138
		Concentration in birds	-0.00586
		Proportion of fish in diet	-0.0757
		Proportion of invertebrates in diet	-0.0751
		Proportion of birds in diet	0.0441
		Body weight	-0.0504
		Proportion of amphibians	0.00000270
		Concentration in amphibians	-0.0142
		Concentration in invertebrates	-0.0000129
	Reach 6	FMR slope term	0.367
		Proportion of birds in diet	0.562
		Proportion of invertebrates in diet	-0.393
		FMR power term	0.367
		Proportion of mammals in diet	-0.293
		Concentration in birds	0.156
		Proportion of fish in diet	0.0809
		Proportion of amphibians	0.0623
		Body weight	-0.0407
		Concentration in amphibians	0.00355
	Upstream Reference Area	FMR slope term	0.819
		FMR power term	0.461
		Proportion of invertebrates in diet	0.197
		Proportion of birds in diet	-0.134
		Proportion of mammals in diet	-0.0820
		Body weight	-0.0493
		Proportion of amphibians	0.167

Table I.2-9

Sensitivity Analysis Results from Monte Carlo Analysis of Mink Exposed to 2,3,7,8-TCDD TEQ (Continued)

Chemical	Location	Exposure Model Input Variable	Correlation Coefficient
2,3,7,8-TCDD TEQ (continued)		Proportion of fish in diet	0.00000630
		Concentration in fish	0.0270
	Threemile Pond Reference Area	FMR slope term	0.598
		FMR power term	0.348
		Proportion of birds in diet	0.513
		Proportion of amphibians	0.222
		Proportion of invertebrates in diet	-0.568
		Concentration in birds	0.0798
		Proportion of fish in diet	0.0119
		Body weight	-0.0300
		Concentration in fish	0.0106
		Proportion of mammals in diet	0.0778

Table I.2-10

Summary of Studies Used in the Selection of River Otter Diet for the Piscivorous Mammals Section of the Housatonic ERA

Study	Percent of Diet			Observations
	Spring	Summer	Fall	
Melquist and Hornocker (1983)				
Fish	100	93	97	Scat analysis reveals the majority of the diet is fish
Other	0	7	3	
Greer (1955)				
Fish	91.4	92.9	100	
Other	8.6	6.9	0	
Andersen and Woolf (1987)				
Fish	97	69	98	
Other	3	31	2	
Woodlot Alternatives (2001)				
Fish			80	Scat analysis of river otter from the Housatonic watershed
Crayfish			20	

Table I.2-11

Input Variables Used in the Monte Carlo Exposure and Probability Bounds Analysis for Total Daily Intake of tPCBs and 2,3,7,8-TCDD TEQ by River Otter Inhabiting the Housatonic River Area

Variable	Distribution	Parameters		
Body weight (BW; g)	Normal	0 = 8,630; s = 1600		
Free metabolic rate (FMR; kcal/kg bw/d)	$Y=aBW^b$	Log a=0.367, s=log 0.223; b=0.850, s=0.055		
Gross energy (GE; kcal/g ww)				
Fish	Point Estimate	1.2		
Crayfish	Point Estimate	1.1		
Assimilation efficiency (AE; unitless)				
Fish	Point Estimate	0.91		
Crayfish	Point Estimate	0.87		
Proportion of Prey Items in Diet	Monte Carlo			
		Alpha	Beta	Scale
Crayfish	Beta	7.67	1.67	0.240
Fish	1-Proportion of crayfish			
	Probability Bounds			
Crayfish	Min=0		Max=0.76	
Fish	1-Proportion of crayfish			

Table I.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of River Otter

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Crayfish	Reaches 5 and 6	40	2.59	6.63	8.15	14.8	52.1	UCL > Max: No	Point Estimate UCL = 14.8	Distribution Free Range LCL = 10.1 UCL = 14.8
	Upstream Reference Area	10	0.025	0.027	0.039	0.052	0.066	UCL > Max: No	Point Estimate UCL = 0.053	Distribution Free Range LCL = 0.034 UCL = 0.053
	Threemile Pond Reference									
Fish	Reaches 5 and 6	523	2.59	25.6	41	77.2	447	UCL > Max: No	Point Estimate UCL = 66.8	Distribution Free Range LCL = 59.4 UCL = 66.8
	Upstream Reference Area	76	0.076	0.149	0.179	0.222	2.36	UCL > Max: No	Point Estimate UCL = 0.227	Distribution Free Range LCL = 0.187 UCL = 0.227
	Threemile Pond Reference	73	0.011	0.025	0.041	0.064	0.193	UCL > Max: No	Point Estimate UCL = 0.06	Distribution Free Range LCL = 0.046 UCL = 0.06

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

Table I.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of 2,3,7,8-TCDD TEQ in Prey of River Otter

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Crayfish	Reaches 5 and 6	12	73.6	141	164	426	905	Detection Limit Issue: No Co-elution Issue: Yes UCL > Max: No	Triangular Distribution Min = 438 (Co-elute=0) Best = 515 Max = 592 (Co-elute=100)	Distribution Free Range LCL = 135 (Co-elute=0) UCL = 592 (Co-elute=100)
	Upstream Reference Area	3	47.7	65.8	83.9	279	473	Detection Limit Issue: No Co-elution Issue: No UCL > Max: Yes	Point Estimate Max = 473	Distribution Free Range Min = 47.7 Max = 473
	Threemile Pond Reference Area									
Fish	Reaches 5 and 6	236	30.2	162	368	749	4103	Detection Limit Issue: No Co-elution Issue: No UCL > Max: No	Point Estimate UCL = 727	Distribution Free Range LCL = 542 UCL = 727
	Upstream Reference Area	69	7.26	11.6	13.4	16.6	70.1	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: No	Triangular Distribution Min = 10.7 (ND=0) Best = 16.2 (ND=DL/2) Max = 21.9 (ND=DL)	Distribution Free Range LCL = 8.75 (ND=0) UCL = 21.9 (ND=DL)
	Threemile Pond Reference Area	67	6.54	8.88	10.8	13.6	24.6	Detection Limit Issue: Yes Co-elution Issue: No UCL > Max: No	Triangular Distribution Min = 7.44 (ND=0) Best = 12.2 (ND=DL/2) Max = 17.6 (ND=DL)	Distribution Free Range LCL = 8.75 (ND=0) UCL = 21.9 (ND=DL)

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution was not an issue when estimating concentrations of congeners in prey tissues. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

Table I.2-14

Exposure Model Results for River Otter Exposed to tPCBs (mg/kg bw/d) in the PSA

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
100% Foraging Time													
Reaches 5 and 6	0.251	111	8.42	6.02	2.15	6.03	17.1	1.59	3.27	5.78	8.12	14.2	53.0
Upstream Reference Area	0.000857	0.379	0.0287	0.0205	0.007321	0.0205	0.0582	0.00791	0.0178	0.0439	0.0127	0.0271	0.0765
Threemile Pond	0.000213	0.0934	0.00713	0.00510	0.00181	0.00510	0.0145	0.00190	0.00421	0.0105	0.00312	0.00666	0.0188

Table I.2-15

Exposure Model Results for River Otter Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d) in the PSA

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
100% Foraging Time													
Reaches 5 and 6	2.20	1,714	104	74.2	263	74.2	212	23.1	52.1	128	47.8	102	288
Upstream Reference Area	0.368	244	17.6	12.4	4.33	12.4	35.8	0.734	1.72	4.34	8.92	19.0	53.6
Threemile Pond	0.0339	30.9	1.49	1.04	0.363	1.04	3.05	0.225	0.499	1.25	0.916	1.95	5.51

Table I.2-16

Sensitivity Analysis Results from Monte Carlo Analysis of River Otter Exposed to tPCBs and 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variable	Correlation Coefficient
tPCBs	Reaches 5 and 6	FMRslope	0.770
		FMRpower	0.592
		Proportion of crayfish in diet	-0.0279
		Body weight	-0.04347
	Upstream Reference Area	FMRslope	0.770
		FMRpower	0.592
		Proportion of crayfish in diet	-0.0273
		Body weight	-0.0434
	Threemile Pond Reference Area	FMRslope	0.770
		FMRpower	0.592
		Proportion of crayfish in diet	-0.0397
		Body weight	-0.0435
2,3,7,8-TCDD TEQ	Reaches 5 and 6	FMRslope	0.779
		FMRpower	0.597
		Body weight	-0.0361
		Ccrayfish	0.00930
		Proportion of crayfish in diet	-0.0275
	Upstream Reference Area	FMRslope	0.768
		FMRpower	0.588
		Proportion of crayfish in diet	0.126
		Body weight	-0.0348
		Cfish	0.0300
	Threemile Pond Reference Area	FMRslope	0.760
		FMRpower	0.585
		Cfish	0.188
		Body weight	-0.0311
		Ccrayfish	-0.00110
		Proportion of crayfish in diet	-0.0583

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals

Chemical	Concentration	Effects
Aroclor 1254	0.0896 mg/kg bw/d 0.640 mg/kg	Whelping reduced; 100% kit mortality at 24 hours; 17% female mortality, no male mortality, 160 days of exposure (Platonow and Karstad 1973)
	0.0967 mg/kg bw/d 0.690 mg/kg	Reduced body weight in whelped kits at 4 weeks (Hornshaw et al. 1983)
	0.140 mg/kg bw/d 1.00 mg/kg	Births, whelping (LOAEL; Aulerich and Ringer 1977), and kit growth reduced after 3 weeks; reduced survival in lactating kits, LOAEL (post-natal development) (Wren et al. 1987b); liver and kidney weight unaffected; thyroid, adrenal, and pituitary structure unaffected; T3 and T4 unaffected (Wren et al. 1987a)
	0.100 to 0.155 mg/kg bw/d	Proposed criteria for the protection of mink (Eisler and Belisle 1996)
	0.210 mg/kg bw/d 1.50 mg/kg	No live kits whelped; 100% kit mortality at 4 weeks, increased liver weight; 7 months total exposure (Hornshaw et al. 1983)
	0.280 mg/kg bw/d 2.00 mg/kg	Births and birth weight reduced; 100% kit mortality at 4 weeks; 12% adult mortality at 10 months (Aulerich and Ringer 1977)
	0.350 mg/kg bw/d 2.50 mg/kg	Whelping reduced, no live births; adult growth unaffected, liver weight increased, BaP, EROD, hepatic P450, norepinephrine, and dopamine activity increased, progesterone reduced (Aulerich et al. 1985)
	0.500 mg/kg bw/d 3.57 mg/kg	No whelping; 100% adult mortality, 105 days of exposure (Platonow and Karstad 1973)
	0.700 mg/kg bw/d 5.00 mg/kg	Adult growth unaffected (Aulerich et al. 1973); 0% adult mortality at 4 months, 29% at 9 months, liver, kidney, and heart weight increased, spleen weight unaffected (Aulerich and Ringer 1977)
	1.28 mg/kg bw/ day	Increased liver weight, reduced number of whelping dams, 100% birth mortality in kits, increased number of interrupted pregnancies, 6 weeks of exposure (Kihlstrom et al. 1992)
	1.40 mg/kg bw/d 10.0 mg/kg	Adult male growth reduced after 28 days of exposure, female weight reduced; 71% adult mortality at 9 months; spleen weight unaffected; liver, kidney, and heart weight increased (Aulerich et al. 1973; Aulerich and Ringer 1977; Hornshaw et al. 1986)
	1.81 mg/kg bw/d 12.9 mg/kg	Adrenal weight increased in males and females after 28 days of exposure (Hornshaw et al. 1986)
	2.10 mg/kg bw/d 15.0 mg/kg	No live births, no females whelped, 31% adult mortality at 3 months (Aulerich and Ringer 1977)
	2.52 mg/kg bw/d 18.0 mg/kg	Growth of young male and female mink reduced (Hornshaw et al. 1986)
	3.23 mg/kg bw/d 23.1 mg/kg	Adult male and female growth reduced after 28 days of exposure (Hornshaw et al. 1986)

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals (Continued)

Chemical	Concentration	Effects
Aroclor 1254 (continued)	4.41 mg/kg bw/d 31.5 mg/kg	35-day LC_{50} (Aulerich et al. 1986)
	4.54 mg/kg bw/d 32.4 mg/kg	Female adult mink growth reduced after 28 days of exposure, heart weight reduced in males and heart and liver weights reduced in females (Hornshaw et al. 1986)
	6.58 mg/kg bw/d 47.0 mg/kg (range 29.6 to 74.6; slope 2.11; 95% CI 1.34 to 3.33)	Dietary LC_{50}, 28 days (Hornshaw et al. 1986; Aulerich et al. 1986)
	6.79 mg/kg bw/d 48.5 mg/kg	35-day LC_{50} (Aulerich et al. 1986)
	8.16 mg/kg bw/d 58.3 mg/kg	Growth reduced after 28 days of exposure, heart weight reduced (Hornshaw et al. 1986)
	11.06 mg/kg bw/d 79.0 mg/kg	28-day LC50 (Aulerich et al. 1986)
	14.7 mg/kg bw/d 105 mg/kg	Liver weight reduced in males after 28 days of exposure, spleen and kidney weights unaffected (Hornshaw et al. 1986)
	140 mg/kg bw/d 1,000 mg/kg	LC_{50}, 48 hours, single intra-peritoneal injection (Aulerich et al. 1973)
Aroclor 1016	0.280 mg/kg bw/d 2.00 mg/kg	Births, birth weight, and survival unaffected (Aulerich and Ringer 1977)
	2.80 mg/kg bw/d 20.0 mg/kg	Whelping (44%) and growth reduced, survival unaffected in males after 247 days of exposure, 25% mortality in females after 247 days of exposure, LOAEL (Bleavins et al. 1980)
Aroclor 1221	0.280 mg/kg bw/d 2.00 mg/kg	Births, birth weight, and survival unaffected (Aulerich and Ringer 1977)
	35.0 – 70.0 mg/kg bw/d 250 - 500 mg/kg	LD_{50}, 48 h, intra-peritoneal injection (Aulerich et al. 1973)
	105 mg/kg bw/d 750 mg/kg	LD_{50}; dietary exposure (Aulerich and Ringer 1977)
Aroclor 1242	0.280 mg/kg bw/d 2.00 mg/kg	Births, birth weight, and survival unaffected (Aulerich and Ringer 1977)
	0.700 mg/kg bw/d 5.00 mg/kg	No whelping, survival of adult females and males unaffected; LOAEL (Bleavins et al. 1980)
	1.20 mg/kg bw/d 8.60 mg/kg	LC_{50}, dietary exposure for 247 days (Bleavins et al. 1980)

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals (Continued)

Chemical	Concentration	Effects
Aroclor 1242 (continued)	1.40 mg/kg bw/d 10.0 mg/kg	67% mortality in males and females, 247 days (Bleavins et al. 1980)
	2.80 mg/kg bw/d 20.0 mg/kg	100% mortality in males and females, 247 days (Bleavins et al. 1980)
	44.1 - 117 mg/kg bw/d 315 - 833 mg/kg	LD_{50}, 247 days; diet (Bleavins et al. 1980)
	70.0 - 140 mg/kg bw/d 500-1,000 (mg/kg)	LD_{50}, 48 h, intra-peritoneal injection (Aulerich et al. 1973)
	420 mg/kg bw/d 3,000 mg/kg	LD_{50}, dietary (Aulerich and Ringer 1977)
tPCBs (dietary)	0.00400 mg/kg bw/d 0.0150 mg/kg fish	Dietary NOAEL, reproductive effects, 15% kit mortality at 3 and 6 weeks (Heaton et al. 1995)
	0.0800 mg/kg bw/d 0.700 mg/kg diet	Decreased fecundity (Brunstrom et al. 1991).
	0.100 mg/kg bw/d 0.720 mg/kg diet	Birth weight unaffected, kit growth reduced after 3 weeks of exposure (Tillitt et al. 1996); 69% kit mortality at 3 weeks, 72% at 6 weeks, food consumption reduced (Heaton et al. 1995)
	0.134 mg/kg bw/d 1.00 mg/kg fish	Reduced gestation length, reduced growth rate after 3 weeks of exposure, in adult females liver, spleen, lung, heart, and thyroid weight increased, in kits brain, liver, spleen, kidney, heart, and adrenal weight decreased, LOAEL (Heaton et al. 1995); reduced kit survival (Restum et al. 1998).
	0.210 mg/kg bw/ day 1.53 mg/kg diet	Birth weight reduced (Tillitt et al. 1996); 71% kit mortality at 3 weeks, 89% at 6 weeks (Heaton et al. 1995)
	0.260 mg/kg bw/d 1.86 mg/kg diet	Reduced body weight of kits at birth (Heaton et al. 1995)
	0.320 mg/kg bw/d 2.29 mg/kg diet	Increased female body weight at whelping (Heaton et al. 1995)
	0.360 mg/kg bw/d 2.56 mg/kg diet	100% kit mortality at 24 hours (Heaton et al. 1995)
	1.54 mg/kg/day 11.0 mg/kg diet	No live births after exposure for 66 days (Jensen et al. 1977)
	4.20 mg/kg bw/d 30.0 mg/kg diet	100% mortality in adults (Ringer et al. 1972)
tPCBs (mink tissue)	0.0900 mg/kg liver tissue	NOAEL (Heaton et al. 1995)
	1.20 mg/kg mink tissue	EC_{50} relative litter size (Leonards et al. 1995)

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals (Continued)

Chemical	Concentration	Effects
tPCBs (mink tissue) (continued)	2.03 mg/kg lipid weight in liver	NOAEL (Heaton et al. 1995)
	2.19 mg/kg liver tissue	LOAEL (Heaton et al. 1995)
	2.36 mg/kg mink tissue	EC_{50} kit survival (Leonards et al. 1995)
	12.0 mg/kg mink tissue	Decreased fecundity (Brunstrom et al. 1991)
	44.6 mg/kg lipid weight in liver	LOAEL (Heaton et al. 1995)
TCDD (dietary)	47.0 ng/kg bw/d 336 ng/kg diet	125-day LC_{50} (Hochstein et al. 1998)
	100 ng/kg bw/d 714 ng/kg diet	Depressed body weight and 62% mortality after 12 days of exposure, intraperitoneal injection (Aulerich et al. 1988)
	140 ng/kg bw/d 1,000 ng/kg diet	12.5 % mortality after 28 days of exposure, 100% mortality after 125 days (Hochstein et al. 1998)
	150 ng/kg bw/d 4,200 ng/kg bw	28-day LD_{50} (Hochstein et al. 1988)
	196 ng/kg bw/d 1,400 ng/kg diet	17% mortality after 132 days of exposure (Hochstein et al. 2001)
	264 ng/kg bw/d	28-day LC_{50} (Hochstein et al. 1998)
	336 ng/kg bw/d 2,400 ng/kg diet	Reduced weight gains after 36 days of exposure (Render et al. 2001)
	700 ng/kg bw/d 5,000 ng/kg diet	Six-month exposure resulted in periodontal lesions (Render et al. 2000)
	1,000 ng/kg bw/d 7,142 ng/kg diet	100% mortality after 12 days of exposure, intraperitoneal injection (Aulerich et al. 1988)
	1,400 ng/kg bw/d 10,000 ng/kg diet	62.5 % mortality after 28 days of exposure, 100% mortality after 125 days (Hochstein et al. 1998); exposure of 101 days resulted in decreased weight gains (Aulerich et al. 2001).
	2,500 ng/kg	Single oral dose was associated with reduced body weights after 3 weeks (Hochstein et al. 1998).
	14,000 ng/kg bw/d 100,000 ng/kg diet	100 % mortality after 28 days of exposure (Hochstein et al. 1998)
	0.0800 ng/kg bw/d	NOAEL (Tillitt et al. 1996)
	0.250 ng/kg bw/d TEQ 1.03 ng/kg fish TEQ	Dietary NOAEL, reproductive effects, 15% kit mortality at 3 and 6 weeks (Heaton et al. 1995)
	0.300 ng/kg bw/d 3.00 ng/kg diet)	NOAEL (reproduction) (Brunstrom et al. 1991)

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals (Continued)

Chemical	Concentration	Effects
TCDD (dietary) (continued)	2.40 ng/kg bw/d 22.3 ng/kg diet	LOAEL (reproduction) (Brunstrom et al. 1991)
	0.420 ng/kg bw/d TEQ 1.90 ng/kg fish TEQ	Threshold daily dose, reproductive effects (Tillitt et al. 1996)
	2.24 ng/kg bw/d	LOAEL (Tillitt et al. 1996)
	3.60 ng/kg bw/d TEQ 19.4 ng/kg fish TEQ	Dietary LOAEL, reproduction; birth weight unaffected, kit growth reduced, 69% kit mortality at 3 weeks, 72% at 6 weeks, food consumption reduced (Heaton et al. 1995)
	6.80 ng/kg bw/d TEQ 40.0 ng/kg TEQ	Birth weight reduced, 71% kit mortality at 3 weeks, 89% at 6 weeks (Heaton et al. 1995)
	10.7 ng/kg bw/d TEQ 80.8 ng/kg TEQ	100% kit mortality at 24 hours (Heaton et al. 1995)
TEQ (mink tissue)	5.00 ng/kg liver tissue in mink TEQ	NOAEL (Heaton et al. 1995)
	11.0 ng/kg liver tissue TEQ	NOAEL (Tillitt et al. 1996)
	60.0 ng/kg liver tissue TEQ	Threshold liver concentration (geometric mean of NOAEL and LOAEL) (Tillitt et al. 1996)
	112 ng/kg liver lipid TEQ	NOAEL (Heaton et al. 1995)
	160 ng/kg TEQ	EC50, tissue concentration, litter size (Leonards et al. 1995)
	200 ng/kg TEQ	EC50, tissue concentration, kit survival (Leonards et al. 1995)
	324 ng/kg liver tissue TEQ	LOAEL (Tillitt et al. 1996)
	496 ng/kg liver tissue TEQ	LOAEL (Heaton et al. 1995)
	10,420 ng/kg liver lipid TEQ	LOAEL (Heaton et al. 1995)
2,3,6,2',3',6'-HCB	0.350 mg/kg bw/d 2.50 mg/kg	Growth unaffected (Aulerich et al. 1985)
	0.700 mg/kg bw/d 5.00 mg/kg	Growth unaffected, norepinephrine and dopamine activity increased, LOAEL (Aulerich et al. 1985)
2,4,5,2',4',5'-HCB	0.350 mg/kg bw/d 2.50 mg/kg	Growth unaffected, decreased survival time, litter size reduced; BaP and hepatic P450 increased, LOAEL (Aulerich et al. 1985)
	0.700 mg/kg bw/d 5.00 mg/kg	Growth unaffected, AP, norepinephrine, and dopamine activity increased (Aulerich et al. 1985)

Table I.3-1

Summary of PCB and 2,3,7,8-TCDD TEQ Effects on Piscivorous Mammals (Continued)

Chemical	Concentration	Effects
3,4,5,3',4',5'-HCB	0.00140 mg/kg bw/d 0.0100 mg/kg	Growth unaffected after 120 days of exposure, no adult mortality, liver weight increased, LOAEL (Aulerich et al. 1987)
	0.00700 mg/kg bw/d 0.0500 mg/kg	Growth reduced after 90 days of exposure, brain, liver, kidney, and adrenal weight increased, AP, BaP, and EGF unaffected, T3 and T4 levels decreased, 50% mortality in 135 days (Aulerich et al. 1987)
	0.0140 mg/kg bw/d 0.100 mg/kg	No whelping, growth reduced, 50% mortality at 88 days, reduced survival time, heart weight decreased, adrenal weight increased, BaP, norepinephrine, hepatic P450, and progesterone increased, dopamine reduced in mid-brain, but increased in cerebral hemisphere (Aulerich et al. 1985)
	0.0700 mg/kg bw/d 0.500 mg/kg	100% mortality at 88 days, growth reduced, reduced survival time, kidney weight increased, heart and spleen weight decreased (Aulerich et al. 1985)

Table I.3-2

Effects of PCBs Derived from Housatonic River Fish on Kit Survival (%) from Birth to 6 Weeks of Age*

Actual Dietary Treatment (mg/kg PCBs)	Birth	Three Weeks of Age	Six Weeks of Age
Control (0.055)	96 (80.1-99.4)	85 (61.2-98.3)	85^{A} (61.2-98.3)
0.34	95 (76.0-99.7)	81 (55.0-97.3)	78^{AB} (50.9-95.8)
0.61	95 (76.8-99.5)	66 (37.9-89.0)	66^{AB} (37.9-89.0)
0.96	91 (71.1-99.7)	68 (42.4-88.5)	68^{AB} (42.4-88.5)
1.6	97 (82.4-99.3)	93 (74.2-100)	93^{A} (74.2-100)
3.7	99 (53.6-96.7)	77 (47.9-96.0)	46^{B} (18.1-74.7)

*Data are presented as mean (95% confidence intervals).

Means with different superscripts are significantly different ($p < 0.05$).

Table I.3-3

Mean Hepatic Microsomal Activities (pmol/min/mg) of Maternal Mink Fed Diets Containing Fish from the Housatonic River

Actual Dietary Treatment (mg PCB/kg)	H.R. Fish Content (%)	N	Hepatic Microsomal Activities (pmol/min/mg)			
			BROD Mean (SD)	PROD Mean (SD)	ECOD Mean (SD)	EROD Mean (SD)
Control (0.055)	0	11	12 (6.7)[A]	13 (11)[A]	65 (100)[A]	67 (32)[A]
0.34	0.22	12	14 (5.3)[A]	9 (7.4)[A]	130 (100)[A]	130 (55)[AB]
0.61	0.44	10	13 (5.4)[A]	8 (5.5)[A]	130 (98)[A]	190 (80)[BC]
0.96	0.89	12	13 (6.2)[A]	10 (9.7)[A]	150 (180)[A]	250 (73)[C]
1.6	1.77	11	12 (6.0)[A]	6 (4.4)[A]	390 (240)[B]	340 (110)[D]
3.7	3.54	11	10 (7.7)[A]	7 (7.4)[A]	480 (290)[B]	490 (160)[E]

BROD - benzyloxyresorufin-O-deethylase
PROD - pentoxyresorufin-O-deethylase
ECOD - ethoxycoumarin-O-deethylase
EROD - ethoxyresorufin-O-deethylase

H.R. fish content is the percentage (%) of the treatment diet contributed by fish from the Housatonic River. The remainder of the fish meal in the diets (30% fish total) was comprised of ocean fish.

N represents the number of livers sampled for each dose.

Means and standard deviations were determined in SAS using individual values and ½ the mean limit of detection (LOD) or limit of quantitation (LOQ) for those values that were < LOD/LOQ.

Values in a column noted with different letters are significantly different ($p \leq 0.05$).

Table I.3-4

Mean Hepatic Microsomal Activities (pmol/min/mg) of 6-Week-Old F1 Mink Offspring Fed Diets Containing Fish from the Housatonic River

Actual Dietary Treatment (mg PCB/kg)	H.R. Fish Content (%)	N	Hepatic Microsomal Activities (pmol/min/mg)			
			BROD Mean (SD)	PROD Mean (SD)	ECOD Mean (SD)	EROD Mean (SD)
Control (0.055)	0	6	14 (12)[A]	3 (4.0)[A]	2 (0)[A]	81 (45)[A]
0.34	0.22	6	14 (5.1)[A]	16 (16)[B]	5 (7.4)[A]	99 (20)[A]
0.61	0.44	6	11 (6.6)[AB]	4 (2.6)[A]	65 (97)[A]	150 (82)[AB]
0.96	0.89	6	8.1 (2.7)[AB]	7 (6.0)[A]	42 (50)[A]	200 (52)[B]
1.6	1.77	6	9.6 (3.6)[AB]	8 (6.7)[A]	90 (110)[A]	280(48)[C]
3.7	3.54	6	6.1 (3.0)[B]	4 (4.7)[A]	79 (130)[A]	350 (89)[C]

BROD - benzyloxyresorufin-O-deethylase
PROD - pentoxyresorufin-O-deethylase
ECOD - ethoxycoumarin-O-deethylase
EROD - ethoxyresorufin-O-deethylase

H.R. fish content is the percentage (%) of the treatment diet contributed by fish from the Housatonic River. The remainder of the fish meal in the diets (30% fish total) was comprised of ocean fish.

N represents the number of livers sampled for each dose.

Means and standard deviations were determined in SAS using individual values and ½ the mean limit of detection (LOD) or limit of quantitation (LOQ) for those values that were < LOD/LOQ.

Values in a column noted with different letters are significantly different (p # 0.05).

Table I.3-5

Mean Hepatic Microsomal Activities (pmol/min/mg) of 6-Month-Old F1 Mink Offspring Fed Diets Containing Fish from the Housatonic River

Actual Dietary Treatment (mg PCB/kg)	Nominal Dose (Φg PCB/g)	H.R. Fish Content (%)	N	Hepatic Microsomal Activities (pmol/min/mg)			
				BROD Mean (SD)	PROD Mean (SD)	ECOD Mean (SD)	EROD Mean (SD)
Control (0.055)	0	0	6	5 (2.7)[A]	4 (1.0)[AC]	18 (21)[A]	80 (30)[A]
0.34	0.25	0.22	6	17 (10)[B]	11 (4.6)[BC]	83 (54)[AB]	150 (44)[AB]
0.61	0.5	0.44	6	16 (6.9)[B]	8.3 (4.4)[ABC]	140 (50)[BC]	300 (130)[BC]
0.96	1	0.89	6	15 (12)[B]	8.9 (4.6)[BC]	150 (110)[BC]	410 (170)[CD]
1.6	2	1.77	6	14 (5.3)[AB]	6.2 (2.0)[AC]	220 (82)[C]	540 (63)[D]
3.7	4	3.54	6	14 (11)[AB]	7.1 (1.7)[ABC]	190 (110)[C]	700 (200)[E]

BROD - benzyloxyresorufin-O-deethylase
PROD - pentoxyresorufin-O-deethylase
ECOD - ethoxycoumarin-O-deethylase
EROD - ethoxyresorufin-O-deethylase

H.R. fish content is the percentage (%) of the treatment diet contributed by fish from the Housatonic River. The remainder of the fish meal in the diets (30% fish total) was comprised of ocean fish.

N represents the number of livers sampled for each dose.

Means and standard deviations were determined in SAS using individual values and ½ the mean limit of detection (LOD) or limit of quantitation (LOQ) for those values that were < LOD/LOQ.

Values in a column noted with different letters are significantly different (p # 0.05).

Table I.4-1

Results of Snow Tracking and Scent Post Station Surveys in the PSA and Reference Areas (EPA Study)

Primary Study Area	Mink	Otter
Hours of survey effort	260.5	260.5
Number of sightings	5	4
No. sightings/hour	0.019	0.015
Reference Areas		
Hours of survey effort	108.0	108.0
Number of sightings	4	14
No. sightings/hour	0.037	0.130

Table I.4-2

Summary of Exceedance Probabilities for Piscivorous Mammals from the Housatonic River Study Area

Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LB		FOMC		UB		LB		FOMC		UB	
	LD	UD	LD	UD	LD	UD	LT	UT	LT	UT	LT	UT
Mink												
Foraging Time: 100%												
Reach 5	98	86	100	100	100	100	52	3	100	97	100	100
Reach 6	59	40	100	100	100	100	28	3	100	83	100	100
Upstream Reference Area	2	0	50	17	95	65	16	0	64	0	100	82
Threemile Pond Reference Area	0	0	91	78	100	98	0	0	96	32	100	90
Foraging Time: 50%												
Reach 5	88	70	100	100	100	100	33	0	100	82	100	100
Reach 6	41	32	100	100	100	100	22	0	100	59	100	100
Upstream Reference Area	19	4	0	0	68	26	7	0	29	0	100	36
Threemile Pond Reference Area	0	0	78	58	99	81	5	0	87	14	100	60
Foraging Time: 25%												
Reach 5	71	62	100	100	100	100	14	0	100	51	100	96
Reach 6	33	30	100	100	100	100	13	0	98	32	100	89
Upstream Reference Area	0	0	4	0	29	3	0	0	8	0	100	11
Threemile Pond Reference Area	0	0	58	35	83	49	0	0	67	5	100	24

Table I.4-2

Summary of Exceedance Probabilities for Piscivorous Mammals from the Housatonic River Study Area (Continued)

Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LB		FOMC		UB		LB		FOMC		UB	
	LD	UD	LD	UD	LD	UD	LT	UT	LT	UT	LT	UT
Foraging Time: 10%												
Reach 5	61	51	100	100	100	100	3	0	97	12	100	52
Reach 6	29	27	100	98	100	100	2	0	82	9	100	48
Upstream Reference Area	0	0	0	0	6	2	0	0	0	0	82	3
Threemile Pond Reference Area	0	0	30	13	40	13	0	0	32	0	90	5
River Otter												
Foraging Time: 100%												
Reaches 5 and 6	100	100	100	100	100	100	100	72	100	82	100	97
Upstream Reference Area	69	26	72	37	89	49	15	0	94	10	100	19
Threemile Pond Reference Area	6	1	13	0	19	5	0	0	7	0	21	0
Foraging Time: 50%												
Reaches 5 and 6	100	100	100	100	100	100	99	31	100	52	100	71
Upstream Reference Area	29	6	39	12	53	17	3	0	75	0	96	6
Threemile Pond Reference Area	1	0	0	0	6	2	0	0	0	0	7	0
Foraging Time: 25%												
Reaches 5 and 6	100	100	100	100	100	100	98	8	98	21	100	31
Upstream Reference Area	7	1	13	0	19	6	0	0	45	0	67	3

Table I.4-2

Summary of Exceedance Probabilities for Piscivorous Mammals from the Housatonic River Study Area (Continued)

Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LB		FOMC		UB		LB		FOMC		UB	
	LD	UD	LD	UD	LD	UD	LT	UT	LT	UT	LT	UT
Threemile Pond Reference Area	0	0	0	0	2	1	0	0	0	0	3	0
Foraging Time: 10%												
Reaches 5 and 6	100	100	100	100	100	100	72	0	82	3	97	7
Upstream Reference Area	0	0	0	0	4	1	0	0	36	0	20	0
Threemile Pond Reference Area	0	0	0	0	1	0	0	0	0	0	0	0

FOMC = First Order Monte Carlo
LB = Lower Probability Bound
LD = Lower Toxicity Dose
LT = Lower Toxicity Threshold
UB = Upper Probability Bound
UD = Upper Toxicity Dose
UT = Upper Toxicity Threshold

Table I.4-3

Summary of Qualitative Risk Statements for Piscivorous Mammals from the Housatonic River Study Area

Mammal/Location	Qualitative Risk Statements				
	tPCBs			TEQ	
	Risk Category	Risk Range		Risk Category	Risk Range
Mink					
Reach 5	High	High		High	Intermediate/High
Reach 6	High	Intermediate/High		High	Intermediate/High
Upstream Reference Area	Intermediate	Low/High		Intermediate	Low/High
Threemile Pond	High	Low/High		Low	Low/High
River Otter					
Reaches 5 and 6	High	High		High	High
Upstream Reference Area	Intermediate	Low/Intermediate		Intermediate	Low/Intermediate
Threemile Pond	Low	Low/Intermediate		Low	Low/Intermediate

Table I.4-4

Weighting of Measurement Endpoints for Piscivorous Mammal Weight-of-Evidence Evaluation

Attributes	Field Surveys		Feeding Study	Modeled PCBs	Modeled TEQ	Rationale
	EPA	GE				
I. Relationship Between Measurement and Assessment Endpoints						
1. Degree of Association	L/M	L/M	H	H	H	Field surveys did not evaluate growth, reproduction or survival, but were specific to piscivorous mammals. Feeding study, exposure models and effects metrics were species-specific.
2. Stressor/Response Correlation	M	L/M	M/H	H	M	Field surveys determined presence and relative abundance, but did not demonstrate an exposure-response relationship. The GE study lacked reference areas. The feeding study was species- and chemical-specific. Exposure modeling was species- and chemical-specific and dose-response models were available for mink. A field-based threshold range was used for TEQ.
3. Utility of Measure	M	L/M	H	M/H	M	Field surveys employed, while standardized and location-specific, had low statistical power and were not capable of detecting subtle effects. Modeled exposure and effects procedures as well as feeding studies used standardized methods that are widely accepted. The use of a threshold range for TEQ lowers the score for this attribute.
II. Data Quality						
4. Data Quality	M/H	L	H	M/H	M/H	Field surveys and feeding studies collected data using appropriate experimental design and methodology. The GE survey had shortcomings related to obtaining tracking data. The modeling of exposure used tissue concentrations data of high quality (Data quality objectives were met).
III. Study Design						
5. Site Specificity	H	M/H	M	L/M	L/M	Surveys were conducted in the PSA and appropriate reference areas (except the GE study, which resulted in a lower value). The feeding study and modeling of exposure and effects did not account for site-specific conditions.

Table I.4-4

Weighting of Measurement Endpoints for Piscivorous Mammal Weight-of-Evidence Evaluation (Continued)

Attributes	Field Surveys		Feeding Study	Modeled PCBs	Modeled TEQ	Rationale
	EPA	GE				
6. Sensitivity	M	L/M	H	H	H	Surveys conducted were not sensitive to potential subtle effects associated with COCs. The field survey by GE had limitations in methodology and lacked a reference area. The feeding study was able to detect subtle differences (at least two-fold). Modeled exposure and effects directly assessed exposure-response relationship. Mink are particularly sensitive to effects from exposure to PCBs. Laboratory studies from which effects data were derived yielded a dose response curve.
7. Spatial Representativeness	H	M/H	M/H	M/H	M/H	The surveys had complete coverage of the study area, measurement locations, stressors, representative species. The coverage of the GE study was reduced, hence the lower rating. The feeding study relied on field-collected fish from some locations. Modeled exposures relied on tissue data collected throughout the study area.
8. Temporal Representativeness	H	M/H	M/H	M/H	M/H	The surveys were conducted over 3 (GE over 2 years) years. In the feeding study, the fish used were from a relatively short sampling period; however, the concentrations were representative of fish concentrations at other time periods. The modeled exposure and effects line of evidence spanned critical life stages and for the most part included tissue data collected during multiple sampling events.
9. Quantitative Measure	L	L	H	H	H	The surveys were primarily qualitative. The feeding study was quantitative. Probabilistic exposure and effects modeling was highly quantitative and propagated uncertainty associated with modeling procedures. Given the use of dose-response curves, results reflect biological significance.
10. Standard Method	H	M/H	H	M/H	M/H	Field surveys followed standard accepted methods, but the study by GE had some problems with methodology. The feeding study followed standard methods. Generally accepted exposure and effects modeling procedures were followed, but were modified specifically for this evaluation.
Overall Endpoint Value	**M/H**	**M**	**H**	**M/H**	**M/H**	

L = Low
M = Moderate
H = High

Table I.4-5

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	EPA	Moderate/High	Yes	Intermediate
	GE	Moderate	No	Low
Feeding Study		High	Yes	Intermediate
Modeled Exposure and Effects		Moderate/High	Yes	High

Table I.4-6

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	EPA	Moderate/High	Yes	Intermediate
	GE	Moderate	No	Low
Feeding Study		High	Yes	Intermediate
Modeled Exposure and Effects		Moderate/High	Yes	High

Table I.4-7

Risk Analysis Summary for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of piscivorous mammals

Harm/Magnitude	Weighting Factors (increasing confidence of weight)				
	Low	**Low/Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High				MEE	
Yes/Intermediate				FS-EPA	MFS
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low			FS-GE		
No/Intermediate					
No/High					

FS-EPA = Field surveys by EPA
FS-GE = Field surveys by GE
MFS = Mink feeding study
MEE = Modeled exposure and effects

Table I.4-8

Risk Analysis Summary for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of piscivorous mammals

Harm/Magnitude	Weighting Factors (increasing confidence of weight)				
	Low	**Low/Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High				MEE	
Yes/Intermediate				FS-EPA	MFS
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low			FS-GE		
No/Intermediate					
No/High					

FS-EPA = Field surveys by EPA
FS-GE = Field surveys by GE
MFS = Mink feeding study
MEE = Modeled exposure and effects

APPENDIX I
ASSESSMENT ENDPOINT – SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS MAMMALS

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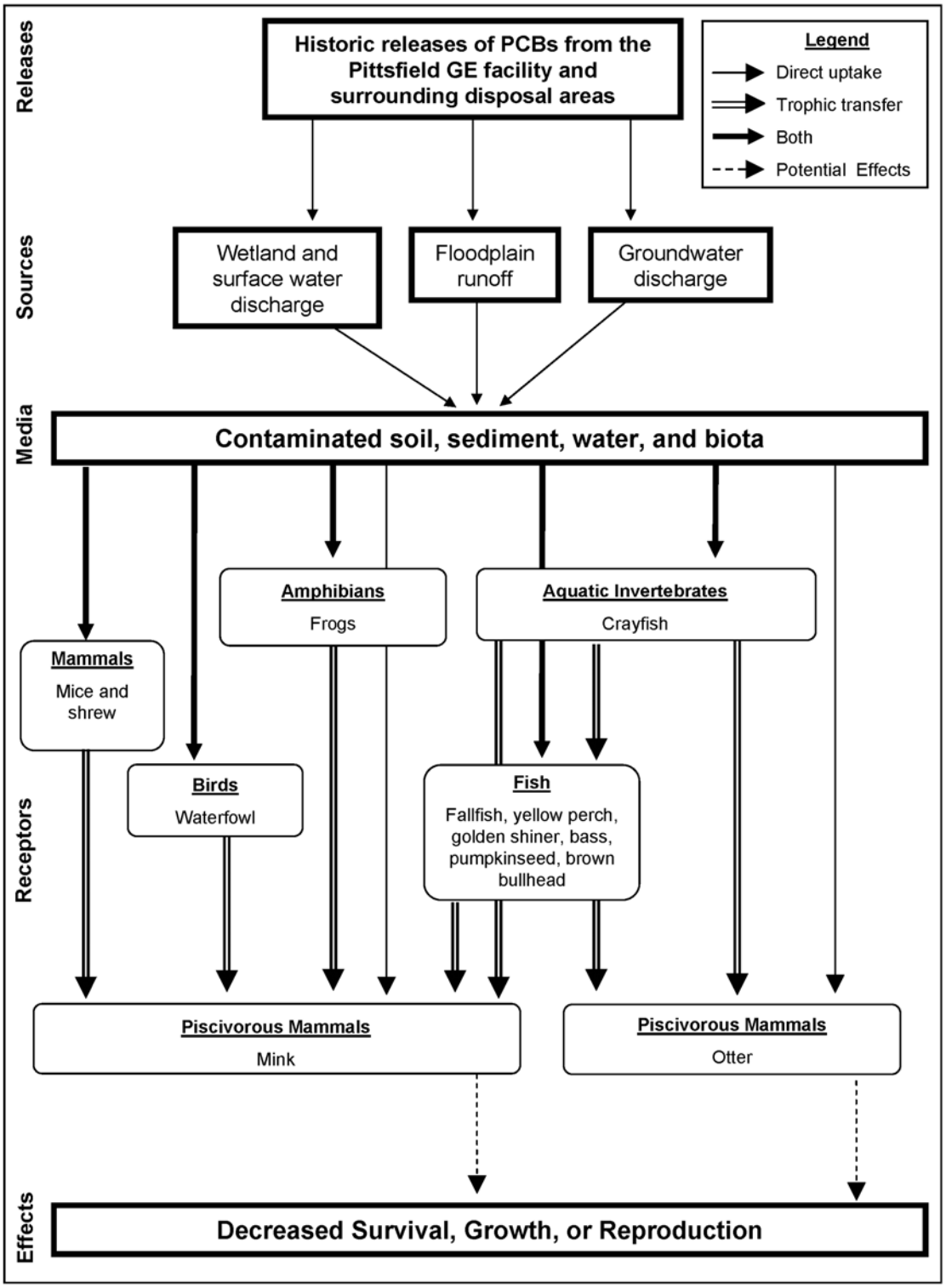


Figure I.1-1 Conceptual Model Diagram: Exposure Pathways for Piscivorous Mammals Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA

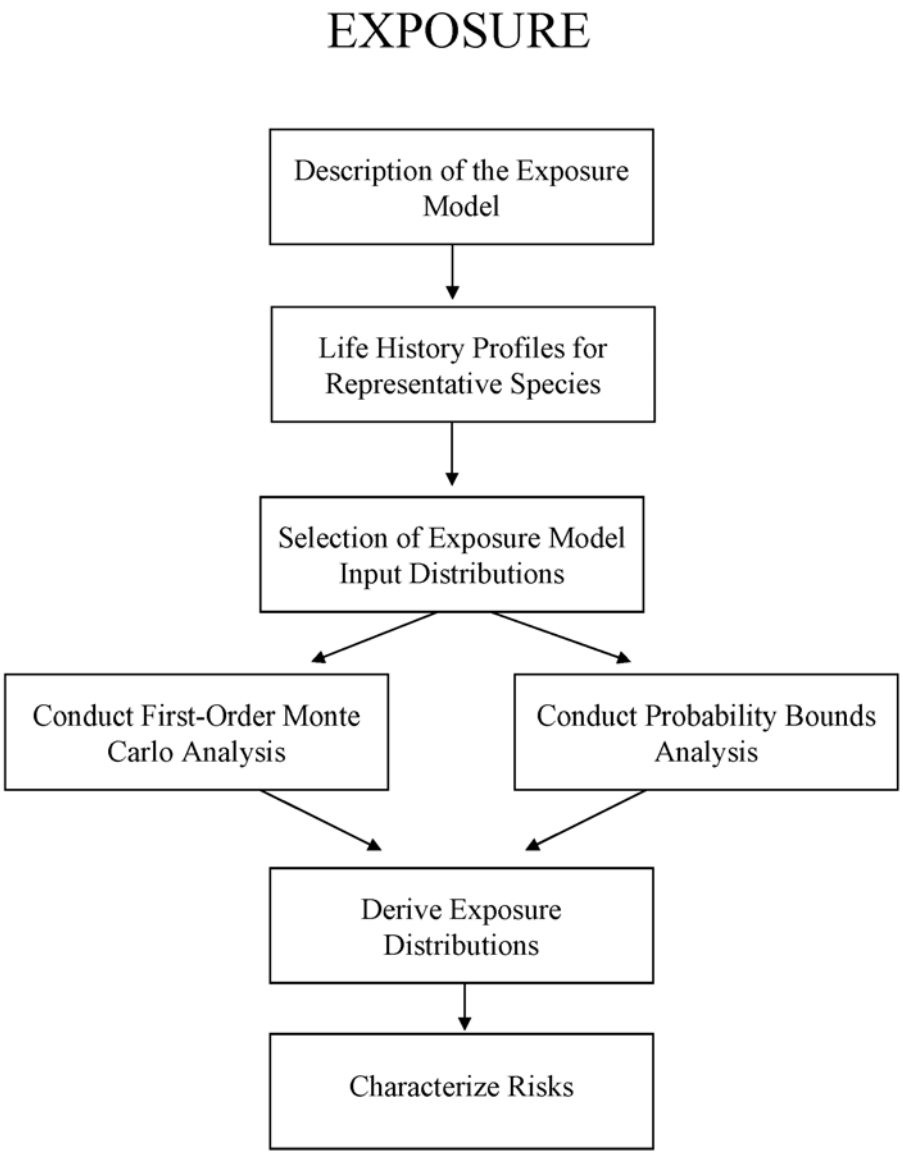


Figure I.1-2 Overview of Approach Used to Assess Modeled Exposure of Piscivorous Mammals to Contaminants of Concern (COCs) in the Housatonic River PSA

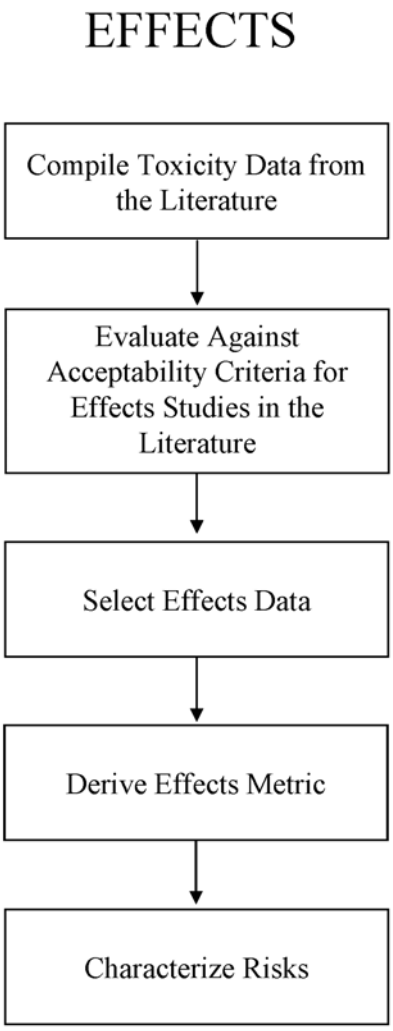


Figure I.1-3 Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA

RISK CHARACTERIZATION

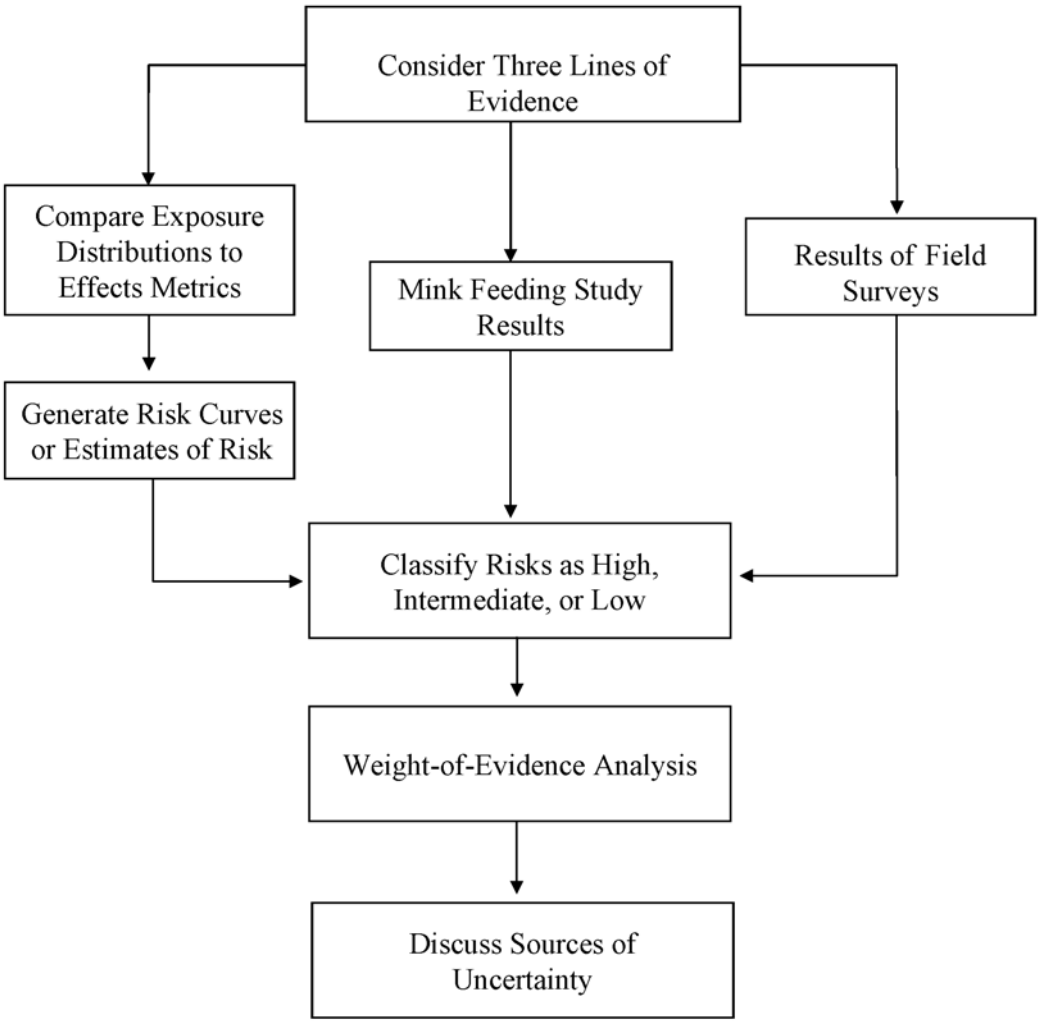
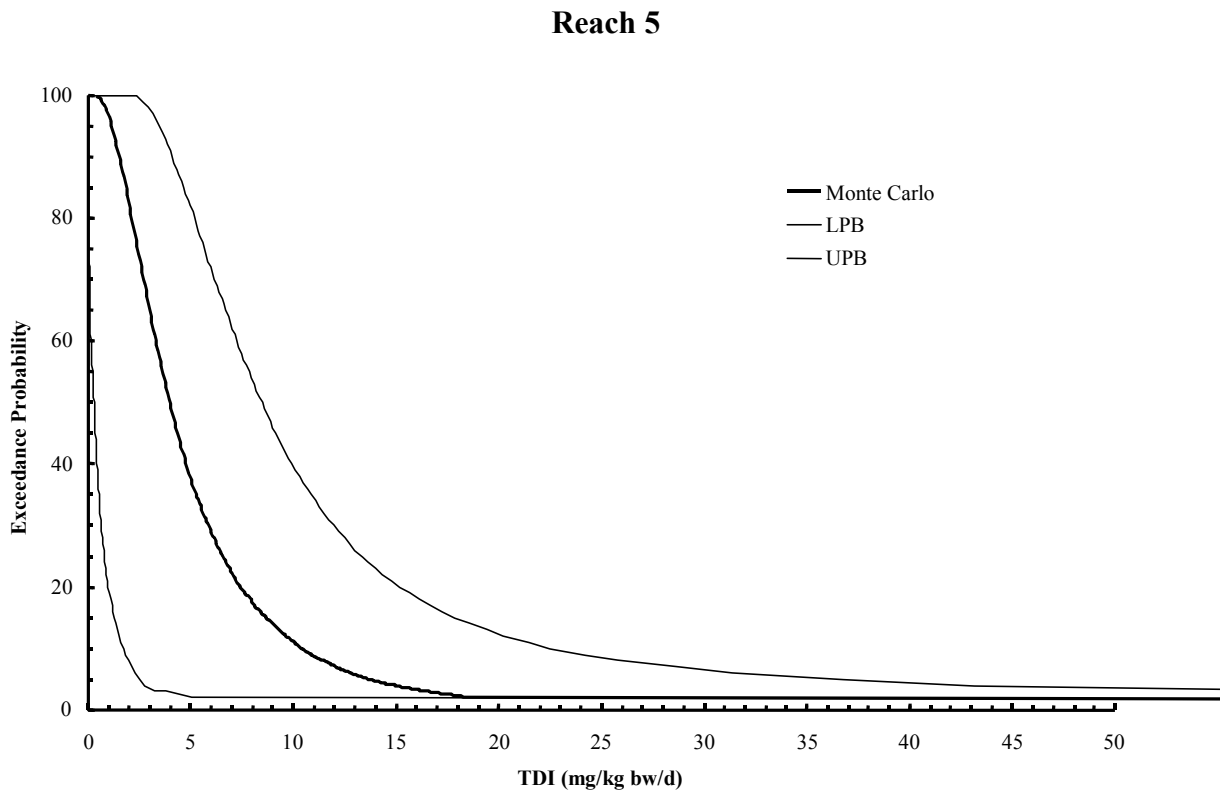


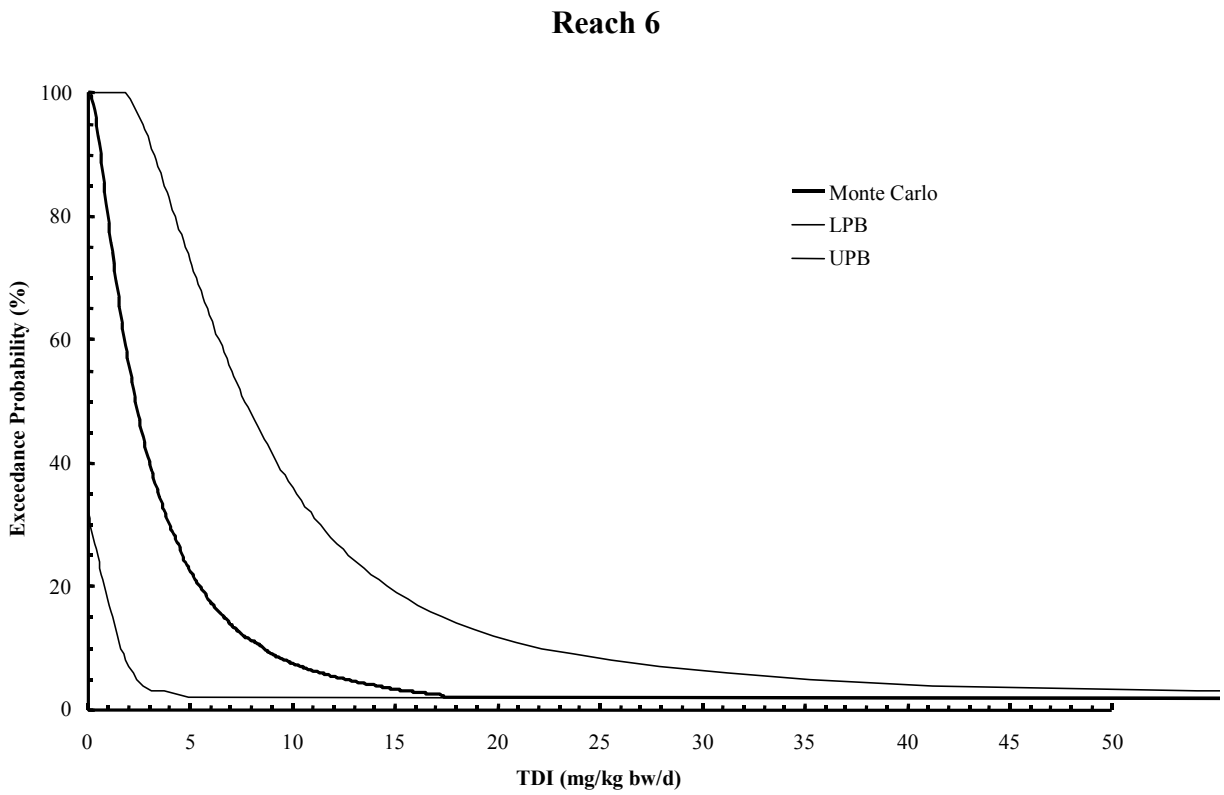
Figure I.1-4 Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA

Figure I.2-1 Mink (*Mustela vison*)



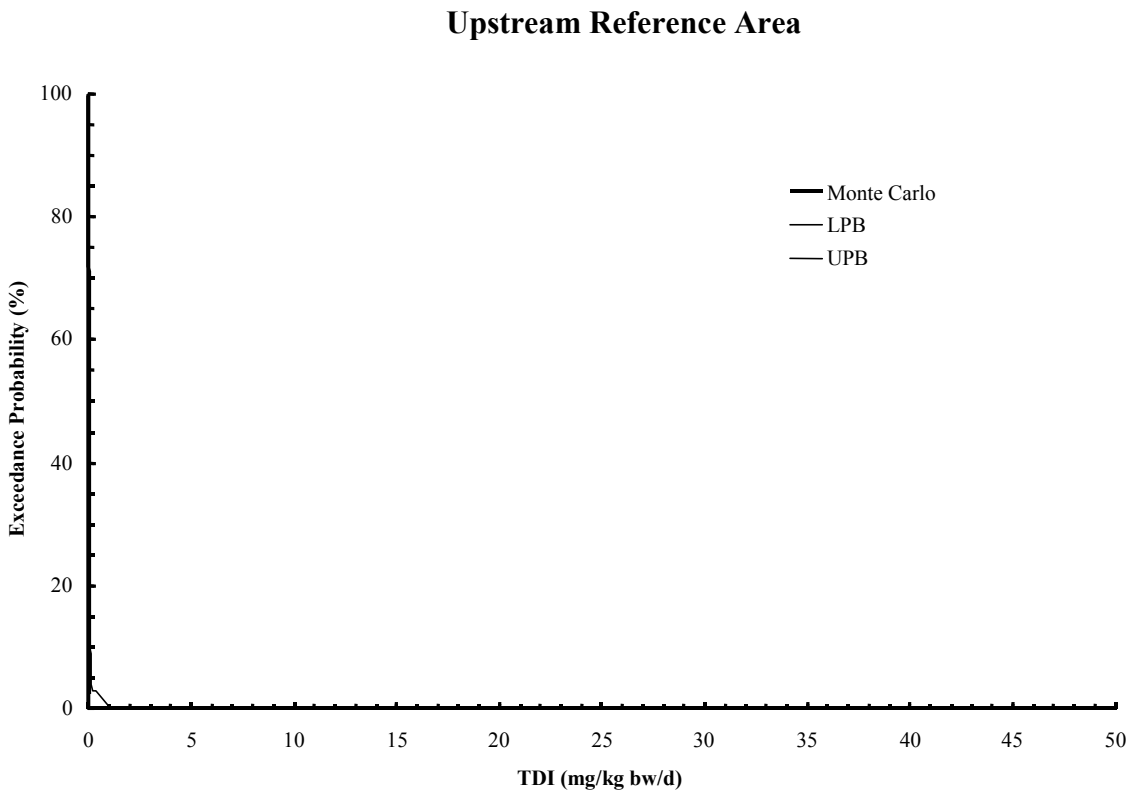
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-2 Exposure of Mink to tPCBs in Reach 5 of the Housatonic River



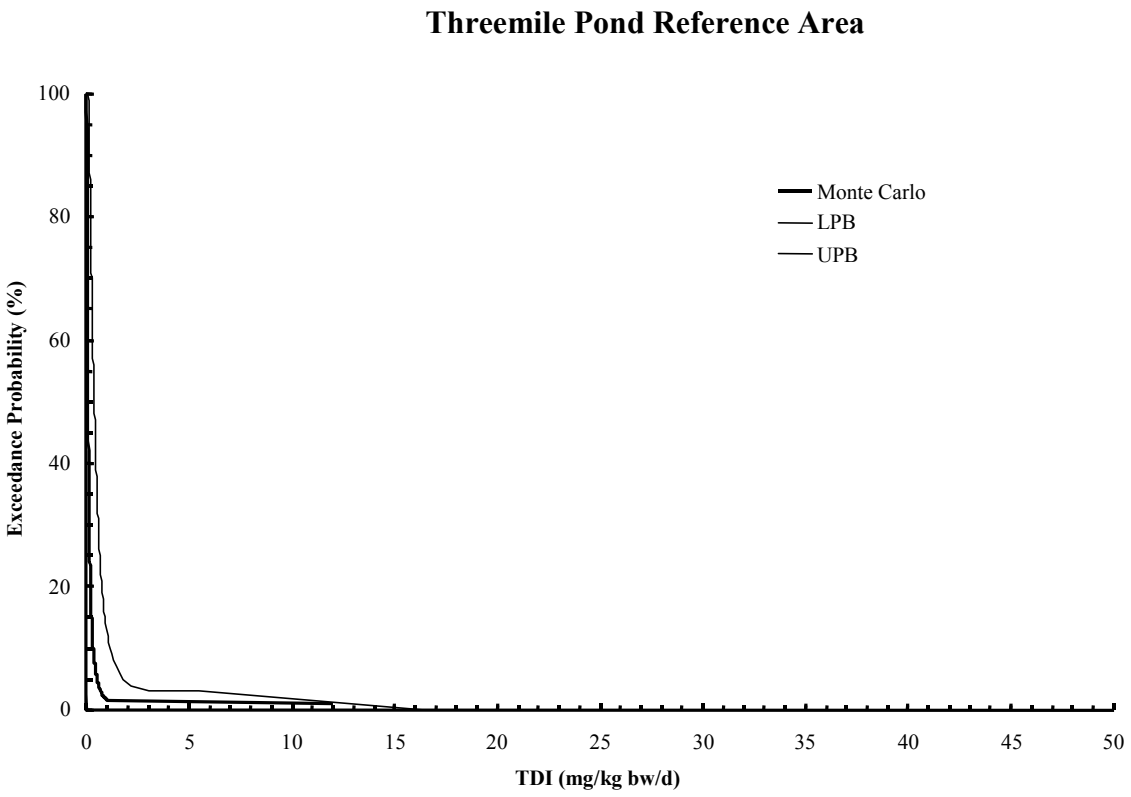
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-3 Exposure of Mink to tPCBs in Reach 6 of the Housatonic River



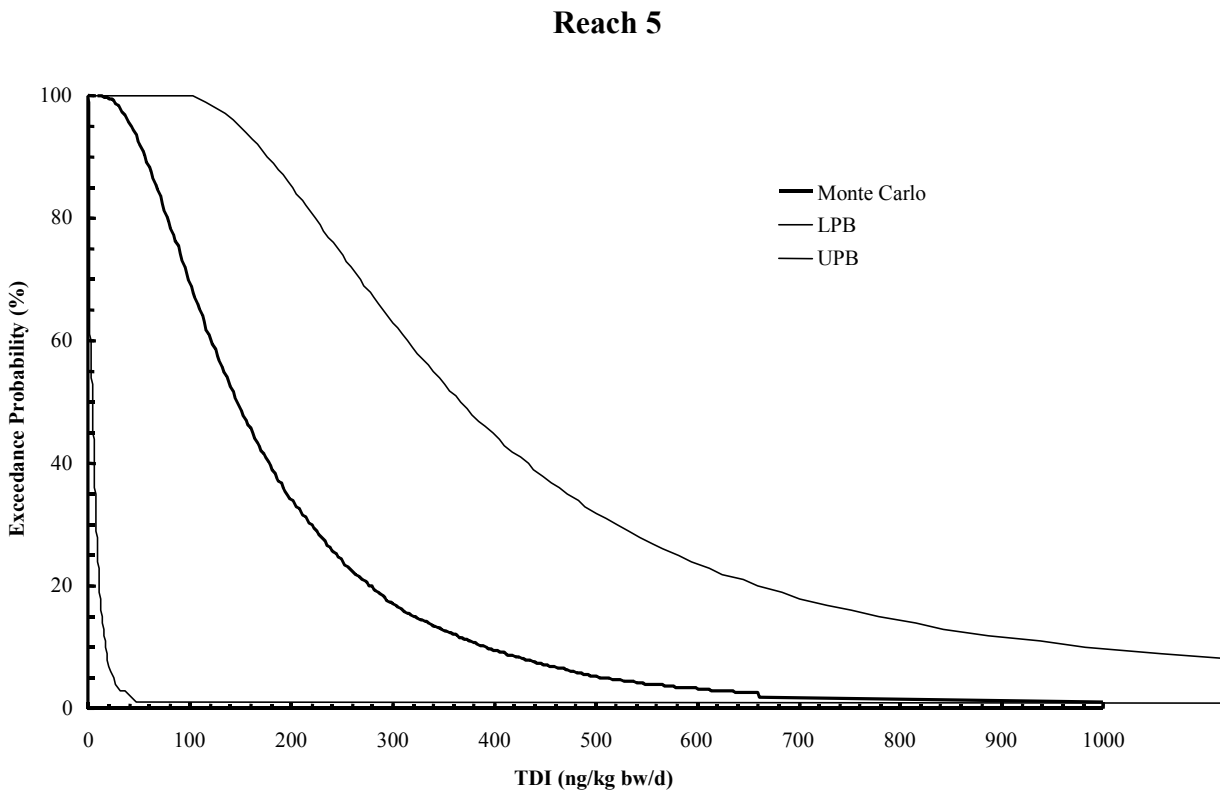
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-4 Exposure of Mink to tPCBs in the Housatonic River Upstream Reference Area



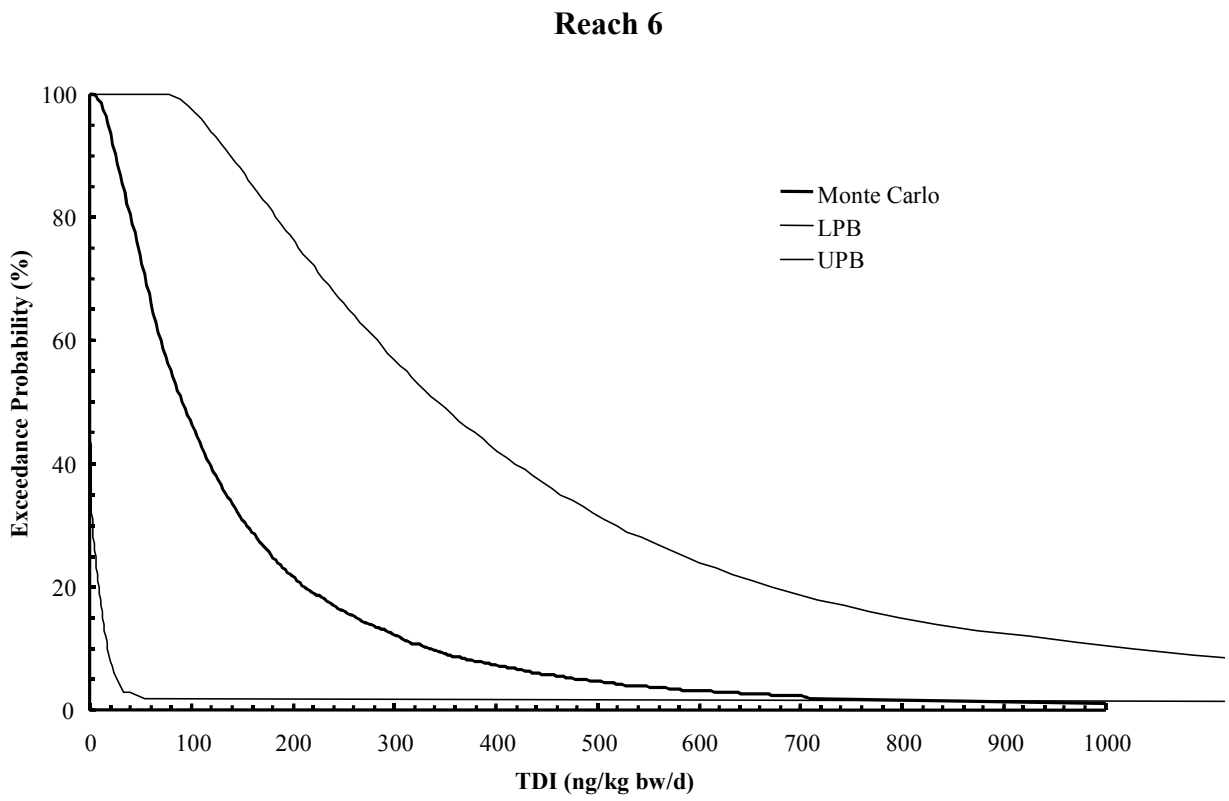
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-5 Exposure of Mink to tPCBs in the Threemile Pond Reference Area



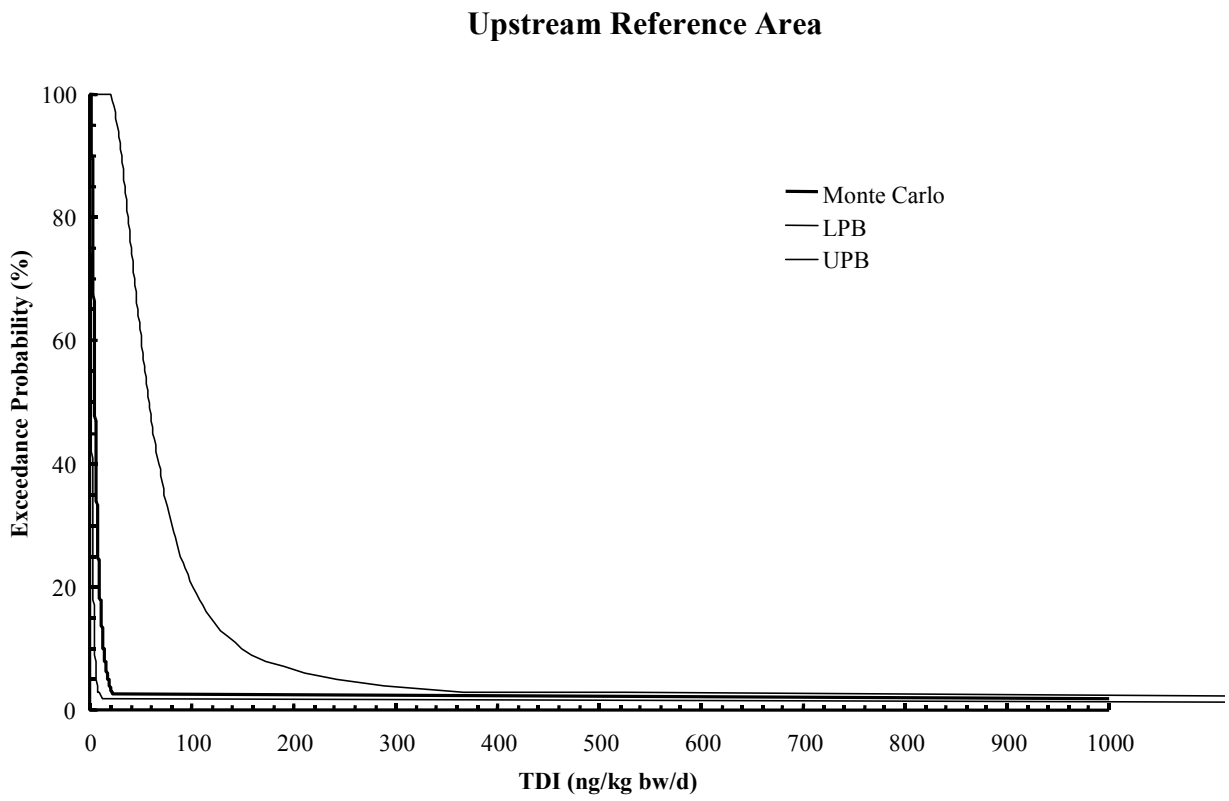
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-6 Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 5 of the Housatonic River



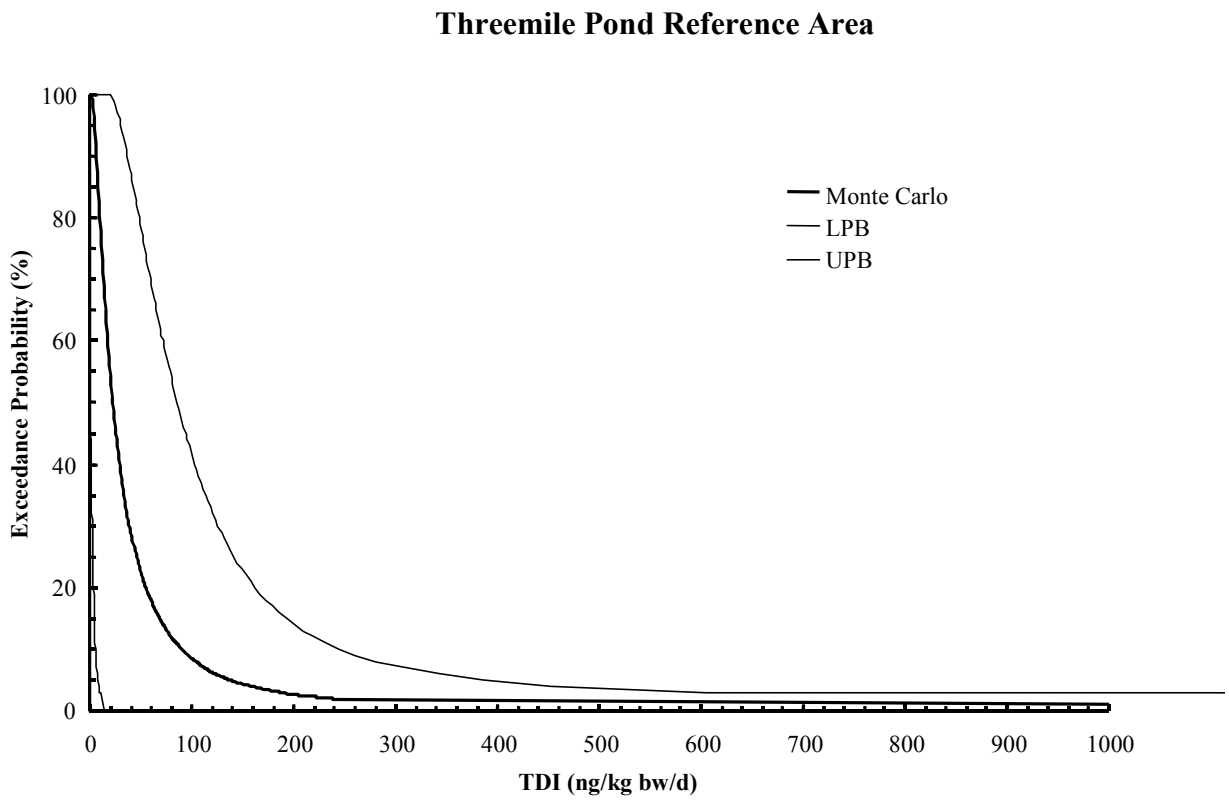
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-7 Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 6 of the Housatonic River



LPB = Lower probability bound
UPB = Upper probability bound

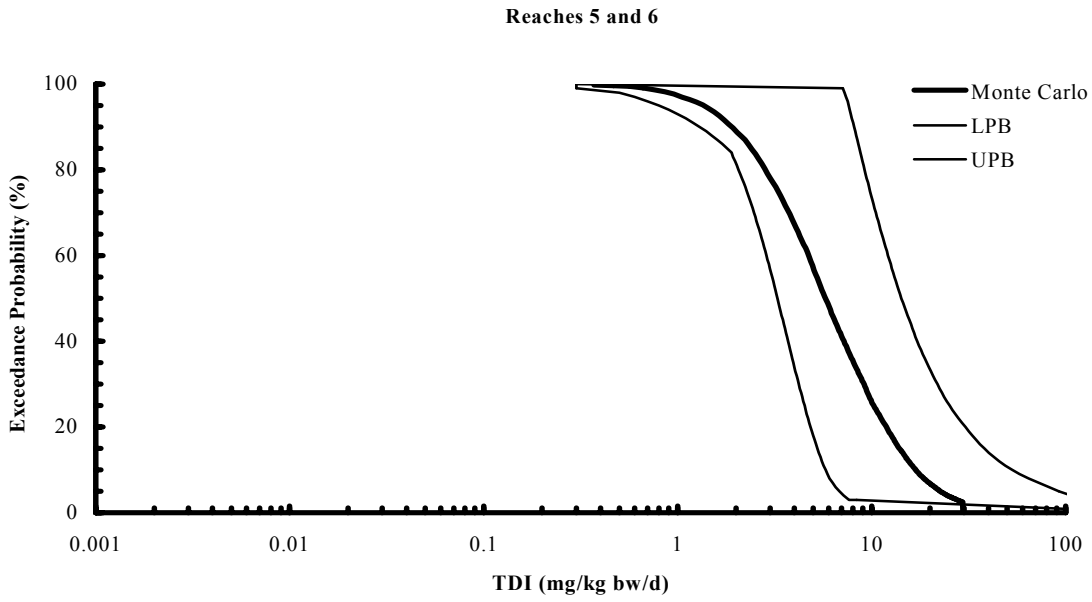
Figure I.2-8 Exposure of Mink to 2,3,7,8-TCDD TEQ in the Housatonic River Upstream Reference Area



LPB = Lower probability bound
UPB = Upper probability bound

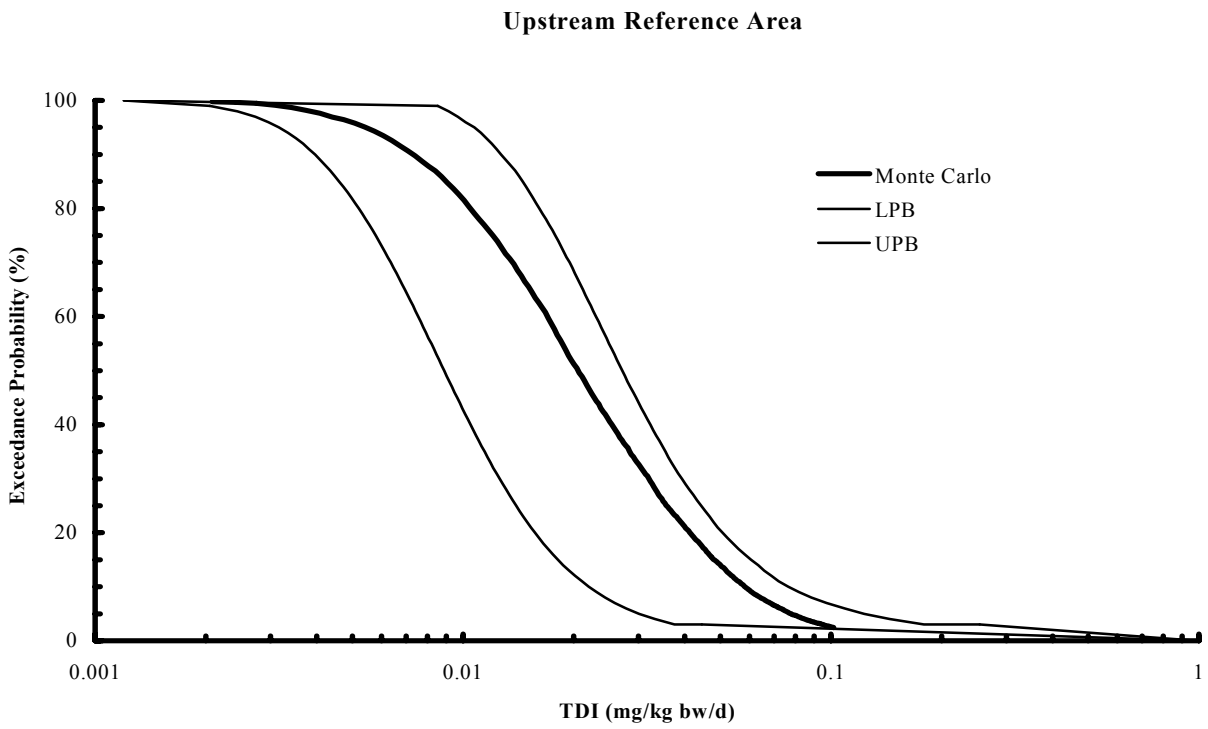
Figure I.2-9 Exposure of Mink to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area

Figure I.2-10 River Otter (*Lutra canadensis*)



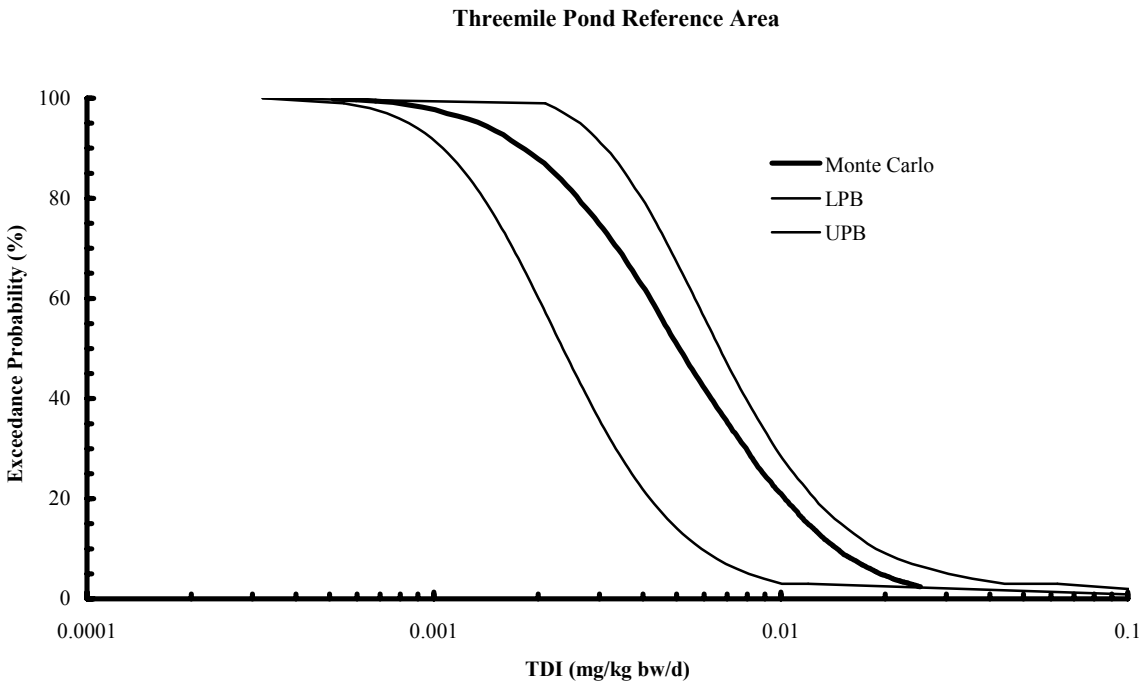
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-11 Exposure of River Otter to tPCBs in Reaches 5 and 6 of the Housatonic River



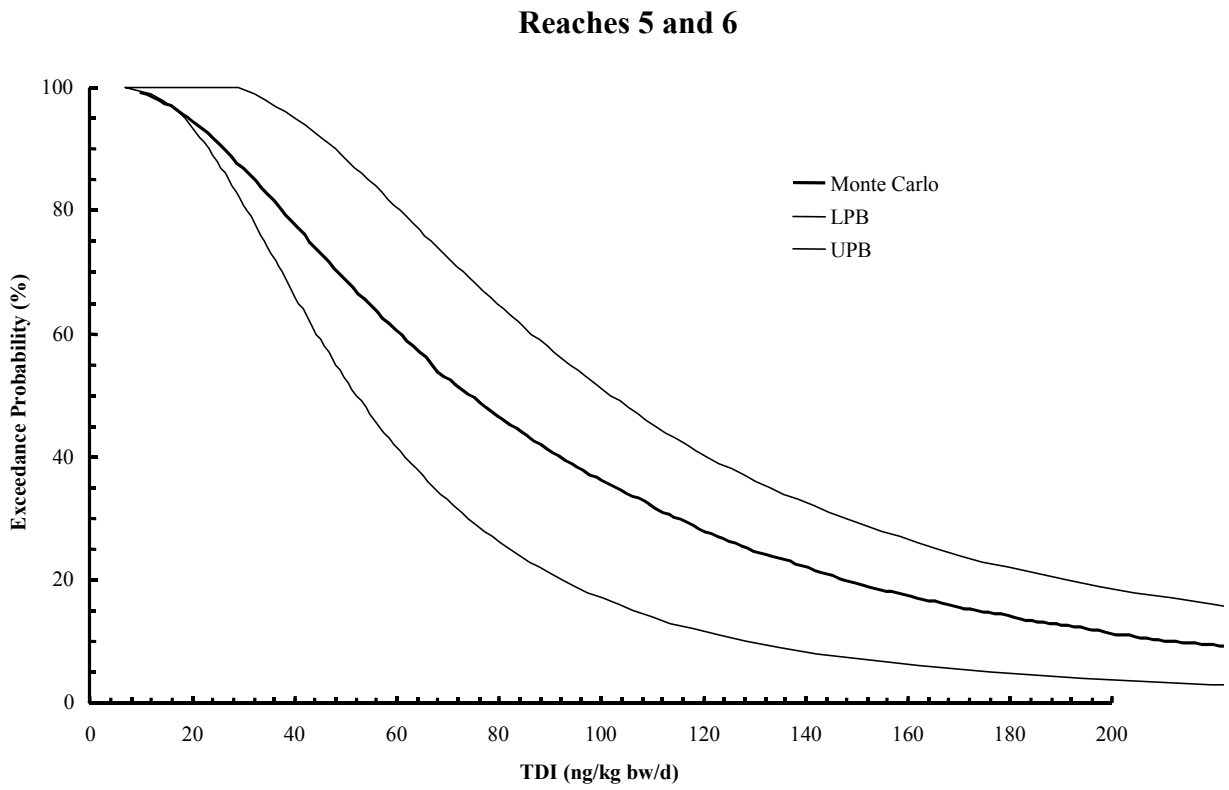
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-12 Exposure of River Otter to tPCBs in the Housatonic River Upstream Reference Area



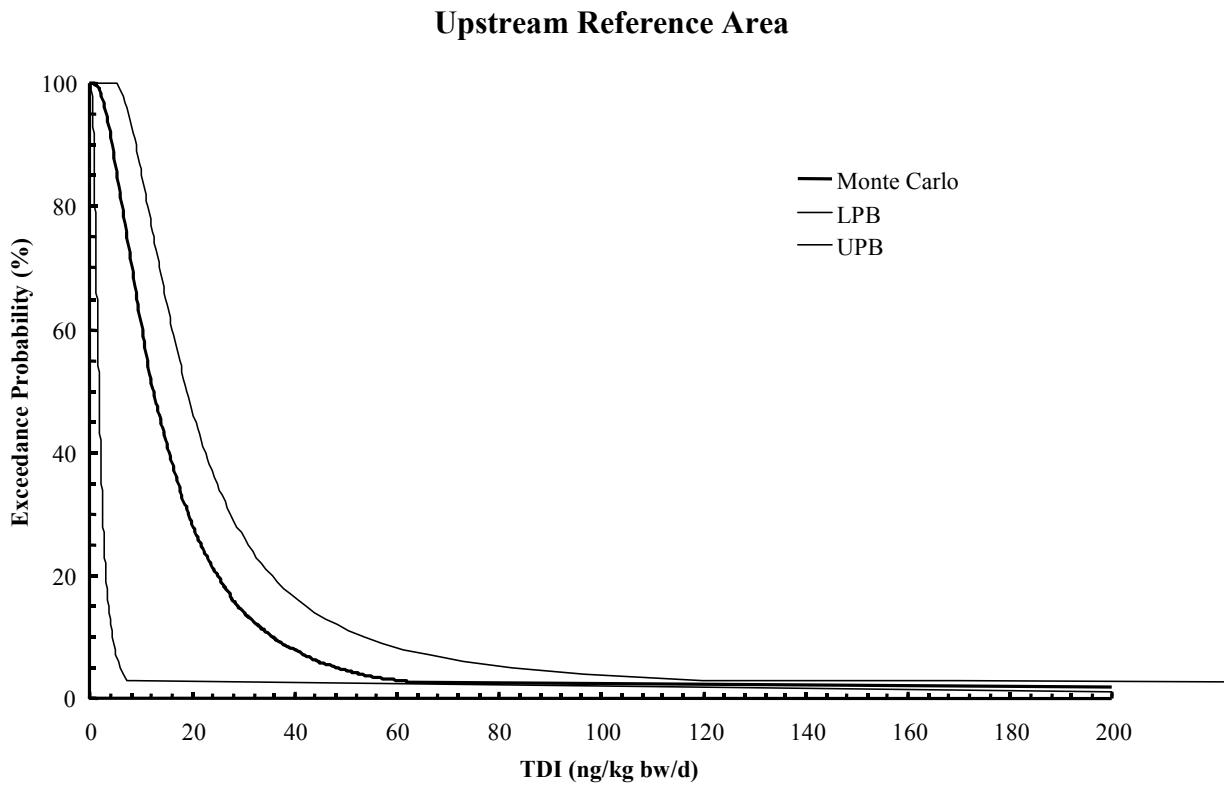
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-13 Exposure of River Otter to tPCBs in the Threemile Pond Reference Area



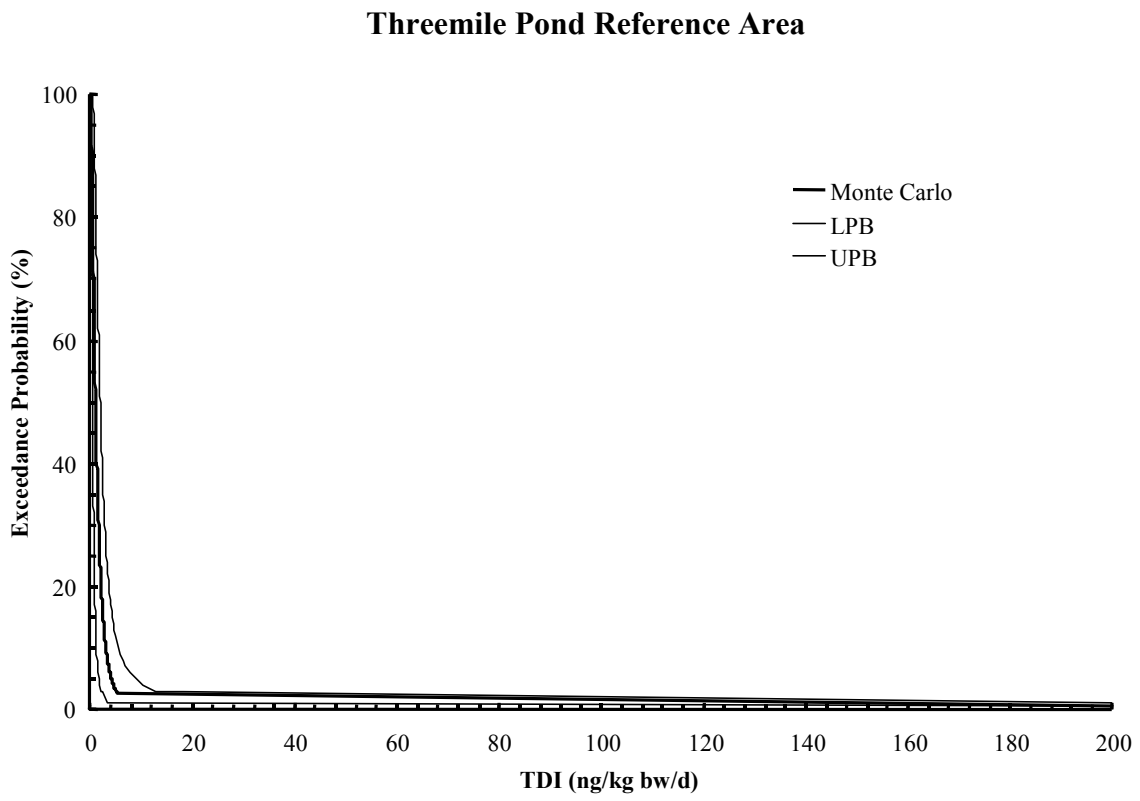
LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-14 Exposure of River Otter to 2,3,7,8-TCDD TEQ in Reaches 5 and 6 of the Housatonic River



LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-15 Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Upstream Reference Area



LPB = Lower probability bound
UPB = Upper probability bound

Figure I.2-16 Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area

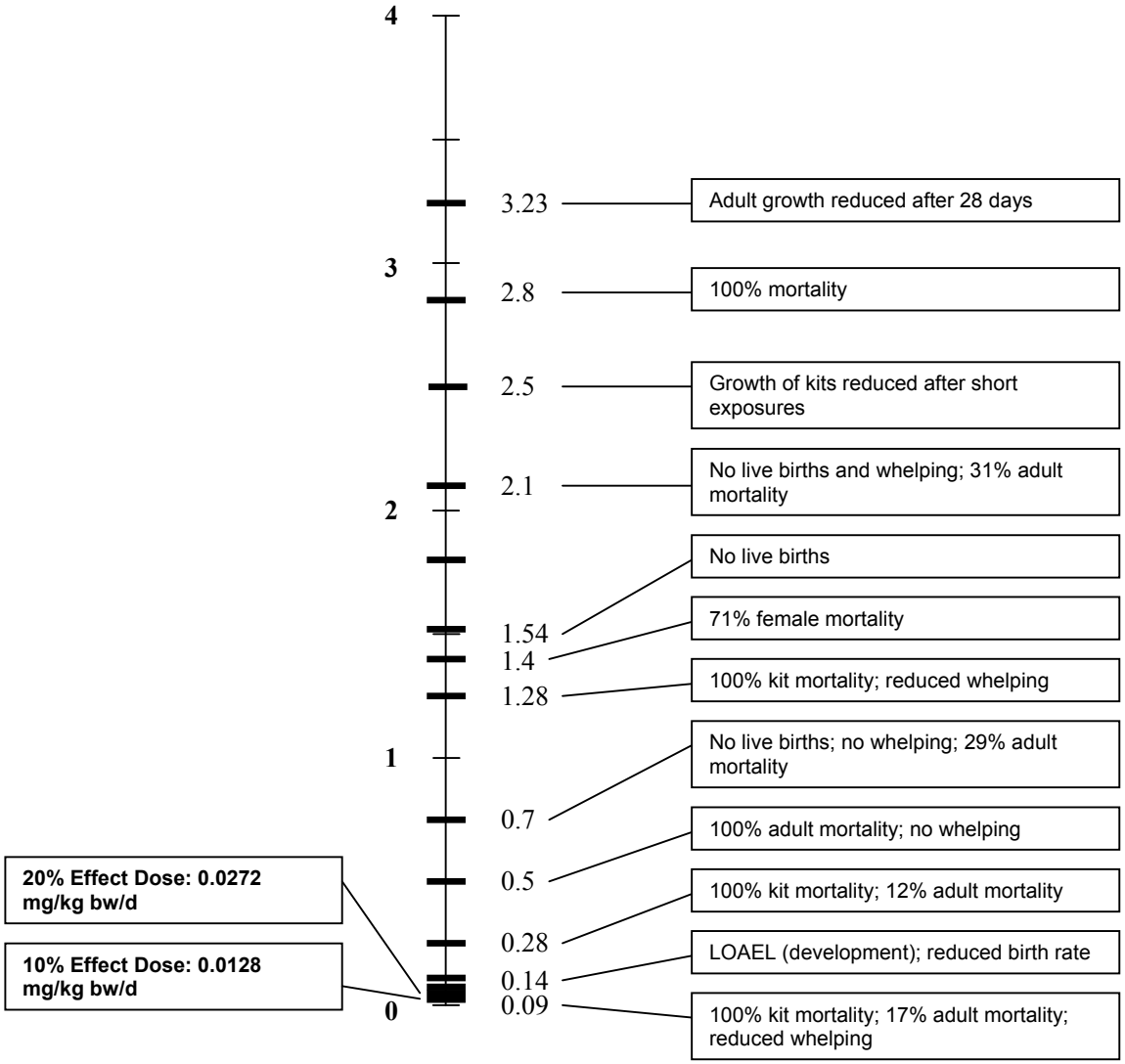


Figure I.3-1 Range of Effects of tPCBs in Diet of Piscivorous Mammals (mg/kg bw/d)

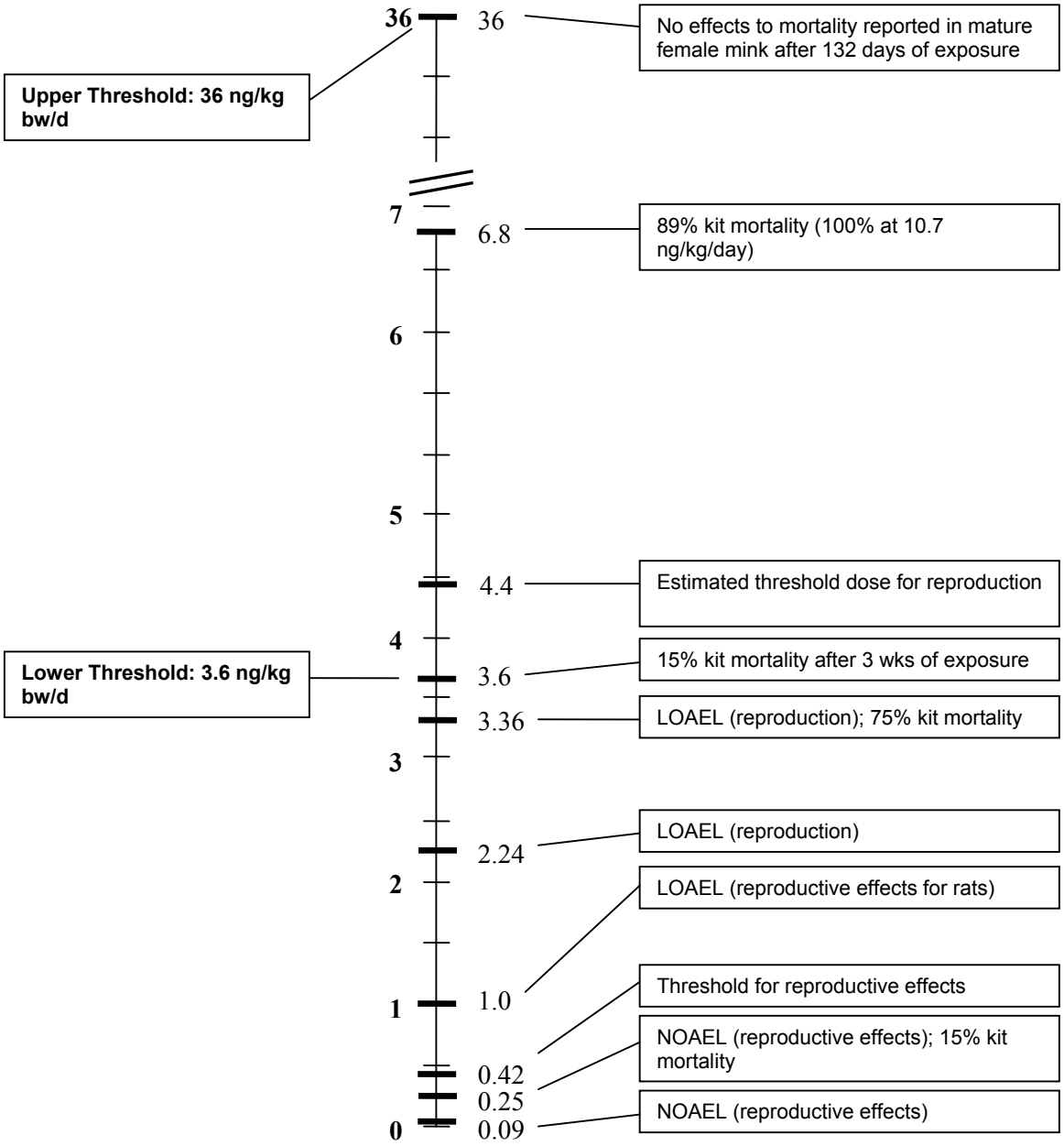


Figure I.3-2 Range of Effects of 2,3,7,8-TCDD TEQ in Diet of Piscivorous Mammals (ng/kg bw/d)

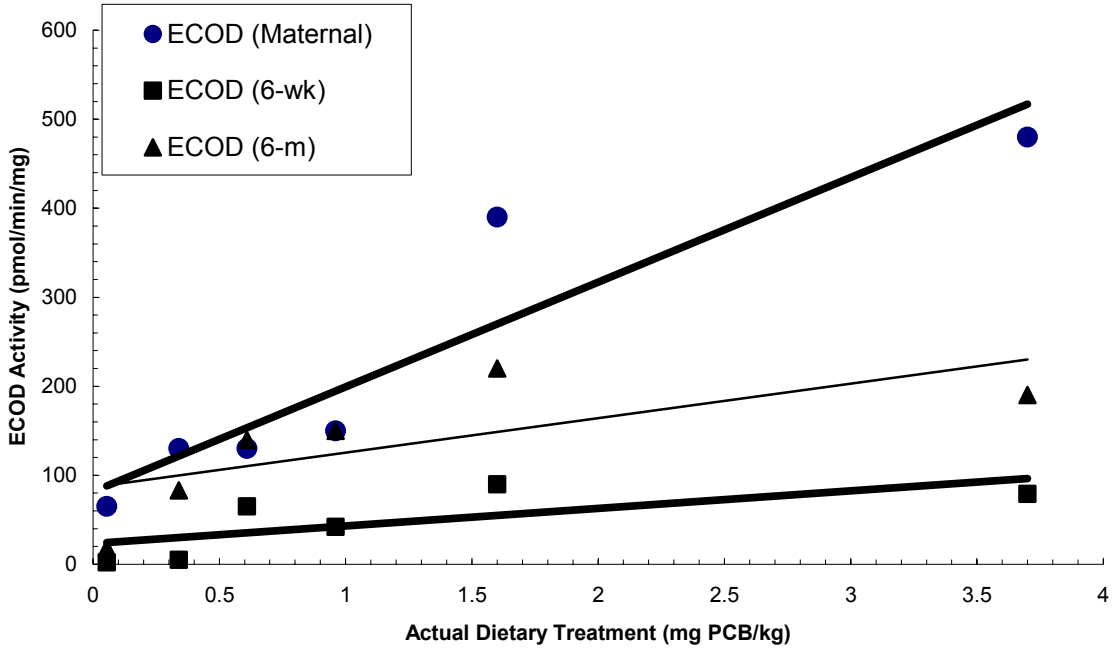


Figure I.3-3 ECOD Activity in Mink Fed Diet Contaminated with tPCBs (mg/kg)

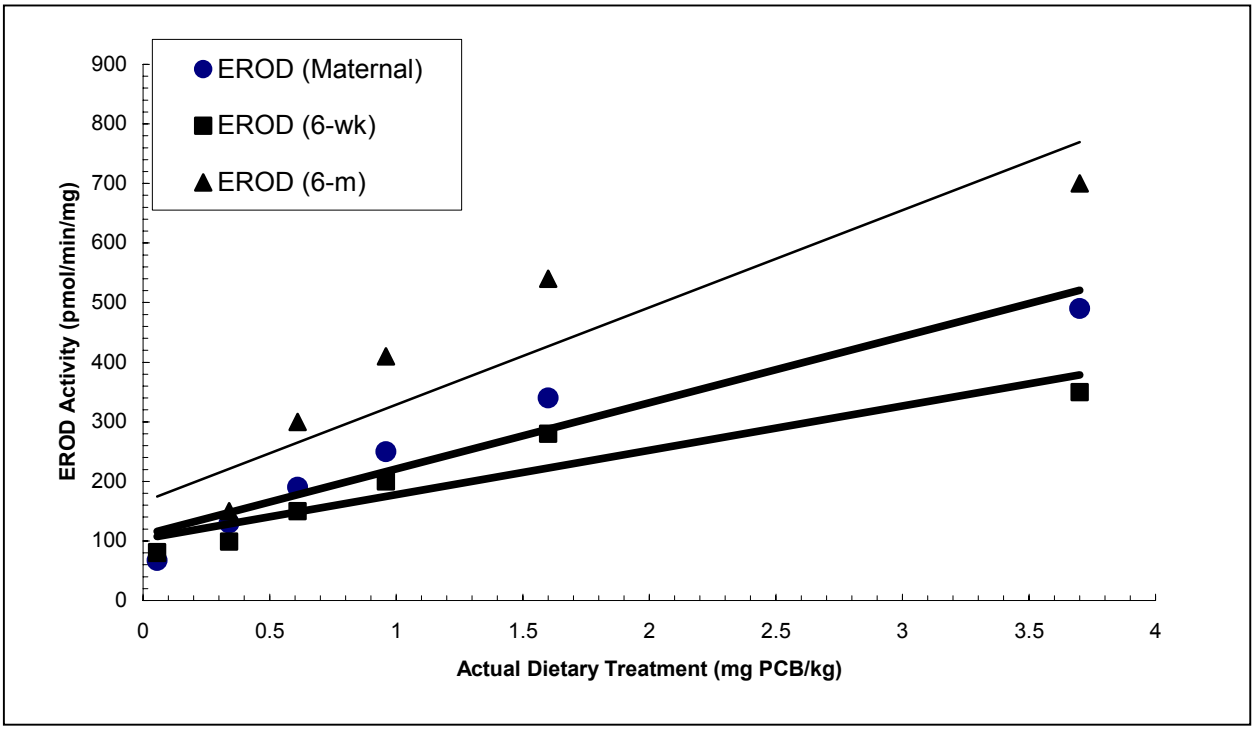


Figure I.3-4 EROD Activity in Mink Fed Diet Contaminated with tPCBs (mg/kg)

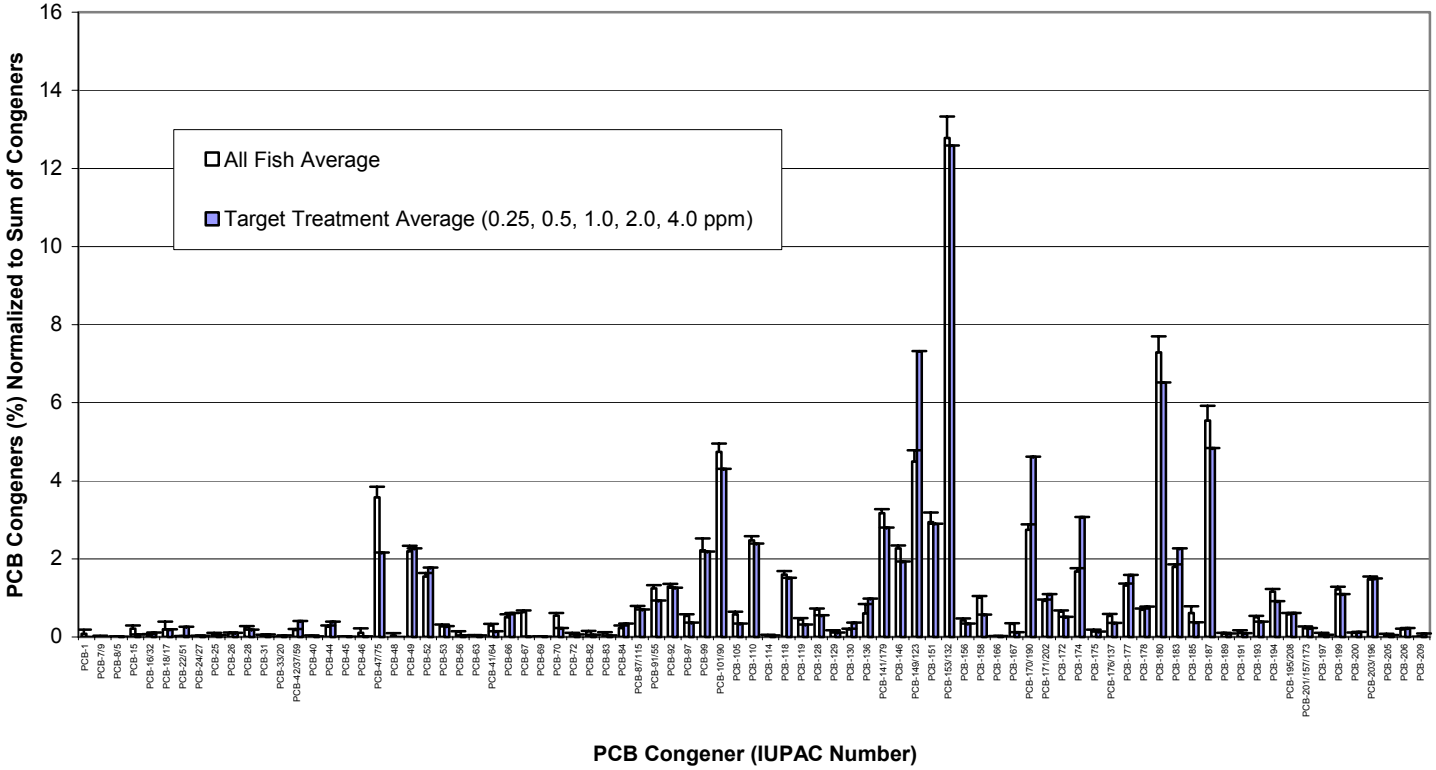


Figure I.3-5 Average Normalized PCB Congeners in Reaches 5 and 6 Fish (Lengths of 7 to 20 cm) and Mink Diet Treatments (2 SE Error Bars)

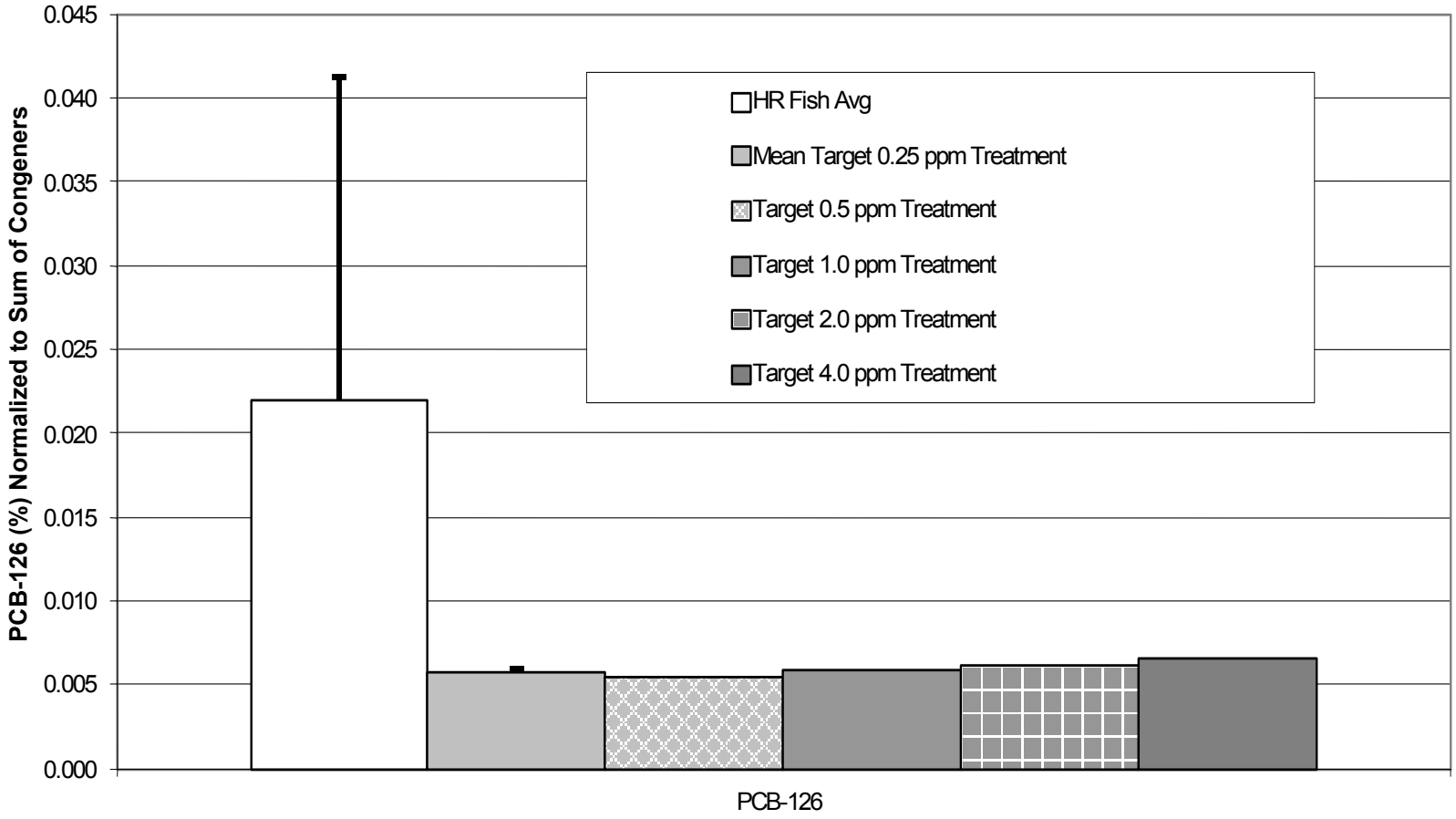


Figure I.3-6 Average Normalized PCB-126 in Reaches 5 and 6 Fish (Lengths of 7 to 20 cm) and Mink Diet Treatments (2 SE Error Bars for Housatonic River Fish and 0.25 ppm Triplicate Diet Treatment)

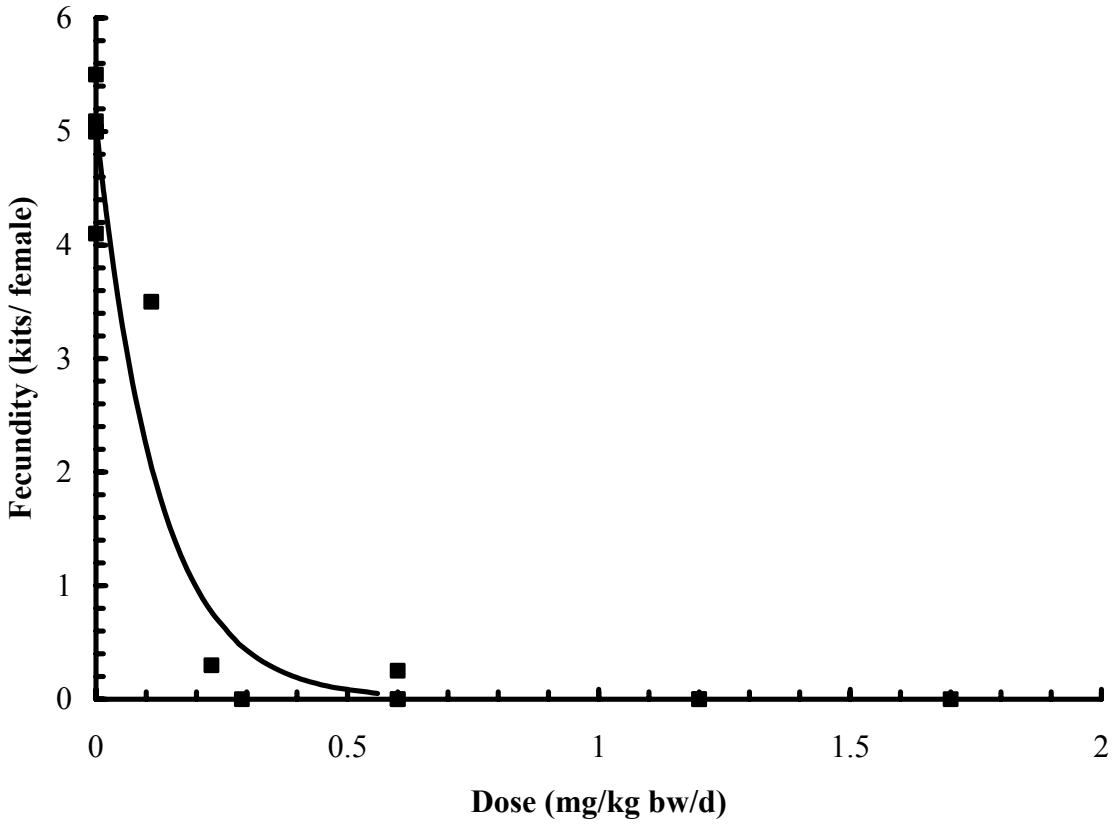


Figure I.3-7 Dose Response Curve for Effects of tPCBs on Fecundity of Mink (Symbols Indicate Raw Data)

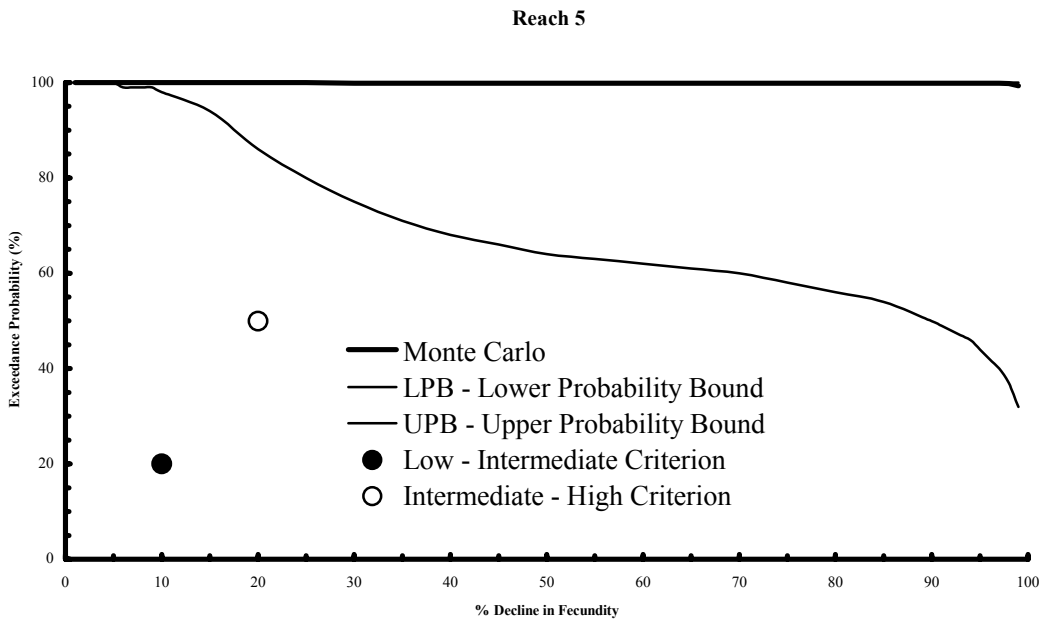


Figure I.4-1 Total PCB Risk to Mink Exposed to tPCBs in Reach 5 of the Housatonic River

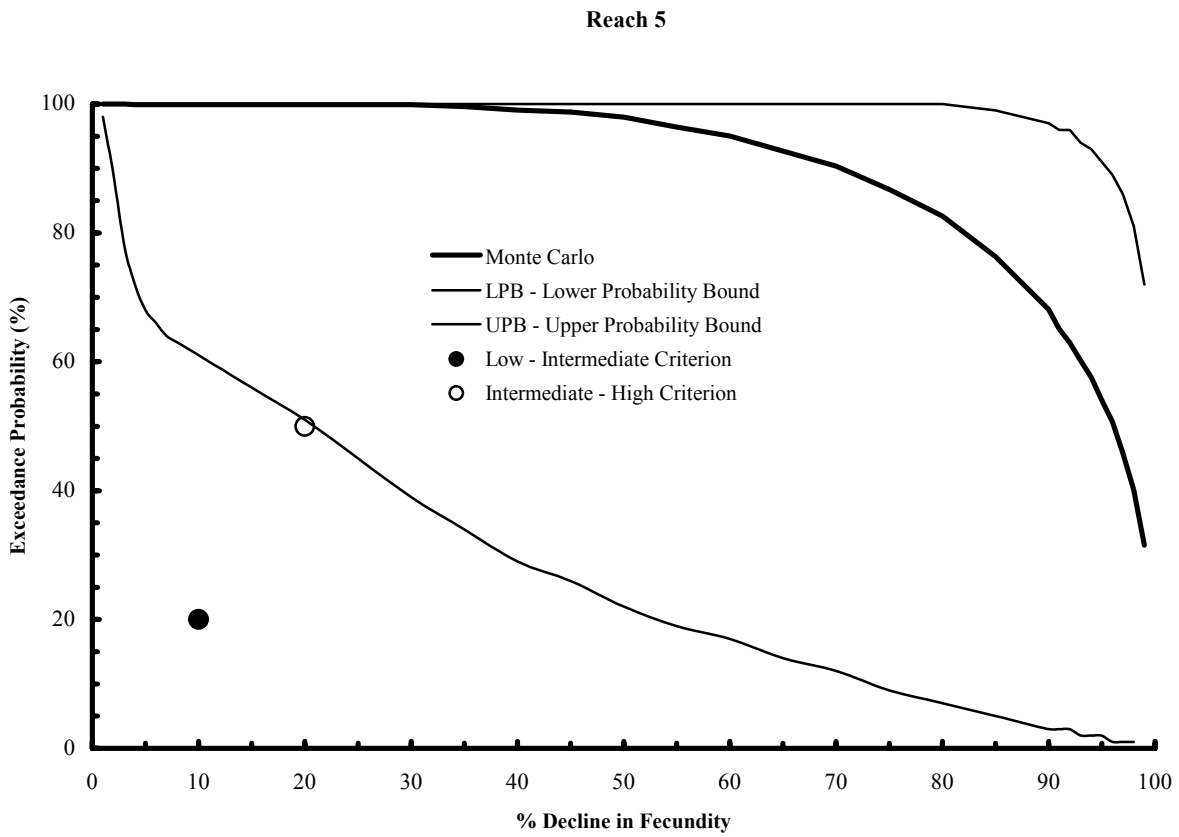


Figure I.4-2 Total PCB Risk to Mink (10% Foraging Time in Reach 5)

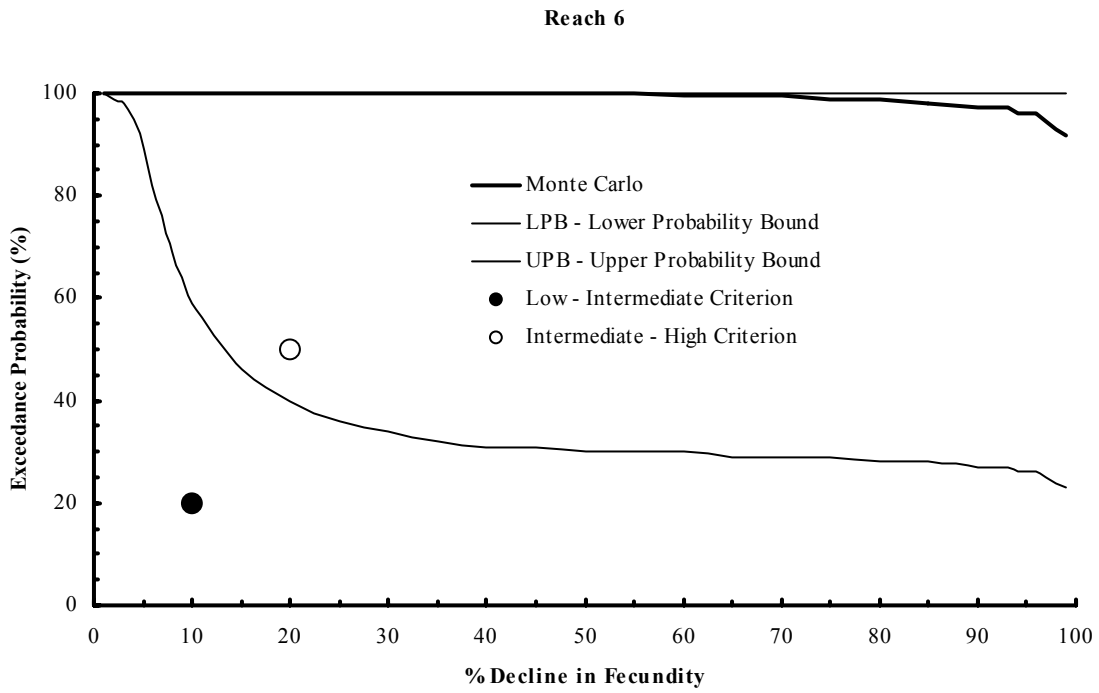


Figure I.4-3 Total PCB Risk to Mink Exposed to tPCBs in Reach 6 of the Housatonic River

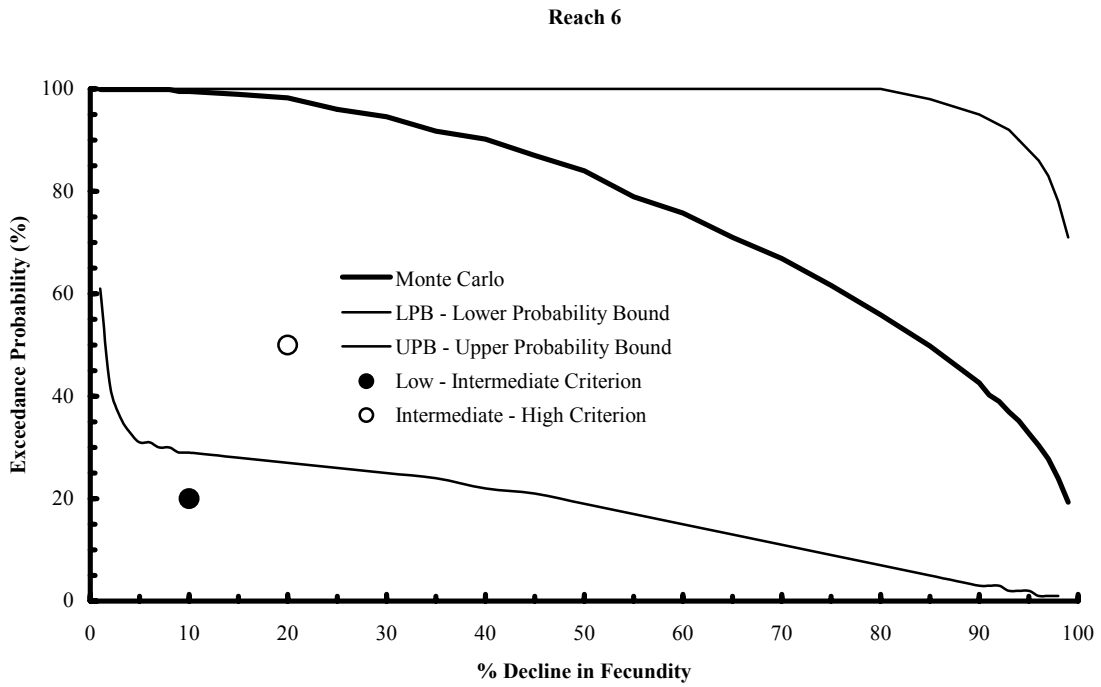


Figure I.4-4 Total PCB Risk to Mink (10% Foraging Time in Reach 6)

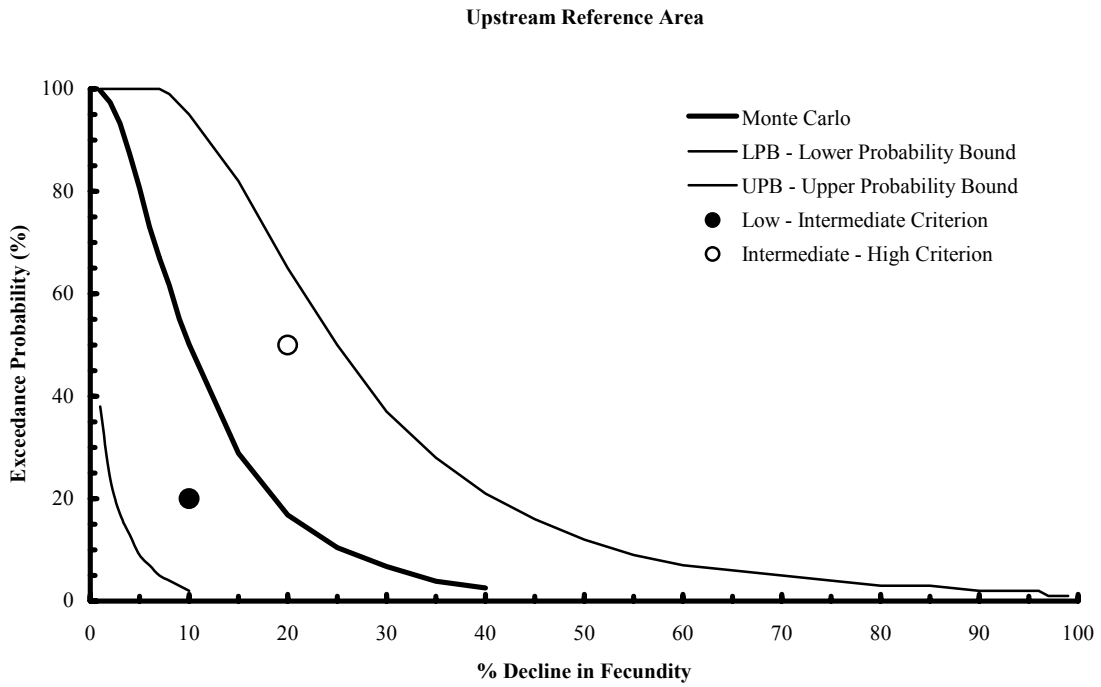


Figure I.4-5 Total PCB Risk to Mink Foraging in the Upstream Reference Area

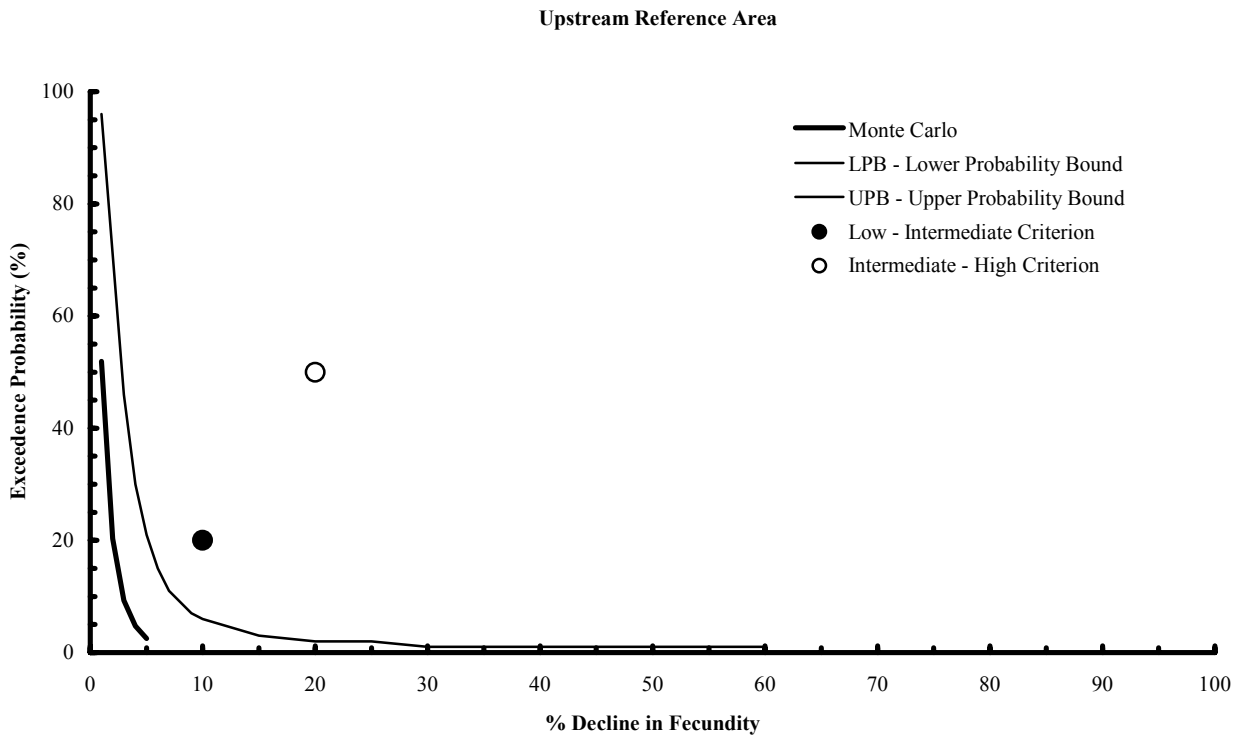


Figure I.4-6 Total PCB Risk to Mink (10% Foraging Time in the Upstream Reference Area)

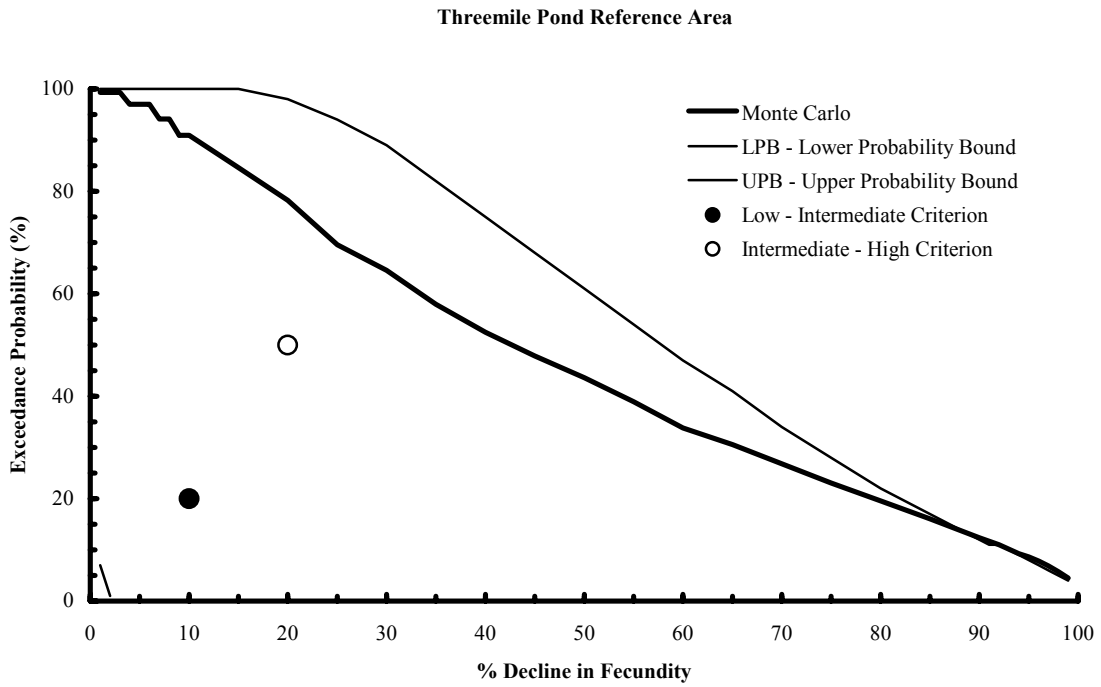


Figure I.4-7 Total PCB Risk to Mink Exposed to tPCBs in the Threemile Pond Reference Area

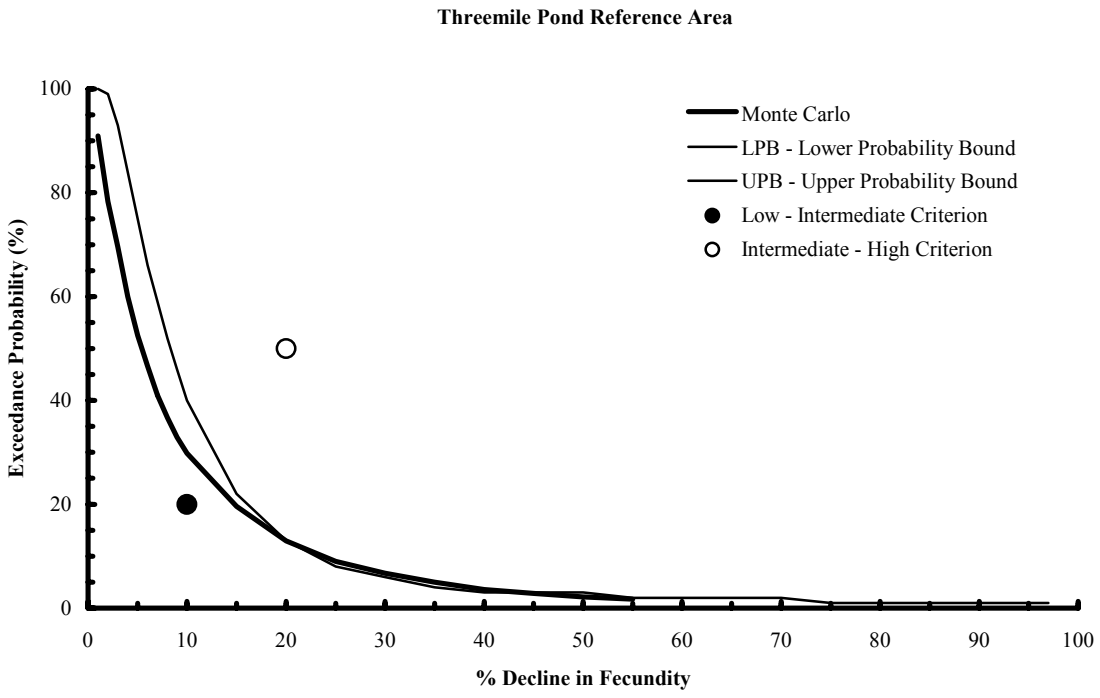
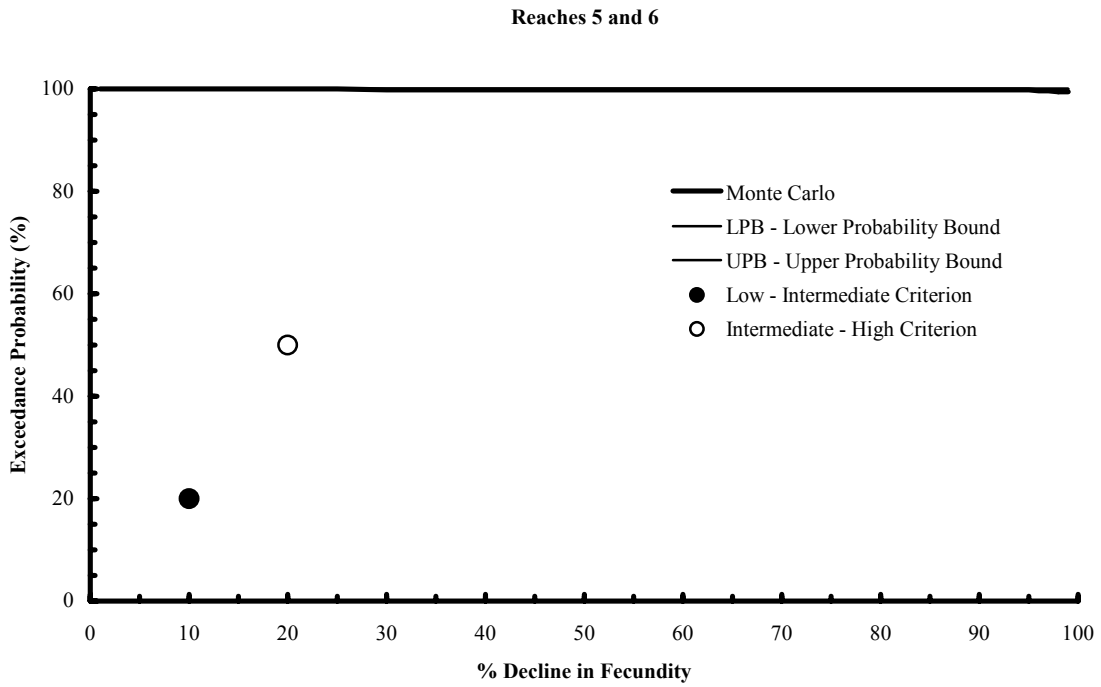


Figure I.4-8 Total PCB Risk to Mink (10% Foraging Time in the Threemile Pond Reference Area)



Note: The LPB and UPB overlap the Monte Carlo line

Figure I.4-9 Total PCB Risk to River Otter Exposed to tPCBs in Reaches 5 and 6 of the Housatonic River

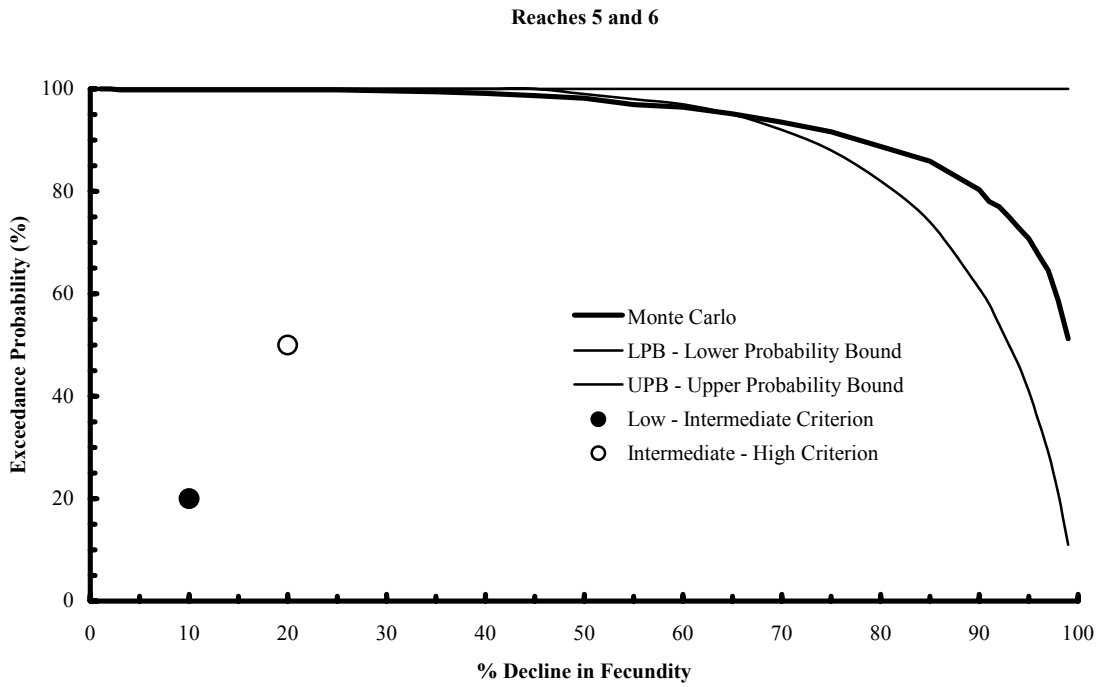


Figure I.4-10 Total PCB Risk to River Otter (10% Foraging Time in Reaches 5 and 6)

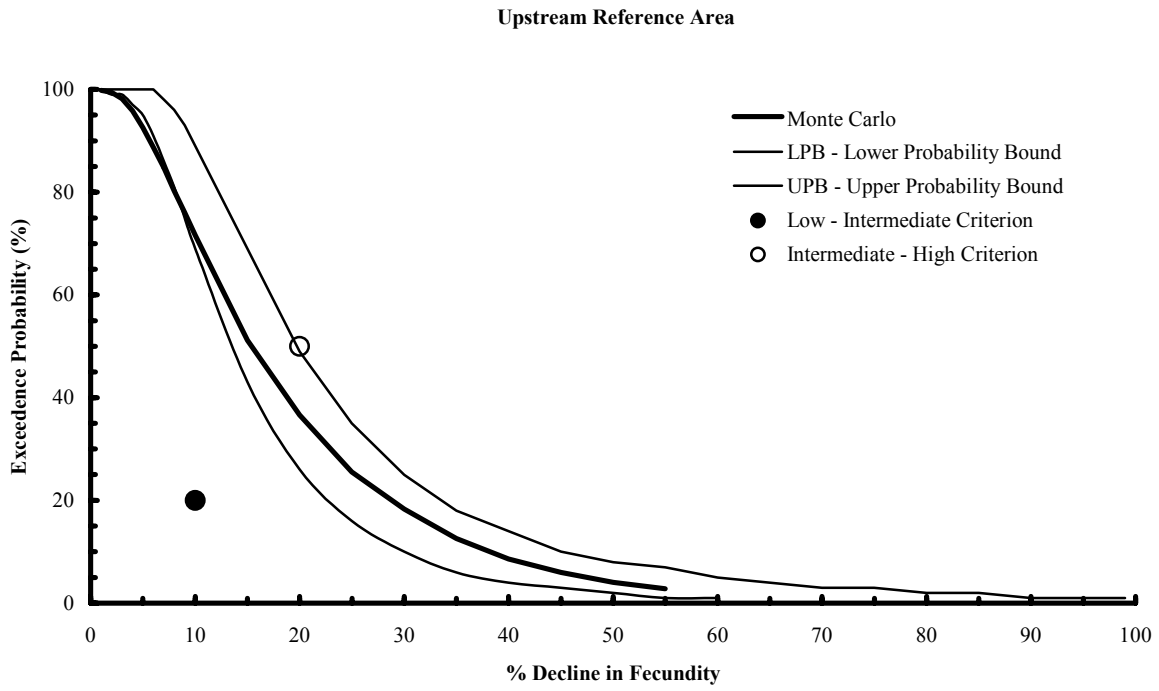


Figure I.4-11 Total PCB Risk to River Otter Foraging in the Upstream Reference Area

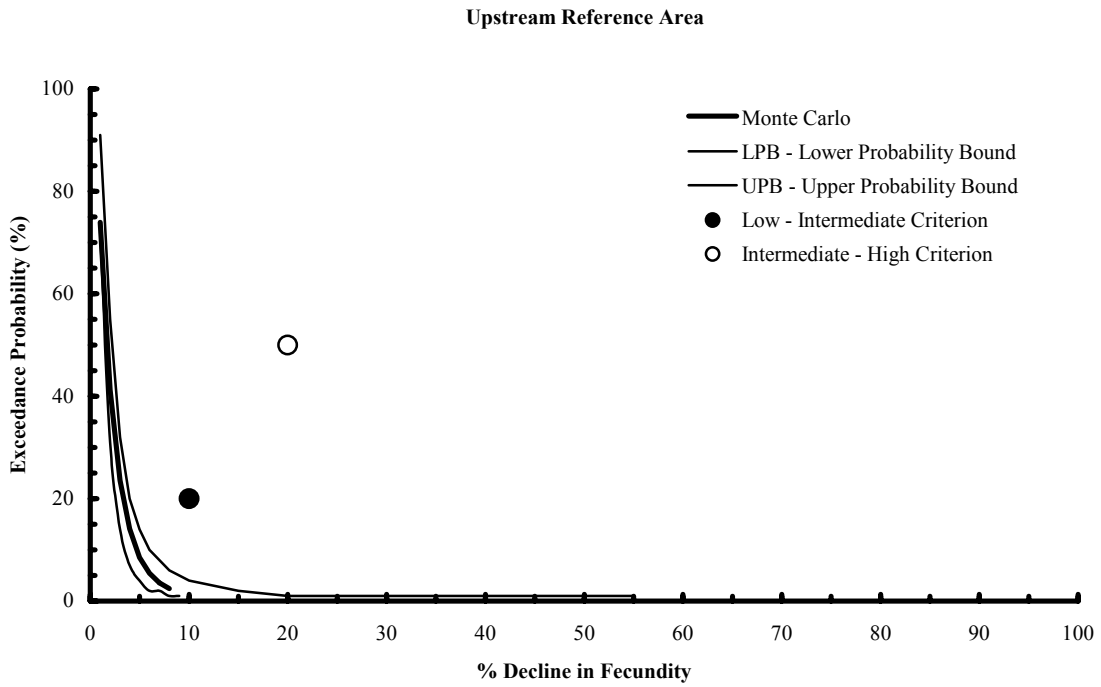


Figure I.4-12 Total PCB Risk to River Otter (10% Foraging Time in the Upstream Reference Area)

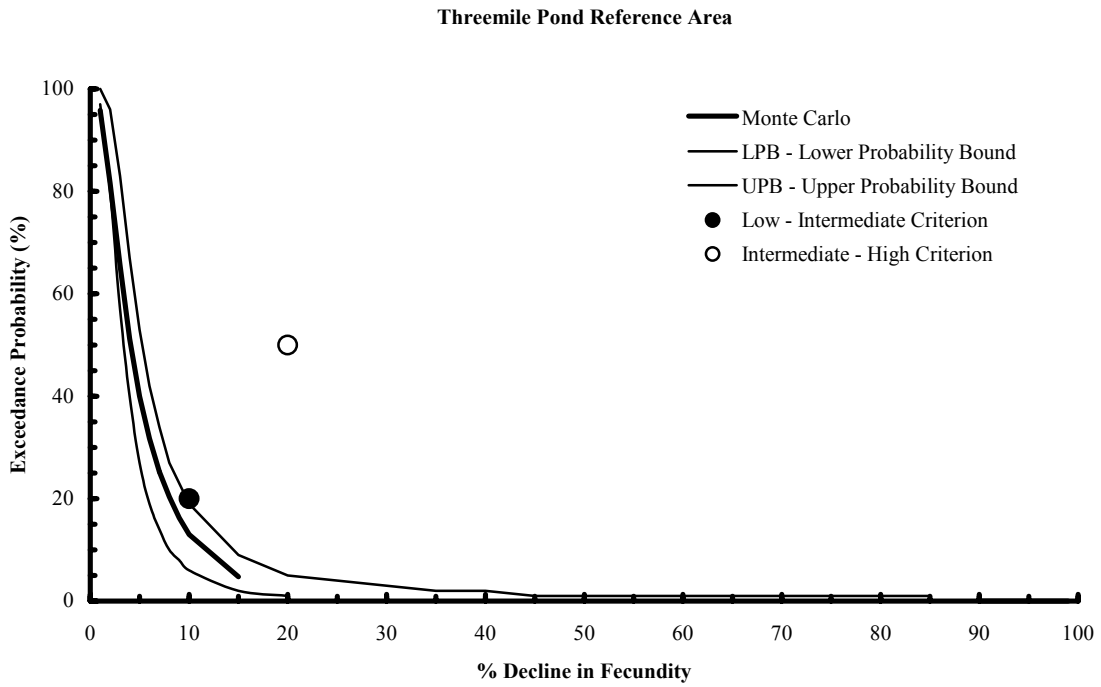


Figure I.4-13 Total PCB Risk to River Otter Exposed to tPCBs in the Threemile Pond Reference Area

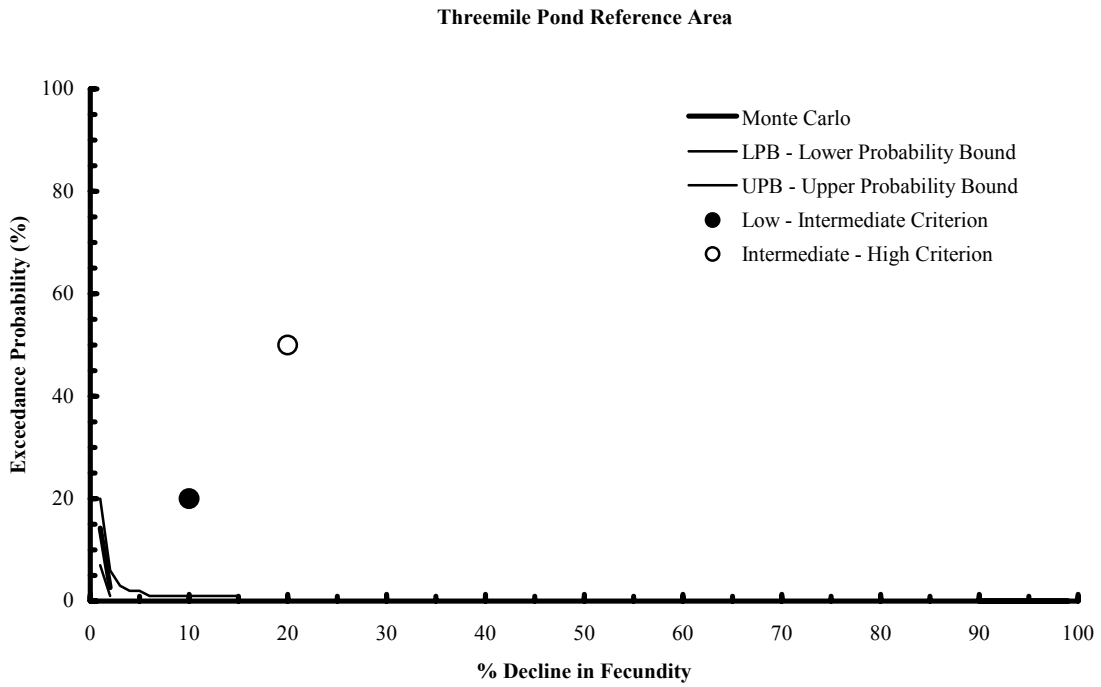
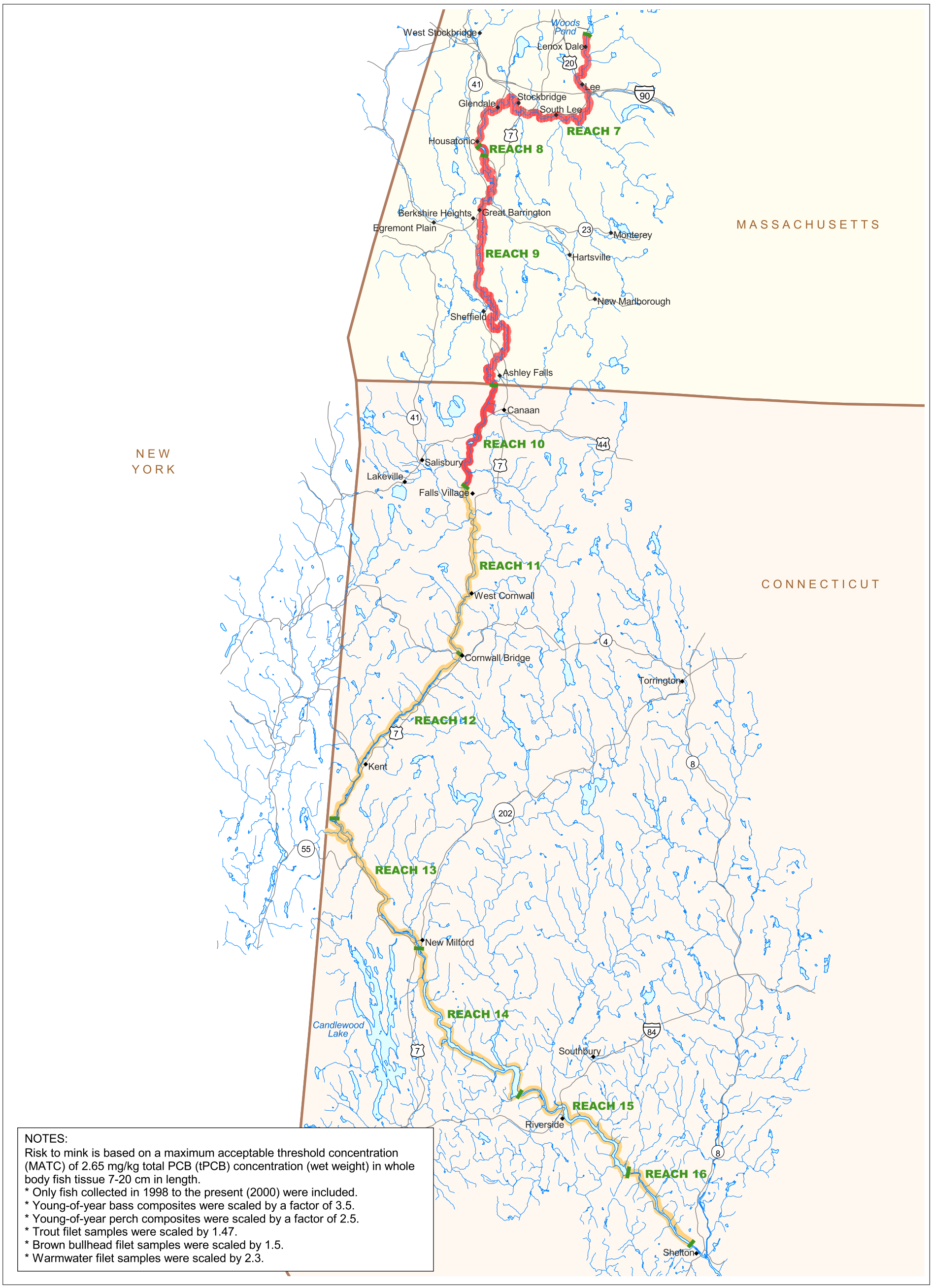


Figure I.4-14 Total PCB Risk to River Otter (10% Foraging Time in the Threemile Pond Reference Area)



NOTES:
Risk to mink is based on a maximum acceptable threshold concentration (MATC) of 2.65 mg/kg total PCB (tPCB) concentration (wet weight) in whole body fish tissue 7-20 cm in length.
* Only fish collected in 1998 to the present (2000) were included.
* Young-of-year bass composites were scaled by a factor of 3.5.
* Young-of-year perch composites were scaled by a factor of 2.5.
* Trout filet samples were scaled by 1.47.
* Brown bullhead filet samples were scaled by 1.5.
* Warmwater filet samples were scaled by 2.3.

LEGEND:

MINK RISK
- < 2.65 mg/kg
- >= 2.65 mg/kg

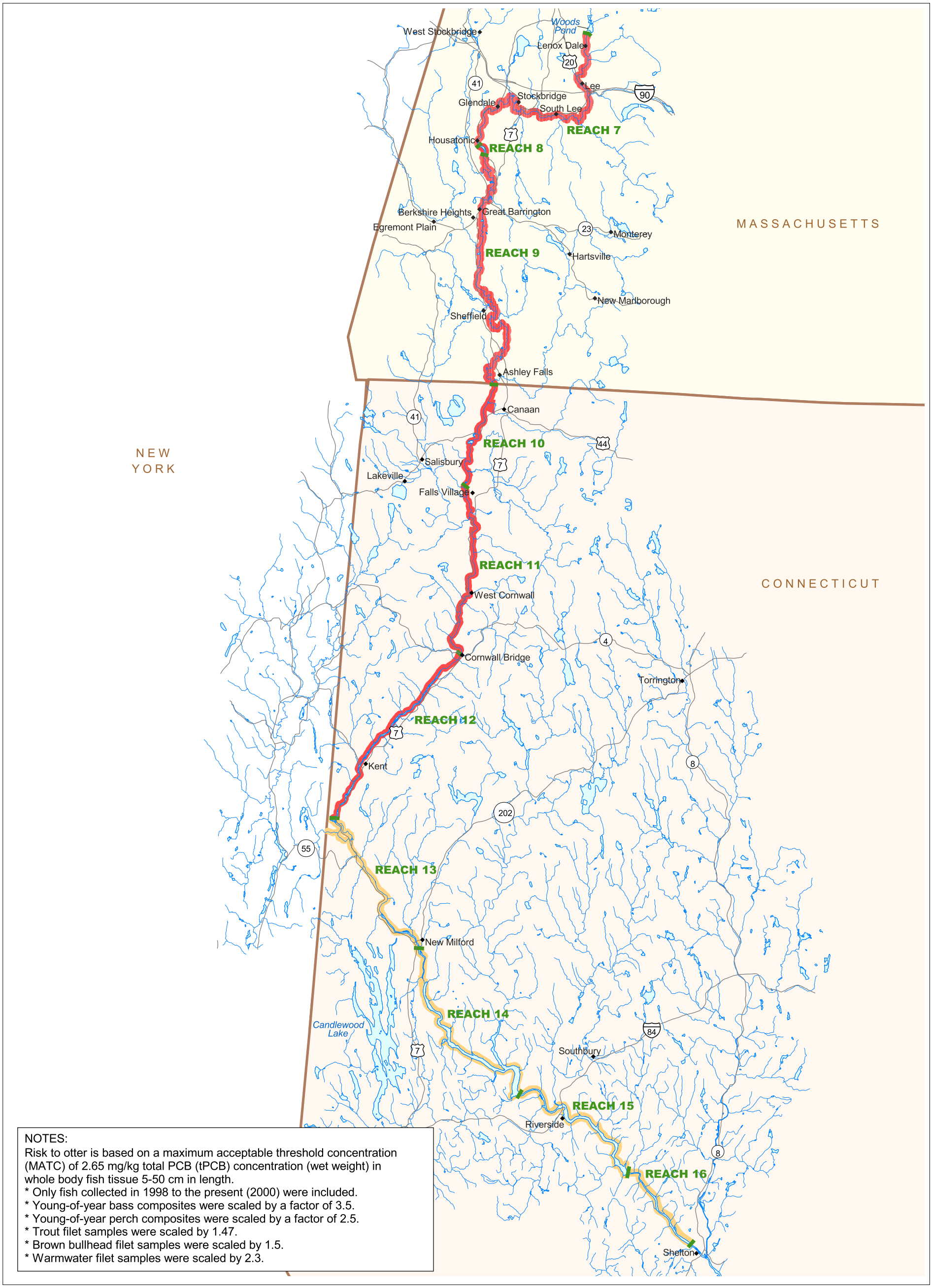
- Town
- Reach Break
- Roads
- Housatonic River Basin Hydrology
- State Boundary

N W E S

2 0 2 4 6 8 Miles

Ecological Risk Assessment
GE/Housatonic River Site
Rest of River

FIGURE I.4-15
ASSESSMENT OF RISK TO MINK EXPOSED TO tPCBs DOWNSTREAM OF WOODS POND



NOTES:
Risk to otter is based on a maximum acceptable threshold concentration (MATC) of 2.65 mg/kg total PCB (tPCB) concentration (wet weight) in whole body fish tissue 5-50 cm in length.
* Only fish collected in 1998 to the present (2000) were included.
* Young-of-year bass composites were scaled by a factor of 3.5.
* Young-of-year perch composites were scaled by a factor of 2.5.
* Trout filet samples were scaled by 1.47.
* Brown bullhead filet samples were scaled by 1.5.
* Warmwater filet samples were scaled by 2.3.

LEGEND:

OTTER RISK
- < 2.65 mg/kg
- >= 2.65 mg/kg

- Town
- Reach Break
- Roads
- Housatonic River Basin Hydrology
- State Boundary

N W E S

2 0 2 4 6 8 Miles

Ecological Risk Assessment
GE/Housatonic River Site
Rest of River

FIGURE I.4-16
ASSESSMENT OF RISK TO OTTER EXPOSED TO tPCBs DOWNSTREAM OF WOODS POND

APPENDIX J

ASSESSMENT ENDPOINT – SURVIVAL, GROWTH, AND REPRODUCTION OF OMNIVOROUS AND CARNIVOROUS MAMMALS

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TABLES

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LIST OF ACRONYMS

BW	body weight
CDDs	chlorinated dibenzo-p-dioxins
CL	confidence limits
COC	contaminant of concern
COPC	contaminant of potential concern
CRCD	Center for Research in Chronic Disorders
FIR	food intake rate
FMR	free metabolic rate
FT	foraging time
GE	General Electric Company
GLiM	generalized linear model
H/W	Han/Wistar
HCDD	1,2,3,4,7,8-hexachlorodibenzo-p-dioxin
IDW	inverse distance weighting
kJ	kilojoules
LCL	lower confidence limit
LE	Long-Evans
LOAEL	lowest observed adverse effect level
NOAEL	no observed adverse effect level
PCB	polychlorinated biphenyl
PCDD	1,2,3,7,8-pentachlorodibenzo-p-dioxin
PSA	Primary Study Area
TCDD	tetrachrlorodibenzo-p-dioxin
TEF	toxic equivalency factor
TEQ	toxic equivalence
TN	trap nights
tPCBs	total PCBs
UCL	upper confidence limit

APPENDIX J

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF OMNIVOROUS AND CARNIVOROUS MAMMALS

J.1 INTRODUCTION

The purpose of this appendix is to characterize and quantify the current and potential risks posed to omnivorous and carnivorous mammals exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain, focusing on total PCBs (tPCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The watershed is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters of the watershed. The Primary Study Area (PSA) includes the river and 10-year floodplain from the confluence of the East and West Branches of the Housatonic River downstream of the GE facility, to Woods Pond (Figure 1.1-2).

A pre-ERA was conducted to narrow the scope of the ecological risk assessment by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A three-tiered deterministic approach was used to screen COPCs. The deterministic assessments compared potential conservative estimates of exposure with conservative adverse effects benchmarks to identify which contaminants are of potential concern to omnivorous and carnivorous mammals in the Housatonic River. A hazard quotient (total daily intake/effect benchmark) greater than 1 in the Housatonic River area resulted in the COPC being screened through to the next tier assessment, and to the probabilistic ecological risk assessment, if necessary. In the COPC screening specific to this endpoint, several other COPCs (primarily organochlorine pesticides) were screened out because their actual concentrations in the PSA were likely much lower than the measured values due to laboratory interference (see Section 2.4). In summary, the COPCs that screened through to the probabilistic risk assessment for omnivorous and carnivorous mammals were the contaminants of concern (COCs), tPCBs and 2,3,7,8-TCDD TEQ (TEQ). Total PCBs detected in Housatonic River media samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using

the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4).

J.1.1 Overview of Approach

A step-wise approach was used to assess the risks of tPCBs and TEQ to omnivorous and carnivorous mammals in the Housatonic River watershed. The four main steps in this process are as follows:

1. Derivation of a conceptual model (Figure J.1-1).
2. Assessment of exposure of mammals to COCs (Figure J.1-2).
3. Assessment of the effects of COCs on mammals (Figure J.1-3).
4. Characterization of risks to the omnivorous and carnivorous mammalian community (Figure J.1-4).

J.1.2 Conceptual Model

The conceptual model presented in Figure J.1-1 illustrates the exposure pathways for omnivorous and carnivorous mammals exposed to tPCBs and TEQ in the PSA.

Total PCBs and TEQ are persistent, hydrophobic, and highly lipophilic. Therefore, they are highly bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001). Small mammals, earthworms, and other invertebrates comprise the major dietary items for omnivorous mammals. Carnivorous mammals primarily feed on mammals, although fruits, birds, and invertebrates can supplement their diet. In summary, omnivorous and carnivorous mammals that reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally through diet as a result of trophic transfer. Other routes of exposure, considered to be less important to overall exposure, include inhalation, water consumption, and soil/sediment ingestion (Moore et al. 1999).

The problem formulation (see Section 2) identified the red fox (*Vulpes vulpes*) as the representative species for carnivorous mammals potentially exposed to tPCBs and TEQ from consumption of contaminated prey. The northern short-tailed shrew (*Blarina brevicauda*) was selected as the representative species for omnivorous mammals. Life history profiles for the red fox and short-tailed shrew are presented in text boxes. Additional life history information on these species is presented in Sections J.2.1.5 and J.2.1.6, respectively.

Life History of Red Fox

The red fox is a common dog-sized canine that occurs in many habitats throughout its range and is the most widely distributed carnivore in the world. In North America, the red fox is found throughout the United States and Canada, but not in the southeast coastal region, extreme southwest, parts of the central states, or the Pacific Coastal regions. The typical pelage color of fox is red and it can be identified by its characteristic bushy, white-tipped tail, pointed muzzle, and prominent ears.

Habitat - Occupies a variety of habitats, but preferred habitat is a matrix of forest, cropland, and pastureland, habitats common in the PSA. The availability of suitable prey as well as suitable den sites is also important. Prefer to locate dens in forested areas, but within a short distance of open areas and usually within 100 meters of a source of open water.

Home Range - Maintains territory throughout the year and is considered nonmigratory. Average home range for adults in Maine was 14.7 km^2 (range = 6.0-27.5 km^2), average home range in Ontario was 9 km^2 with a range of 5 to 20 km^2. Mean territory sizes reported in EPA (1993) ranged from 100 to 2,000 hectares (1 to 20 km^2). Adults traverse most of their territory on a routine basis, but focus activities around dens, preferred hunting areas, food supplies, and resting areas.

Dietary Habit - Diet varies throughout the year depending on food availability. Includes almost all available animals as prey, such as insects, fish, reptiles, amphibians, birds, small mammals, and carrion. Although typically identified as carnivores, can consume considerable amounts of plant materials, particularly in the summer and fall. Plant material in the diet includes berries, apples, and nuts.

Life History of Short-Tailed Shrew

The northern short-tailed shrew is a small energetic mouse-like animal with dark slate-colored pelage found throughout the northcentral and northeastern United States extending into southern Canada. It is easily identified as a shrew by its long pointed snout, small black eyes, concealed ears, and five toes on each foot. The northern short-tailed shrew has a short tail, which is approximately 20% of total animal length.

Habitat - Can be found in a variety of habitats, including wetlands and uplands, and are common in areas with abundant vegetative cover, occur in both forested and open habitats.

Home Range - Home range of 0.06 acre (0.024 ha) in central New York State. Other estimates of home range size vary from 0.25 to 0.5 acres (0.1 to 0.2 ha) in areas of low prey density in winter months during nonbreeding periods to 0.07 to 0.17 acres (0.03 to 0.07 ha) in areas of high prey density with a minimum of territory overlap. Do not seasonally migrate, remaining in home range.

Dietary Habits - Earthworms and insects comprise most of the diet, earthworms reported to be the most important item in the diet. Invertebrates in the diet are mainly obtained from the leaf litter layer, and consist of millipedes, insect larvae, spiders, slugs, snails, and other mollusks. Plant materials, including nuts, berries, roots, and fungi, and occasional small mammals are also a component of the diet.

The assessment endpoint is the survival, growth, and reproduction of omnivorous and carnivorous mammals in the Housatonic River PSA. The measurement endpoints used to evaluate the assessment endpoint include: (1) determining, by comparisons of modeled exposure to doses reported in the literature to cause adverse effects, the extent to which the concentrations of tPCBs and other COPCs ingested in the diet will cause adverse effects to the survival, growth, or reproduction of omnivorous and carnivorous mammals, and (2) determining, by conducting field surveys, the relationship between the concentrations of tPCBs and TEQ and survival, reproduction, and abundance of omnivorous and carnivorous mammals in the Housatonic River floodplain. As part of the EPA field survey, placental scars in small mammals were analyzed as an indication of past reproductive performance.

J.1.3 Exposure Assessment

The exposure assessment estimates the exposure of omnivorous and carnivorous mammals to tPCBs and TEQ in the Housatonic River PSA (Figure J.1-2), beginning with a description of the exposure model. The exposure model was parameterized using life history information on the representative species and concentrations of tPCBs and TEQ in prey collected in the PSA.

Monte Carlo and probability bounds analyses were conducted to estimate exposure for each COC.

J.1.4 Effects Assessment

The effects assessment provides an overview of the literature on the effects of tPCBs and TEQ to survival, growth, and reproduction of representative wildlife species (Figure J.1-3). An extensive literature search was conducted to locate studies with data and information on the survival, growth, and reproduction of the representative and surrogate wildlife species exposed to the COCs. Each study was evaluated using defined acceptability criteria. Studies were then selected and used to derive the most appropriate effects metric.

J.1.5 Risk Characterization

The risk characterization evaluates the likelihood that adverse effects may occur as a result of wildlife exposure to tPCBs and TEQ (Figure J.1-4). Three lines of evidence are available to characterize risks to omnivorous and carnivorous mammals from exposure to tPCBs and TEQ:

- **Modeled Exposure and Effects**—The purpose is to determine the extent to which the concentrations of tPCBs and TEQ ingested in the diet will cause adverse effects to the survival, growth, or reproduction of omnivorous and carnivorous mammals. A probabilistic food web model is used to estimate exposure of fox and shrews to tPCBs and TEQ. The total daily intakes for fox and shrews in the PSA are calculated using equations from the *Wildlife Exposure Factors Handbook* (EPA 1993) and related publications. These estimated exposures are compared to results of toxicological studies reported in the literature to determine if the mammalian species are being exposed to tPCBs and TEQ at concentrations likely to induce adverse effects.

- **Field Surveys (performed by EPA)**—The purpose of the survey was to determine the relationship between the concentrations of tPCBs and TEQ and the relative abundance of omnivorous and carnivorous mammals in the Housatonic River floodplain.

- **Population Demography Field Study (performed by GE)—**The objective of this study was to determine the demography of the short-tailed shrew living in six different locations in the Housatonic PSA on soil contaminated with a range of tPCB concentrations.

A weight-of-evidence assessment was conducted to collectively evaluate the results from each of these lines of evidence. The section concludes with a discussion of sources of uncertainty in

the assessment of risks of COCs to omnivorous and carnivorous mammals, and the conclusions of the risk characterization.

J.1.6 Organization

This appendix is organized as follows:

- Section J.2 describes the exposure model, input parameters, and uncertainty propagation techniques. Also presented in this section are the input data and exposure results for red fox and northern short-tailed shrew.
- Section J.3 describes the effects to mammals exposed to tPCBs and TEQ.
- Section J.4, risk characterization discusses the lines of evidence, the sources of uncertainty regarding risk estimates, and the conclusions regarding risk for omnivorous and carnivorous mammals exposed to tPCBs and TEQ in the Housatonic River.

J.2 EXPOSURE ASSESSMENT

This exposure assessment for omnivorous and carnivorous mammals focuses on the PSA. The representative species for omnivorous and carnivorous mammals are the northern short-tailed shrew and the red fox. These mammals occur in the PSA and feed on prey exposed directly to the COCs and through trophic transfer. Exposure of fox and shrews to tPCBs and TEQ was estimated using a total daily intake model adapted from Nagy (1987), Nagy et al. (1999) and the *Wildlife Exposure Factors Handbook* (EPA 1993). Section 6.4 of the ERA describes how TEQ were estimated.

This section begins with a description of the exposure model used for both of the representative species. Subsequent sections describe the inputs used in the exposure analyses for each representative species. The exposure assessment section concludes with a description of the Monte Carlo and probability bounds analyses conducted to estimate total daily intake of tPCBs and TEQ by each of the representative omnivorous and carnivorous mammal species in the Housatonic River PSA.

J.2.1 Exposure Model for Omnivorous and Carnivorous Mammals

The model used to estimate exposure of omnivorous and carnivorous mammals to tPCBs and TEQ focuses on ingestion of these contaminants through their diet. Other exposure routes (e.g., water ingestion) were considered to be much less important for tPCBs and TEQ. The model used in the exposure analysis was:

$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i$$

where

TDI	=	total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ)
FIR	=	normalized food intake rate (kg/kg bw/d)
FT	=	foraging time in PSA (unitless)
C_i	=	concentration in *i*th food item (mg/kg tPCBs, ng/kg TEQ)
P_i	=	proportion of the *i*th food item in the diet (unitless)

J.2.1.1 Selecting Exposure Distributions

Input distributions were generally assigned as follows: lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue), beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet), normal distributions for variables that were symmetric and not bounded by one (e.g., body weight), and point estimates for minor variables or variables with low coefficients of variation. In certain situations (e.g., poor fit to the data), other distributions were selected or other approaches were used. These latter situations are described in detail where they occurred in the following exposure analysis sections.

J.2.1.2 Incorporation of Spatial Averaging in Derivation of Estimated Prey Concentrations

Concentrations of tPCBs and TEQ vary spatially in prey. Many wildlife species forage over distances ranging from tens of meters to greater than 10 km. An individual integrates the spatial variation in tissue concentrations of their prey. Therefore, estimates of the central tendency were used in the exposure model as an expression of the spatial and temporal averaging of concentrations of COCs in prey tissue. In the probabilistic exposure analyses, it was assumed that the spatially and temporally averaged exposure estimate did not vary between individuals foraging in the same area. To account for uncertainty due to sample size, the measure of centrality in the Monte Carlo analysis was the minimum of: (1) the 95% upper confidence limit calculated using the Land H-statistic, or (2) the maximum measured concentration. The Land H-statistic has the underlying assumption that data are lognormally distributed. Testing of data for lognormality is discussed in Section 6.3. In the probability bounds analyses, however, the uncertainty regarding the arithmetic mean was accounted for with a different procedure. The procedure generally involved using the Land H-statistic to estimate the lower and upper 95% confidence limits on the mean (Gilbert 1987), and then using these lower and upper confidence limits to derive bounds on all possible distributions that exist within this range. This approach results in an expression of the uncertainty about the true value of the arithmetic mean that arises due to limited sample size. In cases where the 95% upper confidence limit could not be estimated or exceeded the maximum, other techniques were used to derive the bounds on the

mean. These techniques and the criteria for their use are described in Section 6.3 and Appendix C.5 of the ERA.

J.2.1.3 Techniques for Propagating Uncertainty

In this assessment, two types of probabilistic analysis were performed: Monte Carlo analysis and probability bounds analysis. The former is the most common probabilistic method employed in ecological risk assessments, and guidance for its use in Superfund assessments is available (EPA 1997). While Monte Carlo methods are appropriate for the determination of exposure risks when input distributions are known precisely, they do not adequately represent the effects of uncertainty around the input distributions (Ferson 1996). In many ecological risk assessments, the available data are limited, and consequently the input distributions used to calculate risks are uncertain. Probability bounds analysis is a tool for separating variability and uncertainty to obtain bounds on the result that explicitly account for the uncertainty about the input distributions. As in Monte Carlo analysis, the overall slopes of the bounds indicate how much variability exists in the system. The distance between the bounds, on the other hand, is an indication of the uncertainty that exists due to lack of knowledge. More detailed descriptions of Monte Carlo analysis and probability bounds analysis are presented in Section 6.5 and Appendix C.4.

For each Monte Carlo analysis, a sensitivity analysis was performed to determine the relative influence each input variable had on the output exposure distribution. This was done by determining the correlation between the input values for a particular variable randomly chosen during the simulation and the corresponding output exposure estimates. Input variables having a strong influence on the output exposure estimate tend to have high correlation coefficients, although this interpretation becomes problematic when there are dependencies between input variables. The results of the sensitivity analyses are included in this exposure assessment.

The exposure models for the ERA contain multiple variables, some of which may be correlated. The assumption of independence can be inappropriate, because even subtle dependencies can have substantial effects on the estimated exposure. If correlations are not accounted for, the variance and the tails of the exposure distribution may be poorly estimated. This assessment uses several approaches to address correlations between variables. These approaches include

simulation of observed correlations, assumption of perfect covariance (e.g., when the diet consists of two prey items, the proportion of one item in the diet is equal to one minus the other item), or no assumptions at all about dependencies (all possible relationships between two variables can occur). The specific approach used depends on the type of data and the application. In cases where independence of variables seemed intuitively obvious (e.g., COC concentration in the prey item and proportion of that item in the diet), independence was assumed.

J.2.1.4 Treatment of Non-Detects

The approach for generating summary statistics or distributions when a data set includes samples with COC concentrations below the detection limit is described in detail in Appendix C.2. In summary, for data where contaminants were not detected, summary statistics were calculated assuming that the contaminant concentrations were zero (ND = 0), and assuming that the contaminant concentrations were equal to the detection limit (ND = DL). If the ratio between the statistic of interest (e.g., the mean) calculated assuming ND = DL and the statistic assuming ND = 0 was less than or equal to 1.3, the analysis was performed assuming that all non-detected chemical concentrations were equal to half the detection limit (ND = 1/2 DL)[1]. If the ratio was greater than 1.3 and sample size and detection frequency were adequate (i.e., n>5, detection frequency >25%), the probability plot method described in Appendix C.2 was used to generate estimated concentrations for samples with COC concentrations below the detection limit. If sample size and detection frequency were not adequate, then the exposure calculations were done assuming ND = 0, ND = 1/2 DL, and ND = DL (Figure 6.4-2).

J.2.1.5 Red Fox

Red fox were selected as one of the representative species for omnivorous and carnivorous mammals because the habitat within the PSA is suitable for fox, they breed in the Housatonic River watershed, and the prey they consume are directly exposed to COCs in the area. Therefore, fox in the PSA could ingest tPCBs and TEQ through their diet. Typical dietary items

[1] This decision criterion supplements the procedures described in Appendix C.2.

for red fox are listed in Table J.2-1. Fox were observed relatively uniformly throughout the PSA from 1998 to 2001 (Appendix A). Fox, or their tracks, scat, or other sign were observed in the study area along the shoreline of the river at point bars and beaches, on mud flats, in hardwood and tamarack swamps, transitional floodplain forest, and high-terrace floodplain forest. Altered habitats, such as agricultural fields and cultural grasslands, are also used by red fox (see Appendix A).

J.2.1.5.1 Exposure Model Input Distributions

The input variable parameterizations used in the Monte Carlo and probability bounds analyses to estimate exposure of red fox to tPCBs and TEQ are summarized in Tables J.2-2, J.2-3, J.2-4, and J.2-5. Additional information on each of the input variables is provided below.

Body Weight (BW)

Body weight is not used in the model directly, but is a required variable in allometric models (e.g., Nagy 1987) to estimate food intake or free metabolic rates. As with many mammalian species, the red fox exhibits sexual dimorphism in body size. Males are typically 10% larger and 20% to 30% heavier than females (Storm et al. 1976; Lariviere and Pasitschniak-Arts 1996; Voigt 1987). In a study conducted in Indiana, males weighed an average of 4.9 kg and females weighed an average of 4.0 kg (Lariviere and Pasitschniak-Arts 1996). Voigt (1987) found that male red fox in Ontario averaged 4.1 kg in weight (n = 37) and females averaged 3.4 kg in weight (n = 37).

In the Monte Carlo analyses, female body weight was assumed to be normally distributed, with a mean of 3.87 kg and a standard deviation of 0.322. Females were selected because the effects endpoints are associated with reproductive failure. The uncertainty in this variable is due to natural variability, rather than lack of knowledge or data (i.e., the variable is easily measured and many studies have been conducted that measured this variable). Accordingly, the same distribution was used in the probability bounds analysis for body weight of red fox.

Food Intake Rate (FIR)

The food intake rate of red fox has been measured in laboratory and captive animals (Sargeant 1978). Because the animals were captive or kept in a laboratory, the measured food intake rates likely underestimated food intake rates of free-living fox (EPA 1993). Free-living fox, unlike captive fox, expend energy foraging for prey, avoiding predators, defending territories, etc. As a result, an allometric modeling approach, described below, was used to estimate food intake rate for red fox rather than the rates measured in the controlled study.

Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating free metabolic rate (*FMR*) of free-living mammals in kilojoules (kJ) per day, using the following general equation:

$$FMR\ (kJ/d) = a \cdot BW(g)^b$$

For both the Monte Carlo and the probability bounds analyses, the free metabolic rate (*FMR*) for fox was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (body weight [*BW*], *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999), assuming an underlying normal and lognormal distribution, respectively. A lognormal distribution was used for the power term (*b*) because the standard error was large and would have generated negative *FMR* values had a normal distribution been assumed. For carnivores, *log a* had a reported mean of 1.67 and a standard error of 2.65 in $\log_{10}$ units, and *b* had a reported mean of 0.869 and a standard error of 0.116 (Nagy et al. 1999). The body weight (*BW*) distribution was described above. The results of the calculation were then converted to kcal/kg bw/d.

Food intake rate (*FIR*) is derived from *FMR* using the following equation:

$$FIR\ (kg/kg\ bw/d) = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$

where:

FMR = normalized free metabolic rate (kcal/kg bw/d)

AE_i = assimilation efficiency of *i*th food item (unitless)

GE_i = gross energy of *i*th food item (kcal/kg)

The gross energies of various wildlife food sources are summarized in the *Wildlife Exposure Factors Handbook* (EPA 1993). The mean gross energy of mammals is 1,700 kcal/kg (SD = 280) (Koplin et al. 1980). The assimilation efficiency for mammals consumed by mammals is 84% (SD = 6.5%) (Castro et al. 1989). Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation.

Proportions of Dietary Items (P_i)

The red fox can occupy a variety of habitats in the PSA and may use a variety of food sources. Available studies reporting the dietary composition of the red fox in North America show that the proportion of dietary items varies according to season (Table J.2-1). Most studies found that mammals constitute the majority of the diet of the red fox, with the percentage in the diet as high as 92% in the spring (Knable 1974). The average proportion of small mammals in the diet of fox was relatively constant across seasons, with the lowest average in the summer when fruits and nuts are available to supplement the diet. Mammals represent approximately 76% of the average diet for all seasons. Other food items including birds, invertebrates, and vegetation were not included as part of the exposure model because the dietary items represent a relatively small portion of the diet (e.g., birds and invertebrates) or the contribution to the overall exposure is negligible (e.g., vegetation).

Proportions of prey items in the diet were assumed to follow a beta distribution. For this assessment, the estimated average dietary proportion for mammals was 76.0% (alpha = 26, beta = 5.7, scale = 0.92).

The beta distribution is not an available option in RiskCalc, the software used for conducting the probability bounds analyses. The minimum, mean, and maximum values were instead included as a distribution-free statement in RiskCalc for each dietary item. The results are bounds that include all possible distributions, given the specified minimum, mean, and maximum values specified for the dietary items.

Foraging Time (FT)

Adult red fox visit all parts of their territory on a regular basis (Ables 1974). The home range used in this assessment is 9 km^2 (Voigt and Tinline 1980). The width of the floodplain for Reach 5 ranges from 200 to 600 m. Therefore, the red fox is expected to spatially and temporally average exposure inside and outside the PSA within its home range, potentially experiencing areas of high contamination along with areas of low or no contamination. As a result, the PSA represents only a portion of a potential red fox home range. Based on the difference between the total home range area for fox and the area of the PSA found within the fox's home range, the average spatial foraging time for red fox in the PSA was estimated to be approximately 10%. However, red fox are likely to spend more of their foraging time closer to the river, within the PSA, because of the availability of food and water in that area. The ecological characterization indicated that red fox were more frequently observed in wetland habitats, including shorelines and palustrine forested areas (Appendix A). As a result, for the purposes of modeling COC exposure, it was assumed that red fox spend 50% of their time foraging in the PSA. The foraging time was specified as a point estimate.

J.2.1.5.2 Exposure Concentrations

Concentration of tPCBs in Mammals in Reach 5 (C_i)

Concentrations of tPCBs were measured in 76 mammal samples collected in Reach 5. The Land H-statistic was used to determine the lower and upper 95% confidence limits (CL) on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 80.6 mg/kg. For the probability bounds analysis, the lower and upper 95% confidence limits, 23.1 and 80.6 mg/kg, respectively, determined from the Land H-statistic were used to parameterize a distribution-free statement in RiskCalc (Table J.2-4).

Concentration of TEQ in Mammals in Reach 5 (C_i)

TEQ concentrations were measured in 12 mammal samples collected in Reach 5. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration.

Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and those that co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat non-detected congeners in the TEQ calculation also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean of ND = DL/2 data. The lower of the upper 95% CL and the maximum value was used as a point estimate in the Monte Carlo analysis. In this analysis, the maximum value was the lower of the two, with a value of 4,038 ng/kg (Table J.2-5).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values, assuming that non-detected congeners were equal to half the DL, were calculated using the greater of the lower 95% CL and the minimum value and the lower of the upper 95% CL and the maximum value, respectively. In this data set, the minimum and maximum values were used, and the resulting values ranged from 14.7 to 4,038 ng/kg (Table J.2-5).

J.2.1.5.3 Exposure Model Results

Exposure to tPCBs in Reach 5

The Monte Carlo analysis indicated that exposure of red fox to tPCBs could range from a minimum of 0.0220 to a maximum of 82.5 mg/kg bw/d. The mean exposure was 6.25 mg/kg bw/d, and the median exposure 2.68 mg/kg bw/d (Table J.2-6). Ninety percent of the exposure estimates were between 0.321 and 25.0 mg/kg bw/d. Figure J.2-1 depicts the cumulative distribution of tPCB intake rates for red fox in Reach 5.

Sensitivity analysis (Table J.2-7) revealed that the *FMR* slope term used in the free metabolic rate equation was the most important variable (Pearson correlation coefficient (r_p) = 0.698) followed by the *FMR* power term (r_p = 0.673) and the proportion of mammals in the diet (r_p = 0.0495). Discussion of the significance of *FMR* slope term and *FMR* power term is provided in

Section 6. The probability bounds estimated for red fox foraging in Reach 5 are shown in Figure J.2-1. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 0.106 and 0.702 mg/kg bw/d. The 50th percentile ranged between 0.607 and 3.54 mg/kg bw/d, and the 90th percentile ranged between 3.52 and 23.5 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 0.434, the 50th percentile was 2.68, and the 90th percentile was 16.7 mg/kg bw/d (Table J.2-6).

Exposure to TEQ in Reach 5

The Monte Carlo analysis indicated that exposure of red fox to TEQ could range from a minimum of 1.40 to a maximum of 3,089 ng/kg bw/d. The mean exposure was 296 ng/kg bw/d and the median exposure 137 ng/kg bw/d (Table J.2-8). Ninety percent of the exposure estimates were between 15.8 and 1,163 ng/kg bw/d. Figure J.2-2 depicts the cumulative distribution of TEQ intake rates for red fox in Reach 5.

Sensitivity analysis demonstrated that the *FMR* slope term and power terms were the most important parameters for the Monte Carlo analysis (Table J.2-7). Discussion of the significance of *FMR* slope term and *FMR* power term is provided in Section 6.

The probability bounds estimated for TEQ exposure to red fox foraging in Reach 5 are shown in Figure J.2-2. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 0.0672 and 35.2 ng/kg bw/d. The 50th percentile ranged between 0.386 and 177 ng/kg bw/d, and the 90th percentile ranged between 2.24 and 1,175 ng/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 16.8, the 50th percentile was 137, and the 90th percentile was 829 ng/kg bw/d (Table J.2-8).

J.2.1.6 Short-Tailed Shrew

The northern short-tailed shrew was selected as a representative species for omnivorous and carnivorous mammals because the habitat within the study area is suitable for shrews, they breed in the Housatonic River watershed, their foraging range is small, and the prey they consume are directly exposed to COCs in the area. Therefore, shrews in the PSA could ingest tPCBs and TEQ through the dietary exposure route. Typical dietary items for northern short-tailed shrews

are listed in Table J.2-9. The short-tailed shrew was the most common of the shrews observed in the PSA (see Appendix A) and was found in shrub and forested habitats, from the confluence downstream to the backwater areas of Woods Pond. In wetland habitats, short-tailed shrews were found in transitional floodplain forests, red maple swamps, black ash–red maple–tamarack calcareous seepage swamps, and high-terrace floodplain forests.

Description of Sampling Locations 13, 14, and 15

Location 13 is a relatively flat area located on the west shore of the river, in the floodplain adjacent to river mile 133.2, situated at an elevation of 965 ft (294 m). The community type is transitional floodplain forest that is flooded seasonally and is moderately well drained, with extensive vegetation cover (80%) and alluvial silt-loam soil. The PCB concentrations in floodplain soil averaged 55.2 mg/kg dw.

Location 14 is a relatively flat low-lying area located on the west shore of the river, in the floodplain adjacent to river mile 129.9, situated at an elevation of 965 ft (294 m). The community type is transitional floodplain forest that is flooded seasonally with extensive vegetation cover (70%) and fluvial silt soil. The PCB concentrations in floodplain soil averaged 26.1 mg/kg dw.

Location 15 is a flat area located on the west shore of the river, in the floodplain adjacent to river mile 126.7, situated at an elevation of 965 ft (294 m). Community types are circumneutral hardwood swamp and transitional floodplain forest that are flooded seasonally. This site has 60% vegetation cover, 40% leaf litter cover and a primarily mineral soil. The PCB concentrations in floodplain soil averaged 0.484 mg/kg dw.

The majority of the soil in the shrew's diet is the result of consuming earthworms (DeGraaf and Yamasaki 2001; Mumford and Whitaker 1982). Earthworms contain a high percentage of soil in their gastrointestinal tract and shrews consume earthworms whole. The acid-insoluble ash content of the earthworms *Lumbricus rubellus* and *Eisenoides lonnbergi*, reported in Beyer et al. (1994), was 13% and 23%, respectively. Consequently, direct soil ingestion was not considered as a separate variable in the exposure model for shrews because the chemical analyses were conducted on earthworms that had not been depurated.

J.2.1.6.1 Exposure Model Input Distributions

The parameterization of the input variables used in the Monte Carlo and probability bounds analyses to estimate exposure of short-tailed shrew to tPCBs and TEQ is summarized in Tables J.2-10, J.2-11, J.2-12, and J.2-13. Exposure analyses were conducted for three locations in the

PSA, Locations 13, 14, and 15 (Figure J.2-3). These locations were selected to provide a range of tPCB and TEQ concentrations.

Body Weight (BW)

The northern short-tailed shrew can weigh over 22 grams (George et al. 1986; Burt and Grossenheider 1980, as cited in EPA 1993). Whitaker and Hamilton (1998) reported mean total length and weight for adult males and females of 124 mm (n = 60) and 19.3 g (n = 50). As part of the ecological characterization of the PSA (Appendix A), 58 adult short-tailed shrews of both sexes were caught during small mammal trapping in 1998 to 2001. The total lengths were 104 to 150 mm (mean = 123 mm), tail lengths were 21 to 34 mm (mean = 25 mm), and weights were 15 to 27 g (mean = 21.9 g, SD = 2.76 g). The average weight of adult female shrews was 22.3 g (SD = 2.87 g). Total lengths, tail lengths, and weights of six juveniles were 102 to 122 mm (mean = 111 mm), 23 to 30 (mean = 25 mm), and 11 to 15 g (mean = 14 g), respectively.

In the Monte Carlo analyses, female body weight was assumed to be normally distributed with a mean of 22.3 g and a standard deviation of 2.87. Females were selected because the effects endpoints are associated with reproductive failure. The uncertainty in this variable is due to natural variability, rather than lack of knowledge or data (i.e., the variable is easily measured). Accordingly, the same distribution was used in the probability bounds analyses for body weight of short-tailed shrews.

Food Intake Rate (FIR)

Food intake rate of the northern short-tailed shrew was measured in laboratory animals (Barrett and Stuek 1976; Morrison et al. 1957). Because the animals were kept in a laboratory, the measured food intake rates likely underestimated food intake rates of free-living shrews (EPA 1993). Free-living shrews, unlike captive shrews, expend energy foraging for prey, avoiding predators, defending territories, etc. As a result, an allometric modeling approach, described below, was used to estimate food intake rate for short-tailed shrew rather than the rates reported from the captive study.

Nagy (1987) and Nagy (1999) derived allometric equations for estimating the free metabolic rate (*FMR*) of free-living mammals in kilojoules (kJ) per day using the following general equation:

$$FMR\ (kJ/d) = a \cdot BW(g)^b$$

For both the Monte Carlo and the probability bounds analyses, *FMR* for shrew was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (body weight [*BW*], *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999), assuming an underlying normal distribution for each. For omnivorous mammals, log *a* had a reported mean of 6.98 and a standard error of 1.32 in $\log_{10}$ units, and *b* had a reported mean of 0.622 and a standard error of 0.0630 (Nagy et al. 1999). The body weight (*BW*) distribution was described above. The results of the calculation were then converted to kcal/kg bw/d.

Food intake rate (*FIR*) is derived from *FMR* using the following equation:

$$FIR\ (kg/kg\ bw/d) = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$

where:

FMR	=	normalized free metabolic rate (kcal/kg bw/d)
AE_i	=	assimilation efficiency of *i*th food item (unitless)
GE_i	=	gross energy of *i*th food item (kcal/kg)

The gross energies of various wildlife food sources have been summarized in the *Wildlife Exposure Factors Handbook* (EPA 1993). The gross energy of earthworms ranges from 780 to 830 kcal/kg (mean = 805; SD = 141) (Cummins and Wuycheck 1971; Thayer et al. 1973). The mean gross energy for grasshoppers and crickets is 1,700 kcal/kg (SD = 260) (Cummins and Wuycheck 1971; Collopy 1975; Bell 1990), and for adult beetles, the mean is 1,500 kcal/kg (Cummins and Wuycheck 1971; Collopy 1975; Bell 1990). Grasshoppers, crickets, and beetles were used as representatives of litter invertebrates; their mean gross energy is 1,600 kcal/kg. Mammals have a gross energy of 1,700 kcal/kg (SD = 280) (Koplin et al. 1980).

The assimilation efficiency of earthworms consumed by mammals is not known. The mean assimilation efficiency for insects consumed by small mammals is 87% (Bryant and Bryant

1988). This value was used to represent the assimilation efficiency for earthworms consumed by mammals. For the consumption of mammals, the mean assimilation efficiency is 84% (Castro et al. 1989). Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation (i.e., CV<10%).

Proportion of Dietary Items (P_i)

Table J.2-9 lists the main food items and their proportion in the diet of the short-tailed shrew for winter and summer. As with the red fox, there is variation in the proportion of dietary items reported for the short-tailed shrew. Composition of the diet of the short-tailed shrew does not differ substantially from winter to summer and consists mainly of earthworms, leaf litter invertebrates (including arthropods and gastropods), small mammals, and plants. Available dietary studies for shrews indicate that the average proportion of food items in winter and summer is comparable for earthworms, but that consumption of small mammals (i.e., other shrews, voles, mice) and insects/gastropods increases in the winter, while plant consumption decreases. Earthworms comprised between 5 and 31% of the diet of short-tailed shrew, whereas insects and small mammals were reported as high as 61% and 24% of the diet, respectively (Hamilton 1941, as cited in EPA 1993; Linzey and Linzey 1973; Eadie 1944).

Averaging the available data for the winter and summer diets of the short-tailed shrew indicates that earthworms, other invertebrates (all combined), and small mammals comprise the major dietary items for the short-tailed shrew. The proportion of a particular prey item in the diet was assumed to follow a beta distribution. For this exposure assessment, the diet of the shrew was on average 19% for earthworms (alpha = 12.0, beta = 4.0, scale = 0.25), 60% for other invertebrates (alpha = 12, beta = 5.2, scale = 0.85), and 12% for small mammals (alpha = 9.0, beta = 8.5, scale = 0.235). To ensure that total diet summed to 1.0 in each iteration of the Monte Carlo analyses, the proportion of each particular dietary item was normalized using the following equation:

$$P_i' = \frac{P_i}{\sum_{i=1}^{3} P_i}$$

Foraging Time (FT)

The foraging range for northern short-tailed shrew is small. Whitaker and Hamilton (1998) and Degraaf and Yamasaki (2001) found that the northern short-tailed shrew had a home range size of 0.06 acre (0.024 ha), while Platt (1976) reported that the home range size varies from 0.25 to 0.5 acres (0.1 to 0.2 ha) in areas of low prey density. Because short-tailed shrews have a small foraging range, the exposure analysis was performed for three locations in the PSA (Locations 13, 14, and 15) where earthworms, insects, and mammals were sampled. The sizes of Locations 13, 14, and 15 are approximately 2 to 3 acres. Therefore, shrews are expected to have 100% of their foraging range within each of Locations 13, 14, and 15 in the PSA.

J.2.1.6.2 Exposure Concentrations

Concentration of tPCBs in Earthworms at Location 13 (C_i)—Concentrations of tPCBs were measured in 10 earthworm composite samples collected at Location 13. The Land H-statistic was used to determine the lower and upper 95% confidence limits on the mean. The lower of the upper 95% CL and the maximum measured concentration was used as a point estimate in the Monte Carlo analysis. In this analysis, the upper 95% CL was the lower of the two, with a value of 24.2 mg/kg. For the probability bounds analysis, the lower and upper 95% confidence limits, 18.0 and 24.2 mg/kg, respectively, determined from the Land H-statistic were used to parameterize a distribution-free statement in RiskCalc**.**

Concentration of TEQ in Earthworms at Location 13 (C_i)—TEQ concentration was measured in one composite earthworm sample (n = 45) collected at Location 13. Using the decision criteria outlined in Section 6.4, the assumption of how to treat co-eluted congeners in the TEQ calculation did have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 0% and 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations; this data set was used to calculate distribution parameters for the Monte Carlo analysis and the probability bounds analyses. The assumption of how to treat non-detected congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, a triangular distribution assuming that non-detected congener

concentrations were equal to half the DL was used in the Monte Carlo analysis to capture the uncertainty on the mean. The minimum and maximum values in the triangular distribution were calculated assuming that PCB-123 and PCB-157 had zero concentration and assuming concentrations of PCB-123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. The best estimate value was the average of the minimum and maximum values. The resulting triangular distribution parameters were minimum = 207, best estimate = 361, and maximum = 516 ng/kg.

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum and maximum values, assuming that non-detected congener concentrations were equal to half the DL, were those calculated above (i.e., 207 to 516 ng/kg).

Concentrations of tPCBs and TEQ in Other Prey Items

Concentrations in other prey items are presented in Tables J.2-12 and J.2-13.

J.2.1.6.3 Exposure Model Results

Exposure to tPCBs at Location 13

The Monte Carlo analysis indicated that exposure of short-tailed shrew to tPCBs could range from a minimum of 3.11 to a maximum of 25.5 mg/kg bw/d. The mean exposure was 12.2 mg/kg bw/d, and the median exposure 11.5 mg/kg bw/d (Table J.2-14). Ninety percent of exposure estimates were between 6.23 and 20.6 mg/kg bw/d. Figure J.2-4 depicts the cumulative distribution of tPCB intake rates for short-tailed shrew at Location 13.

Sensitivity analysis (Table J.2-15) revealed that the *FMR* slope term used in the free metabolic rate equation was the most important variable (Pearson correlation coefficient (r_p) = 0.701) followed by the *FMR* power term (r_p = 0.475) and the proportion of other invertebrates in the diet (r_p = -0.401). Discussion of the significance of *FMR* slope term and *FMR* power term is provided in Section 6.

The probability bounds estimated for short-tailed shrew foraging at Location 13 are depicted in Figure J.2-4. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 2.79 and 15.0 mg/kg bw/d. The 50th percentile ranged between 5.07 and 23.4 mg/kg bw/d, and the 90th percentile ranged between 9.25 and 37.8 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 7.11, the 50th percentile was 11.5, and the 90th percentile was 18.9 mg/kg bw/d (Table J.2-14).

Exposure to Other COC/Location Combinations

Exposures of short-tailed shrew to tPCBs at Locations 14 and 15 were lower than at Location 13, having mean total daily intakes of 9.94 and 1.34 mg/kg bw/d, respectively. Thus, exposure of shrews to tPCBs varies by approximately an order of magnitude at different sites in the PSA. The uncertainty of these exposure estimates, as illustrated by the probability bounds distributions, indicates a similar degree of uncertainty for all three locations (Figures J.2-4 to J.2-6).

Mean exposures of short-tailed shrew to TEQ at Locations 13, 14, and 15 were 288, 143, and 46.1 ng/kg bw/d, respectively. Figures J.2-7 to J.2-9 depict the cumulative distributions for total daily intake of TEQ, as well as their respective probability bounds at the three sites. For each exposure scenario, sensitivity analysis revealed that the *FMR* slope was the most important variable in the Monte Carlo analysis. The exposure model results for the exposure of short-tailed shrew to TEQ in the PSA are presented in Table J.2-16 and the sensitivity analyses are presented in Table J.2-15.

J.3 EFFECTS ASSESSMENT

Studies on the effects of COCs to wildlife were considered in the development of effects metrics if sufficient information was provided to permit evaluation of study design, study execution, chemical analysis methods, statistical analyses, and other key aspects of the study. Those studies with major issues (e.g., incomplete explanation of statistical techniques) were included in the review of the effects literature that follows, but were not considered in the derivation of the effects metrics that will be used in the risk characterization. Further details on the selection criteria for the effects metrics are provided in Section 6.6 of the ERA.

Exposure of mammals to tPCBs and 2,3,7,8-TCDD (TEQ) causes a range of effects including hormone induction (French et al. 2001; Bjerke and Peterson 1994; Bjerke et al. 1994; Mably et al. 1992b), decreases in body and organ weight (Murray et al. 1979; Gray and Ostby 1995; Mably et al. 1992b; Huuskonen et al. 1994), reduced fertility (Gray et al. 1995; Khera and Ruddick 1973; Murray et al. 1979; Gray and Ostby 1995), reduced litter size or survival at birth or weaning (Murray et al. 1979; Giavini et al. 1983; Khera and Ruddick 1973; Bjerke et al. 1994; Mably et al. 1992b; Thomas and Hinsdill 1979), and mortality (Green et al. 1975; Kimbrough et al. 1972; Pohjanvirta et al. 1993; Stahl et al. 1992). This section focuses on effects that have an influence on the long-term maintenance of mammal populations (i.e., mortality, or impairment of reproduction or growth). Studies involving multiple exposure treatments that employed statistical evaluations to determine whether treatment results were different from controls were preferred.

All toxicity estimates presented in this section are reported as daily doses normalized to a body weight of 1.0 kg (mg/kg bw/d for tPCBs and ng/kg bw/d for TEQ, daily dose levels normalized to the body weight of the test animals). When calculating a daily dose, measured body weights and food intake rates of the test animals were preferred. However, when such data were not available, the daily food intake rates and body weights presented in Table J.3-1 were used to convert dietary concentrations to daily doses.

J.3.1 Contaminants of Concern

J.3.1.1 Total PCBs

A summary of mortality and reproduction effects to mammals is presented in Figure J.3-1 and in Table J.3-2.

J.3.1.1.1 Mortality

In an acute multiple dose study, a dose of 300 mg/kg bw/d of Aroclor 1254 for 5 days caused 50% mortality and a mean weight loss of 0.056 kg in Osborne-Mendel male rats (Green et al. 1975).

Oral LD_{50}s for 3- to 4-week-old Sherman-strain male rats exposed to Aroclors 1254 and 1260 were 1,295 and 1,315 mg/kg bw/d, respectively (Linder et al. 1974). Under similar test conditions, groups of 10 female Sherman-strain rats were treated with Aroclor 1260 doses of 7.2, 38.2, and 72.4 mg/kg bw/d for 8 months (Kimbrough et al. 1972). During this time period, one, two, and eight females died, respectively. No mortality was observed in controls and male rats. Bruckner et al. (1973) estimated a 14-day LD_{50} of 4,250 mg/kg bw/d for Aroclor 1242 for rats. The toxicity of a single dose of Aroclor 1254 to Wistar male and female rats at 30, 60, and 120 days of age was investigated by Grant and Phillips (1974). The LD_{50}s ranged from 1,300 to 2,500 mg/kg bw/d.

J.3.1.1.2 Reproduction

Many of the available studies focus on determining effects to offspring of mammals following in utero and/or lactational exposure to tPCBs. Impaired reproductive performance as a result of maternal PCB exposure has been reported for many mammals, including rats and mice. In general, females are administered contaminants by gavage or in the diet prior to or during gestation. Endpoints studied included pre- and post-natal survival and development, fertility, and other effects (Linder et al. 1974; Brezner et al. 1984; Overmann et al. 1987; Linzey 1987, 1988; Masuda et al. 1979; Allen and Barsotti 1976; Bleavins et al. 1981).

Most mammal reproduction studies have been performed on females. There is limited evidence for reproductive effects of tPCBs on male adult animals (Faqi et al. 1998; Smits-van Prooije et al. 1993), although it is well documented that gestational exposure to PCB mixtures and specific congeners can adversely affect morphology and production of sperm and fertility in male offspring of rats and mice (Gray 1993; Sager 1983; Sager et al. 1987, 1994; Huang et al. 1998a).

Aroclor 1254

A two-generation reproduction study was performed in which groups of 20 female and 10 male Sherman rats were exposed to diets of Aroclor 1254 at doses of 0.06, 0.32, 1.5, and 7.6 mg/kg bw/d (Linder et al. 1974). Exposure times ranged from 62 to 274 days. Because exposure schedules were staggered, controls were used each time one or more treated groups were started on the dietary regimen. The F0 rats were started on the diets at 3 to 4 weeks of age and the F1b rats at weaning. Exposure was continuous through mating, gestation, and lactation until the rats were euthanized. The F0 rats were mated when they reached 3 and 7 months old to produce the F1a and F1b generations, respectively. The F1b rats were selected at weaning for mating when 3 months old to produce the F2a generation. The F1b rats were mated a second time when 8 months old to produce the F2b generation.

Exposure to Aroclor 1254 caused significantly reduced litter sizes (13.7%; $p<0.05$) at 7.6 mg/kg bw/d in the F1a generation and at 1.5 mg/kg bw/d in the F1b (15.4%; $p<0.05$), F2a (15.2%; $p<0.05$) and F2b (24.4%; $p<0.005$) generations. Survival to weaning was not affected in the F1a offspring; however, only 73.6% of the 7.6 mg/kg bw/d F1b pups survived to weaning (this latter effect was not statistically significant). In the F2a generation, 77.8% ($p<0.005$) survived to weaning at 7.6 mg/kg bw/d ($p<0.05$). A marked decrease in mating performance was observed in the 7.6 mg/kg bw/d treatment, where only seven and four litters were born in the F2a and F2b generations, respectively. No effects on litter size were found in either generation of rats fed 0.32 mg/kg bw/d or 0.06 mg/kg bw/d of Aroclor 1254. Although reproduction was not affected at lower dietary concentrations of tPCBs, a significant increase in liver weight in 21-day-old weanlings was found at all concentrations of exposure.

Spencer (1982) investigated reproductive effects of Aroclor 1254 using eight treatment concentrations on Sprague Dawley strain rats fed treated diets on days 6 through 15 of gestation.

Statistically significant reductions were observed in fetal weight at birth (11.8%; $p<0.05$) and fetal survival (28%; $p<0.05$) for rats fed diets of Aroclor 1254 at 7.47 and 17.05 mg/kg bw/d, respectively. Overmann et al. (1987) found that Wistar rats whose mothers were exposed to Aroclor 1254 at 2.83 mg/kg bw/d were significantly lighter (p value not provided) compared to controls on days 14 and 21. Similarly, Brezner et al. (1984) and Sager and Girard (1994) observed that rats fed Aroclor 1254 had reduced weight gain compared to controls. In contrast to these studies, no effects were observed on birth weight or body weight growth in other studies on mice and rats (Shiota 1976; Curley et al. 1973; Baker et al. 1977).

Reproductive success was impaired in white-footed mice (*Peromyscus leucopus*) exposed to a chronic dose of 1.55 mg/kg bw/d of Aroclor 1254. PCB-exposed laboratory mice had statistically significant ($p<0.05$) longer intervals between births, smaller litter sizes at birth (25%, averaging one less individual), and smaller litter sizes at weaning (control = 98% survival; PCB treatment = 56% survival) (Linzey 1987). The offspring were used in a second-generation study (Linzey 1988). The reproductive success of the second-generation PCB-treated mice was reduced compared to the parental generation. For example, 42% of the PCB-treated pairs in the parental generation produced litters, whereas only 4% of the PCB-treated pairs in the second generation produced litters. In addition, only 47% of the second-generation mice survived to weaning compared to 56% in the parental generation. The second-generation PCB-treated mice exhibited poor growth of reproductive organs, with the females being most affected. The data in the second-generation study were too few to test for statistical significance. However, these results suggest that effects of chronic exposure to tPCBs are cumulative through generations.

This result was supported by McCoy et al. (1995) who maintained oldfield mice over three generations (P1, F1, and F2 generations) on an Aroclor 1254 dose of 0.678 mg/kg bw/d to investigate effects on fertility, growth, and survival. Mean birth weight (13% decrease) in offspring and weaning weight (13% decrease) of first generation were significantly lower ($p<0.05$ and $p<0.01$, respectively) than that of the P1 control. Greater differences between PCB-exposed and control mice occurred in the second generation, including lower mean birth weight (14.8% decrease, $p<0.01$) and lower weaning weight (15% decrease, statistical testing not possible). The percentage survival to weaning was 23% for the second generation, compared to 55% for the first generation. Whole body tissue concentrations of Aroclor 1254 with each

generation of exposure increased significantly ($p<0.01$ and $p<0.05$ for second and third generations, respectively).

Aroclor 1254 was fed at a dietary concentration of 30.9 mg/kg bw/d to pairs of white-footed mice for 60 days, during which time litters were produced. The controls had a litter production rate of 84.6%, whereas the PCB-treated group had a litter production rate of only 29.6% (Merson and Kirkpatrick 1976). No offspring of the PCB-treated group survived beyond 21 days.

Aroclor 1260

A two-generation reproduction study was performed in which groups of 20 female and 10 male Sherman rats were exposed to diets of Aroclor 1260 at doses of 0.39, 1.5, and 7.4 mg/kg bw/d (Linder et al. 1974). Exposure ranged from 68 to 188 days. No effects on litter size were found in either generation of rats fed >0.390 mg/kg bw/d of Aroclor 1260. An increase in liver-to-body weight ratios was observed at all dietary concentrations in F1b males between 5 and 7 months old, but only at 7.4 mg/kg bw/d in the F1b females. In the same study, there was no significant difference from controls for number of litters born and survival at weaning. In a one-generation reproduction study using a higher dose of 35.4 mg/kg bw/d, Linder et al. (1974) observed a significant ($p<0.005$) decrease in litter size at birth.

J.3.1.1.3 Growth

Sprague Dawley rats were administered by oral intubation doses of Aroclor 1254 of 50 and 500 mg/kg bw/d (Komives 1979). A significant ($p<0.05$) decrease in body weight and food intake was observed in rats given the dose of 50 mg/kg bw/d after 11 days and 4 days of exposure, respectively. At 500 mg/kg bw/d, a significant ($p<0.05$) decrease in body weight was observed at 5 days of exposure. A significant decrease in body weight of male rats was also observed by Grant et al. (1974) when rats were fed a dose of 0.139 mg/kg bw/d of Aroclor 1254 after 60 days of exposure. Body weight gain was inhibited in male and female Sprague Dawley rats at a dose of 28.5 mg/kg bw/d and 8.2 mg/kg bw/d after a 39-day exposure (Kerkvliet and Kimeldorf 1977).

J.3.1.1.4 Other Effects

Prolonged estrous cycles have been observed in mice fed 0.8 mg/kg bw/d of Clophen A60 (PCB mixture containing about 59% chlorine) for a 10-week period (Orberg and Kihlstrom 1973) and in rats fed 10 mg/kg bw/d of Aroclor 1254 for 6 weeks (Brezner et al. 1984). Decreased implantation and embryonic survival in rats are associated with a prolonged estrous cycle (Butcher and Pope 1979).

Jonsson et al. (1976) reported reduced circulating progesterone levels and distinctive histological change in ovarian stromal cells in rats fed 3.7 and 7.5 mg/kg bw/d of Aroclor 1242 for 36 weeks, although estrous cycle length was not affected. In the same study, there were fewer matings after exposure of males and females to 7.5 mg/kg bw/d compared to controls. Aroclor 1242, administered in the diet at 12 mg/kg bw/d on alternative days for 3 weeks, significantly altered the weights of livers in rats (Bruckner et al. 1973). Liver weights were also significantly different for rats exposed to Aroclor 1254 and 1260 (Kimbrough et al. 1972). Similar effects have been reported for offspring of exposed individuals. PCB mixtures affected weight gain, liver weight, uterine weight during proestrus, delayed puberty, and impaired thyroid function in rats and mice including offspring of rats (Linzey 1987; Collins and Capen 1980; Grant and Phillips 1974; Pantaleoni et al. 1988; Linder et al. 1974; Overmann et al. 1987; Storm et al. 1981; Kerkvliet and Kimeldorf 1977; Sanders and Kirkpatrick 1977; Kimbrough et al. 1972; French et al. 2001; Kasza et al. 1978; Talcott and Koller 1983; Sager and Girard 1994).

Voltura and French (2000) conducted a study on white-footed mice, in which the energetic cost of exposure to tPCBs was determined. Exposure to harmful substances is likely to be metabolically expensive and may result in a trade-off between energy spent to detoxify and excrete contaminants and energy allocated to growth and reproduction. Four-month-old mice were fed diets for 6 weeks containing a mixture of 2:1 Aroclor 1242:1254 at concentrations ranging from 0 to 5.4 mg/kg bw/d. At 6 weeks of exposure, there were no differences in food intake or body mass. The diets were continued for 1 year. The results showed that mice fed the highest dose had higher food intake and oxygen consumption ($p<0.05$).

J.3.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

A summary of mortality and reproductive effects attributed to TEQ is presented in Figure J.3-2 and Table J.3-3.

J.3.1.2.1 Mortality

The acute lethality of 2,3,7,8-TCDD, 1,2,3,7,8-pentachlorodibenzo-p-dioxin (PCDD), and 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin (HCDD) were investigated for Long-Evans (LE) rats and Han/Wistar (H/W) rats dosed by gavage at doses ranging from 4,000 to 20,000 ng/kg bw/d TEQ for TCDD and 1,200,000 to 7,200,000 ng/kg bw/d TEQ for 1,2,3,7,8-PCDD and 1,2,3,4,7,8-HCDD (Pohjanvirta et al. 1993). The LD_{50} values for exposure of female and male LE rats were 9,800 and 17,700 ng/kg bw/d TEQ, respectively, whereas LD_{50}s for H/W female rats were greater than 7,200,000 ng/kg bw/d TEQ. The H/W rats were approximately 1,000-fold more resistant to TCDD than LE rats. A similar difference in strains was observed for PCDD. Female H/W rats had a LD_{50} of >1,620,000 ng/kg bw/d TEQ compared to between 20,000 and 60,000 ng/kg bw/d TEQ for LE female rats. HCDD had a LD_{50} for H/W female rats of 187,100 ng/kg bw/d TEQ compared to between 12,000 and 36,000 ng/kg bw/d TEQ for LE rats.

Stahl et al. (1992) conducted a similar acute toxicity study of 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 1,2,3,4,7,8-HCDD and 1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin (hepta-CDD) on Sprague Dawley rats. The LD_{50}s were 43,000, 206,000, 88,700, and 6,325,000 ng/kg bw/d TEQ, respectively. Mortality in treated animals was caused by a dose-dependent reduction in feed intake, followed by weight loss, exhaustion of energy reserves, and death. Van Miller et al. (1977) fed 10 different doses of TCDD ranging from 0.0428 to 71,400 ng/kg bw/d TEQ to Sprague Dawley rats over a period of 78 days. None of the animals survived to week 4 after exposure to doses ranging from 3,400 to 71,000 ng/kg bw/d TEQ while after 31 weeks 100% mortality occurred at 57.1 ng/kg bw/d TEQ.

Kociba et al. (1978) conducted a 2-year study by feeding diets containing TCDD at 1, 10, and 100 ng/kg bw/d TEQ to male and female Sprague Dawley rats. At 100 ng/kg bw/d TEQ, they observed a cumulative increase in mortality ($p<0.05$) in the latter half of the study period and a decrease in mean body weight ($p<0.05$) from 6 to 24 months compared to controls. No

significant effect was reported for endpoints at the lower doses. There was no significant alteration in the food consumption rate at any dose.

J.3.1.2.2 Reproduction

PCB Congeners

Among the 209 possible PCB congeners, the non-ortho-substituted (planar) congeners are the most toxic because of their structural similarity to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The most toxic of these congeners include 3,3',4,4'-tetrachlorobiphenyl (PCB-77), 3,3'4,4',5,5'-hexachlorobiphenyl (PCB-169), and 3,3',4,4',5-pentachlorobiphenyl (PCB-126) (Safe 1984). When wildlife species tissues were analyzed for specific congeners, PCB-77 was found at the greatest concentration (Tanabe et al. 1987).

The prenatal toxicity of PCB-77 was determined in rats and mice fed contaminated diets between days 6 and 18 of gestation (Marks et al. 1989; d'Argy et al. 1987; Wardell et al. 1982; Rands et al. 1982a; 1982b). Mammals were sacrificed on day 18 or 19 of gestation and necropsies were performed on the females. PCB-77 caused embryotoxic effects to the fetuses of rats and mice, including death and resorption (Marks et al. 1989; d'Argy et al. 1987; Wardell et al. 1982). Marks et al. (1989) reported a PCB-77 dose-related increase in the percentage of implants that resorbed, at concentrations ranging from 400 (7% increase) to 6,400 (82.5% increase) ng/kg bw/d TEQ; a significant increase (16.4%) was determined at 1,600 ng/kg bw/d TEQ and above. Eight treatment levels were investigated. In addition, the average number of live fetuses per female was significantly reduced (21.5%) at 1,600 ng/kg bw/d TEQ and above (Marks et al. 1989). Rands et al. (1982a) observed that pregnant rats dosed with PCB-77 at 300 ng/kg bw/d TEQ on day 6 to 18 of gestation experienced a statistically significant increase in mortality of offspring. The results showed a trend toward decreased viability with increasing gestational time; this result was also observed by Linzey (1987). A significant number of resorptions (37%) was reported after pregnant C57BL/6 (B6) mice were administered a dose of 2,500 ng/kg bw/d TEQ of PCB-126 on day 11 of gestation (d'Argy et al. 1987). Wardell et al. (1982) observed significant mortality (14% resorption) of embryos of Sprague Dawley rats when females were exposed on day 6 through 8 of gestation to a dose of 300 ng/kg bw/d TEQ of PCB-77.

Huang et al. (1998b) fed female C57BL/6J mice (F0) estimated dietary doses of PCB-77 of 60 and 700 ng/kg bw/d TEQ 2 weeks before mating with untreated males, and subsequently throughout gestation and lactation. Female offspring (F1) were fed the same diets as the dams from weaning until 7 weeks of age, and were then mated with untreated males. Fecundity (percentage of mated females that gave birth) in F0 females after mating was 80%, 71%, and 47% in the 0, 60, and 700 ng/kg bw/d TEQ treatment groups, respectively. Pup survival at ages 4 and 21 days was significantly reduced ($p<0.05$) in the F0 females at 700 ng/kg bw/d TEQ. There were no effects on fecundity or litter size in the F1 females, although all of their offspring died before 4 days of age at 60 and 700 ng/kg bw/d TEQ. Other effects included a significant reduction of in vitro fertilizing ability of the eggs and a significant increase in number of degenerated eggs in the F1 females at ≥ 60 ng/kg bw/d TEQ.

2,3,7,8-TCDD

Fetal survival in Holtzman rats exposed to TCDD at a dose of 1,000 ng/kg bw/d TEQ was significantly reduced on day 15 of gestation (6%; $p<0.05$) and on days 20 and 21 of gestation (19%; $p<0.05$) (Mably et al. 1992b). A significant increase (19.5%; $p<0.05$,) in pre-implantation loss was observed when Center for Research in Chronic Disorders (CRCD) rats were fed a dose of TCDD of 2,000 ng/kg bw/d TEQ for 2 weeks before mating (Giavini et al. 1983). At 500 and 2,000 ng/kg bw/d TEQ, a significant (10.2% [$p<0.01$] and 30.3% [$p<0.001$], respectively) dose-related increase in post-implantation loss was observed. A significant ($p<0.01$) reduction in fetal weight was recorded at 2,000 ng/kg bw/d TEQ. Giavini et al. (1983) also observed a significant reduction ($p<0.01$) in live fetuses at 21 days of gestation when CRCD rats were exposed to 2,000 ng/kg bw/d TEQ; the response was dose related and resulted in a 45% reduction compared to controls.

In LE rats, there was a significant ($p<0.05$) decrease in the number of living fetuses per litter when rats were fed a dose of TCDD of 5,000 ng/kg bw/d TEQ on day 8 of gestation (Huuskonen et al. 1994). Over 70% of implantations were resorbed, and a further 5% died at a late fetal stage. Khera and Ruddick (1973) treated pregnant Wistar rats with doses of TCDD of 125, 250, 500, 1,000, 2,000, 4,000, 8,000, and 16,000 ng/kg bw/d TEQ on gestation days 6 to 15. Animals were sacrificed on day 22 of gestation. A dose-related decrease in live fetuses was observed;

100% embryonic lethality was reported when animals were exposed to a dose of 4,000 ng/kg bw/d TEQ or higher. Similar observations were made by Sparschu et al. (1971) in Sprague Dawley rats fed doses of TCDD of 30, 125, 500, 2,000, and 8,000 ng/kg bw/d TEQ on days 6 through 15 of gestation. The number of viable fetuses decreased and the total number of resorptions increased starting at 125 ng/kg bw/d TEQ. Maternal body weight gains decreased ($p < 0.01$) at 500 ng/kg bw/d TEQ and fetal body weight decreased ($p<0.01$) at 125 ng/kg bw/d TEQ.

Naval Medical Research Institute (NMRI) mice were fed doses of TCDD ranging from 0 to 9,000 ng/kg bw/d TEQ on gestation days 6 through 15 (Neubert and Dillman 1972). High embryonic lethality resulted at 9,000 ng/kg bw/d TEQ, and two-thirds of the litters showed 100% resorption. Sprague Dawley rats and Golden Syrian hamsters received a single dose of TCDD ranging from 0 to 18,000 ng TEQ/kg bw on gestation day 10 (rats), and 7 or 9 (hamsters), respectively (Olson and McGarrigle 1992). At 18,000 ng/kg bw TEQ, TCDD produced 73% fetal mortality in rats and 58% fetal mortality in hamsters.

A number of studies have reported mortality of rats and mice at birth when females were fed TCDD during gestation. Sprague Dawley rats were maintained on diets of 1, 10, or 100 mg TEQ/kg bw/d for 90 days prior to mating and throughout three generations (Murray et al. 1979). A dose-related reduction in the average litter size (live and dead) was reported; litter size was significantly reduced ($p<0.05$) for the F1a generation at 100 ng/kg bw/d TEQ and produced 100% stillborn pups in the F1a generation. Similarly, the litter size of the single F1b litter at the high dose was smaller than the control and all pups were stillborn. At 10 ng/kg bw/d TEQ, there was a significant ($p<0.05$) reduction in litter size for the F2 and F3 generations, but not the F1. At that dose, there was a significant decrease ($p<0.05$) in the gestational survival index in the F2 and F3 generations but not the F1a and F1b generations.

Holtzman rats were administered a single dose of TCDD of 1,000 ng/kg bw/d TEQ on day 15 of gestation (Bjerke and Peterson 1994; Mably et al. 1992b). The mean percentage of offspring alive at birth was reduced ($p<0.05$) by 30% (Bjerke and Peterson 1994) and 15% (Mably et al. 1992b). In both these studies there was a significant reduction ($p<0.05$) in body weight of offspring at birth and 5 days postpartum. In a similar study, a dose of TCDD of 700 ng/kg bw/d

TEQ produced significant mortality (23%) in offspring Holtzman rats based on number of uterine implantation sites (Bjerke et al. 1994). Mean body weight of male offspring was decreased 89 to 92% of controls by in utero and lactational TCDD exposure between birth and 7 days postpartum. Significant mortality ($p<0.05$) in Holtzman rats at birth was also observed by Flaws et al. (1997) when pregnant females were exposed to a single dose of TCDD of 1,000 ng TEQ/kg bw on days 11, 15, or 18 of gestation. Mortality reached as high as 39.6% compared to 5.5% in one of the controls. Khera and Ruddick (1973) treated Wistar rats with doses of TCDD of 125, 250, 500, and 1,000 ng/kg bw/d TEQ on days 6 through 15 of gestation. A dose-related reduction in the average number of viable pups per litter and mean pup birth weight was reported.

Postnatal effects to offspring have been determined via two routes of exposure: (1) in utero, in which females are administered TEQ during gestation, and (2) lactation, in which TEQ are transferred through the mother's milk to pups. Survival of newborn pups to 21 days of age significantly decreased ($p<0.05$) in Sprague Dawley rats in the F1a generation maintained on diets of TCDD of 1, 10, and 1,000 ng/kg bw/d TEQ and for the F2 generation maintained on the 10 ng TEQ/kg bw diet for 90 days prior to mating and throughout three generations (Murray et al. 1979). In the F1a generation, the decreases were dose-related and 100% mortality occurred in the highest dose.

Pregnant LE rats were dosed by gavage with TCDD at 1,000 ng/kg bw/d TEQ on gestation day 8 or 15 (Gray et al. 1995). This dose significantly reduced ($p<0.05$) pup survival by postnatal day 22 by approximately 22% for the group that received the dose on day 15 of gestation compared to 100% survival for the control litters. Growth of offspring was retarded throughout lactation, but the effect disappeared after puberty and was no longer significant during adulthood. In a similar study, Gray and Ostby (1995) observed that 50% of female offspring of Holtzman rats died at 16 days of age compared to control rats (2.5%). Swiss-Webster mice fed diets of 1,000, 2,500, 5,000, 10,000, and 20,000 ng TCDD/kg food also exhibited a dose-related increased in post-natal mortality, with most deaths occurring during the first week of nursing (Thomas and Hinsdill 1979).

Survival of offspring of Wistar rats until weaning (21 days of age) and average pup weight of the weanlings were reduced compared to controls in females administered a dose of TCDD of 500 or 1,000 ng/kg bw/d TEQ during days 6 through 15 of gestation (Khera and Ruddick 1973). Mortality of 100% and 60% of pups until weaning (21 days of age) was observed when females were treated with 1,000 and 500 ng TEQ/kg bw/d, respectively. Cross-fostering experiments were performed where newborn pups from treated mothers with 1,000 ng/kg bw/d TEQ were transferred to control mothers for nursing, while newborn pups from control mothers were transferred to treated mothers. At weaning, 86% of the pups born to treated mothers died, whereas only 4% of the control pups died. Postnatal mortality seemed to result from in utero exposure to TCDD and not transfer of TCDD via lactation. Nau et al. (1986) found similar cross-fostering results when NMRI mice were fed TCDD at a dose of 12,500 ng/kg bw/d TEQ for 4 consecutive days starting on gestation day 14 (Nau et al. 1986). The newborns were nursed by the control mothers. On postnatal days 1, 14, and 22, offspring mortality was 45%, 68.5%, and 75%, respectively.

Murray et al. (1979) found that TCDD reduced fertility of the F0 generation in a three-generation study. When a dose of 100 ng/kg bw/d TEQ was fed to Sprague Dawley rats starting 90 days before mating, fertility was significantly ($p<0.05$) reduced and only 3 of 31 females delivered a litter. When the F0 generation was re-mated, only 1 of 30 females delivered a litter ($p<0.05$). When fed a dose of 10 ng/kg bw/d TEQ, there was no effect on the fertility among the F0 rats, but a significant reduction ($p<0.05$) in fertility was apparent for the F1 and F2 rats. There was no significant difference in fertility at 1 ng/kg bw/d TEQ in F1 and F2 rats.

Long Evan female rats were dosed by gavage with TCDD at 1,000 ng/kg bw/d TEQ on gestation day 8 (Gray and Ostby 1995). There was a significant reduction in fertility as observed by the number of offspring compared to control. Female offspring were allowed to continuously breed for 140 days starting at 223 days of age. Only 19% of female offspring produced a fifth litter compared to 61% for the control. Khera and Ruddick (1973) also found a decrease in the incidence of pregnancy and in the average litter size from offspring born from dams administered a dose of TCDD of 500 ng TEQ/kg bw during gestation days 6 through 15. There was no effect at 250 ng/kg bw/d TEQ. It is possible that TCDD-induced reductions in ejaculated sperm numbers are, in part, responsible for the reduced fertility of treated offspring seen in several

studies (Gray et al. 1995; Khera and Ruddick 1973; Murray et al. 1979). Mably et al. (1992a) found that fertility of male rats born from mothers administered a single dose of TCDD up to 1,000 ng/kg bw/d TEQ on day 15 of gestation had a fertility index of 78% compared to 100% for controls. Litter size, live birth index, and 21-day survival of offspring were not affected. Results were inconsistent for daily sperm production and epididymal sperm reserves because rats produce and ejaculate more sperm than required for normal fertility. Sparschu et al. (1971) observed little effect on fertility when Sprague Dawley rats were administered doses ranging from 30 to 8,000 ng/kg bw/d TEQ of TCDD on gestation day 6 through 15.

In utero and/or lactational exposure of TCDD when administered as a single dose of 1,000 ng/kg bw TEQ during gestation feminizes sexual behavior in adult male rats (Mably et al. 1992c; Gray 1993). Castrated males primed with estradiol benzoate and treated with progesterone displayed dose-related increases in lordosis quotient and lordosis intensity in response to being mounted by other males. Gray et al. (1995) found no difference among groups for quality of lordosis response.

Furans

Couture et al. (1989) observed an increase in fetal mortality when Fisher 344 rats were treated with a single dose of 2,3,4,7,8-pentachlorodibenzofuran ranging from 0 to 150,000 ng/kg bw/d TEQ on days 8, 10, or 12 of gestation. Animals were sacrificed on day 20 of gestation. When treated on day 8 of gestation, there was significant ($p<0.05$) fetal mortality at 50,000 ng/kg bw/d TEQ while significant fetal mortality ($p<0.05$) occurred at the highest dose of 150,000 ng/kg bw/d TEQ when administered on gestation day 10 or 12. Mean fetal weight was correlated with fetal toxicity and was dose related compared to controls.

J.3.1.2.3 Growth

PCB Congeners

The toxic and biochemical potencies of 2,3,3',4,4',5-hexachlorobiphenyl (PCB-156) and 3,3',4,4',5-pentachlorobiphenyl (PCB-126) were investigated in a 13-week feeding study in female rats (Van Birgelen et al. 1994a, 1994b). PCB-156 and PCB-126 caused a dose-related

decrease in body weight gain. At 13 weeks, a significant reduction in body weight gain was observed at a dose of 182 ng/kg bw/d TEQ of PCB-156 ($p<0.01$) and 318 ng/kg bw/d TEQ of PCB-126 ($p<0.05$). In a similar study, a significant increase ($p<0.05$) in body weight was found in male rats at a dose of PCB-118 of 68.3 ng/kg bw/d TEQ (Chu et al. 1995).

2,3,7,8-TCDD

Kociba et al. (1976, 1978) exposed Sprague Dawley rats to doses of TCDD ranging from 1 to 1,000 ng/kg bw/d TEQ for a period of 13 weeks in one study and 2 years in another. Rats given doses of 100 or 1,000 ng/kg bw/d TEQ had decreased body weight and decreased food consumption, respectively.

J.3.1.2.4 Other Effects

PCB Congeners

The toxicity of 3,3',4,4'-tetrachlorobiphenyl (PCB-77), 2,3'4,4',5-pentachlorobiphenyl (PCB-118), 2,3,3',4,4',5-hexachlorobiphenyl (PCB-156), and 3,3',4,4',5-pentachlorobiphenyl (PCB-126) were investigated in a 13-week feeding study in male and female rats. These congeners caused a significant increase in liver, spleen, and kidney weight and EROD activity, and a decrease in thymus weight and other biochemical parameters in doses ranging from 7.5 to 1,010 ng/kg bw/d TEQ (Van Birgelen et al. 1994a,b; Chu et al. 1995). Teratogenic effects were also reported in fetuses of rats and mice including malformed fetuses, cleft palate, dilated kidney pelvis, and decreased length of tibia (Marks et al. 1989; d'Argy et al. 1987; Wardell et al. 1982). Marks et al. (1989) reported a significant increase in the average percentage of malformed fetuses per litter in mice at a 400 (7.2%), 800 (9.8%), 1,600 (25.4%), 3,200 (50.0%), and 6,400 (75%) ng/kg bw/d TEQ of PCB-77 compared to the control.

2,3,7,8-TCDD

A number of studies have found impaired development of the reproductive system in male and female rats and mice exposed to TCDD during gestation. In utero and/or lactational exposure to rats and mice administered a single dose of TCDD during gestation produced adverse developmental effects on the reproductive system of male and female offspring (Bjerke and

Peterson 1994; Bjerke et al. 1994; Gray and Ostby 1995; Gray et al. 1995, 1997; Flaws et al. 1997; Mably et al. 1992a,b,c; Giavini et al. 1983; Huuskonen et al. 1994; Murray et al. 1979; Olson and McGarrigle 1992; Heimler et al. 1998; Nau et al. 1986; Neubert and Dillman 1972; Smith et al. 1976; Thomas and Hinsdill 1979). Most of these studies showed significant effects at a dose equal to or less than 1,000 ng/kg bw/d TEQ. Effects included the following:

- A decrease in plasma testosterone concentrations (Bjerke and Peterson 1994; Mably et al. 1992b).
- Increased incidence of cystic hyperplasia of the endometrium (Gray and Ostby 1995).
- Histopathological alterations of the female reproductive tract (Gray et al. 1997).
- Decreased accessory sex organ weights (Bjerke and Peterson 1994; Mably et al. 1992a,b).
- Decreases in body, brain, ovarian, testis, thymus, liver, and ventral prostate weight (Murray et al. 1979; Gray and Ostby 1995; Mably et al. 1992b; Huuskonen et al. 1994).
- Decreased daily sperm production by 83% (Bjerke and Peterson 1994; Mably et al. 1992a).
- Decreased epididymal sperm reserves by 32 to 52% (Bjerke and Peterson 1994; Gray et al. 1995; Mably et al. 1992a).
- Decreased ejaculated sperm counts by 58 to 65% (Gray et al. 1995).
- Constant estrus (Gray and Ostby 1995).
- Reduction in ovulation rate (Giavini et al. 1983).
- Formation of a persistent vaginal membrane or thread of tissue across the opening of the vagina (Gray and Ostby 1995; Flaws et al. 1997).
- Delayed time to pregnancy (Gray et al. 1997).
- Reduction in maternal weight gain (Giavini et al. 1983; Huuskonen et al. 1994).
- Decrease in anogenital distance in male rats (Mably et al. 1992b).
- Complete or partial clefting of the phallus (Gray and Ostby 1995; Gray et al. 1997).
- Cleft clitoris (Flaws et al. 1997).

- Cleft palate (Huuskonen et al. 1994; Olson and McGarrigle 1992; Neubert and Dillmann 1972; Smith et al. 1976).

- Delayed male and female puberty (e.g., delays in testicular descent, vaginal opening delayed) (Gray and Ostby 1995; Gray et al. 1995; Gray et al. 1997; Mably et al. 1992b).

- Other fetal malformations including cystic kidney, subcutaneous edema, incomplete ossification, gastrointestinal hemorrhage and skeletal malformations (Sparschu et al. 1971; Giavini et al. 1983; Huuskonen et al. 1994).

Van Miller et al. (1977) observed neoplastic alterations in liver, kidneys, lungs, and brain after exposure of Sprague Dawley rats to doses of TCDD as low as 0.14 ng/kg bw/d TEQ for 78 weeks. Other effects included atrophy of the thymus and spleen, dilatation of the common bile ducts, and hemorrhage in the gastrointestinal tract. Kociba et al. (1976; 1978) exposed Sprague Dawley rats to doses of TCDD ranging from 1 to 1,000 ng/kg bw/d TEQ for a period of 13 weeks in one study and 2 years in another. Rats given doses of 100 or 1,000 ng/kg bw/d TEQ had slight depression of certain hematologic parameters, increased urinary excretion of porphyrins, and morphological changes in several tissues.

Furans

Couture et al. (1989) observed several effects on the developmental system of female Fisher 344 rats and fetuses when females were treated with a single dose of 2,3,4,7,8-pentachlorodibenzofuran ranging from 0 to 150,000 ng/kg bw/d TEQ on days 8, 10, or 12 of gestation. Significant increases in liver weight and significant decreases in thymus weight in dams were observed at the lower doses. Fetuses had a significant decrease in the size of thymus and lungs compared to controls. Incidence of cleft palate was significantly increased at the highest dose.

J.3.2 Effects Metrics for Characterizing Risk

J.3.2.1 Selection of Effects Metrics

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to concentration- or dose-response curves for the functional group of interest (e.g., benthic invertebrates, mammalian piscivores). In this ERA, the

effects characterization preferentially relied on concentration- or dose-response curves, but defaulted to benchmarks or other estimates of effect (e.g., no observed adverse effect level [NOAEL], lowest observed adverse effect level [LOAEL]) when insufficient data were available to derive dose-response curves. Effects associated with growth, survival, and reproduction are generally the preferred measures of effect. Further details on the decision criteria used in selecting effects metrics are provided in Section 6.6 of the ERA.

The decision criteria were applied to each COC-receptor combination. For PCBs and dioxins/furans, two measures of concentration were considered: tPCBs and 2,3,7,8-TCDD TEQ (TEQ). For tPCBs, the focus was on studies using mixtures with a higher degree of chlorination (i.e., 54 to 60%) because these mixtures most closely resemble the mixtures occurring in the PSA. For TEQ, the toxicity caused by coplanar PCBs, dioxins, and furans was considered together using the approach described by Van den Berg et al. (1998) (see Section 6.4 and Appendix C.10 for more detail).

For some COC-receptor combinations, several effects metrics were developed to account for different routes of exposure. In all cases, the effects metrics were consistent with the metrics used in the exposure analysis.

Dose-response relationships were combined with the corresponding exposure distribution in risk characterization to derive risk curves that characterize the relationship between probability and magnitude of effect.

It would be desirable to assess the effects of tPCBs and TEQ to the representative species in the Housatonic River using the results of controlled investigations that treated short-tailed shrew and red fox with at least five dose levels and measured for effects on growth, survival, or reproduction. However, controlled toxicity studies on these endpoints for short-tailed shrew and red fox were not available for tPCBs or TEQ. As a result, surrogate mammals for these representative species were used to assess toxicity. For the short-tailed shrew, the mouse was used as a surrogate mammal, when data were available. Otherwise the rat was used. For red fox, the rat served as the surrogate mammal.

J.3.2.2 tPCBs for Red Fox and Short-Tailed Shrew

The dose-response curve for tPCBs was generated using the Spencer (1982) study. This study investigated effects on reproduction of rats by administering Aroclor 1254 on gestation days 6 through 15. The study investigated effects on mortality at birth using eight treatment levels. The curve fitting of dose-response data to a generalized linear model (GLiM) was performed using SAS® (SAS Institute, Cary, NC). Mortality data required a logit link function and a binomial error distribution was assumed (Bailer and Oris 1997). Figure J.3-3 presents the dose-response curve for mortality at birth of rats. In this analysis, Abbott's formula was used to correct for control mortality (Newman 1995). Because mean responses were used in the analysis (the raw data were not available), fiducial limits and goodness of fit were not estimated. The model parameters were β_0 = -15.3804, β_1 = 3.7136, seβ_0 = 1.1897, seβ_1 = 0.2865, and corr$\beta_0\beta_1$ = -0.9971. The dose-response model was significant at $p<0.0007$ (F value = 30.75, 6 degrees of freedom).

J.3.2.3 2,3,7,8-TCDD (TEQ)

J.3.2.3.1 Red Fox

The Khera and Ruddick (1973) and Sparschu et al. (1971) studies were combined for the derivation of the dose-response curve for rats exposed to TEQ. Both these studies investigated reproduction effects in rats by administering TCDD on gestation days 6 to 15. The studies used similar protocols and when combined yielded a data set with 17 treatment levels. The curve fitting of dose-response data to a GLiM was performed using SAS®. Fecundity data required a log link function and a Poisson error distribution was assumed (Bailer and Oris 1997). Figure J.3-4 presents the dose-response curve for reproductive fecundity of rats exposed to TEQ.

Because mean responses were used in the analysis (the raw data were not available), fiducial limits or goodness of fit were not estimated. The model parameters were β_0 = 2.4869, β_1 = -0.6765, seβ_0 = 0.1073, seβ_1 = 0.1469, and corr$\beta_0\beta_1$ = -0.5046. The dose-response model was significant at $p<0.0001$ (F value = 88.64, 15 degrees of freedom).

J.3.2.3.2 Short-Tailed Shrew

The effects curve for TEQ was generated using the Marks et al. 1989 study. This study investigated reproductive effects in mice by administering TCDD on gestation days 6 through 15. The studies investigated effects on fecundity using eight treatment levels. The curve fitting of dose-response data to a GLiM was performed using SAS®. Fecundity data required a log link function, and a Poisson error distribution was assumed (Bailer and Oris 1997). Figure J.3-5 presents the dose-response curve for reproductive fecundity of mice exposed to TEQ.

Because mean responses were used in the analysis (the raw data were not available), fiducial limits and goodness of fit were not estimated. The model parameters were β_0 = 2.5287, β_1 = -0.1848, seβ_0 = 0.1338, seβ_1 = 0.0737, and corr$\beta_0\beta_1$ = -0.5455. The dose response model was significant at $p<0.0001$ (F value = 858.89, 6 degrees of freedom).

J.4 RISK CHARACTERIZATION

This section characterizes risk to omnivorous and carnivorous mammals exposed to tPCBs and 2,3,7,8-TCDD TEQ (TEQ) in the PSA of the Housatonic River. The risk characterization includes a weight-of-evidence assessment consistent with the methodology developed by MDEP (Menzie et al. 1996) and which is described in detail in Section 2.9 of the ERA.

The risk characterization for omnivorous and carnivorous mammals in the PSA used three lines of evidence (probabilistic exposure and effects modeling, semi-quantitative field surveys, and a population demography field study on short-tailed shrews) to determine potential ecological risks to this group of organisms. The red fox and the short-tailed shrew were the representative species selected for this evaluation. The three major lines of evidence are considered to be independent and will be combined at the end of this section in the weight-of-evidence assessment.

Section J.4.1 presents a brief overview of the methodology, results, and interpretation of the mammal surveys conducted from 1998 to 2001. A more detailed presentation of this information is presented in Appendix A. Section J.4.2 compares the quantitative probabilistic exposure estimates calculated for the red fox and short-tailed shrew (see Section J.2) with the relevant ecological effect doses for tPCBs and TEQ presented in Section J.3. Section J.4.3 presents a brief overview of the methodology, results, and interpretation of the population demography field study conducted in 2001 (Boonstra 2002). Section J.4.4, Weight-of-Evidence Evaluation, summarizes the findings of the three lines of evidence, provides an analysis of the relative significance of the lines of evidence, and discusses the overall findings of the risk assessment. The sources of uncertainty are presented in Section J.4.5, and the extrapolation of risk to other species is presented in Section J.4.6. Section J.4.7 presents the conclusions of this assessment endpoint.

The purpose of this risk characterization is to address the risk questions:

- Are the concentrations of tPCBs and TEQ present in the prey of omnivorous and carnivorous mammals sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?

- If so, how severe are the risks and what are their potential consequences?

J.4.1 Field Surveys (Performed by EPA)

J.4.1.1 Introduction

The mammalian community in the PSA was studied over a 4-year period by EPA, from 1998 to 2001. Surveys were conducted to record presence, relative abundance, and habitat usage for small and large mammals including red fox and short-tailed shrew and used a variety of field survey techniques (e.g., small mammal trapping, snow tracking, and scent-post station surveys, bat echolocation recordings). Additional information on the use of the PSA by these and other mammalian species was obtained from field notes from other studies and from incidental observations. To help characterize the mammalian community, a literature review was performed to locate information on local species and populations. General and technical references on habitat requirements and use, seasonality of occurrence, and relative abundance in the region (Whitaker and Hamilton 1998; DeGraaf and Yamasaki 2001) were then used to create a matrix of those species that could potentially occur in the PSA and surrounding areas (see Attachment C of Appendix A). Field observations were used to refine parameters such as habitat use, relative abundance, and seasonality of occurrence for all mammal species observed in the primary and reference study areas.

Small mammal trapping was performed in 1998 and 1999 to verify the relative abundance of different species in the PSA, to provide tissue samples for PCB analysis (for use in exposure modeling), and to determine the reproductive status of females.

J.4.1.2 Small Mammal Trapping Methods

Semi-quantitative small mammal trapping was conducted at three locations in September 1998 and at three additional locations in August to September 1999. At each site, trap arrays were set on one day and run for five consecutive nights for a total of 580 trap nights (TN) (116 traps times 5 nights equals 580 TN) per trap site.

Species, sex, weight (g), total length (mm), tail length (mm), hind foot length (mm), and ear length (mm) were recorded for each small mammal collected. Each individual was also aged

(adult versus juvenile) and inspected for abnormalities or deformities, which were described on data forms. Length (mm) and width (mm) of the testes were measured for all males. During the 1999 surveys, each female was checked for milk production, and the uterus was removed for placental scar analysis (see Section J.4.1.3.3).

In addition to small mammal trapping, small mammals were captured in pitfall traps installed for amphibian studies, and tracks of many of these species were observed during snow tracking and scent post station surveys.

J.4.1.3 Results and Discussion

Forty-two mammal species were documented in the PSA during the 4 years of field surveys. An additional 10 species are likely to occur but were not verified, for a total of 52 species potentially occurring in the area (see Attachment C in Appendix A). Many species were observed throughout the PSA in a variety of habitats. Forested communities, such as red maple swamp, black ash-red maple-tamarak, transitional floodplain forest, and high-terrace floodplain forest, supported the greatest number of species. Common omnivorous and carnivorous mammals included coyotes (*Canis latrans*), red fox (*Vulpes vulpes*), raccoons (*Procyon lotor*), white-footed mice (*Peromyscus leucopus*), short-tailed shrews (*Blarina brevicauda*), and little brown bats (*Myotis lucifugus*), all of which were observed in forested and nonforested habitats as well as riverine, shoreline, wetland, upland, and residential habitats.

Other carnivorous mammals observed in the PSA included bobcats (*Lynx rufus*), fishers (*Martes pennanti*), and long-tailed weasels (*Mustela frenata*). Omnivorous mammals were one of the most abundant groups of mammals observed in the PSA. Common omnivores included white-footed mice, raccoons, striped skunks (*Mephitis mephitis*), Virginia opossums (*Didelphis virginiana*), and black bears (*Ursus americanus*).

J.4.1.3.1 Occurrence and Abundance of Shrews

Masked shrews (*Sorex cinereus*), smoky shrews (*Sorex fumeus*), northern short-tailed shrews (*Blarina brevicauda*), and northern water shrews (*Sorex palustris*) all were observed in the PSA (see Attachment C in Appendix A). Short-tailed shrews were the most abundant shrew caught

during trapping in the PSA. Thirty-two short-tailed shrews were trapped in the PSA in 1998 and 1999, accounting for 14 and 26% of small mammal captures, respectively. They were also routinely captured in pit traps during wood frog and leopard frog studies. Masked shrews were frequently observed, but in smaller numbers than the short-tailed shrew. Three masked shrews were trapped in the PSA in 1998 and five were trapped in 1999, representing 1 and 4% of the small mammal trapping results. This tiny shrew is the smallest mammal occurring in the PSA. Smoky shrews were less common than the masked and short-tailed shrews. Only a few were captured in pit traps during the wood frog and leopard frog studies, although they are generally common throughout New England. In the PSA, smoky shrews were found only in red maple swamp communities. The northern water shrew is rare in the PSA, having been observed only once. This observation was of a dead individual washed up on the shoreline of the river in the southern part of the PSA. The northern water shrew is a species of conservation concern in Massachusetts and is uncommon throughout New England.

J.4.1.3.2 Occurrence and Abundance of Other Small Mammals

Other small mammals, such as white-footed mice, while traditionally categorized as herbivores, have diets that vary greatly depending upon seasonal and regional availability of food sources and can include a high percentage of animal matter. Large amounts of insects (primarily ground beetles, caterpillars, cutworms, snails, and centipedes) are taken during the spring and summer. As the season progresses, the diet of these mammals shifts more toward seeds, nuts, berries, and fungus. White-footed mice are active year-round and often cache large amounts of seeds and nuts to last through the winter. Of the total, 221 small mammals (representing 5 species) and 122 small mammals (representing 6 species) were captured during 1998 and 1999, respectively. White-footed mice were by far the most abundant, making up 64 and 62% of captures in 1998 and 1999, respectively. White-footed mice were captured at all small mammal-trapping locations and at numerous vernal pools and were observed at all locations during snow tracking surveys.

In the PSA, woodland jumping mice (*Napaeozapus insignis*) were observed in red maple swamp, black ash-red maple-tamarack calcareous seepage swamp, transitional floodplain forest, and high-terrace floodplain forest communities. Meadow jumping mice (*Zapus hudsonius*) were

observed in the PSA in red maple swamp and transitional floodplain forest communities. Norway rats (*Rattus norvegicus*), first introduced to the United States in the late 1700s, are now abundant in residential and agricultural areas throughout the country. This species was also captured during amphibian pit trapping in red maple swamp and transitional floodplain forest communities, primarily at locations close to residential areas.

J.4.1.3.3 Small Mammal Reproductive Status

Trapped small female mammals were checked for evidence of lactation and the uterus was removed for placental scar analysis. During placental scar analysis, the uterus was placed on a microscope slide and the number of placental scars and embryos were counted with the aid of a dissecting microscope. Placental scars were grouped and counted based on the size, shape, and opacity (Harder and Kirkpatrick 1996). Although the number of placental scars has been used in a variety of mammals to estimate litter sizes (Hensel et al. 1969; Sanderson 1950; Oleyar and McGinnes 1974; Nixon et al. 1975), a major limitation of the technique is that reabsorbed fetuses can also leave placental scars that are usually indistinguishable from those left by fetuses developing to term (Conaway 1955).

The evaluation of placental scars was performed on four species including short-tailed shrew, white-footed mouse, meadow jumping mouse, and masked shrew at Locations 13, 14, and 15. Sample sizes were small for each species. White-footed mouse had the largest number of samples, ranging from 6 to 11 females among the sites. The average number of placental scars per white-footed mouse female was 6.33, 6.27, and 6.50 at Locations 13, 14, and 15, respectively. Mean soil tPCB concentrations vary over 50-fold between these three sites. The number of females for the other three species combined ranged from two to six, and had a lower average number of scars, ranging from zero to 2.50 (Table J.4-1). In some cases, placental scars were difficult to identify, particularly for the white-footed mouse. There were no differences in numbers of placental scars among sites (Table J.4-1), although there are uncertainties associated with these data because of the limitations described above.

J.4.2 Comparison of Estimated Exposures to Laboratory-Derived Effects Doses

For omnivorous and carnivorous mammals, exposure for red fox was assessed including all of Reach 5. Exposure of short-tailed shrews in the PSA was estimated in three areas (Locations 13, 14, and 15, Figure J.2-3) in the PSA.

After a review of the potential exposure pathways and relative contribution to overall exposure to tPCBs and TEQ, ingestion of food items (mammals for red fox; mammals, litter invertebrates, and earthworms for short-tailed shrew) was determined to be the only pathway of concern for these species. A detailed presentation of the exposure analyses for representative omnivorous and carnivorous mammals species is provided in Section J.2.

The effects characterization for mammals (see Section J.3) provides a detailed overview of the current toxicological literature on the potential effects of tPCBs and TEQ to mammalian species. There were no studies in the literature that directly quantified the effects of tPCBs or TEQ to either of the representative species selected. Dose-response curves were developed to characterize effects of tPCBs and TEQ using surrogate mammal species.

The following discussion presents an integration of the exposure distributions and effects metrics developed for each species and exposure area. For each receptor-COC combination, a category of low, intermediate, or high risk was assigned using the following guidance:

- If the probability of 10% or greater effect was less than 20%, then the risk to omnivorous and carnivorous mammals was considered low.
- If the probability of 20% or greater effect was greater than 50%, then the risk to omnivorous and carnivorous mammals was considered high.
- All other outcomes were considered to have intermediate risk.

This evaluation was done separately for the results of the Monte Carlo analyses and the lower and upper bounds from the probability bounds analyses. The “risk category” refers to the level of risk based on the results of the Monte Carlo analysis. The “risk range” refers to the levels of risk based on the results of the probability bounds analyses.

J.4.2.1 PCB Risk Characterization – Red Fox and Short-Tailed Shrew

The effects metric for tPCBs was derived using the results of Spencer (1982), for both the red fox and short-tailed shrew. Spencer (1982) investigated reproductive effects of Aroclor 1254 using eight treatment levels on Sprague Dawley rats fed treated diets on days 6 through 15 of gestation. Doses of 7.47 and 17.1 mg/kg bw/d produced a statistically significant reduction in fetal weight at birth (11.8%) and reduced fetal survival (28%), respectively. Sufficient data were available to generate a dose-response curve. The dose-response curve indicated that 10% and 20% mortality at birth would be expected at doses of 3.05 and 5.37 mg/kg bw/d, respectively.

J.4.2.1.1 Red Fox – Reach 5

The Monte Carlo predictions for total daily intake of tPCBs by red fox in the PSA indicated that there was a 44.6% probability of exceeding the 10% effect dose, and a 31.1% probability of exceeding the 20% effect dose (Figure J.4-1, Table J.4-2). Further, there was a 50% probability of an 8% or greater increase in mortality at birth and a 10% probability of a 58% or greater increase in mortality at birth. Thus, the risk category for tPCBs for red fox feeding and reproducing in the PSA of the Housatonic River is intermediate (Table J.4-3).

The lower bound from the probability bound analysis indicated that there was a 12% probability of exceeding the 10% effect dose, and a 6% probability of exceeding the 20% dose. The upper bound from the probability bounds analysis indicated that there was a 54% probability of exceeding the 10% effect dose, and a 38% probability of exceeding the 20% dose (Figure J.4-1, Table J.4-2). The corresponding risk range for red fox in the PSA is, therefore, categorized as low for the lower probability bound and intermediate for the upper probability bound (Table J.4-3).

In summary, the Monte Carlo analysis indicates a risk category of intermediate for fox exposed to tPCBs. The risk range, as determined by the probability bounds analysis, is low to intermediate.

J.4.2.1.2 Short-Tailed Shrew – Locations 13, 14, and 15

The risk category for short-tailed shrew at Locations 13 and 14 is high. The risk range for these two sites ranges from intermediate to high (Figures J.4-2 and J.4-3, Table J.4-3). The risk category for Location 15 for shrews exposed to tPCBs is low; the risk range is low to intermediate (Figure J.4-4, Table J.4-3). Location 15 was selected to represent the lower bound of tPCB concentrations in soil in the PSA.

J.4.2.2 TEQ Risk Characterization – Red Fox and Short-Tailed Shrew

The effects metric for TEQ was derived using the rat study results of Khera and Ruddick (1973) and Sparschu et al. (1971). Khera and Ruddick (1973) treated pregnant Wistar rats with several doses of TEQ on gestation days 6 to 15. Animals were sacrificed on day 22 of gestation. A dose-related decrease in live fetuses was observed; 100% embryonic lethality was reported when animals were exposed to a dose of 4,000 ng/kg bw/d TEQ. Similar observations were made by Sparschu et al. (1971) in Sprague Dawley rats fed several doses of TCDD on days 6 to 15 of gestation. The number of viable fetuses decreased and the total number of resorptions increased with dose, starting at 125 ng/kg bw/d TEQ. These two studies were combined to generate a dose-response curve. The dose-response curve indicated that 10% and 20% declines in fecundity would be expected at doses of 156 and 330 ng/kg bw/d TEQ, respectively.

For short-tailed shrew, the TEQ effects metric was derived using the study by Marks et al. (1989). Marks et al. (1989) reported a dose-related increase in the percentage of implants that resorbed when mice were treated with PCB-77; a significant increase in resorption (16.4%) compared to controls was observed at 1,600 ng/kg bw/d TEQ. The average number of live fetuses per dam was also significantly reduced (21.5%) at 1,600 ng/kg bw/d TEQ and above compared to controls. In addition, Marks et al. (1989) reported a significant increase in the average percentage of malformed fetuses at several doses. This study was used to generate a dose-response curve. The dose-response curve developed indicated that 10% and 20% declines in reproductive fecundity would be expected at doses of 570 and 1,207 ng/kg bw/d TEQ, respectively.

J.4.2.2.1 Red Fox – Reach 5

The Monte Carlo predictions for total daily intake of TEQ by red fox in the PSA indicated that there was a 44.8% probability of exceeding the 10% effect dose, and a 25.6% probability of exceeding the 20% effect dose (Figure J.4-5, Table J.4-2). Further, there was a 50% probability of a 9% or greater decrease in fecundity and a 10% probability of a 43% or greater decrease in fecundity. Thus, the risk category for exposure to TEQ of red fox feeding and reproducing in the PSA of the Housatonic River is intermediate (Table J.4-3).

The lower bound from the probability bounds analysis indicated that there was a 0% probability of exceeding the 10% and 20% effect doses. The upper bound from the probability bounds analysis indicated that there was a 54% probability of exceeding the 10% effect dose, and a 33% probability of exceeding the 20% effect dose (Figure J.4-5, Table J.4-2). The corresponding risk range for red fox exposed to TEQ in the PSA is, therefore, categorized as low for the lower probability bound and intermediate for the upper probability bound (Table J.4-3).

In summary, the Monte Carlo analysis indicates a risk category of intermediate for fox exposed to TEQ. The risk range, as determined by the probability bounds analysis, is low to intermediate.

J.4.2.2.2 Short-Tailed Shrew – Locations 13, 14, and 15

The risk category for exposure of short-tailed shrew to TEQ at Location 13 is low; the risk range is low to intermediate (Figure J.4-6, Table J.4-3). Short-tailed shrew exposed to TEQ at Locations 14 and 15 have a risk category of low. Both the upper and lower bound of the risk ranges for Locations 14 and 15 are low (Figures J.4-7 and J.4-8, Table J.4-3).

J.4.3 Population Demography Field Study (Performed by GE)

A population demography field study was performed in 2001 along a 16-km (10-mile) reach of the Housatonic River between Pittsfield and Woods Pond (Boonstra 2002). The study objectives included evaluating population density, survival, rates of reproduction, sex ratio, and growth rates of short-tailed shrew. More information on this study is provided in Boonstra (2002).

J.4.3.1 Methodology

Six locations were selected based on tPCB concentrations, habitat uniformity, and sufficient area to permit a 1-hectare (ha) trapping grid to be located within each location. The report states that no reference location was used because there were no suitable areas with similar vegetation types. Instead, results from the literature were used for comparison with the results from the study. Two grid classes were selected, designated as high and low concentrations of tPCBs, with three locations in each class (Table J.4-4). The top 15.2 cm (6 in) of soil in the trapping grids, and a 33-m (108-ft) buffer around the grid were used to calculate tPCB concentrations. The report states that the buffer around the grid represents the maximum distance short-tailed shrews would typically move during their daily activities. All locations were within the eastern deciduous temperate forest biome in primarily palustrine habitat, with portions of two grids also including upland habitat (see Figure J.4-9). Habitat varied across the grids, particularly between the northern locations and the southern locations (i.e., the former had much more vegetative biomass than did the latter). Comparison of the northern and southern locations is an indirect way of determining whether habitat quality has an effect on the survival, growth and reproduction of shrews. The six areas trapped in this study, in fact, varied in habitat quality. Without habitat and microhabitat data at the six trapping locations, it is difficult to tell if differences in habitat explain variation in population parameters between the six locations.

Three trapping sessions were conducted in spring, summer, and fall (trapping sessions one, two, and three, respectively) over the course of the study, with each session lasting three consecutive days. However, because of flooding during the spring session, trapping activity (and collection of data) could be carried out only twice at three locations and three times at the other three locations. Some of the statistical analyses could be conducted only for locations having three trapping session data sets.

All sampling locations were laid out with 100 grid points, with a Longworth live trap placed on every other grid point (total of 50 traps per location). The traps were baited using crimped oats. When first captured, short-tailed shrews were marked by toeclipping; and, on the first capture in each trapping session, sex, sexual condition (female lactating or not, male breeding or not), mass, and location were recorded.

Population densities and standard errors were estimated using the mark-recapture heterogeneity model from the program Capture. This population estimator assumes no mortality, and no immigration and emigration during the sampling period.

Survival was assessed using two approaches. If an animal disappeared from a trapping grid, it was unknown whether the animal died or emigrated, and mortality was assumed. In the first approach, survival rates were measured by counting the marked animals. These rates were calculated as the percentage of animals recaptured at time 2 that were released at time 1. Rates are expressed as the minimum survival rates per 30 days, to permit comparisons to other live-trapping studies of short-tailed shrew. In the second approach, a logistic regression was used for the period between the summer and autumn trapping session based on recapture.

Reproductive condition was assessed based on the body weight of animals, and the presence of prominent nipples or nipple scars in females, or the presence of enlarged testes in males. Females that were pear-shaped were considered pregnant. Males weighing 19 g or less were excluded from the analysis, whereas all females weighing 18 g or more were included as potentially breeding. Only three females could be positively identified as pregnant. A logistic regression approach was used to analyze differences between trapping sessions and grids and high/low tPCBs areas.

Growth was assessed using two approaches. The first approach involved calculating instantaneous growth rates per day for all animals caught in more than one trapping session. This analysis required an ANCOVA with initial weight as the covariate. The second approach compared mean body weights among the grids.

J.4.3.2 Statistical Analysis – Boonstra (2002)

Analyses were conducted by Boonstra (2002) to:

1. Assess the exposure-response relationships (different levels of tPCB exposure on trapping grids) for demographic parameters using simple correlation analyses.
2. Determine whether demographic parameters differed between high and low tPCB soil concentration grids. Each grid was considered a replicate of one of these two categories.

3. Assess whether there was an area-effect (two northern grids versus four southern grids) as the vegetation and undergrowth was more lush in the northern grids. In this analysis, each grid was treated as a replicate in one of these two categories.

ANOVAs, post-hoc tests (Tukey-Kramer), and power analyses were performed with Statsview. Binary data were analyzed by logistic regression using log-likelihood ratios (Boonstra 2002).

J.4.3.3 Supplemental Analyses (Performed by EPA)

For the ERA, concentrations of tPCBs in soil (using measured data and spatially weighted data) were estimated in grids 1-6 (Table J.4-5). The spatially weighted data were estimated using the inverse distance weighting (IDW) approach presented in Appendix C.3. As no geographic coordinates were available in the Boonstra (2002) report describing the extent of each of the six grids from which shrews were sampled, the grids were digitized (in ArcView 3.3) by visual inspection of the maps provided in the Boonstra (2002) report. A 33-m buffer (33 m from the outer boundary) was created around each of the grids using ArcView Spatial Analyst 2.0 (Figure J.4-9). Boonstra (2002) used the 33-m boundary, as a conservative estimate of how far short-tailed shrews would typically move during their daily activities. The soil data set was then imported into ArcView, and data points were isolated in each of the grid and buffer polygons for use in the analysis. In some instances, the buffer zone extended into the main channel of the river, or across a tributary. In these cases, these data points were removed from the data set for that grid (e.g., Grid 1 and Grid 4, Figure J.4-9). The arithmetic mean of soil concentrations was generated using point data found within each grid and buffer (Table J.4-5).

To derive the spatially weighted PCB concentration statistics, map calculator (Spatial Analyst) was used to overlay the spatially weighted surface with the polygon of the buffer for each grid. The grid polygon was assigned a value of one and multiplied by the spatially weighted surface grid. The end result was a grid polygon containing the weighted-surface values (Figure J.4-10; Table J.4-5). Arithmetic means of soil concentrations of the new grid polygon surface were calculated. Where the spatially weighted surface crossed the river, backwater, or a tributary, the weighted surface was revised to eliminate the estimated soil concentrations at these points.

Using both the spatially weighted and measured soil concentrations, and Boonstra's (2002) survival data from July to September (Table J.4-5), a probit model was used to determine if there

was a relationship between arithmetic mean tPCB concentrations and mortality of shrews. The curve fitting of data to a generalized linear model (GLiM) was performed using SAS® (SAS Institute, Cary, NC). Arithmetic means of tPCB concentrations were log transformed prior to the analyses.

J.4.3.4 Results

The results of the Boonstra (2002) study and the supplemental analyses are presented below.

J.4.3.4.1 Population Density

Population densities of short-tailed shrews per hectare were constant for all the southern grids, whereas the two northern grids showed pronounced population fluctuations over the trapping sessions (Table J.4-6).

Boonstra (2002) found no significant relationship between population densities and concentrations of tPCBs in soil in either summer or autumn ($p = 0.56$ and 0.27, respectively). Similarly, when the results of the summer and autumn trapping sessions were pooled into high and low tPCB concentrations, there was no significant relationship with population densities ($p = 0.55$); however, in these analyses the power was low. There was a significantly higher density of short-tailed shrew in areas with lush vegetation (grids 1 and 2) ($p = 0.01$).

J.4.3.4.2 Survival

Boonstra (2002) performed a two-factor (grid and sex) logistic regression of survival, and found that there were significant differences in overall survival among grids ($p = 0.008$), but that males and females from the same grid had similar rates of survival ($p = 0.24$). To test whether these observed differences in survival were due to tPCB concentrations, the sites were pooled into low (<2.6 mg/kg) and high (>17.5 mg/kg) concentration sites. A Chi-square analysis showed no difference in survival between high and low sites ($p = 0.31$). Additional analyses, conducted as part of this ERA, did not corroborate the findings of Boonstra (2002) regarding the lack of relationship between tPCB soil concentration and survival of shrews (see J.4.3.4.6).

J.4.3.4.3 Sex Ratio

Boonstra (2002) found no difference in sex ratio in either spring or autumn trapping sessions ($p = 0.71$ and 0.18, respectively). There was, however, a significant difference among grids for the summer trapping session. When the grids were pooled into high and low tPCB categories, there was no significant difference in sex ratio as a function of tPCB concentrations in either the summer or fall sessions ($p=0.17$ and $p = 0.96$, respectively). In contrast, when the two northern sites were pooled and compared with the four pooled southern sites, there was a significant different in sex ratio in both the summer session ($p = 0.005$) and the fall session ($p = 0.02$).

J.4.3.4.4 Reproduction

A two-factor logistic regression was carried out comparing trapping session versus high and low tPCB sites (Boonstra (2002). The dependent variable was "reproductive intensity," which is defined by Boonstra (2002) as animals potentially being in breeding condition based on their body mass. Males weighing 19 g or less were excluded from the analysis, whereas all females weighing 18 g or more were included as potentially breeding. There was a strong trapping session effect ($p < 0.0001$), but no evidence of a tPCB effect. In the spring, summer, and autumn trapping sessions, 31.2%, 61.3%, and 5%, respectively, of the males were in breeding condition. The analysis was carried out for females and the results were similar. Female lactation, which is better evidence for reproduction than body weight, was observed predominantly in spring, and summer. In spring, summer, and autumn trapping sessions, 75%, 39.7%, and 12.3%, respectively, of the females were lactating. The use of body weight to imply reproductive fitness may not be appropriate because it is insensitive to potential reproductive impairments. This point is further discussed in the Section J.4.3.5.

J.4.3.4.5 Growth

Based on statistical analyses, Boonstra (2002) demonstrated that smaller males and females grow faster than larger animals, but that short-tailed shrews did not differ in growth rates among the grids. For males, there was a significant effect of initial body weight on growth ($p = 0.003$), but no evidence of a tPCB effect ($p = 0.31$). For females, there was a significant effect of initial

body weight on growth (p = 0.04), no grid effect on growth rate (p = 0.84) and no interaction effect (p = 0.88). In the pooled analysis, all effects were not significant.

J.4.3.4.6 Supplemental Analyses

Comparison of data from Boonstra (2002) and the spatially weighted arithmetic mean concentrations of tPCBs derived as part of this ERA (assuming Boonstra's estimates are expressed as arithmetic means) showed that the order of the grids from highest to lowest concentrations changed but that the same three "high" contaminated sites remained "high" (Table J.4-5). The Boonstra (2002) estimates of tPCB concentrations in soil in the six grids, however, appear to be in error. Several factors may have contributed to the error including: (1) Boonstra (2002) included samples from areas that are not shrew habitat (e.g., sediment samples from aquatic areas); (2) the Boonstra spatial weighting approach relied solely on data internal to the grids – with small sample sizes in some of the grids, this likely produced poor estimates of the spatially weighted arithmetic means; and (3) some soil samples used in the analyses do not exist in the Housatonic River database (which includes historical and recent data from both EPA and GE) or soil samples were not included in the Boonstra (2002) study that are in the database. These issues prompted the supplemental analyses of the relationship between mean soil tPCB concentrations and shrew survival. These analyses were conducted using two different estimates of mean soil tPCB concentration in each grid: (1) the arithmetic mean derived from measured soil concentrations in the grid, and (2) the arithmetic mean using spatially weighted soil concentrations.

The supplemental analyses indicated a significant relationship between the spatially weighted mean concentration of tPCBs in soil and survival of shrews from summer to autumn for males (p = 0.0131) and females (p = 0.0006), and males and females combined (p = 0.0068). Similar analyses conducted without spatial weighting of the soil concentrations data (i.e., arithmetic means of measured concentrations) produced very similar results. Figures J.4-11 and J.4-12 present the results of both analyses for females and males combined. Although the results of the analyses indicated a significant relationship between soil concentrations of tPCBs and shrew survival, the confidence limits indicate that the relationships are not strong. Some of the "noise" in the relationships may be attributed to the influence of habitat differences among the grids,

small sample sizes, the effects of flooding, the analytical methods used to measure tPCBs, and the relatively small number of treatments. The slope of the regression models is not steep, indicating that survival was only slightly reduced at the "high" contaminated grids compared to the "low" contaminated grids.

J.4.3.5 Discussion of Results

The stated objectives of the population demography field study (Boonstra 2002) were to collect population demographics information, including survival, growth, and reproduction of short-tailed shrews in the Housatonic River floodplain. The conclusions of the Boonstra (2002) study suggest that population characteristics of short-tailed shrew living on more contaminated tPCB sites are not negatively affected compared to those living on less contaminated sites in the PSA. The study concluded that exposure of short-tailed shrew to tPCBs had no apparent effect on population density, sex ratio, reproduction, and growth rate. In general, the analyses presented in the study showed high monthly survival. Although there was grid-to-grid survival variability, this variability could not be explained by differences in tPCB concentrations among the grids. The only significant effect was on mass of males but, in this case, the males living in the high-contaminated sites weighed more, not less, than those living in the low-contaminated sites. In summary, variations in tPCB concentrations among the sites resulted in no differences in population demography variables of short-tailed shrew according to Boonstra (2002).

In comparison to the analyses reported in Boonstra (2002), the results of the analyses conducted in support of this ERA using data reported in Boonstra indicated a significant relationship between concentrations of tPCBs in soil and survival of shrews from summer to autumn for males, females, and males and females combined. The differences in the results are likely due to differences in the statistical methods used and, more importantly, to selection of the soil samples for inclusion in the analyses. The results of the analyses performed for the ERA show that tPCBs could have an effect on the survival of short-tailed shrews. These analyses do not support the conclusion in Boonstra (2002) that "there is no evidence that this variability [in shrew survival] can be explained by differences in tPCB concentrations among the grids."

There were a number of uncertainties associated with the study. Flooding of the grids occurred during the course of the study. Water levels rose in early June and prevented trapping on three

of the six grids during the first of three trapping events (the other three grids had already been trapped). Although spring flooding is a natural phenomenon in the Housatonic River, with the study design, it is difficult to determine the impact of flooding on the study results. In addition, the population estimator used by Boonstra assumed no mortality, emigration, or immigration, which do occur, particularly during a flooding event.

The six areas trapped in this study varied in habitat quality. Without habitat and microhabitat data at the six trapping sites, it is difficult to tell if differences in habitat explain variation in population parameters between the six sites. Boonstra (2002) acknowledges how area (i.e., location within the floodplain) and quality of habitat are likely considerations for explaining variation observed at the six trapping sites, but provides only a qualitative discussion of this issue.

Rates of production and survival of young were not calculated because of the tendency for shrews not to enter traps until they are nearly adult size, and annual flooding possibly resulting in young not being produced on site. Therefore, the assessment of breeding condition relied primarily on body weight to assess reproductive readiness or capability in individual shrews, rather than more definitive indicators of reproductive status. The lack of reproduction rate data in the Boonstra (2002) study results in uncertainty regarding population maintenance. Even with high adult survivorship, without reproduction rate data, it is difficult to know whether shrew populations are maintaining themselves through natural production or by immigration.

The use of body weight to imply reproductive fitness may not be appropriate because it is insensitive to potential reproductive impairments. For example, 6 of 10 female short-tailed shrews greater than 18 grams in weight trapped by EPA in August 1999 possessed no evidence of breeding history upon placental scar analysis. Although some of these six could have been young animals that had not bred yet, others could have been animals with reproductive impairments due to various factors, including PCB exposure. Additionally, during the 1999 trapping, of those specimens submitted for tissue analysis (8 of the 10 females over 18 grams), five females with no evidence of breeding history had body burdens up to 135 mg/kg (average 74 mg/kg), whereas those with direct evidence of breeding had body burdens up to 93 mg/kg (average 57 mg/kg). This sample size is quite small; however, along with laboratory evidence of

PCB-induced reproductive impairments to mammals, it suggests that animal weight alone may not be representative of the reproductive status of individual animals. The lack of direct measures of individual or population-level reproduction relative to PCB exposure limits the utility of the Boonstra (2002) study in evaluating the influence of COC concentrations on shrew reproduction in the PSA.

J.4.4 Weight-of-Evidence Analysis

A weight-of-evidence analysis was used to combine the two and three major lines of evidence described in the preceding sections for red fox and short-tailed shrew, respectively. The goal of this analysis is to determine whether significant risk is posed to omnivorous and carnivorous mammals in the Housatonic River PSA as a result of exposure to tPCBs and TEQ. The analysis follows the methodology proposed by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996; see Section 2.9 for details). The analyses were conducted separately for tPCBs and TEQ.

J.4.4.1 Evaluating Measurement Endpoints

Measurement endpoints are assigned a relative weight for each of the attributes that account for the strength of association between the assessment and measurement endpoints and the study design and execution. The measurement endpoint values are presented in Table J.4-7 for tPCBs and TEQ. The measurement endpoint values for each attribute are discussed in more detail below.

J.4.4.1.1 Relationship Between Measurement and Assessment Endpoints

Degree of Association

This attribute quantifies the degree of association between each measurement and assessment endpoint. The assessment endpoint is the survival, growth, and reproduction of omnivorous and carnivorous mammals. For tPCBs and TEQ, the measurement endpoints for the field surveys, the population demography field study (Boonstra 2002), and for the modeled exposure and effects lines of evidence were given values of low, moderate/high, and moderate, respectively.

The field surveys were conducted to record presence, relative abundance, and habitat usage for small and large mammals including red fox and short-tailed shrew. The field surveys were not designed to provide a quantitative evaluation of survival, growth, and reproduction of omnivorous and carnivorous mammals in the PSA, hence, the low value. The population demography field study was performed in situ and attempted to measure population demography of short-tailed shrew including reproduction, growth, and survival in the PSA. Reproduction was not measured, but was assessed using body weight and the presence of enlarged nipples or testes. As a result, a moderate/high value was given to the population demography field study.

The modeled exposure and effects line of evidence was given a moderate value for both tPCBs and TEQ. The exposure assessment was conducted on two omnivorous and carnivorous mammals known to occur in the Housatonic River watershed using dietary concentrations of COCs measured in prey tissue from the PSA, but the effects metrics used in the risk characterization were derived from surrogate species. Ideally, laboratory animals would be closely related in taxonomy, metabolism, diet, and behavior to the actual species being modeled. Because toxicity data were not available for the representative species, rats were used to model risk to red fox, whereas mice and rats were used to model risk to short-tailed shrew. These surrogate species are different from shrew and fox. For example, the metabolic rate of shrews is very high compared to other mammals of similar size, and the diet of rats is different from fox. As a result, the assessment and measurement endpoints are not directly linked. The modeled exposure and effects line of evidence was thus given a moderate value.

Stressor/Response

This attribute measures the ability to correlate the magnitude of response with the degree of exposure. For tPCBs and TEQ, the measurement endpoints for the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given values of moderate, moderate, and moderate/high, respectively.

The field surveys determined the presence, relative abundance, size, and reproductive status (for small mammals only) of omnivorous and carnivorous mammals. However, the study was not designed to test for an exposure-response relationship, hence the moderate value for the field surveys.

The population demography field study estimated exposure using soil samples from the PSA and measured the degree of response including reproduction, growth, and survival. The responses were quantitatively correlated with magnitude of exposure, and statistically significant regressions were demonstrated. However, confounding factors such as flooding, area (i.e., location within the floodplain), and habitat quality may have influenced the results of the population demography field study. In addition, the use of body weight to imply reproductive status may also be a confounding factor when interpreting potential PCB-related effects on shrew population demographics. Supplemental analyses revealed that tPCB concentrations in soil estimated by Boonstra (2002) in the six grids were in error. These analyses of the relationship between mean soil tPCB concentrations and shrew survival produced different results than those provided in the Boonstra (2002) study. Therefore, a moderate value was given to this line of evidence.

The exposure modeling was species and stressor specific. Although responses were quantitatively correlated with magnitude of exposure estimated using species-specific exposure factors and concentrations of COCs measured in prey tissue from the PSA, the dose-response relationship was derived from a surrogate species. As a result, a value of moderate/high was given to the modeled exposure and effects lines of evidence.

Utility of Measure

This attribute reflects the quality of the accepted criteria, standards, or performance-based measures used to judge environmental harm. For tPCBs and TEQ, the measurement endpoints for the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given values of low/moderate, moderate/high, and moderate/high, respectively.

Although the field surveys were standardized and location specific, they were not designed to have high statistical power or to be capable of detecting subtle effects. As a result, this line of evidence was given a value of low/moderate.

The population demography field study attempted to measure reproduction, growth, and survival of short-tailed shrew living in the PSA. Although the study was well executed, the study habitat

quality differences and flooding were factors that may have observed the interpretation of PCB-related effects. Rates of production and survival of young were not calculated. Therefore, the assessment of breeding condition relied on body weight as a surrogate for reproductive status or capability in individual shrews rather than more definitive measurements. Without reproduction rate data, it is impossible to know if these shrew populations are maintaining themselves through natural production or immigration. The supplemental analyses did not reach the same conclusion on the relationship of PCB concentrations and shrew survival. This line of evidence was given a moderate/high value.

The techniques used to model exposure and effects are well established and accepted by the scientific community. These measurement endpoints are sensitive (given the species-specific exposures and the dose/response relationship used to estimate effects) and should be capable of detecting effects to survival of omnivorous and carnivorous mammals. However, although the effects metric used in the risk characterization was a dose-response curve, it was based on studies of surrogate species. Therefore, this endpoint was given a value of moderate/high.

J.4.4.1.2 Data Quality

Data Quality

This attribute reflects the degree to which data quality objectives (DQOs), QA/QC procedures, and other recognized characteristics of high quality studies are met for each line of evidence. For tPCBs and TEQ, the measurement endpoints for the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given values of high, moderate/high, and moderate/high, respectively.

The field surveys were performed according to well-defined and documented protocols for semi-qualitative field surveys to demonstrate relative numbers of individuals per unit effort, and reproductive history and status of individuals captured and therefore, was assigned a value of high. Similarly, the population demography field study was performed according to a well-defined and documented protocol; however, the protocol was not available for review by EPA before the study was conducted for input as to utility of the data quality objectives for use in the ERA. Habitat parameters in this latter study were not measured, and rates of reproduction and

immigration were not determined. In addition, estimates of tPCB concentrations in soil in the six grids appear to be in error. The population demography field study line of evidence was given a moderate/high value.

The DQOs established for the sampling and analysis of tissue samples were met for the tissue residue data used in the exposure analyses for both tPCBs and TEQ. Although the effects metrics were derived from peer-reviewed scientific literature, details on the DQOs for these studies were not available. A value of moderate/high was given to this line of evidence.

J.4.4.1.3 Study Design

Site Specificity

The value for this attribute reflects the site specificity of data, media, species, environmental conditions, and habitat for each measurement endpoint. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and the modeled exposure and effects lines of evidence were given values of high, high, and low/moderate, respectively. The mammal surveys were conducted in the PSA across a gradient of COC concentrations and, in the case of snow tracking and scent posts, had appropriate reference areas. As a result, this line of evidence was given a high value.

The population demography field study was given a high value because the investigation took place in situ and used local species. Published studies were used as benchmarks rather than a reference area.

The modeling of exposure and effects line of evidence was assigned a value of low/moderate. The tissue analyses used in the exposure models were site specific, and other exposure parameters were representative of site conditions. The effects data, however, were laboratory based and did not reflect the site-specific environmental conditions found in the Housatonic River area.

Sensitivity

This attribute refers to the ability of the measurement endpoint to detect changes caused by a stressor against a background of natural variability and other sources of uncertainty. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given values of moderate, moderate, and high, respectively.

The field surveys were not designed to be sensitive to detecting subtle effects associated with COCs, but would be expected to detect effects an order of magnitude or greater. One of the objectives stated in the population demography field study report was to detect effects from exposure to COCs. However, there was a large amount of "noise" affecting the studied populations as a result of flooding, variation in habitat quality, and using body weight to measure reproductive status. These factors introduced uncertainty in the results of this study. Therefore, both the field surveys and population demography field study were given a moderate value.

The modeled exposure and effects line of evidence directly assessed the exposure-response relationship. In addition, laboratory studies from which effects data were derived were stressor specific, hence the high value.

Spatial Representativeness

This attribute reflects the degree of spatial overlap of study area, measurement locations, locations of stressors, locations of representative species, and points of potential exposure to those receptors. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given a value of high, high, and moderate, respectively.

The mammal survey and population demography field study had complete coverage of the study area, measurement locations, stressors, receptors, and points of exposure and therefore, both were assigned a value of high.

The modeled exposures relied on concentrations in prey tissue collected throughout the PSA and areas of exposure. However, some of the exposure parameters used in the modeling were not site-specific, but taken from the published literature (e.g., body weight, gross energy,

assimilation efficiency, etc.). The effects assessment used toxicity studies conducted in laboratories. Overall, four of the five criteria overlap spatially for this attribute, hence, a moderate value.

Temporal Representativeness

This attribute describes the degree of coincidence in time between data collection and exposures being assessed, hence the moderate/high value. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given values of moderate/high, high and moderate, respectively.

The mammal surveys were conducted during two field seasons when effects were most likely to be observed, hence, the moderate/high value. The shrew population demography field study was performed over a 1-year period, but included three trapping sessions covering spring, summer, and autumn conditions. Measurements were collected during the breeding season. Moderate to high seasonal variability was observed in the results. This line of evidence was given a value of high.

The exposure model was designed to span the reproductive cycle of red fox and short-tailed shrew. Although multiple tissue samples were used to parameterize the model, the samples were collected in August-September, which does not coincide with the breeding season for red fox (January-February) but is within that of the short-tailed shrew (March-September). These samples were also collected in only 1 year. The effects studies selected for tPCBs and TEQ spanned the reproductive cycle of mammals. Therefore, the value assigned for this endpoint was moderate.

Quantitative Measure

This attribute indicates the degree to which the response can be quantified by a given measurement endpoint. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and the modeled exposure and effects lines of evidence were given values of moderate, moderate/high, and high, respectively.

The mammal field surveys were semi-quantitative but were not amenable to statistical tests between sites because of the lack of replication and the small sample numbers. The population demography field study included statistical tests using site-specific data. However, supplemental analyses performed by EPA cast doubt on the study results and conclusions. Therefore, the mammal field survey was assigned a value of moderate, and the demography field study was assigned a value of moderate/high.

The results from the modeled exposure and effects line of evidence are probabilistic and highly quantitative, and propagated uncertainty associated with modeling procedures, hence, the high value.

Standard Method

This attribute quantified the extent to which measurement endpoint studies adhered to recognized scientific protocols. For both tPCBs and TEQ, the field surveys, the population demography field study (Boonstra 2002), and modeled exposure and effects lines of evidence were given a value of high, high, and moderate/high, respectively.

For all lines of evidence, generally accepted methods and guidance were used and were applicable for the assessment endpoint and site; however, the probability bounds analyses used in the modeled exposure and effects line of evidence is a relatively new technique for propagating uncertainty. Therefore, a high value was given to the field surveys and population demography field study lines of evidence, but a lower value of moderate/high was given to the modeled exposure and effects endpoint.

J.4.4.1.4 Total Value

The total value assigned to each measurement endpoint is a weighted average of the values from the attributes (Table J.4-7). For both tPCBs and TEQ, the field surveys, the population demography field study for short-tailed shrews, and the modeled exposure and effects lines of evidence were given a moderate/high value.

J.4.4.2 Magnitude of Response in Measurement Endpoints

The measurement endpoint values are used together with the magnitude of response in the measurement endpoints to judge the weight of evidence for adverse effects on representative species. The magnitude of response can be presented as qualitative or quantitative measures. Qualitative measures are used here.

The total attribute weighting values, evidence of harm, and magnitude of response for each measurement endpoint are presented in Table J.4-8 for tPCBs and Table J.4-9 for TEQ.

The field surveys indicated that red fox and short-tailed shrew commonly were observed in the PSA. The objectives of the population demography field study (Boonstra 2002) were to measure population demography of short-tailed shrews directly, including reproduction, growth, and survival in the PSA. The responses were quantitatively compared with magnitude of exposure. However, confounding factors such as flooding, area (i.e., location within the floodplain), habitat quality, and the use of body weight to imply reproductive status were either not assessed or controlled and may have had significant effects on population demographics and the results of the field study. Additional analyses of data generated in the population demography field study showed that tPCBs may be having effects on survival of short-tailed shrews. Other demographic parameters, however, do not appear to be affected by tPCB concentrations in soil.

The results from the modeled exposure and effects line of evidence suggest that there is a high risk to short-tailed shrew exposed to tPCBs at Locations 13 and 14, and a low risk at Location 15. There is an intermediate risk for fox exposed to tPCBs foraging in Reach 5 (Table J.4-8). There is an intermediate risk to red fox exposed to TEQ in the PSA, and low risk to short-tailed shrew exposed to TEQ at Locations 13, 14, and 15 (Table J.4-9).

J.4.4.3 Concurrence Among Measurement Endpoints

The final component in the weight-of-evidence approach examines the agreement, logical connections, interdependencies, and correlations among the measurement endpoints as they relate to each assessment endpoint. The methodology for detecting concurrence involves the use of a graphical method where measurement endpoints are plotted on a matrix that also includes

the weight of each endpoint and degree of response. Tables J.4-10 and J.4-11 depict the outcome for omnivorous and carnivorous mammals exposed to tPCBs and TEQ, respectively.

The results from the modeled exposure and effects line of evidence suggest that tPCBs pose a high risk to short-tailed shrew inhabiting Locations 13 and 14 of the PSA, and an intermediate risk to fox exposed to tPCBs and TEQ in the PSA. The uncertainty concerning the modeled exposure and effects line of evidence for tPCBs and TEQ, particularly because surrogate species were used for estimating effects, could mean that risks of these COCs are being under or over-estimated for the PSA. The field survey line of evidence, although inconclusive in terms of demonstrating effects and risk, indicated that omnivorous and carnivorous mammals, including short-tailed shrew and red fox, were commonly observed in hardwood forests, palustrine forested areas, and shorelines of the PSA. In addition, according to Boonstra (2002), the population demography field study line of evidence suggests that no effects attributable to tPCBs are occurring to short-tailed shrews. However, the results of the latter study are confounded by factors such as flooding, quality of habitat and using body weight to imply reproductive fitness, all of which likely introduced a large amount of uncertainty. In addition, the soil concentrations data used in the Boonstra (2002) study appear to be in error. Additional analyses with revised soil concentration data do not support the conclusion in the Boonstra (2002) study that shrew survival in the study grids was not affected by soil tPCB concentration, but indicates that there is statistically significant relationship between PCB concentrations and survival, although not strong.

Therefore, the WOE assessment suggests an intermediate risk for short-tailed shrews exposed to tPCBs and TEQ in the contaminated areas of the PSA. This conclusion, however, is uncertain because of the lack of definitive findings as to whether effects are occurring in the field surveys and population demography field study, and the lack of species-specific measures of effects.

The WOE analysis also suggests, based on two lines of evidence for red fox, an intermediate risk to fox exposed to tPCBs and TEQ in the PSA with a degree of uncertainty associated with this conclusion because of the relatively common observations of fox in the field surveys, and the lack of species-specific measures of effects.

J.4.5 Sources of Uncertainty

The assessment of risk to omnivorous and carnivorous mammals contains uncertainties. Each source of uncertainty can influence the estimates of risk; therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to omnivorous and carnivorous mammals are described below:

- In this assessment, it was assumed that dietary exposure represents the most important pathway of exposure for omnivorous and carnivorous mammals exposed to COCs. Although unlikely to provide a major contribution to the risk, other pathways could increase the exposure and perhaps increase risk slightly (Moore et al. 1999). When including drinking water and vegetation as part of the diet for omnivorous and carnivorous mammals, the exposure model showed negligible increases in exposure.

- The Monte Carlo sensitivity analyses suggest that the free metabolic rate (*FMR*) slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. However, no measurements of free metabolic rate were available for the representative wildlife species. Similarly, measured food intake rates were not available for red fox or northern short-tailed shrew or reasonable surrogate species. Therefore, *FMRs* were estimated using allometric equations. The use of allometric equations introduces some uncertainty into the exposure estimates because they have model-fitting error, and are based on species different from the representative species used in this assessment. For northern short-tailed shrew, the insectivore model of Nagy et al. (1999) was selected as the most appropriate allometric model to estimate *FMR*. Only two shrew species were included in the derivation of this model. Examples of other species used to derive the insectivore model include bat, mole, mouse, and wolf species. For red fox, the carnivore model of Nagy et al. (1999) was used. Although the model was developed using three species of fox, dog, cat, and wolf species were also used to derive the model. Given the lack of data on representative species used in the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as inputs for the allometric slope and power terms.

- The free-living metabolic rate equations require body weight as an input variable. Data on the body weight of red fox living in the PSA are not available. As a result, body weights of red fox were estimated from the literature. The potential magnitude and direction of the uncertainty associated with lack of site-specific information on body weights of red fox are unknown, but are likely small.

- Because no stomach contents or other dietary analyses are available for the northern short-tailed shrew and red fox in the PSA, dietary compositions were derived from those reported in the literature for mammals collected from other geographical

locations. The potential magnitude and direction of the uncertainty associated with lack of information on diet are unknown. The uncertainty due to lack of knowledge on diet of red fox and short-tailed shrews in the PSA was partially addressed by using distributions to represent variability in diets observed at other similar sites.

- Sample sizes were limited for the analyses of COC concentrations in some prey items. One composite sample of earthworm (comprising between 20 and 45 worms) and four samples of mammals were available to estimate exposure of shrews to TEQ at each site. Similarly, only two to three samples of litter invertebrates were available to estimate exposure to shrews to tPCBs. Uncertainty due to sample size was explicitly addressed in the uncertainty analyses. In the Monte Carlo analysis, sample size uncertainty was addressed by use of the 95% upper confidence limit (UCL) on the mean. Use of the UCL addressed uncertainty, but is biased toward overestimating exposure. In the probability bounds analysis, uncertainty was addressed by specifying concentration variables as a range from the 5% lower confidence limit (LCL) to the UCL. This treatment of uncertainty is unbiased.

- Data on concentrations of TEQ in litter invertebrate prey were not available to estimate exposure to short-tailed shrew. In this case, the concentrations of TEQ in prey were extrapolated using measured concentrations in other prey items. Concentrations of TEQ in litter invertebrates were estimated at the three locations using the ratio of concentrations of tPCBs in earthworms to the concentrations of tPCBs in litter invertebrates. This ratio was then applied to the measured TEQ concentration in earthworms at the site of interest to estimate the TEQ concentration in litter invertebrates. This type of extrapolation introduces some uncertainty regarding the concentration of COCs in prey tissue, although the magnitude and direction of this uncertainty is difficult to judge.

- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). This source of uncertainty was addressed in the uncertainty analyses by estimating prey concentrations assuming concentrations of PCB-123 and-157 were equal to zero, and assuming that concentrations of PCB-123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. The resulting TEQ estimates were then compared. If the ratio of the upper to lower bound TEQ estimates was less than 1.3, this source of uncertainty was deemed unimportant and disregarded. If the ratio exceeded 1.3, the uncertainty due to the co-elution was propagated through the uncertainty analyses.

- The foraging time of the short-tailed shrew was assumed to be 100% in each of Locations 13, 14, and 15. This assumption is reasonable because short-tailed shrews have a small foraging range. However, there may be periods when they forage outside these sites (e.g., during flooding). The red fox has a larger foraging range and could, therefore, forage in areas outside the PSA, thus reducing exposure to tPCBs and TEQ. Therefore, it was assumed that red fox spend 50% of their time in the PSA, with the remainder of their time in areas that are not contaminated. This area use factor was based on a qualitative comparison of fox foraging range to available fox habitat within and outside the 10-year floodplain and professional judgment. The

uncertainty associated with this assumption could result in either an overestimate or underestimate of the exposure of fox to COCs.

- The largest source of uncertainty in the effects assessment was associated with the lack of toxicity studies involving the representative species. There were no toxicity studies available for red fox and short-tailed shrew exposed to tPCBs or TEQ. As a result, laboratory studies involving surrogate species were used to estimate effects to these species. For short-tailed shrew and red fox, the rat was used as a surrogate species for effects caused by exposure to tPCBs. In the case of exposure to TEQ, the mouse was used as a surrogate species for short-tailed shrew while the rat was used for red fox. These extrapolations introduced uncertainty in the effects assessment because of the variations in physiological and biochemical differences between the species such as uptake, metabolism, and disposition that can alter the potential toxicity of a contaminant. The sensitivity of wildlife to an environmental contaminant may differ from that of a laboratory or domestic species because of behavioral and ecological parameters, including stress factors (e.g., competition, seasonal changes in temperature or food availability), disease, and exposure to other contaminants. Inbred laboratory animal strains may also have an unusual sensitivity or resistance to a tested substance.

- For rats exposed to TEQ, two toxicity studies, Khera and Ruddick (1973) and Sparschu et al. (1971) were combined to derive a dose-response curve. Both studies investigated reproduction effects in rats by administering TCDD on gestation days 6 to 15. The studies used similar protocols, and when combined, yielded a data set with 17 treatment levels. Although these studies were similar in terms of endpoints, duration of exposure, quality, and acceptability of protocols, there is some uncertainty in the data set generated from combining the studies.

- The effects metrics used to estimate risk to omnivorous and carnivorous mammals were derived for Aroclor 1254 mixtures. Some uncertainty is inherent in extrapolating from those mixtures to the specific congener patterns observed in the PSA of the Housatonic River. The potential magnitude and direction of the uncertainty associated with this extrapolation are unknown for omnivorous and carnivorous mammals.

- The lack of reproduction rate data in the Boonstra (2002) study creates uncertainty regarding population maintenance. Even with high adult survivorship, without reproduction rate data, it is difficult to determine whether shrew populations are maintaining themselves through natural production or immigration.

- In the Boonstra (2002) study, the use of body weight to imply reproductive status is a limitation because it is a potentially insensitive indicator of reproductive condition. For example, 6 of 10 female short-tailed shrews greater than 18 grams in weight trapped in August of 1999 possessed no evidence of breeding history upon placental scar analysis. Although some of these six could have been young animals that simply had not bred, others could have been animals with reproductive impairments due to various factors, including PCB exposure. Additionally, of those specimens submitted

for tissue analysis (eight of the ten females over 18 grams), five females with no evidence of breeding history had body tPCB concentrations up to 135 ppm (average 74 ppm), whereas those with direct evidence of breeding had tPCB concentrations up to 93 ppm (average 57 ppm). This sample size is quite small; however, along with laboratory evidence of PCB-induced reproductive impairments to mammals, it provides evidence that body weight alone may not be representative of the reproductive preparedness of individual animals.

J.4.6 Comparisons to Other Omnivorous and Carnivorous Mammals

The northern short-tailed shrew and red fox were chosen to represent omnivorous and carnivorous mammals inhabiting the Housatonic River area. Other omnivorous and carnivorous species common to the area include smoky shrews, masked shrews, coyotes, gray fox, fishers, short-tailed shrews, and long-tailed shrews (see Appendix A). Exposure and sensitivity to the COCs are the two factors that are used to estimate risk to omnivorous and carnivorous mammals. As noted in this ERA, effects studies conducted on short-tailed shrew and red fox are not available. Similarly, effects data are not available for other omnivorous and carnivorous species living in the Housatonic River area. As a result, the same surrogate effects data used to estimate effects to short-tailed shrew and red fox would be used for other omnivorous and carnivorous species.

The purpose of this section is to qualitatively compare exposure to tPCBs and TEQ estimated for the representative species to other omnivorous and carnivorous mammals. The major factors that influence mammalian exposure to tPCBs and TEQ include the following:

1. Foraging behavior and dietary composition.
2. Foraging and home range.
3. Species body weight and other life history characteristics.

Representative species and other mammals were compared using these factors. The comparison highlights similarities and differences, and their potential to influence exposure and risks to tPCBs and TEQ (see Table J.4-12).

J.4.6.1 Comparison of Risks to Northern Short-Tailed Shrew to Other Species

Smoky Shrew (Sorex fumens)

The smoky shrew's life history, foraging behavior, habitat preferences, and feeding habits are similar to the short-tailed shrew. The smoky shrew is much smaller than the short-tailed shrew and thus has a higher metabolism, leading to increased exposure to tPCBs and TEQ. The foraging range and feeding habits of these two shrews is similar. Therefore, the smoky shrew is expected to have a higher level of risk from exposure to tPCBs and TEQ than do short-tailed shrews because of the higher metabolic rate.

Masked Shrew (Sorex cinereus)

The masked shrew and short-tailed shrew have similar life expectancies, foraging behaviors, and ranges. Differences between the two include body size and habitat preferences. The masked shrew is considerably smaller than the short-tailed shrew. Thus, the masked shrew has a higher metabolism that results in increased exposure to tPCBs and TEQ. In terms of habitat preferences, the short-tailed shrew is primarily found in damp woodlands and fields while the masked shrew prefers drier upland areas. Exposure to tPCBs and TEQ in the Housatonic River PSA is higher in damp woodlands and fields than in the drier uplands. Therefore, the masked shrew is likely to experience approximately similar levels of risk from exposure to tPCBs and TEQ.

J.4.6.2 Comparison of Risks to Red Fox to Other Species

Coyote (Canis latrans)

The coyote and red fox have a similar diet, but their foraging range and body size are very different. The foraging range of the coyote is larger than the red fox, resulting in less time spent in the PSA and lower exposure to tPCBs and TEQ. Coyotes are more than twice the size of red fox. Therefore, they have a slower metabolism than the red fox, which results in lower exposure to tPCBs and TEQ. Considering these characteristics, coyotes are expected to experience lower risks from exposure to tPCBs and TEQ.

Gray Fox (Urocyon cinereoargenteus)

The gray fox and red fox share similar physical and life history characteristics. The gray fox has a larger foraging range, which may result in less time spent in the PSA and, therefore, lower exposure to tPCBs and TEQ. In regard to diet, the gray fox relies more heavily on animal matter than the red fox. Thus, the gray fox is expected to accumulate higher concentrations of tPCBs and TEQ through its diet. Based on these considerations, the gray fox is likely to experience a roughly similar level of risk from exposure to tPCBs and TEQ.

Fisher (Martes pennanti)

Because the fisher and red fox have similar body weights they likely consume similar amounts of prey. The fisher has a longer life span than the red fox. This will increase the fisher's lifetime exposure to tPCBs and TEQ. The fisher's foraging range is much larger than the red fox and could result in the fisher spending less time in the PSA. Animal matter constitutes nearly 100% of the fisher's diet compared to 76% of the red fox's diet. Therefore, although the fisher may spend less time in the PSA, its greater consumption of animal matter and longer life expectancy are expected to result in a similar to higher level of risk from exposure to tPCBs and TEQ.

Long-Tailed Weasel (Mustela frenata)

The long-tailed weasel's body weight and foraging range are considerably smaller than the red fox. The long-tailed weasel's smaller body weight results in a higher metabolism that increases accumulation of contaminants. Similarly, the smaller foraging range increases the long-tailed weasel's exposure to tPCBs and TEQ in the Housatonic River area. The red fox and long-tailed weasel forage for similar prey items (e.g., voles, shrews, rabbits, birds) and are therefore exposed to comparable concentrations of contaminants through diet. Based on these considerations, the long-tailed weasel is likely to experience similar to higher levels of risk from exposure to tPCBs and TEQ.

Short-Tailed Weasel (Mustela erminea)

The red fox and short-tailed weasel are both year-round inhabitants of the Housatonic River area. The short-tailed weasel is much smaller than the red fox. This results in a higher metabolism,

leading to greater accumulation of contaminants from prey items. The short-tailed weasel's foraging range is smaller than the red fox's, increasing exposure to tPCBs and TEQ for short-tailed weasels inhabiting the Housatonic River area. The short-tailed weasel and red fox forage for similar prey items (e.g., mice, shrews, rabbits, birds) and are therefore exposed to comparable concentrations of tPCBs and TEQ through diet. Considering these variables, the short-tailed weasel is expected to experience similar to higher levels of risk from exposure to tPCBs and TEQ.

J.4.7 Conclusions

For omnivorous and carnivorous mammals, data from three major lines of evidence were available, including field surveys, a population demography field study of short-tailed shrew, and exposure and effects modeling. The weight-of-evidence analysis indicates an intermediate risk for short-tailed shrews exposed to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because of the lack of definitive findings about whether effects are occurring in the field surveys and population demography field study, and the lack of species-specific measures of effects.

The WOE suggests, based on two lines of evidence for red fox, an intermediate risk to fox exposed to tPCBs and TEQ in the PSA. This finding is also uncertain because although fox were commonly observed during the field surveys, a foraging rate of 50% in Reach 5 was used, and species-specific measures of effects were not available.

Field surveys were conducted (in part) to determine which omnivorous and carnivorous mammal species were present in the PSA. The surveys were not designed to provide a quantitative evaluation of the relationship between exposure to COCs and the survival, growth, and reproduction of omnivorous and carnivorous mammals in the PSA. Instead, the surveys determined the presence, relative abundance, size, and reproductive status of omnivorous and carnivorous mammals. Red fox, short-tailed shrew, and other omnivorous and carnivorous mammals were observed frequently in several areas in the PSA.

The objectives of the population demography field study were to determine population density, survival, rates of reproduction, sex ratio, and growth rates of short-tailed shrew measured at six

sites having different concentrations of tPCBs in the PSA (Boonstra 2002). The Boonstra (2002) results found that variation in tPCB concentrations among the sites resulted in no differences in population demographic parameters of short-tailed shrew. However, confounding factors such as flooding, area (i.e., location within the floodplain), and habitat quality may have had significant effects on population demographics. The lack of reproduction rate data in the Boonstra (2002) study creates uncertainty regarding the ability of the shrew population to maintain itself. Even with high adult survivorship, without reproduction rate data, it is difficult to know whether shrew populations are maintaining themselves through natural production or immigration. In addition, the use of body weight to imply reproductive status may not be appropriate because it is a potentially insensitive indication of reproductive condition.

In contrast to the findings in the Boonstra study, the results of the supplemental analyses conducted for this ERA indicated a significant relationship between tPCB spatially weighted and measured concentrations in soil and survival of shrews from summer to autumn for males, females, and males and females combined, although the confidence limits indicate that the relationships are not strong. Some of the "noise" in the relationships may be attributed to factors listed above. The additional analyses do not support the Boonstra (2002) conclusion that "there is no evidence that this variability [in shrew survival] can be explained by differences in tPCB concentrations among the grids."

The modeling of exposure and effects line of evidence was used to determine the level of risk to the representative mammal species, short-tailed shrew, and red fox. The effects characterization developed dose-response curves to describe the potential effects of tPCBs and TEQ to omnivorous and carnivorous mammals. There were no toxicity studies available for red fox and short-tailed shrew. Surrogate species were used to estimate effects with the assumption that representative omnivorous and carnivorous mammal species would experience adverse effects similar to the surrogate species.

The dose-response curve for effects of tPCBs to omnivorous and carnivorous mammals indicated that 10% and 20% declines in mortality at birth would be expected at doses of 3.05 and 5.37 mg/kg bw/d, respectively. The modeled exposure results indicated that the daily intake of tPCBs by red fox fell within this range, whereas the daily intake of tPCBs by northern short-tailed

shrew was greater than 5.37 mg/kg bw/d at Locations 13 and 14. This means that shrews, and possibly red fox in the PSA, are likely to receive tPCB doses that would cause adverse reproductive effects. The daily intakes of short-tailed shrews at Location 15 were below the 10% effects dose, meaning that shrews are likely not at risk from exposure to TEQ at that site.

For TEQ, the dose-response curve for red fox indicated that 10% and 20% declines in reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d, respectively. The modeled exposure results indicated that the daily intake of TEQ by red fox fell within this range. It is, therefore, difficult to make definitive conclusions about the risks of TEQ to this species. For northern short-tailed shrews, the dose-response curve indicated that 10% and 20% declines in reproductive fecundity would be expected at doses of 570 and 1,207 ng/kg bw/d TEQ, respectively. The daily intakes of short-tailed shrews at Locations 13, 14, and 15 were below the 10% effects dose, meaning that shrews are likely not at risk from exposure to TEQ in the PSA.

The field surveys and the conclusions made in the Boonstra (2002) study contradict the results from the modeling of exposure and effects line of evidence. However, the results of the supplemental analyses of the data from the Boonstra (2002) study on survival of short-tailed shrews are in agreement with the modeling results, suggesting that there are intermediate effects from exposure to COCs in the contaminated areas of the PSA.

Population dynamics of mammals are affected by processes such as growth, reproduction, death of predators, immigration, and emigration. As a result, a number of mechanisms exist to possibly compensate for the adverse effects of a toxic chemical. For example, a toxic chemical may lead to an increase of a mammal population by reducing abundance of competitors or by eliminating predators. Other mechanisms could also compensate for the direct effects of a toxic chemical (e.g., increased immigration from uncontaminated sites). In the Housatonic River PSA, such compensating mechanisms could exist for the local populations of short-tailed shrew, and red fox. Thus, a possible explanation for the lack of concordance between the field survey results, the Boonstra (2002) field study results, the additional analyses on survival of shrew, and the modeling results is that other mechanisms (e.g., reduced competition, elimination of predators) compensated for the direct effects due to tPCBs. No information, however, is available to support or test this supposition.

ERA Summary

The weight-of-evidence analysis indicates an intermediate risk for short-tailed shrews exposed to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because of the uncertainty about whether effects are occurring in two of the lines of evidence (i.e., field survey and population demography field study).

Risk to carnivorous mammals, such as red fox, exposed to tPCBs and TEQ are intermediate in the PSA.

Other omnivorous and carnivorous mammal species common to the PSA include smoky shrew, masked shrew, coyote, gray fox, fisher, short-tailed weasel, and long-tailed weasel. A qualitative analysis of risk on these species indicates that smoky shrew, short-tailed weasel, and long-tailed weasel have higher levels of risk; masked shrew, gray fox, and fisher have similar levels of risk; and coyote has a lower level of risk compared to the representative species.

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APPENDIX J

ASSESSMENT ENDPOINT— SURVIVAL, GROWTH, AND REPRODUCTION OF OMNIVOROUS AND CARNIVOROUS MAMMALS

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Table J.2-1

Dietary Studies for the Red Fox

Dietary Composition	Spring (%)	Summer (%)	Fall (%)	Winter (%)	All Year Average	Location Habitat	Reference
Rabbits				44.4		Nebraska	Powell and Case 1982, in EPA 1993
Small mammals				33			
Pheasant				8.4			
Other birds				11.2			
Miscellaneous				2			
Unknown				1			
Mammals	92.2	37.1	61.7	65		Illinois	Knable 1974, in EPA 1993
Birds	2.4	43.2	0.2	8.6			
Arthropods	0.2	11.6	4.2	0.1			
Plants	4.6	6.3	31.1	26.1			
Other	0.6	1.8	2.8	0.3			
Rabbits	24.8	10.7	36.5	38.7		Missouri	Korschgen 1959, in EPA 1993
Mice/rats	24.2	6.2	21.3	22.5			
Other mammals	4	1.4	8.1	8.2			
Poultry	21	45	16.3	11.6			
Carrion	12.9	13	6.5	7.4			
Livestock	9.8	0.3	2	5.4			
Birds	0.6	1.2	1.1	3.8			
Invertebrates	trace	15.3	1.6	trace			
Plants	2.7	6.9	6.6	2.1			

Table J.2-1

Dietary Studies for the Red Fox
(Continued)

Dietary Composition	Spring (%)	Summer (%)	Fall (%)	Winter (%)	All Year Average	Location Habitat	Reference
Mammals				81.4		Maryland/ Appalachian	Hockman and Chapman 1983, in EPA 1993
Birds				4.8			
Arthropods				2.8			
Plants				7			
Other				4			
Hare		30.8	37	55.6		Pierce Pond, Maine	Dibello et al. 1990
Deer (scavenge)		2.31	0.00356	13.9			
Small mammals		34.6	48.4	29.6			
Fruit		32.3	14.2	0.00926			
Vegetation					3.64		
Invertebrates					4.72		
Birds					6.96		
Hare		16.3	15.1	59.1		Cherry Hill, Maine	Dibello et al. 1990
Deer (scavenge)		12.1	1.81	14.5			
Small mammals		27	33.7	21.8			
Fruit		44.7	49.4	4.5			
Vegetation					6.03		
Invertebrates					5.59		
Birds					6.72		

Table J.2-2

Input Variables Used in the Monte Carlo Exposure Analysis for Total Daily Intake of tPCBs and TEQ by Red Fox Inhabiting the Primary Study Area

Variable	Distribution	Parameters
Body weight (*BW*; kg)	Normal	0 = 3.87; s = 0.322
Free metabolic rate (*FMR*; kcal/kg bw/d) a = *FMR*-slope b = *FMR*-power	$FMR = aBW^b$ Normal Lognormal	 *log a*: 0 = 1.67; s = 2.65 0 = 0.869; s = 0.116
Gross energy mammals (*GE*; kcal/kg)	Point Estimate	1700
Assimilation efficiency mammals (*AE*; unitless)	Point Estimate	0.84
Proportion mammals in diet (P_m; unitless)	Beta	α = 26.0; β = 5.7; scale = 0.92
Proportion forage time in Reach 5 (*FT*; unitless)	Point Estimate	0.5

Table J.2-3

Input Variables Used in the RiskCalc Probability Bounds Analysis for Total Daily Intake of tPCBs and TEQ by Red Fox Inhabiting the Primary Study Area

Variable	Distribution	Parameters
Body weight (*BW*; kg)	Normal	0 = 3.87; s = 0.322
Free metabolic rate (*FMR*; kcal/kg bw/d) a = *FMR*-slope b = *FMR*-power	$FMR = aBW^b$ Normal Lognormal	 *log a*: 0 = 1.67; s = 2.65 0 = 0.869; s = 0.116
Gross energy mammals (*GE*; kcal/kg)	Point Estimate	1700
Assimilation efficiency mammals (*AE*; unitless)	Point Estimate	0.84
Proportion mammals in diet (P_m; unitless)	Minmaxmean	0.518, 0.898, 0.761
Proportion forage time in Reach 5 (*FT*; unitless)	Point Estimate	0.5

Table J.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Red Fox

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Mammals	Reach 5	76	0.151	1.78	4.98	29.3	148	UCL > Max: No	Point Estimate UCL=80.6	Distribution-Free Range LCL=23.1 UCL=80.6

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

Table J.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Red Fox

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Mammals	Reach 5	12	14.7	179	290	1107	4,038	Co-elution issue: No Detection limit issue: No UCL > Max: Yes	Point Estimate Max=4,038	Distribution-Free Range Min=14.7 Max=4,038

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] If assumptions about concentrations for samples below the detection limit or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL and UCL were used in the probability bounds analysis.

Table J.2-6

Exposure Model Results for Red Fox Exposed to Total PCBs (mg/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
PSA	0.0220	82.5	6.25	0.434	2.68	16.7	0.106	0.607	3.52	0.702	3.54	23.5

Table J.2-7

Sensitivity Results From Monte Carlo Analysis of Red Fox Exposed to tPCBs and 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variables	Sensitivity Data*
tPCBs	Reach 5	FMR - Log10 Slope	0.698
		FMR Power Term	0.673
		Proportion of Mammals in the Diet	0.0495
		Body Weight (g)	-0.00233
2,3,7,8-TCDD TEQ	Reach 5	FMR - Log10 Slope	0.697
		FMR Power Term	0.677
		Proportion of Mammals in the Diet	0.0744
		Body Weight (g)	-0.0123

*Sensitivity determined by absolute value.

Table J.2-8

Exposure Model Results for Red Fox Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Reach 5	1.40	3,089	296	16.8	137	829	0.0672	0.386	2.24	35.2	177	1,175

Table J.2-9

Dietary Studies for the Northern Short-Tailed Shrew

Dietary Composition	Spring (%)	Summer (%)	Fall (%)	Winter (%)	All Year Average	Location Habitat	Reference
Centipedes		1.8				New York	Whitaker and Ferraro 1963, in EPA 1993
Earthworms		31.4					
Snails and slugs		27.1					
Insects		4.3					
Beetles		5.9					
Miscellaneous animals		8.1					
Endegon (fungi)		7.7					
Vegetation		5.4					
Other		8.6					
Crustaceans		2.22				Eastern U.S., primarily NY	Hamilton 1941, in EPA 1993
Centipedes		4.44					
Spiders and mites		3.66					
Earthworms		25.1					
Snails and slugs		3.42					
Insects		46.6					
Small Mammals		3.12					
Vegetation		10.3					
Other		1.62					

Table J.2-9

Dietary Studies for the Northern Short-Tailed Shrew (Continued)

Dietary Composition	Spring (%)	Summer (%)	Fall (%)	Winter (%)	All Year Average	Location Habitat	Reference
Earthworms		5		16.3		N. Car./Tenn.	Linzey and Linzey 1973
Snails and slugs		13		6.4			
Insects		60.8		23.3			
Endegon (fungi)		5.9					
Vegetation		0.3		5			
Crustaceans				8.95		Ithaca, NY Open fields	Eadie 1944
Centipedes				11.7			
Earthworms				15.6			
Insects				37.2			
Small Mammals				23.5			
Vegetation				3.11			
Crustaceans				2.07		Ithaca, NY Open fields	Eadie 1948
Centipedes				23.3			
Earthworms				11.4			
Snails and slugs				13			
Insects				35.8			
Small Mammals				9.33			
Vegetation				5.18			

Table J.2-10

Input Variables Used in the Monte Carlo Exposure Analysis for Total Daily Intake of tPCBs and TEQ by Northern Short-Tailed Shrews Inhabiting the Primary Study Area

Variable	Distribution	Parameters
Body weight (*BW*; kg)	Normal	0 = 0.0223; s = 0.00287
Free metabolic rate (FMR; kcal/kg bw/d) a = *FMR*-slope b = *FMR*-power	$FMR = aBW^b$ Normal Normal	 *log a*: 0 = 6.98; s = 1.32 0 = 0.622; s = 0.0630
Gross energy (*GE*; kcal/kg)		
Litter Invertebrates (GE_i)	Point Estimate	1,600
Earthworms (GE_w)	Point Estimate	805
Mammals (GE_m)	Point Estimate	1,700
Assimilation efficiency (*AE*; unitless)		
Litter Invertebrates (AE_i)	Point Estimate	0.87
Earthworms (AE_w)	Point Estimate	0.87
Mammals (AE_m)	Point Estimate	0.84
Proportion in diet (*P*; unitless)		
Litter invertebrates (P_i)	Beta	α = 12; β = 5.2; scale = 0.85
Earthworms (P_e)	Beta	α = 12; β = 4; scale = 0.25
Mammals (P_m)	Beta	α = 9; β = 8.5; scale = 0.235
Proportion forage time at locations 13, 14 and 15 (*FT*; unitless)	Point Estimate	1.0

Table J.2-11

Input Variables Used in the RiskCalc Probability Bounds Analysis for Total Daily Intake of tPCBs and TEQ by Northern Short-Tailed Shrews Inhabiting the Primary Study Area

Variable	Distribution	Parameters
Body weight (*BW*; kg)	Normal	0 = 0.0223; s = 0.00287
Free metabolic rate (*FMR*; kcal/kg bw/d) a = *FMR*-slope b = *FMR*-power	$FMR = aBW^b$ Normal Normal	 *log a*: 0 = 6.98; s = 1.32 0 = 0.622; s = 0.0630
Gross energy (kcal/kg)		
Litter invertebrates (*GE*)	Point Estimate	1600
Earthworms (GE_w)	Point Estimate	805
Mammals (GE_m)	Point Estimate	1700
Assimilation efficiency (unitless)		
Litter invertebrates (AE_i)	Point Estimate	0.87
Earthworms (AE_w)	Point Estimate	0.87
Mammals (AE_m)	Point Estimate	0.84
Proportion in diet (unitless)		
Litter invertebrates (P_i)	Minmaxmean	0.0910, 0.246, 0.190
Earthworms in diet (P_e)	Minmaxmean	0.269, 0.816, 0.600
Mammals in diet (P_m)	Minmaxmean	0.0376, 0.202, 0.121
Proportion forage time at locations 13, 14 and 15 (*FT; unitless*)	Point Estimate	1.0

Table J.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Northern Short-Tailed Shrews

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Earthworms	Location 13	10	11.7	17.5	20.7	24.3	26.7	UCL > Max: No	Point Estimate UCL=24.2	Distribution-Free Range LCL=18.0 UCL=24.2
	Location 14	10	10.3	14.6	17.3	19.7	23.3	UCL > Max: No	Point Estimate UCL=20.4	Distribution-Free Range LCL=15.0 UCL=20.4
	Location 15	10	1.10	1.15	1.47	1.62	2.91	UCL > Max: No	Point Estimate UCL=1.93	Distribution-Free Range LCL=1.33 UCL=1.93
Litter Invertebrates	Location 13	3	3.58	3.83	4.09	4.47	4.86	UCL > Max: N/A[c]	Point Estimate Max=4.86	Distribution-Free Range Min=3.58 Max=4.86
	Location 14	3	2.32	2.91	3.50	3.68	3.85	UCL > Max: N/A[c]	Point Estimate Max=3.85	Distribution-Free Range Min=2.32 Max=3.85
	Location 15	2	1.39	1.73	2.08	2.42	2.76	UCL > Max: N/A[c]	Point Estimate Max=2.76	Distribution-Free Range Min=1.39 Max=2.76

Table J.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Northern Short-Tailed Shrews (Continued)

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Mammals	Location 13	30	1.63	2.74	13.9	93.0	139	UCL > Max: Yes	Point Estimate Max=139	Distribution-Free Range Min=1.63 Max=139
	Location 14	30	0.151	2.22	4.98	45.8	148	UCL > Max: No	Point Estimate UCL=112	Distribution-Free Range LCL=17.7 UCL=112
	Location 15	16	0.192	0.396	0.573	2.47	10.7	UCL > Max: No	Point Estimate UCL=6.65	Distribution-Free Range LCL=1.24 UCL=6.65

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean.

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis. When the number of samples is below 5, the maximum concentration was used in the Monte Carlo analyses and the minimum and maximum concentrations were used in the probability bounds analysis.

[c] The maximum measured concentration was used as a point estimate in the Monte Carlo analysis because the sample size was too small to calculate confidence limits using the Land H-statistic.

Table J.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Northern Short-Tailed Shrews

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Earthworms	Location 13	1[c]	361	361	361	361	361	Co-elution issue: Yes Detection limit issue: No UCL > Max: N/A[d]	Triangular Distribution Min=207 (co-elutes=0%) Best=361 (co-elutes=50%) Max=516 (co-elutes=100%)	Distribution-Free Range Min=207 (co-elutes=0%) Max=516 (co-elutes=100%)
	Location 14	1[e]	249	249	249	249	249	Co-elution issue: Yes Detection limit issue: Yes UCL > Max: N/A[d]	Triangular Distribution Min=120 (ND=0, co-elutes=0%) Best=249 (ND=DL/2, co-elutes=50%) Max=377 (ND=DL, co-elutes=100%)	Distribution-Free Range Min=120 (ND=0, co-elutes=0%) Max=377 (ND=DL, co-elutes=100%)
	Location 15	1[f]	130	130	130	130	130	Co-elution issue: No Detection limit issue: Yes UCL > Max: N/A[d]	Triangular Distribution Min=87.4 (ND=0) Best=130 (ND=DL/2) Max=173 (ND=DL)	Distribution-Free Range Min=87.4 (ND=0) Max=173 (ND=DL)
Litter Invertebrates[g]	Location 13	N/A[g]	99.6	99.6	99.6	99.6	99.6	Co-elution issue: N/A Detection limit issue: N/A UCL > Max: N/A	Triangular Distribution Min=57.1 Best=99.6 Max=142	Distribution-Free Range Min=57.1 Max=142
	Location 14	N/A[g]	68.7	68.7	68.7	68.7	68.7	Co-elution issue: N/A Detection limit issue: N/A	Triangular Distribution Min=33.1	Distribution-Free Range Min=33.1

Table J.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Northern Short-Tailed Shrews (Continued)

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
								UCL > Max: N/A	Best=68.7 Max=104	Max=104
	Location 15	N/A[g]	35.9	35.9	35.9	35.9	35.9	Co-elution issue: N/A Detection limit issue: N/A UCL > Max: N/A	Triangular Distribution Min=24.1 Best=35.9 Max=47.7	Distribution-Free Range Min=24.1 Max=47.7
Mammals	Location 13	4	208	385	1,873	3,486	4,038	Co-elution issue: No Detection limit issue: No UCL > Max: N/A[b]	Point Estimate Max=4,038	Distribution-Free Range Min=208 Max=4,038
	Location 14	4	14.7	197	584	1,107	1,698	Co-elution issue: No Detection limit issue: No UCL > Max: N/A[b]	Point Estimate Max=1,698	Distribution-Free Range Min=14.7 Max=1,698
	Location 15	4	21.5	74.4	171	268	321	Co-elution issue: No Detection limit issue: No UCL > Max: N/A[b]	Point Estimate Max=321	Distribution-Free Range Min=21.5 Max=321

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] If assumptions about concentrations for samples below the detection limit or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL

Table J.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Northern Short-Tailed Shrews (Continued)

and UCL were used in the probability bounds analysis. When the number of samples was below 5, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis.

[c] Composite of 45 earthworms.

[d] Confidence limits on the mean could not be calculated because of inadequate sample size.

[e] Composite of 20 earthworms.

[f] Composite of 41 earthworms.

[g] Concentrations of TEQ were not measured in litter invertebrates sampled at Locations 13, 14, and 15. TEQ concentrations in litter invertebrates were estimated from earthworms based on the ratio of tPCBs in earthworms to litter invertebrates. Using concentrations from Locations 13, 14, and 15, the ratios of the geometric mean tPCBs concentrations in earthworms to litter invertebrates was 3.63. This ratio was applied to the measured TEQ concentration in earthworms to derive an estimated TEQ concentration in litter invertebrates.

Table J.2-14

Exposure Model Results for Northern Short-Tailed Shrews Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
13	3.11	25.5	12.2	7.11	11.5	18.9	2.79	5.07	9.25	15.0	23.4	37.8
14	2.56	20.8	9.94	5.82	9.36	15.4	2.55	4.57	8.16	12.4	19.3	31.2
15	0.419	2.62	1.34	0.84	1.29	2.01	0.260	0.457	0.793	1.15	1.78	2.87

Table J.2-15

Sensitivity Results From Monte Carlo Analysis of Northern Short-Tailed Shrews Exposed to tPCBs and 2,3,7,8-TCDD TEQ

Chemical	Location	Exposure Model Input Variables	Sensitivity Data*
tPCBs	13	FMR - Log10 Slope	0.701
		FMR - Power Term	0.475
		Proportion of litter invertebrates in diet	-0.401
		Proportion of mammals in diet	0.389
		Body Weight (g)	-0.125
		Proportion of worms in diet	0.00462
	14	FMR - Log10 Slope	0.702
		FMR - Power Term	0.475
		Proportion of litter invertebrates in diet	-0.401
		Proportion of mammals in diet	0.385
		Body Weight (g)	-0.125
		Proportion of worms in diet	0.00143
	15	FMR - Log10 Slope	0.785
		FMR - Power Term	0.530
		Body Weight (g)	-0.143
		Proportion of mammals in diet	0.109
		Proportion of litter invertebrates in diet	-0.104
		Proportion of worms in diet	0.00415
2,3,7,8-TCDD TEQ	13	FMR - Log10 Slope	0.667
		Proportion of mammals in diet	0.471
		FMR - Power Term	0.468
		Proportion of litter invertebrates in diet	-0.443
		Body Weight (g)	-0.136

Table J.2-15

Sensitivity Results From Monte Carlo Analysis of Northern Short-Tailed Shrews Exposed to tPCBs and 2,3,7,8-TCDD TEQ (Continued)

Chemical	Location	Exposure Model Input Variables	Sensitivity Data*
2,3,7,8-TCDD TEQ (Continued)		Concentration in worms (ng/kg)	0.107
		Proportion of worms in diet	-0.0594
		Concentration in litter invertebrates (ng/kg)	0.00395
	14	FMR - Log10 Slope	0.684
		FMR - Power Term	0.480
		Proportion of litter invertebrates in diet	-0.413
		Proportion of mammals in diet	0.401
		Body Weight (g)	-0.144
		Concentration in worms (ng/kg)	0.137
		Concentration in litter invertebrates (ng/kg)	0.0738
		Proportion of worms in diet	-0.00633
	15	FMR - Log10 Slope	0.730
		FMR - Power Term	0.508
		Proportion of litter invertebrates in diet	-0.323
		Proportion of mammals in diet	0.225
		Concentration in worms (ng/kg)	0.181
		Body Weight (g)	-0.150
		Proportion of worms in diet	0.104
		Concentration in litter invertebrates (ng/kg)	0.0808

* Sensitivity determined by absolute value.

Table J.2-16

Exposure Model Results for Northern Short-Tailed Shrews Exposed to 2,3,7,8-TCDD TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis						Probability Bounds Analysis					
							Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
13	73.0	635	288	162	271	458	35.5	63.5	113	369	574	928
14	41.8	304	143	82.6	136	225	19.0	34.3	62.4	218	338	547
15	12.2	94.4	46.1	27.4	43.8	71.2	14.0	25.2	45.7	81.4	126	202

Table J.3-1

Reference Values for Body Weight and Food Intake Rate for Mammals

Species	Body Weight (kg)	Food Intake Rate (kg/day)	Reference
White-footed mouse	0.022	0.0034	Green and Millar 1987; EPA 1985; Sample et al. 1996
Oldfield mouse	0.014	0.0019*	Silva and Downing 1995; Linzey 1987; Sample et al. 1996
Laboratory rat	0.35	0.028	EPA 1985; Sample et al. 1996
Mouse	0.03	0.0055	EPA 1985; Sample et al. 1996
Red fox	4.5	0.45	Storm et al. 1976; Sargent 1978; Vogtsberger and Barrett 1973; Sample et al. 1996
Short-tailed shrew	0.015	0.009	Schlesinger and Potter 1974; Barrett and Stueck 1976; Buckner 1964; Sample et al. 1996

*Assumed that food intake (0.135 g food/g b w/d) is comparable to that reported by Linzey (1987) for white-footed mouse (*Peromyscus leucopus*) and corrected for body weight by multiplying food intake with body weight.

Table J.3-2

Toxicity of tPCBs in Mammals

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Aroclor 1242						
Osborne-Mendel Rats		M	0	1	Mortality at 10 wks (0%) – control	Green et al. 1975
Osborne-Mendel Rats		M	625	1	Mortality at 10 wks (0%); changes in number of pregnancies, implantations, or dead implantations through 10 wks of mating with untreated females – NS	Green et al. 1975
Osborne-Mendel Rats		M	1,250	1	Mortality at 10 wks (10%); mean weight loss (5g); changes in number of pregnancies, implantations, or dead implantations through 10 wks of mating with untreated females – NS	Green et al. 1975
Osborne-Mendel Rats		M	2,500	1	Mortality at 10 wks (20%); mean weight loss (20g); changes in number of pregnancies, implantations, or dead implantations through 11 wks of mating with untreated females – NS	Green et al. 1975
Osborne-Mendel Rats		M	125	5	Mortality at 11 wks (10%); changes in number of pregnancies, implantations, or dead implantations through 11 wks of mating with untreated females – NS	Green et al. 1975
Osborne-Mendel Rats		M	250	5	Mortality at 11 wks (0%); mean weight loss (21g); changes in number of pregnancies or implantations through 11 wks of mating with untreated females – NS; increase in dead implantations at wk 11 – S	Green et al. 1975
Osborne-Mendel Rats		M	0	1	Control	Green et al. 1975
Osborne-Mendel Rats		M	0	5	Control	Green et al. 1975
Mill-Hill Rats		M	0	30	Weight gain (116%) – control	Neskovic et al. 1984
Mill-Hill Rats		F	0	30	Weight gain (110%) – control	Neskovic et al. 1984
Mill-Hill Rats		M	75	30	Weight gain (113%) – NS	Neskovic et al. 1984
Mill-Hill Rats		F	75	30	Weight gain (112%) – NS	Neskovic et al. 1984

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley Rats			4,250	14	LD_{50}	Bruckner et al. 1973
Aroclor 1254						
Osborne-Mendel Rats		M	0	5	Mortality (0%) at 10 wks – control	Green et al. 1975
Osborne-Mendel Rats		M	75	5	Mortality (0%) at 10 wks; mean weight loss (7g); change in number of pregnancies or implantations through 10 wks of mating with untreated females – NS; increase in dead implantations only at wk 5 – S; change in number of pregnancies or dead implantations through 11 wks of mating with untreated females – NS; decrease in implantations only at wks 3 and 5 – S	Green et al. 1975
Osborne-Mendel Rats		M	150	5	Mortality (0%) at 10 wks; mean weight loss (23g) Change in number of pregnancies or implantations through 10 wks of mating with untreated females – NS; increase in dead implantations only at wk 1 – S; change in number of pregnancies or dead implantations through 11 wks of mating with untreated females – NS; decrease in implantations only at wks 3 – S	Green et al. 1975
Osborne-Mendel Rats		M	300	5	Mortality (50%) at 10 wks; mean weight loss (56g); change in number of pregnancies or implantations through 10 wks of mating with untreated females – NS; increase in dead implantations only at wk 8 – S	Green et al. 1975
Wistar Rats	Adult	M	0	63	Mortality (0%); body weight (444+/-34g); pup weight at d 20 (33+/-4g) – control	Baker et al. 1977
Wistar Rats	Adult	M	6.40	63	Mortality (0%); body weight (440+/-33g); pup weight at d 20 (34+/-7g) – NS	Baker et al. 1977
Wistar Rats	Adult	F	0	63	Body weight (266+/-33g); pup weight at d 20 (41+/-11g) – control	Baker et al. 1977
Wistar Rats	Adult	F	6.40	63	Body weight (270+/-27g); pup weight at d 20 (34+/-7g) – NS	Baker et al. 1977

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Wistar Rats	30 d	M	1,400	1	LD_{50}	Grant and Phillips 1974
Wistar Rats	30 d	F	1,300	1	LD_{50}	Grant and Phillips 1974
Wistar Rats	60 d	M	1,400	1	LD_{50}	Grant and Phillips 1974
Wistar Rats	60 d	F	1,400	1	LD_{50}	Grant and Phillips 1974
Wistar Rats	120 d	M	2,000	1	LD_{50}	Grant and Phillips 1974
Wistar Rats	120 d	F	2,500	1	LD_{50}	Grant and Phillips 1974
Sherman-strain	3-4 wks	M	1,295	1	LD_{50}	Linder et al. 1974
Rats		M	0	20	Weight gain (114.9 +/- 18.0 g) – control	Garthoff et al. 1977
Rats		M	0.417[a]	20	Weight gain (108.0+/-13.1g) – NS	Garthoff et al. 1977
Rats		M	3.35	20	Weight gain (97.2+/-18.2g) – S	Garthoff et al. 1977
Rats		M	39.3	20	Weight gain (60.3+/-17.6g) – S	Garthoff et al. 1977
Sprague Dawley Rats	~ 30 d	F	0	39	Weight gain (89+/-5g) – control	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	0	39	Weight gain (181+/-4g) – control	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	0.600	39	Weight gain (184+/-4g) – NS	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	0.299	39	Weight gain (194+/-6g) – NS	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	7.70	39	Weight gain (188+/-9g) – NS	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	F	8.20	39	Weight gain (71+/-5g) – S	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	F	28.3	39	Weight gain (35+/-5g) – S	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	28.5	39	Weight gain (117+/-14g) – S	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	M	52.9	39	Weight gain (70+/-8g) – S	Kerkvliet and Kimeldorf 1977
Sprague Dawley Rats	~ 30 d	F	54.4	39	Weight gain (22+/-2g) – S	Kerkvliet and Kimeldorf 1977

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley Rats	~ 7 wks	M	0	5	Body weight (250.2 +/ -7.2g); feed intake (24.9 +/- 1.9g) – control	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	0	11	Body weight (280.7 +/ 2g); feed intake (23.3 +/- 2.0g) – control	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	0	19	Body weight (296.3 +/ -3.5g); feed intake (17.0 +/- 0.6g) – control	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	50	4	Feed intake (20.4 +/- 2.1g) – S	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	50	5	Body weight (261.0 +/ -9.1g); feed intake (21.2 +/- 3.1g) – NS	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	50	11	Body weight (257.4 +/ -5.7g); feed intake (15.0 +/- 1.1g) – S	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	50	19	Body weight (243.5 +/ -7.5g); feed intake (11.0 +/- 1.6g) – S	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	500	5	Body weight (225.3 +/- 3.3g); feed intake (9.5 +/- 1.5g) – S	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	500	11	Body weight (190.3 +/ -5.2g); feed intake (12.3 +/- 1.5g) – S	Komives 1979
Sprague Dawley Rats	~ 7 wks	M	500	19	Body weight (176.2 +/- 12.9g); feed intake (9.0 +/- 2.1g) – S	Komives 1979
Albino Rats		M	0	12	Weight gain (32.2+/-3.43g) – control	Chakraborty et al. 1978
Albino Rats		M	10	12	Weight gain (23.6+/-2.78g) – S	Chakraborty et al. 1978
Fischer-344 Rats	10 wks	M	0	35	Weight at d 35 (257+/-4g) – control	Carter and Koo 1984
Fischer-344 Rats	10 wks	M	3.64[a]	35	Weight at d 35 (269+/-8g) – NS	Carter and Koo 1984
Sherman Strain Rats	3-4 wks	F	0	240	Mean weight gain (484g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	6.80	240	Mean weight gain (459g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	36.4	240	Mean weight gain (315g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	0	240	Mean weight gain (244g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	7.50	240	Mean weight gain (235g)	Kimbrough et al. 1972

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats	3-4 wks	F	37.6	240	Mean weight gain (158g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	0	240	Mean weight gain (453g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	1.40	240	Mean weight gain (439g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	0	240	Mean weight gain (238g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	1.60	240	Mean weight gain (249g)	Kimbrough et al. 1972
Wistar Rats	30 d	M	0	60	Body weight (363 +/-6g)	Grant et al. 1974
Wistar Rats	30 d	M	0.139	60	Body weight (404 +/- 15g) – S	Grant et al. 1974
Wistar Rats	30 d	M	1.44	60	Body weight (390 +/- 8g) – S	Grant et al. 1974
Wistar Rats	30 d	M	8.78	60	Body weight (319 +/- 17g) – S	Grant et al. 1974
Wistar Rats	30 d	M	6.91	121	Body weight (405 +/- 15g) – S	Grant et al. 1974
Wistar Rats	30 d	M	5.78	246	Body weight (484 +/- 37g) – S	Grant et al. 1974
One generation study						
Sherman Strain Rats	3-4 wks	M/F	0	67[b]	# litters born (8); # litters weaned (7); litter size at birth (11.1); at weaning (10.6); survival (95.5%); mean bw at weaning (39.2g) – control	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	0	186[b]	# litters born (7); # litters weaned (7); litter size at birth (11.6); at weaning (11.6); survival (100%); mean bw at weaning (37.9g) – control	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	7.20	67[b]	# litters born (9) – NS; # litters weaned (8) – NS; litter size at birth (9.4) – NS; at weaning (8.1) – NS; survival (85.9%) – NS; mean bw at weaning (31.4g) – NS	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	7.20	186[b]	# litters born (8) – NS; # litters weaned (6) – NS; litter size at birth (11.8) – NS; at weaning (8.0) – NS; survival (68.1%) – S; mean bw at weaning (27.9g) – NS	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats	3-4 wks	M/F	37.0	67[b]	# litters born (4)[a] – NS; # litters weaned (0) – NS; litter size at birth (4.0) – S; survival (0%)	Linder et al. 1974
One generation study						
Sherman Strain Rats-F1a	3-4 wks	M/F	0	62	Survival (95.3%); mean bw at weaning (38.7g) – control	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0	67	Survival (99.0%); mean bw at weaning (38.5g) – control	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0.0600	67	Survival (98.6%) – NS; mean bw at weaning (42.3g) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0.320	67	Survival (99.5%) – NS; mean bw at weaning (42.1g) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	1.50	62	Survival (98.6%) – NS; mean bw at weaning (38.3g) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	7.60	62	Survival (96.1%) – NS; mean bw at weaning (32.9g) – NS	Linder et al. 1974
Two generation study						
Sherman Strain Rats-F1a	3-4 wks	M/F	0	62	# litters born (17); # litters weaned (17); litter size at birth (12.4); at weaning (11.8) – control	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0	67	# litters born (18); # litters weaned (18); litter size at birth (11.2); at weaning (11.1) – control	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0.0600	67	# litters born (16) – NS; # litters weaned (15) – NS; litter size at birth (9.2) – NS; at weaning (9.1) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0.320	67	# litters born (17) – NS; # litters weaned (17) – NS; litter size at birth (10.8) – NS; at weaning (10.8) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	1.50	62	# litters born (19) – NS; # litters weaned (19) – NS; litter size at birth (11.7) – NS; at weaning (11.5) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	7.60	62	# litters born (19) – NS; # litters weaned (19) – NS; litter size at birth (10.7) – S; at weaning (10.3) – NS	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats-F1b	3-4 wks	M/F	0	188	# litters born (17); # litters weaned (17); litter size at birth (12.3); at weaning (11.1); survival (90.4%); mean bw at weaning (37.1g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0	201	# litters born (18); # litters weaned (18); litter size at birth (12.3); at weaning (11.0); survival (89.2%); mean bw at weaning (40.7g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0.060	201	# litters born (18) – NS; # litters weaned (18) – NS; litter size at birth (11.1) – NS; at weaning (9.8) – NS; survival (88.0%) – NS; mean bw at weaning (38.0g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0.320	201	# litters born (20) – NS; # litters weaned (19) – NS; litter size at birth (11.1) – NS; at weaning (10.2) – NS; survival (91.4%) – NS; mean bw at weaning (38.9g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	1.50	188	# litters born (18) – NS; # litters weaned (18) – NS; litter size at birth (10.4) – S; at weaning (10.1) – NS; survival (96.3%) – NS; mean bw at weaning (39.0g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	7.60	188	# litters born (20) – NS; # litters weaned (20) – NS; litter size at birth (9.5) – S; at weaning (7.0) – NS; survival (73.6%) – NS; mean bw at weaning (32.1g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	0	129	# litters born (18); # litters weaned (18); litter size at birth (12.5); at weaning (12.2); survival (97.8%); mean bw at weaning (35.5g) – control	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	0	125	# litters born (19); # litters weaned (19); litter size at birth (11.7); at weaning (11.5); survival (97.8%); mean bw at weaning (37.4g) – control	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats-F2a	3-4 wks	M/F	0.0600	125	# litters born (15) – NS; # litters weaned (15) – NS; litter size at birth (11.7) – NS; at weaning (11.5) – NS; survival (98.9%) – NS; mean bw at weaning (37.2g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	0.320	125	# litters born (17) – NS; # litters weaned (17) – NS; litter size at birth (12.1) – NS; at weaning (11.7) – NS; survival (96.6%) – NS; mean bw at weaning (35.7g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	1.50	129	# litters born (17) – NS; # litters weaned (17) – NS; litter size at birth (10.6) – S; at weaning (10.1) – NS; survival (94.5%) – NS; mean bw at weaning (36.8g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	7.60	129	# litters born (7)[c] – NS; # litters weaned (4) – NS; litter size at birth (7.2) – S; at weaning (5.6) – NS; survival (77.8%) – S; mean bw at weaning (35.2g) – NS	Linder et al. 1974
Sherman Strain Rats-F2b	3-4 wks	M/F	0	274	# litters born (17); # litters weaned (16); litter size at birth (12.7); at weaning (11.3); survival (88.4%); mean bw at weaning (31.9g) – control	Linder et al. 1974
Sherman Strain Rats-F2b	3-4 wks	M/F	1.50	274	# litters born (12) – NS; # litters weaned (12) – NS; litter size at birth (9.6) – S; at weaning (8.5) – NS; survival (88.7%) – NS; mean bw at weaning (36.1g) – NS	Linder et al. 1974
Sherman Strain Rats-F2b	3-4 wks	M/F	7.60	274	# litters born (4)[c] – NS; # litters weaned (2) – NS; litter size at birth (3.5) – S; at weaning (3.5) – NS; survival (100%) - NS; mean bw at weaning (38.3g) - NS	Linder et al. 1974
Two generation study						
Sherman Strain Rats	Adult	F	0	9[d]	# litters born (9); # litters weaned (9); litter size at birth (12.3); at weaning (12.1); total pups/group born: dead (0), alive (111); total pups/group alive: at d 3 (110), at weaning (109); survival (98.2%); mean bw at weaning (37.9g) – control	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats	Adult	F	0	9[d]	# litters born (10); # litters weaned (10); litter size at birth (11.9); at weaning (11.8); total pups/group born: dead (0), alive (119); total pups/group alive: at d 3 (119), at weaning (118); survival (99.1%); mean bw at weaning (40.6g) – control	Linder et al. 1974
Sherman Strain Rats	Adult	F	10.0	9[d]	# litters born (9); # litters weaned (9); litter size at birth (12.1); at weaning (12.0); total pups/group born: dead (0), alive (109); total pups/group alive: at d 3 (109), at weaning (108); survival (99.1%); mean bw at weaning (37.6g)	Linder et al. 1974
Sherman Strain Rats	Adult	F	50.0	9[d]	# litters born (10); # litters weaned (10); litter size at birth (13.4); at weaning (12.7); total pups/group born: dead (0), alive (134); total pups/group alive: at d 3 (131), at weaning (127); survival (94.8%); mean bw at weaning (30.5g)	Linder et al. 1974
Sherman Strain Rats	Adult	F	100	9[d]	# litters born (7); # litters weaned (5); litter size at birth (11.9); at weaning (3.6); total pups/group born: dead (8), alive (83); total pups/group alive: at d 3 (64), at weaning (25); survival (30.1%) – S; mean bw at weaning (30.8g)	Linder et al. 1974
Sprague Dawley Rats		F	0	9	Fetal survival rate/litter at birth (79.54 +/- 3.84%); avg. fetal weight/litter at birth (7.02 +/- 0.12g) – control	Spencer 1982
Sprague Dawley Rats		F	1.87	9	Fetal survival rate/litter at birth (66.21 +/- 9.42%) – NS; avg. fetal weight/litter at birth (6.84 +/- 0.08g) – NS	Spencer 1982
Sprague Dawley Rats		F	3.74	9	Fetal survival rate/litter at birth (65.87 +/- 10.33%) – NS; avg. fetal weight/litter at birth (6.67 +/- 0.06g) – NS	Spencer 1982
Sprague Dawley Rats		F	7.47	9	Fetal survival rate/litter at birth (65.38 +/- 12.20%) – NS; avg. fetal weight/litter at birth (6.19 +/- 0.15g) – S	Spencer 1982
Sprague Dawley Rats		F	15.2	9	Fetal survival rate/litter at birth (63.05 +/- 8.44%) – NS; avg. fetal weight/litter at birth (5.61 +/- 0.10g) – S	Spencer 1982
Sprague Dawley Rats		F	17.0	9	Fetal survival rate/litter at birth (28.03 +/- 12.29%) – S; avg. fetal weight/litter at birth (5.58 +/- 0.19g) – S	Spencer 1982

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley Rats		F	25.7	9	Fetal survival rate/litter at birth (17.14 +/- 11.28%) – S; no data reported for avg. fetal weight	Spencer 1982
Sprague Dawley Rats		F	31.7	9	Fetal survival rate/litter at birth (0.00 +/- 0.00); no survivors	Spencer 1982
Wistar Rats	Adult	F	0.00033	7	Body weight (292 +/- 11g) – control	Overmann et al. 1987
Wistar Rats	Adult	F	0.0435	7	Body weight (271 +/- 11g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.426	7	Body weight (271 +/-10g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	2.83	7	Body weight (283 +/- 8g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.00033	20	Body weight (374 +/- 13g) – control	Overmann et al. 1987
Wistar Rats	Adult	F	0.0435	20	Body weight (253 +/- 14g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.426	20	Body weight (347 +/- 12g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	2.83	20	Body weight (346 +/- 13g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.00033	20	# litters born: alive (10.3 +/- 0.9), dead (1.7 +/- 0.7); bw of live pups (5.5 +/- 0.1g) – control	Overmann et al. 1987
Wistar Rats	Adult	F	0.0435	20	# litters born: alive (9.6 +/- 1.2), dead (1.6 +/- 1.1); bw of live pups (5.7 +/- 0.1g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.426	20	# litters born: alive (8.5 +/- 1.4), dead (0.9 +/- 0.4); bw of live pups (5.7 +/- 0.2) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	2.83	20	# litters born: alive (10.5 +/- 1.6) – NS, dead (1.2 +/- 0.6) – NS; bw of live pups (4.9 +/- 0.3) – S	Overmann et al. 1987
Wistar Rats	Adult	F	0.00033	7	Pre-weaning bw (13.3 +/- 0.4g) – control	Overmann et al. 1987
Wistar Rats	Adult	F	0.0435	7	Pre-weaning bw (12.8 +/- 0.5g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.426	7	Pre-weaning bw (12.1 +/- 0.4g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.00033	21	Pre-weaning bw (37.3 +/- 1.0g) – control	Overmann et al. 1987

Table J.3-2

Toxicity of tPCBs in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Wistar Rats	Adult	F	0.0435	21	Pre-weaning bw (35.8 +/- 1.3g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0.426	21	Pre-weaning bw (34.1 +/- 1.4g) – S	Overmann et al. 1987
Wistar Rats	Weanlings	M/F	0.00033	42	82% survival to weaning	Overmann et al. 1987
Wistar Rats	Weanlings	M/F	0.0435	42	87% survival to weaning	Overmann et al. 1987
Wistar Rats	Weanlings	M/F	0.426	42	85% survival to weaning	Overmann et al. 1987
Wistar Rats	Weanlings	M/F	2.83	42	3/4 pups died at 7 d of age	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.00033	21	Body weight (37.2 +/- 1.3g) – control	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.00033	21	Body weight (36.0 +/- 1.9g) – control	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.00033	150	Body weight (255 +/- 10g) – control	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.00033	150	Body weight (465 +/- 19g) – control	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.0435	21	Body weight (33.5 +/- 3.2g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.0435	21	Body weight (37.6 +/- 2.3g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.0435	150	Body weight (245 +/- 7g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.0435	150	Body weight (439 +/- 13g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.426	21	Body weight (35.0 +/- 2.1g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.426	21	Body weight (31.8 +/- 2.8g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	F	0.426	150	Body weight (264 +/- 11g) – NS	Overmann et al. 1987
Wistar Rats	Weanlings	M	0.426	150	Body weight (432 +/- 11g) – NS	Overmann et al. 1987
Wistar Rats	Adult	F	0	9	Avg. litter size (9); avg. litter weight (4.80g); resorption sites/litter (0.33) – control	Villeneuve et al. 1971
Wistar Rats	Adult	F	6.25	9	Avg. litter size (10) – NS; avg. litter weight (4.70g) – NS; resorption sites/litter (1.20) – NS	Villeneuve et al. 1971

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Wistar Rats	Adult	F	12.5	9	Avg. litter size (10) – NS; avg. litter weight (4.78g) – NS; resorption sites/litter (0.40) – NS	Villeneuve et al. 1971
Wistar Rats	Adult	F	25.0	9	Avg. litter size (10) – NS; Avg. litter weight (4.70g) – NS; resorption sites/litter (0.70) – NS	Villeneuve et al. 1971
Wistar Rats	Adult	F	50.0	9	Avg. litter size (9) – NS; avg. litter weight (4.56g) – NS; resorption sites/litter (0.44) – NS	Villeneuve et al. 1971
Wistar Rats	Adult	F	100	9	Avg. litter size (7) – NS; avg. litter weight (4.44g) – S; resorption sites/litter (0.71) – NS	Villeneuve et al. 1971
Oldfield mice (P1)	6-10 wks	M/F	0	365	Mean litter size (3.62 +/- 0.32); birth weight (1.85 +/- 0.08g); offspring born/m: all pairs (0.62), fertile pairs (1.07); litters born/m: all pairs (0.17), fertile pairs (0.31); weaning weight (10.27 +/- 0.96g); survival to weaning (60%); weaned/m: all pairs (0.36), fertile pairs (0.59) – control	McCoy et al. 1995
Oldfield mice (P1)	6-10 wks	M/F	0.679	365	Mean litter size (3.80 +/- 0.31); birth weight (1.61 +/- 0.07g) – S; offspring born/m: all pairs (0.71), fertile pairs (1.11); litters born/m: all pairs (0.19), fertile pairs (0.29); weaning weight (8.94 +/- 0.30g) – S; survival to weaning (55%); weaned/m: all pairs (0.37), fertile pairs (0.57)	McCoy et al. 1995
Oldfield mice (F1)	Weanlings	M/F	0	365[e]	Mean litter size (3.85 +/- 0.36); birth weight (1.96 +/- 0.08g); offspring born/m: all pairs (0.67), fertile pairs (1.33); litters born/m: all pairs (0.13), fertile pairs (0.31); weaning weight (9.76 +/- 0.29g); survival to weaning (94%); weaned/m: all pairs (0.49), fertile pairs (1.11) – control	McCoy et al. 1995
Oldfield mice (F1)	Weanlings	M/F	0.679	365[e]	Mean litter size (3.78 +/- 0.37); birth weight (1.67 +/- 0.11g) – S; offspring born/m: all pairs (0.20), fertile pairs (0.47); litters born/m: all pairs (0.09), fertile pairs (0.15); weaning weight (8.29 +/- 0.44g); survival to weaning (23%); weaned/m: all pairs (0.19), fertile pairs (0.30)	McCoy et al. 1995
Oldfield mice (P1 and F1)	Adult	M/F	0.679	365	Whole body residues (0.31 +/- 0.13 mg/kg) – controls	McCoy et al. 1995

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Oldfield mice (P1)	Adult	M/F	0.679	365	Whole body residues (3.28 +/- 2.00 mg/kg)	McCoy et al. 1995
Oldfield mice (F1)	Adult	M/F	0.679	365	Whole body residues (7.51 +/- 3.36 mg/kg)	McCoy et al. 1995
Oldfield mice (F2)	Adult	M/F	0.679	365	Whole body residues (14.69 +/- 7.64 mg/kg)	McCoy et al. 1995
Swiss-Webster Mice	6-8 wks	F	0	126	Mean weight (33.95 +/- 1.41g); dams producing litters (78%); litter size (8.40 +/ -0.51); number of litters (25); pup weight/litter from treated dams at weaning (3 wk): (13.62 +/- 0.43); mean body weight of offspring of treated dams (31.09 +/- 0.86g) – control	Talcott and Koller 1983
Swiss-Webster Mice	6-8 wks	F	0.167	126	Mean weight (33.97 +/- 1.41g) – NS; dams producing litters (82%) – NS; litter size (8.35 +/ -0.69) – NS; number of litters (14) – NS; pup weight/litter from treated dams at weaning (3 wks): (13.28 +/- 0.57) – NS; mean body weight of offspring of treated dams (29.56 +/- 1.00g) – NS	Talcott and Koller 1983
Swiss-Webster Mice	6-8 wks	F	16.7	126	Mean weight (34.25 +/- 1.41g) – NS; dams producing litters (83%) – NS; litter size (6.40 +/ -0.58) – NS; number of litters (20) – NS; pup weight/litter from treated dams at weaning (3 wks): (14.08 +/- 0.48) – NS; mean body weight of offspring of treated dams (32.11 +/- 0.91g) – NS	Talcott and Koller 1983
Swiss-Webster Mice	6-8 wks	F	41.7	126	Mean weight (38.38 +/- 1.41g) – S; dams producing litters (77%) – NS; litter size (7.65 +/- 0.54) – NS; number of litters (23) – NS; pup weight/litter from treated dams at weaning (3 wks): (12.65 +/- 0.44) – NS; mean body weight of offspring of treated dams (30.12 +/- 0.97g) – NS	Talcott and Koller 1983
White-footed mice	Wild-caught adults	M/F	0	540	Birth weight (1.9 +/- 0.03g) at 4 wks (13.0 +/- 0.10g), at 8 wks (18.1 +/-0.47g), at 12 wks (19.9 +/- 0.32g) – control	Linzey 1988
White-footed mice	Wild-caught adults	M/F	1.55	540	Birth weight (1.8 +/- 0.04g) at 4 wks (11.0 +/- 0.14g), at 8 wks (15.9 +/- 0.33g), at 12 wks (17.2 +/- 0.21g)	Linzey 1988

Table J.3-2

Toxicity of tPCBs in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
White-footed mice	Lab-raised 12 wks	M/F	0	450	Birth weight (1.8 +/- 0.04g) at 4 wks (12.6 +/- 0.07g), 8 wks (18.1 +/- 0.18g), at 12 wks (19.4 +/- 0.34g) – control	Linzey 1988
White-footed mice	Lab-raised 12 wks	M/F	1.55	450	Birth weight (1.8 +/- 0.07g) at 4 wks (11.0 +/- 0.42g), at 8 wks (17.2 +/- 0.36g), at 12 wks (16.7 +/- 0.43g)	Linzey 1988
White-footed mice	Lab-raised 16 wks	M/F	0	270	Birth weight (1.8 +/- 0.03g) at 4 wks (12.8 +/- 0.17g), at 8 wks (18.2 +/- 0.24g)	Linzey 1988
White-footed mice	Lab-raised 16 wks	M/F	1.55	270	Birth weight (1.8 +/- 0.03g) at 4 wks (11.2 +/- 0.74g), at 8 wks (17.4 +/- 0.55g)	Linzey 1988
White-footed mice	Wild-caught adults	M/F	0	540	Pairs breeding (60%); # litters/breeding female (8.8 +/- 1.20) – control; # young/litter at birth (4.4 +/- 0.22); # young/litter at 28 d (3.6 +/- 0.30) – control	Linzey 1987
White-footed mice	Wild-caught adults	M/F	1.55	540	Pairs breeding (70%); # litters/breeding female (9.9 +/- 2.27); # young/litter at birth (4.0 +/- 0.12); # young/litter at 28 d (2.6 +/- 0.22) – S	Linzey 1987
White-footed mice	Lab raised 12 wks	M/F	0	450	Pairs breeding (53%); # litters/breeding female (6.5 +/- 1.64); birth interval: 33.0 +/- 1.06 d; # young/litter at birth (4.8 +/- 0.15); # young/litter at 28 d (4.7 +/- 0.18) – control	Linzey 1987
White-footed mice	Lab raised 12 wks	M/F	1.55	450	Pairs breeding (28%); # litters/breeding female (3.8 +/- 0.97); birth interval: 44.7 +/- 6.2 d – S; # young/litter at birth (3.6 +/- 0.23) – S; # young/litter at 28 d (2.0 +/- 0.38) – S	Linzey 1987
White-footed mice	Lab raised 16 wks	M/F	0	225-270	Pairs breeding (68%); # litters/breeding female (3.8 +/- 0.76); # young/litter at birth (4.6 +/- 0.20); # young/litter at 28 d (2.8 +/- 0.30) – control	Linzey 1987
White-footed mice	Lab raised 16 wks	M/F	1.55	225-270	Pairs breeding (40%); # litters/breeding female (3.6 +/- 1.02); # young/litter at birth (4.6 +/- 0.28); # young/litter at 28 d (0.5 +/- 0.21) – S	Linzey 1987

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
White-footed mice	44-222 d	M/F	0	60	Survival of pairs at d 60 (100%); total pairs having at least one litter (84.6%); total pairs having two litters (42.3%); total litters produced in 60 d (33); mean # litters for all pairs at beginning of experiment (1.31 +/- 0.14); mean # litters for all pairs surviving 60 d (1.31 +/- 0.14); avg. # young/litter of known size (3.82 +/- 0.21) – control	Merson and Kirkpatrick 1976
White footed mice	44-222 d	M/F	30.9	60	Survival of pairs at d 60 (66.7%); total pairs having at least one litter (29.6%); total pairs having two litters (3.7%); total litters produced in 60 d (9); mean # litters for all pairs at beginning of experiment (0.33 +/- 0.06); mean # litters for all pairs surviving 60 d (0.28 +/- 0.14); avg. # young/litter of known size (3.33 +/- 0.33)	Merson and Kirkpatrick 1976
Aroclor 1260						
Sherman Strain Rats	3-4 wks	F	0	240	Mortality (0%); mean weight gain (256g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	0	240	Mortality (0%); mean weight gain (451g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	6.50	240	Mortality (0%); mean weight gain (463g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	7.20	240	Mortality (10%); mean weight gain (253g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	32.8	240	Mortality (0%); mean weight gain (398g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	38.2	240	Mortality (20%); mean weight gain (185g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	71.4	240	Mortality (0%); mean weight gain (351g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	72.4	240	Mortality (80%); mean weight gain (148g) – only 2 animals survived	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	0	240	Mean weight gain (435g) – control	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	M	1.40	240	Mean weight gain (455g)	Kimbrough et al. 1972
Sherman Strain Rats	3-4 wks	F	0	240	Mean weight gain (226g) – control	Kimbrough et al. 1972

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats	3-4 wks	F	1.60	240	Mean weight gain (233g)	Kimbrough et al. 1972
Sherman-strain	3-4 wks	M	1,315	1	LD50	Linder et al. 1974
One generation study						
Sherman Strain Rats	3-4 wks	M/F	0	67[f]	# litters born (9); # litters weaned (9); litter size at birth (12.3); at weaning (12.2); survival (99.1%); mean bw at weaning (37.0g) – control	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	0	186[f]	# litters born (9); # litters weaned (8); litter size at birth (12.1); at weaning (10.3); survival (85.3%); mean bw at weaning (40.9g) – control	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	6.90	67[f]	# litters born (8); # litters weaned (8); litter size at birth (11.6) ; at weaning (11.6) – NS; survival (100%) – NS; mean bw at weaning (35.6g) – NS	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	6.90	186[f]	# litters born (5); # litters weaned (5); litter size at birth (11.0); at weaning (10.8) – NS; survival (98.2%) – NS; mean bw at weaning (36.2g) – NS	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	35.4	67[f]	# litters born (8) – NS; # litters weaned (3) – NS; litter size at birth (8.5) – S; at weaning (3.3) – NS; survival (38.2%) – S; mean bw at weaning (36.3g) – NS	Linder et al. 1974
Sherman Strain Rats	3-4 wks	M/F	35.4	186[f]	# litters born (6) – NS; # litters weaned (2) – NS; litter size at birth (6.7) – S; at weaning (2.3) – NS; survival (35.0%) – NS; mean bw at weaning (40.7g) – NS	Linder et al. 1974
Two generation study						
Sherman Strain Rats-F1a	3-4 wks	M/F	0	68	# litters born (19); # litters weaned (19); litter size at birth (11.7); at weaning (11.2); survival (95.5%); mean bw at weaning (40.9g) – control	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats-F1a	3-4 wks	M/F	0	71	# litters born (19); # litters weaned (19); litter size at birth (11.7); at weaning (11.4); survival (97.3%); mean bw at weaning (33.8g) – control	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	0.390	71	# litters born (18); # litters weaned (18); litter size at birth (12.1); at weaning (11.8); survival (97.7%); mean bw at weaning (38.5g) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	1.50	68	# litters born (17); # litters weaned (17); litter size at birth (11.8); at weaning (11.3); survival (95.0%); mean bw at weaning (39.8g) – NS	Linder et al. 1974
Sherman Strain Rats-F1a	3-4 wks	M/F	7.40	68	# litters born (17); # litters weaned (17); litter size at birth (11.6) – NS; at weaning (11.2); survival (97.0%); mean bw at weaning (38.0g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0	187	# litters born (17); # litters weaned (17); litter size at birth (11.1); at weaning (11.0); survival (98.9%); mean bw at weaning (40.8g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0	188	# litters born (17); # litters weaned (16); litter size at birth (10.3); at weaning (9.2); survival (89.1%); mean bw at weaning (36.1g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	0.390	188	# litters born (16); # litters weaned (15); litter size at birth (11.2); at weaning (10.6); survival (95.0%); mean bw at weaning (39.3g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	1.50	187	# litters born (16); # litters weaned (16); litter size at birth (11.9); at weaning (11.4); survival (95.8%); mean bw at weaning (41.0g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	3-4 wks	M/F	7.40	187	# litters born (13); # litters weaned (13); litter size at birth (10.4); at weaning (10.1); survival (97.0%); mean bw at weaning (39.2g) – NS	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats-F2a	3-4 wks	M/F	0	127	# litters born (13); # litters weaned (13); litter size at birth (11.8); at weaning (11.6); survival (98.7%); mean bw at weaning (34.8g) – control	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	0	128	# litters born (18); # litters weaned (18); litter size at birth (10.9); at weaning (10.3); survival (93.9%); mean bw at weaning (40.3g) – control	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	0.390	127	# litters born (18); # litters weaned (18); litter size at birth (11.8); at weaning (11.6); survival (98.1%); mean bw at weaning (38.5g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	1.50	128	# litters born (19); # litters weaned (19); litter size at birth (11.3); at weaning (11.1); survival (98.1%); mean bw at weaning (38.7g) – NS	Linder et al. 1974
Sherman Strain Rats-F2a	3-4 wks	M/F	7.40	128	# litters born (20); # litters weaned (20); litter size at birth (10.1); at weaning (9.9); survival (98.0%); mean bw at weaning (38.6g) – NS	Linder et al. 1974
Two generation Study						
Sherman Strain Rats-F1b	5 m – old	F	0	179	Terminal bw (297g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	5 m – old	M	0	179	Terminal bw (472g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	5 m – old	F	1.50	179	Terminal bw (304g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	5 m – old	M	1.50	179	Terminal bw (494g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	5 m – old	F	7.40	179	Terminal bw (304g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	5 m – old	M	7.40	179	Terminal bw (509g) – NS	Linder et al. 1974
Sherman Strain Rats-F1b	7 m – old	F	0	190	Terminal bw (279g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	7 m - old	M	0	190	Terminal bw (492g) – control	Linder et al. 1974
Sherman Strain Rats-F1b	7 m – old	F	0.390	190	Terminal bw (286g) – NS	Linder et al. 1974

Table J.3-2

Toxicity of tPCBs in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (mg/kg bw/d)	Exposure	Endpoint	Reference
Sherman Strain Rats-F1b	7 m – old	M	0.390	190	Terminal bw (505g) – NS	Linder et al. 1974
Sherman Strain Rats-Fo	9 m – old	F	0	250	Terminal bw (322g) – control	Linder et al. 1974
Sherman Strain Rats-Fo	9 m – old	M	0	250	Terminal bw (563g) – control	Linder et al. 1974
Sherman Strain Rats-Fo	9 m – old	F	0.390	250	Terminal bw (331g) – NS	Linder et al. 1974
Sherman Strain Rats-Fo	9 m - old	M	0.390	250	Terminal bw (567g) – NS	Linder et al. 1974
Sherman Strain Rats	Adult	F	0	9	# litters born (12); # litters weaned (12); litter size at birth (12.0); at weaning (11.5); total pups/group: born dead (4), alive (144); total pups/group: alive at 3 d (140), at weaning (138), survival (95.8%); mean bw at weaning (33.9g) – control	Linder et al. 1974
Sherman Strain Rats	Adult	F	100	9	# litters born (12); # litters weaned (11); litter size at birth (11.7); at weaning (10.3); total pups/group: born dead (10), alive (140); total pups/group: alive at 3 d (135), at weaning (124), survival (88.6%); mean bw at weaning (33.9g)	Linder et al. 1974

BW = Body weight; M = male; F = female; NS = not significant; S = significant

[a] Calculated from food ingestion and BWs provided in the text.

[b] Exposure ended at time of mating.

[c] Two litters had no live offspring.

[d] Dosed on days 7-15 of pregnancy.

[e] Offspring from control and exposed parents were maintained on parental diets for 12 months.

[f] Secondary exposure prepared from tissues of two Jersey cows orally dosed 1 or 10 mg Aroclor 1254/kg/d for 10 d.

Table J.3-3

Toxicity of TEQ in Mammals

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
2,3,7,8-tetrachrolorodibenzo-p-dioxin (TCDD)						
Long Evans rats	10-14 wks	M	4,000	21	Mortality (0%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	M	8,000	21	Mortality (17%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	M	12,000	21	Mortality (33%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	M	16,000	21	Mortality (43%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	M	20,000	21	Mortality (57%)	Pohjanvirta et al. 1993
Long Evans rats			9,800		LD_{50}	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	F	4, 000	21	Mortality (0%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	F	8, 000	21	Mortality (17%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	F	12,000	21	Mortality (83%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	F	16,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats	10-14 wks	F	20,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats			17,700		LD_{50}	Pohjanvirta et al. 1993
Han/Wistar		M	1,200 000	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar		M	2,400 000	21	Mortality (20%)	Pohjanvirta et al. 1993
Han/Wistar		M	4,800 000	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar		M	6,000 000	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar		M	7,200 000	21	Mortality (20%)	Pohjanvirta et al. 1993
Han/Wistar		F	1,200 000	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar		F	2,400 000	21	Mortality (20%)	Pohjanvirta et al. 1993

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Han/Wistar		F	4,800 000	21	Mortality (20%)	Pohjanvirta et al. 1993
Han/Wistar		F	6,000 000	21	Mortality (20%)	Pohjanvirta et al. 1993
Han/Wistar		F	7,200 000	21	Mortality (40%)	Pohjanvirta et al. 1993
Han/Wistar			>7,200 000		LD_{50}	Pohjanvirta et al. 1993
Sprague Dawley rats		M	20,000	1	Mortality (0%)	Stahl et al. 1992
Sprague Dawley rats		M	30,000	1	Mortality (20%)	Stahl et al. 1992
Sprague Dawley rats		M	40,000	1	Mortality (40%)	Stahl et al. 1992
Sprague Dawley rats		M	60,000	1	Mortality (80%)	Stahl et al. 1992
Sprague Dawley rats					LD_{50}	Stahl et al. 1992
Sprague Dawley rats			0	546	Mortality (60%)	Van Miller et al. 1977
Sprague Dawley rats			0.0428	546	Mortality (20%)	Van Miller et al. 1977
Sprague Dawley rats			0.143	546	Mortality (40%)	Van Miller et al. 1977
Sprague Dawley rats			1.43	546	Mortality (40%)	Van Miller et al. 1977
Sprague Dawley rats			14.3	546	Mortality (50%)	Van Miller et al. 1977
Sprague Dawley rats			57.1	546	Mortality (100%)	Van Miller et al. 1977
Sprague Dawley rats			286	546	Mortality (100%)	Van Miller et al. 1977
Sprague Dawley rats			3,430	546	Mortality (100%)	Van Miller et al. 1977
Sprague Dawley rats			34,300	546	Mortality (100%)	Van Miller et al. 1977
Sprague Dawley rats			71,400	546	Mortality (100%)	Van Miller et al. 1977
Holtzman rats	70	M	0	1	Fertility index[a] (100%)	Mably et al. 1992a
Holtzman rats	70	M	64	1	Fertility index[a] (100%)	Mably et al. 1992a

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Holtzman rats	70	M	160	1	Fertility index[a] (100%)	Mably et al. 1992a
Holtzman rats	70	M	400	1	Fertility index[a] (89%)	Mably et al. 1992a
Holtzman rats	70	M	1,000	1	Fertility index[a] (89%)	Mably et al. 1992a
Holtzman rats		F	0	1	NR	Mably et al. 1992a
Holtzman rats		F	64	1	NR	Mably et al. 1992a
Holtzman rats		F	160	1	NR	Mably et al. 1992a
Holtzman rats		F	400	1	NR	Mably et al. 1992a
Holtzman rats		F	1,000	1	Fetus mortality at day 20 of gestation (6%); fetus mortality at day 21 of gestation (19%); mortality at birth (15%)	Mably et al. 1992a
Holtzman rats		F	0	1	NR	Mably et al. 1992a
Holtzman rats		F	64	1	NR	Mably et al. 1992a
Holtzman rats		F	160	1	NR	Mably et al. 1992a
Holtzman rats		F	400	1	NR	Mably et al. 1992a
CRCD rats	6-7 wks	F	0	14	Pre-implantation loss (11.7%); live fetuses at 21d of gestation (15.1+/- 2.1) ; post-implantation loss (2.9%); fetal weight 3.5 +/- 0.26	Giavini et al. 1983
CRCD rats	6-7 wks	F	125	14	Pre-implantation loss (8.5%); live fetuses at 21d of gestation (14.2 +/- 2.2); post-implantation loss (3.6%); fetal weight (3.6 +/- 0.23)	Giavini et al. 1983

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
CRCD rats	6-7 wks	F	500	14	Pre-implantation loss (13.7%); live fetuses at 21d of gestation (13.4 +/- 3.3); post-implantation loss (10.2%); fetal weight 3.5 +/-0.36	Giavini et al. 1983
CRCD rats	6-7 wks	F	2,000	14	Pre-implantation loss (19.5%); live fetuses at 21d of gestation (8.3 +/- 5.7); post-implantation loss (30.3%); fetal weight 2.8 +/- 0.6 - S	Giavini et al. 1983
Long Evans rats	12-13 wks		0	1	Resorptions (17.1%)	Huuskonen et al. 1994
Long Evans rats	12-13 wks		1,000	1	Resorptions (11.7%)	Huuskonen et al. 1994
Long Evans rats	12-13 wks		5,000	1	Resorptions (70.2%)	Huuskonen et al. 1994
Wistar rats		F	0 untreated	10	Live fetuses at 22 d gestation (10.7); mean viable pups per litter (12)	Khera and Ruddick 1973
Wistar rats		F	0 treated	10	Live fetuses at 22 d gestation (11); mean viable pups per litter (11.6)	Khera and Ruddick 1973
Wistar rats		F	125	10	Live fetuses at 22 d gestation (10.6); mean viable pups per litter (10.9)	Khera and Ruddick 1973
Wistar rats		F	250	10	Live fetuses at 22 d gestation (10.9); mean viable pups per litter (10.2)	Khera and Ruddick 1973
Wistar rats		F	500	10	Live fetuses at 22 d gestation (10.5); mean viable pups per litter (9.9)	Khera and Ruddick 1973

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Wistar rats		F	1,000	10	Live fetuses at 22 d gestation (9.3); mean viable pups per litter (5.9)	Khera and Ruddick 1973
Wistar rats		F	0 untreated	10	Live fetuses at 22 d gestation (11.5)	Khera and Ruddick 1973
Wistar rats		F	0 treated	10	Live fetuses at 22 d gestation (9.8)	Khera and Ruddick 1973
Wistar rats		F	1,000	10	Live fetuses at 22 d gestation (6.5)	Khera and Ruddick 1973
Wistar rats		F	2,000	10	Live fetuses at 22 d gestation (6)	Khera and Ruddick 1973
Wistar rats		F	4,000	10	Live fetuses at 22 d gestation (0)	Khera and Ruddick 1973
Wistar rats		F	8,000	10	Live fetuses at 22 d gestation (0)	Khera and Ruddick 1973
Wistar rats		F	16,000	10	Live fetuses at 22 d gestation (0)	Khera and Ruddick 1973
Wistar rats		F	0 untreated	10	Mean # stillborn (0.4); mean pup birth weight (6.38 g); pup survival at 22 d of age (96%); mean weight at 21 d of age (31.99 g)	Khera and Ruddick 1973
Wistar rats		F	0 treated	10	Mean # stillborn (0.2); mean pup birth weight (6.54 g); pup survival at 22 d of age (88%); mean weight at 21 d of age (30.22 g)	Khera and Ruddick 1973

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Wistar rats		F	125	10	Mean # stillborn (0.3); mean pup birth weight (6.51 g); pup survival at 22 d of age (93%); mean weight at 21 d of age (29.91 g)	Khera and Ruddick 1973
Wistar rats		F	250	10	Mean # stillborn (0.4); mean pup birth weight (5.97 g); pup survival at 22 d of age (90%); mean weight at 21 d of age (30.96 g)	Khera and Ruddick 1973
Wistar rats		F	500	10	Mean # stillborn (1); mean pup birth weight (5.70 g); pup survival at 22 d of age (60%); mean weight at 21 d of age (24.99 g)	Khera and Ruddick 1973
Wistar rats		F	1,000	10	Mean # stillborn (2.4); mean pup birth weight (5.18 g); pup survival at 22 d of age (0%); pup mortality at 21 d of age (100%)	Khera and Ruddick 1973
Sprague Dawley rats	130 d	F	0	10	Total viable fetuses at 20 d of gestation (246, mean: 10 +/- 4.3); mean # of resorption (63 (3 +/-2.8); mean maternal bw (0.423 kg)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	30	10	Total viable fetuses at 20 d of gestation (115, mean: 12 +/- 3.8); mean # of resorptions (31 (3 +/- 2.0); mean maternal bw (0.416 kg)	Sparschu et al. 1971

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley rats	130 d	F	125	10	Total viable fetuses at 20 d of gestation (124, mean: 12 +/- 2.9); mean # of resorptions (19 (2 +/- 1.1); mean maternal bw (0.435 kg)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	500	10	Total viable fetuses at 20 d of gestation (93, mean 8 +/- 4.1) – S; mean # of resorptions (58 (5 +/- 2.3) – S; mean maternal bw (0.388 kg) – S	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	2,000	10	Total viable fetuses at 20 d of gestation (7); mean # of resorptions (129 (12 +/- 3.9) – S; mean maternal bw (0.327 kg) - S	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	8,000	10	Total viable fetuses at 20 d of gestation (0); mean # of resorptions (110 (14 +/- 1.9) – S; mean maternal bw (0.284 kg) – S	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	0	10	Fetal bw (3.89 +/- 0.39 g)	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	30	10	Fetal bw (3.98 +/- 0.35 g)	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	125	10	Fetal bw (3.71 +/- 0.37 g)	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	500	10	Fetal bw (3.78 +/- 0.54 g)	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	2,000	10	Fetal bw (2.69 +/- 0.19 g) – S	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	F	8,000	10	No live fetuses	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	M	0	10	Fetal bw (4.03 +/- 0.37 g)	Sparschu et al. 1971

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley rats	20 d of gestation	M	30	10	Fetal bw (4.14 +/- 0.26 g)	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	M	125	10	Fetal bw (3.85 +/- 0. 35 g) – S	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	M	500	10	Fetal bw (3.86 +/- 0.61 g) – S	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	M	2,000	10	Fetal bw (2.72 +/- 0.25 g) – S	Sparschu et al. 1971
Sprague Dawley rats	20 d of gestation	M	8,000	10	No live fetuses	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	0	10	Fertility index[b]: 24/31 (77%)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	30	10	Fertility index[b]: 10/13 (77%)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	125	10	Fertility index[b]: 10/13 (77%)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	500	10	Fertility index[b]: 12/14 (86%)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	2,000	10	Fertility index[b]: 11/14 (78%)	Sparschu et al. 1971
Sprague Dawley rats	130 d	F	8,000	10	Fertility index[b]: 8/10 (80%)	Sparschu et al. 1971
Three generation study						
Sprague Dawley rats F1a	6-7 wks	M/F	0	90 d +[c]	Avg. litter size (8); pups alive at birth 106/111 (95%); pups surviving to 21 d of age 99/106 (93%)	Murray et al. 1979
Sprague Dawley rats F1a	6-7 wks	M/F	1	90 d +[c]	Avg. litter size (10); pups alive at birth 87/97 (90%); pups surviving to 21 d of age 73/87 (84%) – S	Murray et al. 1979
Sprague Dawley rats F1a	6-7 wks	M/F	10	90 d +[c]	Avg. litter size (8); pups alive at birth 93/103 (90%); pups surviving to 21 d of age 63/93 (68%) – S	Murray et al. 1979

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley rats F1a	6-7 wks	M/F	100	90 d +[c]	Avg. litter size (4) – S; pups alive at birth 0/7 – S; pups surviving to 21 d of age 0	Murray et al. 1979
Sprague Dawley rats F1b	6-7 wks	M/F	0	90 d +[c]	Avg. litter size (11); pups alive at birth 215/225 (96%); pups surviving to 21 d of age 160/215 (74%)	Murray et al. 1979
Sprague Dawley rats F1b	6-7 wks	M/F	1	90 d +[c]	Avg. litter size (10); pups alive at birth 120/135 (89%); pups surviving to 21 d of age 110/120 (92%)	Murray et al. 1979
Sprague Dawley rats F1b	6-7 wks	M/F	10	90 d +[c]	Avg. litter size (10); pups alive at birth 138/144 (96%); pups surviving to 21 d of age 93/138 (71%)	Murray et al. 1979
Sprague Dawley rats F1b	6-7 wks	M/F	100	90 d +[d]	Avg. litter size (one litter only) (6); pups alive at birth 5/6 (83%); pups surviving to 21 d of age 4/5 (80%)	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	0	90 d +[d]	Avg. litter size (11); pups alive at birth 235/244 (96%); pups surviving to 21 d of age 205/235 (87%)	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	1	90 d +[d]	Avg. litter size (11); pups alive at birth 148/166 (89%) – S; pups surviving to 21 d of age 126/148 (85%)	Murray et al. 1979

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley rats F2	6-7 wks	M/F	10	90 d +[d]	Avg. litter size (8); pups alive at birth 87/101 (86%) – S; pups surviving to 21 d of age 51/87 (59%) – S	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	100	90 d +[d]	Avg. litter size (0) – S; pups alive at birth 0; pup mortality at birth (100%)	Murray et al. 1979
Sprague Dawley rats F3	6-7 wks	M/F	0	90 d +[d]	Avg. litter size (11); pups alive at birth 296/318 (93%); pups surviving to 21 d of age 235/296 (79%)	Murray et al. 1979
Sprague Dawley rats F3	6-7 wks	M/F	1	90 d +[d]	Avg. litter size (11); pups alive at birth 208/224 (93%); pups surviving to 21 d of age 163/208 (78%)	Murray et al. 1979
Sprague Dawley rats F3	6-7 wks	M/F	10	90 d +[d]	Avg. litter size (9); pups alive at birth 83/100 (83%) – S; pups surviving to 21 d of age 64/83 (77%)	Murray et al. 1979
Sprague Dawley rats F3	6-7 wks	M/F	100	90 d +[c]	Avg. litter size (0) – S; pups alive at birth 0; pup mortality at birth (100%)	Murray et al. 1979
Sprague Dawley rats F0-first mating	6-7 wks	M/F	0	90 d +[c]	14/32 (44%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F0-first mating	6-7 wks	M/F	1	90 d +[c]	10/20 (50%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F0-first mating	6-7 wks	M/F	10	90 d +[c]	12/20 (60%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F0-first mating	6-7 wks	M/F	100	90 d +[c]	3/31 (10%): fertility index[d] – S	Murray et al. 1979
Sprague Dawley rats F0-2nd mating	6-7 wks	M/F	0	90 d +[c]	21/32 (66%): fertility index[d]	Murray et al. 1979

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Sprague Dawley rats F0-2nd mating	6-7 wks	M/F	1	90 d +[c]	14/19 (74%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F0-2nd mating	6-7 wks	M/F	10	90 d +[c]	15/20 (75%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F0-2nd mating	6-7 wks	M/F	100	90 d +[c]	1/30 (3%): fertility index [d] – S	Murray et al. 1979
Sprague Dawley rats F1	6-7 wks	M/F	0	90 d +[c]	22/26 (87%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F1	6-7 wks	M/F	1	90 d +[c]	15/17 (85%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F1	6-7 wks	M/F	10	90 d +[c]	12/21 (59%): fertility index[d] – S	Murray et al. 1979
Sprague Dawley rats F1	6-7 wks	M/F	100	90 d +[c]	0 :fertility index[d]	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	0	90 d +[c]	28/32 (88%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	1	90 d +[c]	20/20 (100%): fertility index[d]	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	10	90 d +[c]	11/20 (55%): fertility index[d] – S	Murray et al. 1979
Sprague Dawley rats F2	6-7 wks	M/F	100	90 d +[c]	0: fertility index[d]	Murray et al. 1979
Holtzman rats		F	0	1		Bjerke and Peterson 1994
Holtzman rats		F	1,000	1	30% mortality at birth – S	Bjerke and Peterson 1994
Holtzman rats		F	0	1	7% mortality based on number of implantation sites	Bjerke et al. 1994
Holtzman rats		F	700	1	23% mortality based on number of implantation sites; 89-92% decreased bw of male offspring at 7 d of age	Bjerke et al. 1994
Holtzman rats		F	0	1	2.8 - 18.1% pup mortality at birth	Flaws et al. 1997
Holtzman rats		F	1,000	1	15.8 - 39.6% pup mortality at birth	Flaws et al. 1997

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Long Evans rats		F	0	1	0% reduction in pup survival at 22 d of age	Gray et al. 1995
Long Evans rats		F	1,000	1	22% reduction in pup survival at 22 d of age	Gray et al. 1995
Holtzman rats		F	0	1	2.5% reduction in pup survival at 22 d of age	Gray and Ostby 1995
Holtzman rats		F	1,000	1	50% reduction in pup survival at 22 d of age	Gray and Ostby 1995
Long Evans rats		F	0	1	Litter 1: 88% +/- 6% produced a litter	Gray and Ostby 1995
Long Evans rats		F	0	1	Litter 2: 78% +/- 9% produced a litter	Gray and Ostby 1995
Long Evans rats		F	0	1	Litter 3: 76% +/- 10% produced a litter	Gray and Ostby 1995
Long Evans rats		F	0	1	Litter 4: 61% +/- 10% produced a litter	Gray and Ostby 1995
Long Evans rats		F	0	1	Litter 5: 61% +/- 10% produced a litter	Gray and Ostby 1995
Long Evans rats		F	1,000	1	Litter 1: 64% +/- 16% produced a litter	Gray and Ostby 1995
Long Evans rats		F	1,000	1	Litter 2: 51% +/- 13% produced a litter	Gray and Ostby 1995
Long Evans rats		F	1,000	1	Litter 3: 47% +/- 14% produced a litter	Gray and Ostby 1995
Long Evans rats		F	1,000	1	Litter 4: 28% +/- 16% produced a litter	Gray and Ostby 1995

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Long Evans rats		F	1,000	1	Litter 5: 19% +/- 11% produced a litter	Gray and Ostby 1995
NMRI mice		F	0	4		Nau et al. 1986
NMRI mice		F	12,500	4	45% reduction in pup survival at 1 d of age; 68.5% reduction in pup survival at 14 d of age; 75% reduction in pup survival at 22 d of age	Nau et al. 1986
1,2,3,7,8-pentachlorodibenzo-p-dioxin						
Long Evans rats	16-18 wks	F	0	21	Mortality (0%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	20,000	21	Mortality (0%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	60,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	180,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats			20,000-60,000		LD_{50}	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	0	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	180,000	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	1,620 000	21	Mortality (17%)	Pohjanvirta et al. 1993
Han/Wistar			> 1,620 000		LD_{50}	Pohjanvirta et al. 1993
Sprague Dawley rats		M	100,000	1	Mortality (0%)	Stahl et al. 1992
Sprague Dawley rats		M	150,000	1	Mortality (30%)	Stahl et al. 1992
Sprague Dawley rats		M	200,000	1	Mortality (40%)	Stahl et al. 1992
Sprague Dawley rats		M	300,000	1	Mortality (80%)	Stahl et al. 1992
Sprague Dawley rats			206,000		LD_{50}	Stahl et al. 1992

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
1,2,3,4,7,8-hexachlorodibenzo-p-dioxin						
Long Evans rats	16-18 wks	F	0	21	Mortality (0%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	12,000	21	Mortality (17%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	36,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats	16-18 wks	F	108,000	21	Mortality (100%)	Pohjanvirta et al. 1993
Long Evans rats			12,000-36,000		LD_{50}	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	0	21	Mortality (0%)	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	108,000	21	Mortality (17%)	Pohjanvirta et al. 1993
Han/Wistar	16-18 wks	F	324,000	21	Mortality (83%)	Pohjanvirta et al. 1993
Han/Wistar			187,100		LD_{50}	Pohjanvirta et al. 1993
Sprague Dawley rats		M	70,000	1	Mortality (20%)	Stahl et al. 1992
Sprague Dawley rats		M	100,000	1	Mortality (80%)	Stahl et al. 1992
Sprague Dawley rats		M	140,000	1	Mortality (80%)	Stahl et al. 1992
Sprague Dawley rats			88,700		LD_{50}	Stahl et al. 1992
1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin						
Sprague Dawley rats		M	30,000	1	Mortality (100%)	Stahl et al. 1992
Sprague Dawley rats		M	50,000	1	Mortality (20%)	Stahl et al. 1992
Sprague Dawley rats		M	80,000	1	Mortality (80%)	Stahl et al. 1992
Sprague Dawley rats			63,250		LD_{50}	Stahl et al. 1992
3,3',4,4'-tetrachlorobiphenyl (PCB 77)						
Albino mice (CD-1)	60-90 d	F	0	10	Implant resorptions (8.4%); Mean # live fetuses/dam (11.6)	Marks et al. 1989

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Albino mice (CD-1)	60-90 d	F	100	10	Implant resorptions (8.5%); Mean # live fetuses/dam (11.9)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	200	10	Implant resorptions (7.4%); Mean # live fetuses/dam (11.9)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	400	10	Implant resorptions (7.0%); Mean # live fetuses/dam (12.6)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	800	10	Implant resorptions (10.2%); Mean # live fetuses/dam (11.4)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	1,600	10	Implant resorptions (16.4%); Mean # live fetuses/dam (9.1)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	3,200	10	Implant resorptions (57.1%); Mean # live fetuses/dam (7.5)	Marks et al. 1989
Albino mice (CD-1)	60-90 d	F	6,400	10	Implant resorptions (82.5%); Mean # live fetuses/dam (3.5)	Marks et al. 1989
Sprague Dawley rats		F	0	13	Resorptions (8%)	Wardell et al. 1982
Sprague Dawley rats		F	100	13	Resorptions (3%)	Wardell et al. 1982
Sprague Dawley rats		F	300	13	Resorptions (14%)	Wardell et al. 1982
Sprague Dawley rats		F	1,000	13	Resorptions (8%)	Wardell et al. 1982
Two generation study						
C57BL/6J mice (F0)	4 d	F	0	37	Weanling survival (47.4 +/-34.7)	Huang 1998b
C57BL/6J mice (F0)	4 d	F	60	37	Weanling survival (41.6+/-39.1)	Huang 1998b
C57BL/6J mice (F0)	4 d	F	700	37	Weanling survival (30.9 +/- 30.9)	Huang 1998b

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
C57BL/6J mice (F0)	21 d	F	0	54	Weanling survival (96.3 +/- 11.1[a])	Huang 1998b
C57BL/6J mice (F0)	21 d	F	60	54	Weanling survival (88 +/- 31.6[a])	Huang 1998b
C57BL/6J mice (F0)	21 d	F	700	54	Weanling survival (75.8 +/- 37.7)[e]	Huang 1998b
C57BL/6J mice (F1)	4 d	F	0	est. 91	Weanling survival (46.4 +/-43.0)	Huang 1998b
C57BL/6J mice (F1)	4 d	F	60	est. 91	Weanling survival (0)	Huang 1998b
C57BL/6J mice (F1)	4 d	F	700	est. 91	Weanling survival (0)	Huang 1998b
C57BL/6J mice (F0)	4 d	F	0	31	Fecundity (80%)	Huang 1998b
C57BL/6J mice (F0)	4 d	F	60	31	Fecundity (71%)	Huang 1998b
C57BL/6J mice (F0)	4 d	F	700	31	Fecundity (47%)	Huang 1998b
C57BL/6J mice (F1)	4 d	F	0	est. 87	Fecundity (50%)	Huang 1998b
C57BL/6J mice (F1)	4 d	F	60	est. 87	Fecundity (40%)	Huang 1998b
C57BL/6J mice (F1)	4 d	F	700	est. 87	Fecundity (70%)	Huang 1998b
3,3',4,4',5-pentachlorobiphenyl (PCB-126)						
C57BL/6 (B6) mice	3 m	F	0	1	# of resorptions: 7 (9%)	d'Argy et al. 1987
C57BL/6 (B6) mice	3 m	F	2,500 000	1	# of resorptions 26 (37%) – S	d'Argy et al. 1987
C57BL/6 (B6) mice	3 m	F	5,000 000	1	# of resorptions 48 (53%) – S	d'Argy et al. 1987
C57BL/6 (B6) mice	3 m	F	10,000 000	1	# of resorptions 25 (34%) – S	d'Argy et al. 1987
2,3,4,7,8-pentachlorodibenzofuran						
Fischer 344 rats	2 m	F	0	1	Fetal mortality at 20 d gestation (2.78% +/- 2.78%)	Couture et al. 1989

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Fischer 344 rats	2 m	F	15,000	1	Fetal mortality at 20 d gestation (0%)	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Fetal mortality at 20 d gestation (9.93% +/- 3.95)	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Fetal mortality at 20 d gestation (90.53% +/- 5.3%) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	0	1	Mean fetal weight (3.31 +/- 0.06 g)	Couture et al. 1989
Fischer 344 rats	2 m	F	15,000	1	Mean fetal weight (3.13 +/- 0.05 g) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Mean fetal weight (2.88 +/- 0.03 g) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Mean fetal weight (2.22 +/- 0.07 g) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	0	1	Fetal mortality at 20 d of gestation (0%)	Couture et al. 1989
Fischer 344 rats	2 m	F	15,000	1	Fetal mortality at 20 d of gestation (7.83% +/- 4.84%)	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Fetal mortality at 20 d of gestation (3.5% +/- 2.59%)	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Fetal mortality at 20 d of gestation (91.67% +/- 5.69%) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	0	1	Mean fetal weight 3.34 +/- 0.15 g	Couture et al. 1989

Table J.3-3

Toxicity of TEQ in Mammals (Continued)

Species	Life Stage	Sex	Daily Dose (ng TEQ/kg bw/d)	Exposure	Endpoint	Reference
Fischer 344 rats	2 m	F	15,000	1	Mean fetal weight 3.28 +/- 0.13 1 g	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Mean fetal weight 2.76 +/- 0.03 g – S	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Mean fetal weight 2.59 +/- 0.26 g – S	Couture et al. 1989
Fischer 344 rats	2 m	F	5,000	1	Fetal mortality at 20 d of gestation (4.24% +/- 3.35%)	Couture et al. 1989
Fischer 344 rats	2 m	F	15,000	1	Fetal mortality at 20 d of gestation (1.11% +/- 1.11%)	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Fetal mortality at 20 d of gestation (8.64% +/- 2.11%)	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Fetal mortality at 20 d of gestation (81.14% +/- 6.44%) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	0	1	Mean fetal weight (3.23 +/- 0.06 g)	Couture et al. 1989
Fischer 344 rats	2 m	F	5,000	1	Mean fetal weight (3.31 +/- 0.07 g)	Couture et al. 1989
Fischer 344 rats	2 m	F	15,000	1	Mean fetal weight (3.05 +/- 0.04 g) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	50,000	1	Mean fetal weight (2.91 +/- 0.03 g) – S	Couture et al. 1989
Fischer 344 rats	2 m	F	150,000	1	Mean fetal weight (2.67 +/- 0.14 kg) – S	Couture et al. 1989

Table J.3-3

Toxicity of TEQ in Mammals
(Continued)

M = male; F = female; NS = not significant; S = significant; *NR = not reported*

[a] Number of males that impregnated females/number of males that mated x 100.

[b] Number of pregnancies/number of rats bred.

[c] Exposure for 90 days before first mating then continuous exposure for three generations.

[d] Number of females delivering a litter/number of females placed with a male x 100.

[e] Compared to pups viable at day 4.

Table J.4-1

1999 Small Mammal Reproduction Data Collected from the Biological Survey

Species	Location	Number of Adult Females	Total Number of Placental Scars	Placental Scars/Female
White-footed mouse (*Peromyscus leucopus)*	Location 13	9	57	6.33
	Location 14	11	69	6.27
	Location 15	6	39	6.50
Short-tailed shrew (*Blarina brevicauda)*	Location 13	4	3	0.75
	Location 14	6	15	2.50
	Location 15	2	0	0
Meadow jumping mouse (*Zapus hudsonius)*	Location 14	2	2	1
Masked shrew (*Sorex cinereus)*	Location 15	4	2	0.50

Table J.4-2

Summary of Exceedance Probabilities for Omnivorous and Carnivorous Mammals from the Housatonic River Study Area

Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LB		*FOMC*		*UB*		*LB*		*FOMC*		*UB*	
	LD	UD	LD	UD	LD	UD	LD	UD	LD	UD	LD	UD
Red Fox												
Reach 5	12	6	44.6	31.1	54	38	0	0	44.8	25.6	54	33
Short-Tailed Shrew												
Location 13	86	45	99.8	93.3	100	100	0	0	3.76	0	52	3
Location 14	81	37	100	97.8	100	100	0	0	0	0	8	1
Location 15	0	0	0	0	8	1	0	0	0	0	0	0

LB = Lower Probability Bound.

FOMC = First Order Monte Carlo.

UB = Upper Probability Bound.

LD = Lower Toxicity Dose (10% effect).

UD = Upper Toxicity Dose (20% effect).

Table J.4-3

Summary of Qualitative Risk Statements for Omnivorous and Carnivorous Mammals from the Housatonic River Study Area

Mammal/Location	Qualitative Risk Statements				
	TPCBs			TEQ	
	Risk Category	Risk Range		Risk Category	Risk Range
Red Fox					
Reach 5	Intermediate	Low/Intermediate		Intermediate	Low/Intermediate
Short-Tailed Shrew					
Location 13	High	Intermediate/High		Low	Low/Intermediate
Location 14	High	Intermediate/High		Low	Low/Low
Location 15	Low	Low/Intermediate		Low	Low/Low

Table J.4-4

Grids, tPCB Classification, and PSA Area

Grid	Level of tPCB Contamination	Area
1	High	Northern
2	Low	Northern
3	High	Southern
4	High	Southern
5	Low	Southern
6	Low	Southern

Table J.4-5

Soil tPCB Concentrations from Shrew Trapping Grids and Corresponding Survival Data per 30 Days[a] from Trapping Sessions

tPCB Concentrations in Soil				Shrew Survival Data For Trapping Sessions From July To September[b]			
				Males		Females	
Grid	Spatially Averaged Concentrations[b]	Supplemental Analysis – Arithmetic Mean[c]	Supplemental Analysis – Spatially Weighted Arithmetic Mean[c]	% Survival	Number	% Survival	Number
1	33.5	22.6	27.2	74.5	21	67.7	17
2	2.5	3.55	8.09	63.9	19	53.1	17
3	17.6	22.4	43.5	100.0	1	63.5	14
4	38.3	34.3	34.7	38.6	13	40.5	6
5	2.2	4.7	1.63	79.5	11	74.2	9
6	1.5	1.07	2.58	76.2	8	83.2	11

[a] A survival rate of 75% per 30 days means that half the population disappears every 72.3 days.

[b] Boonstra 2002.

[c] Derived as part of this ERA.

Table J.4-6

Population Densities (± 1 SE) per Hectare of Short-Tailed Shrew Using the Jackknife Estimator From CAPTURE for Trapping Grids on the Housatonic River Below Pittsfield, Massachusetts, 2001 (Boonstra 2002)

	Trapping Session		
Grid	**May – June**	**July**	**September**
1	8 ±2.6	67 ± 7.2	39 ±
2	18 ± 3.4	61 ± 7.1	26 ± 4.1
3		24 ± 4.5	25 ± 3.5
4		31 ± 5.2	36 ± 5.2
5	21 ± 3.6	22 ± 2.8	19 ± 1.8
6		23 ± 3.1	28 ± 3.5

Table J.4-7

Weighting of Measurement Endpoints for Omnivorous and Carnivorous Mammals Weight-of-Evidence Evaluation

Attributes	Field Surveys	Population Demography Field Study*	Modeled Exposure and Effects for tPCBs and TEQ	Rationale
I. Relationship Between Measurement and Assessment Endpoints				
1. Degree of Association	L	M/H	M	Field surveys did not quantitatively evaluate survival, growth, and reproduction of omnivorous and carnivorous mammals. The population demography field study measured population demography during in situ exposure. Exposure models were species specific, but effects data were for surrogate mammalian species.
2. Stressor/Response	M	M	M/H	Field surveys determined presence, relative abundance, size, and reproductive status; however, exposure-response relationship was likely relatively insensitive to magnitude of exposure. The population demography field study determined stressor-response for short-tailed shrew. However, confounding factors were not assessed and may have had significant effects on population demographics and the results of the field study. Additional analyses also cast doubt on this field study. Exposure modeling was species and stressor specific; dose-response models for representative species were available, but the effects line of evidence evaluated different receptors.
3. Utility of Measure	L/M	M/H	M/H	The field surveys used, while standardized and location specific, had low statistical power and were incapable of detecting subtle effects. The population demography field study was location specific, species specific, and key measurements were documented. However, additional analyses cast doubt on results for survival to shrew parameter. Modeled exposure and effects procedures used are standardized and widely accepted, and dose-response models were well defined; the primary limitation was lack of species-specific effects data.

Table J.4-7

Weighting of Measurement Endpoints for Omnivorous and Carnivorous Mammals Weight-of-Evidence Evaluation (Continued)

Attributes	Field Surveys	Population Demography Field Study*	Modeled Exposure and Effects for tPCBs and TEQ	Rationale
II. Data Quality				
4. Data Quality	H	M/H	M/H	The field surveys and the population demography field study were performed according to well-defined and documented protocols. However, for the population demography field study, some confounding factors such as habitat quality were likely considerations for explaining variation in response observed at the six trapping locations. The DQOs established for the sampling and analysis of tissue samples were met for the tissue residue data used in the exposure analyses for both tPCBs and TEQ. Other model parameters were derived from EPA (1993) and other published journal articles, and well defined effects metrics (i.e., dose-response models) were derived from peer reviewed scientific literature.
III. Study Design				
5. Site Specificity	H	H	L/M	Mammal surveys were conducted in the PSA and, in the case of snow tracking and scent posts, had appropriate reference areas. The population demography field study was performed in situ. Published studies were used as benchmarks. Biological tissue data used in exposure models were site specific, and other exposure parameters were representative of site conditions. However, effects measures were laboratory based and not site specific.
6. Sensitivity	M	M	H	Field surveys and the population demography field study were not sensitive to potential subtle effects associated with COCs, but would be expected to detect effects and order of magnitude or greater. Modeled exposure and effects directly assess exposure-response relationship. Laboratory studies from which effects data were derived were stressor specific.

Table J.4-7

Weighting of Measurement Endpoints for Omnivorous and Carnivorous Mammals Weight-of-Evidence Evaluation (Continued)

Attributes	Field Surveys	Population Demography Field Study*	Modeled Exposure and Effects for tPCBs and TEQ	Rationale
7. Spatial Representativeness	H	H	M	Mammal surveys and the population demography field study had complete coverage of the study area, measurement locations, stressors, receptors, and points of exposure. Modeled exposures relied on tissue data collected throughout the study area, and areas of actual exposure. Effects assessment used toxicity studies conducted in laboratories.
8. Temporal Representativeness	M/H	H	M	Mammal surveys were conducted during two field seasons when effects were most likely to be observed. The population demography field study was performed over one year only, but covered three seasons. Modeled exposure and effects lines of evidence spanned critical life stages. In general, tissue data used for modeling were not collected during the breeding season.
9. Quantitative Measure	M	M/H	H	Mammal surveys were quantitative, but not amenable to statistical tests between sites because of low replication. The population demography field study was quantitative. Probabilistic exposure and effects modeling were highly quantitative and propagated uncertainty associated with modeling procedures.
10. Standard Method	H	H	M/H	Mammal surveys were conducted followed standard accepted methods, and were applicable to the assessment endpoint. Field study followed standard accepted methods. Generally accepted exposure and effects modeling procedures were followed, but probability bounds analysis is a relatively new technique for propagating uncertainty.
Overall Endpoint Value	**M/H**	**M/H**	**M/H**	---

* Field study only for short-tailed shrew.
L = low
M = moderate
H = high

Table J.4-8

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	Moderate/High	Undetermined	Low
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate
Modeled Exposure and Effects	Moderate/High	Yes (Shrew) Undetermined (Red Fox)	High Intermediate

NA = not applicable

Table J.4-9

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	Moderate/High	Undetermined	Low
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate
Modeled Exposure and Effects	Moderate/High	No (Shrew) Undetermined (Red Fox)	Low Intermediate

NA = not applicable

Table J.4-10

Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint:	Survival, growth, and reproduction of omnivorous and carnivorous mammals

Harm/Magnitude	**Weighting Factors (increasing confidence of weight)**				
	Low	**Low/Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High				MEE-S	
Yes/Intermediate					
Yes/Low					
Undetermined/High					
Undetermined/Intermediate				MEE-F, PDFS	
Undetermined/Low				FS	
No/Low					
No/Intermediate					
No/High					

FS=Field surveys
MEE-S = Modeled exposure and effects – shrew
MEE-F = Modeled exposure and effects – red fox
PDFS = Population demography field study for short-tailed shrew only

Table J.4-11

Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint:	Survival, growth, and reproduction of omnivorous and carnivorous mammals

Harm/Magnitude	Weighting Factors (increasing confidence of weight)				
	Low	**Low/Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High					
Yes/Intermediate					
Yes/Low					
Undetermined/High					
Undetermined/Intermediate				MEE-F, PDFS	
Undetermined/Low				FS	
No/Low				MEE-S	
No/Intermediate					
No/High					

FS = Field surveys
MEE-S = Modeled exposure and effects-shrew
MEE-F = Modeled exposure and effects-red fox
PDFS = Population demography field study for short-tailed shrew only

Table J.4-12
Comparison of Representative and Other Species of Omnivorous and Carnivorous Mammals in the Housatonic River PSA

Representative Species	Other Species	Size	Residency	Longevity	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
Short-tailed Shrew		20.5 g (Housatonic data)	Year-round	94% live <1 yr in wild. 33 months in captivity.	0.0242-0.202	23.3-60.8% insects, 11.4-15.6% earthworms, 6.4-13.0% snails and slugs, 0-8.9% crustaceans, 023.5% sm. mammals, 5.9% *Endogone* (fungi), 0.3-5% vegetation. Summer diet for all seasons: 19% worms, 60% insects, 12% mammals.	Occurs in damp woodlands and fields.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995; EPA, 1993; Whitaker and Ferraro, 1963; Hamilton, 1941; Linzey and Linzey, 1973; Eadie, 1944; Eadie, 1948
	Smoky shrew	3.5-5.5 g	Year-round	18 months	0.0404	Insectivorous; also salamanders, young mice, vegetable matter (winter).	Habitat preferences and feeding habits similar to short-tailed shrew.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995
	Masked shrew	4.0-6.5 g	Year-round	21 months in captivity.	0.162-0.283	Predominantly insectivorous; also mollusks, annelids, dead bodies of larger animals, salamanders, young mice, *Endogone*, vegetable matter (winter).	Similar diet but different habitat than short-tailed shrews. Most common in dryer uplands; meadows, old fields, and fencerows.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995

Table J.4-12

Comparison of Representative and Other Species of Omnivorous and Carnivorous Mammals in the Housatonic River PSA (Continued)

Representative Species	Other Species	Size	Residency	Longevity	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
Red Fox		3.4-6.4 kg	Year-round	3-7 years max.	60-600	As much as 30% plants in summer and fall, the remainder being small mammals, birds, insects. Mammals 76% of average diet for all seasons.	Prefers open agricultural land and forest edges.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995; EPA, 1993; Martin et al., 1951; Powell and Case, 1982; Knable, 1974; Korschgen, 1959; Hockman and Chapman, 1983; Dibello et al., 1990
	Coyote	9.1-22.7 kg	Year-round	6-8 years	1000-4000	78% mammals, 21% fruit, 10% insects, and 3% birds by frequency in 1500 scats from Adirondacks.	Broad habitat requirements, open fields, agricultural land, forested areas.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995; Martin et al., 1951
	Gray Fox	3.2-5.9 kg	Year-round	6-10 years max.	85-3200	85-95% animal matter throughout the year (e.g., rabbit, squirrel).	Most die by age 2, longevity record is for captive animals. Most common in forested areas.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995; Martin et al., 1951

Table J.4-12

Comparison of Representative and Other Species of Omnivorous and Carnivorous Mammals in the Housatonic River PSA (Continued)

Representative Species	Other Species	Size	Residency	Longevity	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
Red Fox (Continued)	Fisher	3.6-5.5 kg	Year-round	10 years	1500-3500	Nearly 100% animal matter, including small mammals, squirrels, rabbits, porcupine, birds, reptiles, and amphibians.	Prefer forested areas with closed canopies.	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta, 1995; Martin et al., 1951
	Long-tailed Weasel	85-270 g	Year-round	3 years	31.9-160	78% small mammals (mice, voles, shrews), 17% rabbits; also birds (up to 10%), squirrels, snakes, invertebrates.	Terrestrial	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta 1995
	Short-tailed Weasel	50-150 g	Year-round	Avg. 2 years, max. 7years	Males: 17.0-25.0 Females:10.1-14.9	75% small mammals (mice, voles, shrews); also squirrels, rabbits, birds, amphibians, snakes, invertebrates.	Terrestrial	DeGraaf and Yamasaki, 2001; Whitaker and Hamilton, 1998; Kurta 1995

APPENDIX J

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF OMNIVOROUS AND CARNIVOROUS MAMMALS

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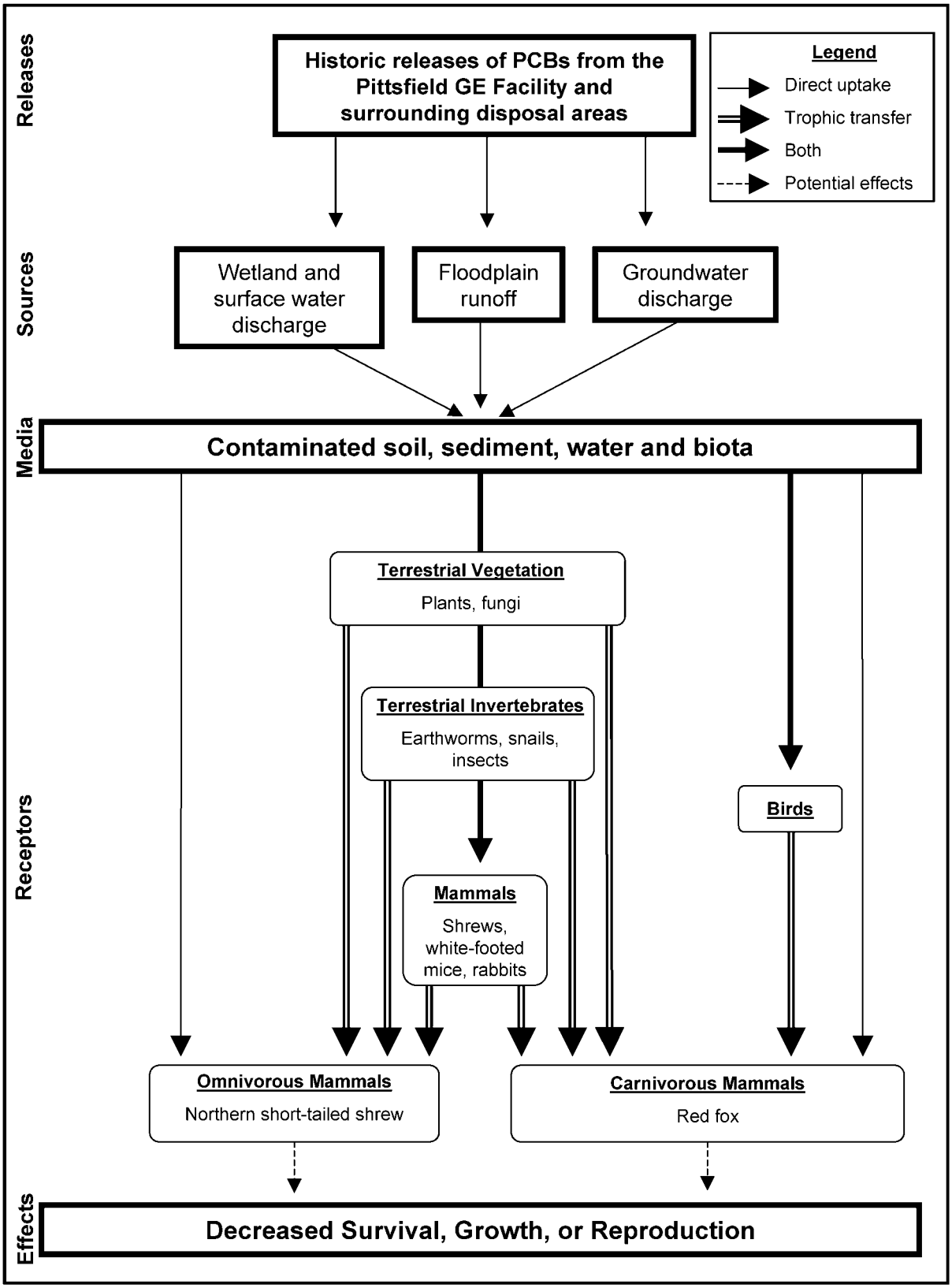


Figure J.1-1 Conceptual Model Diagram: Exposure Pathways for Omnivorous and Carnivorous Mammals Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA

EXPOSURE

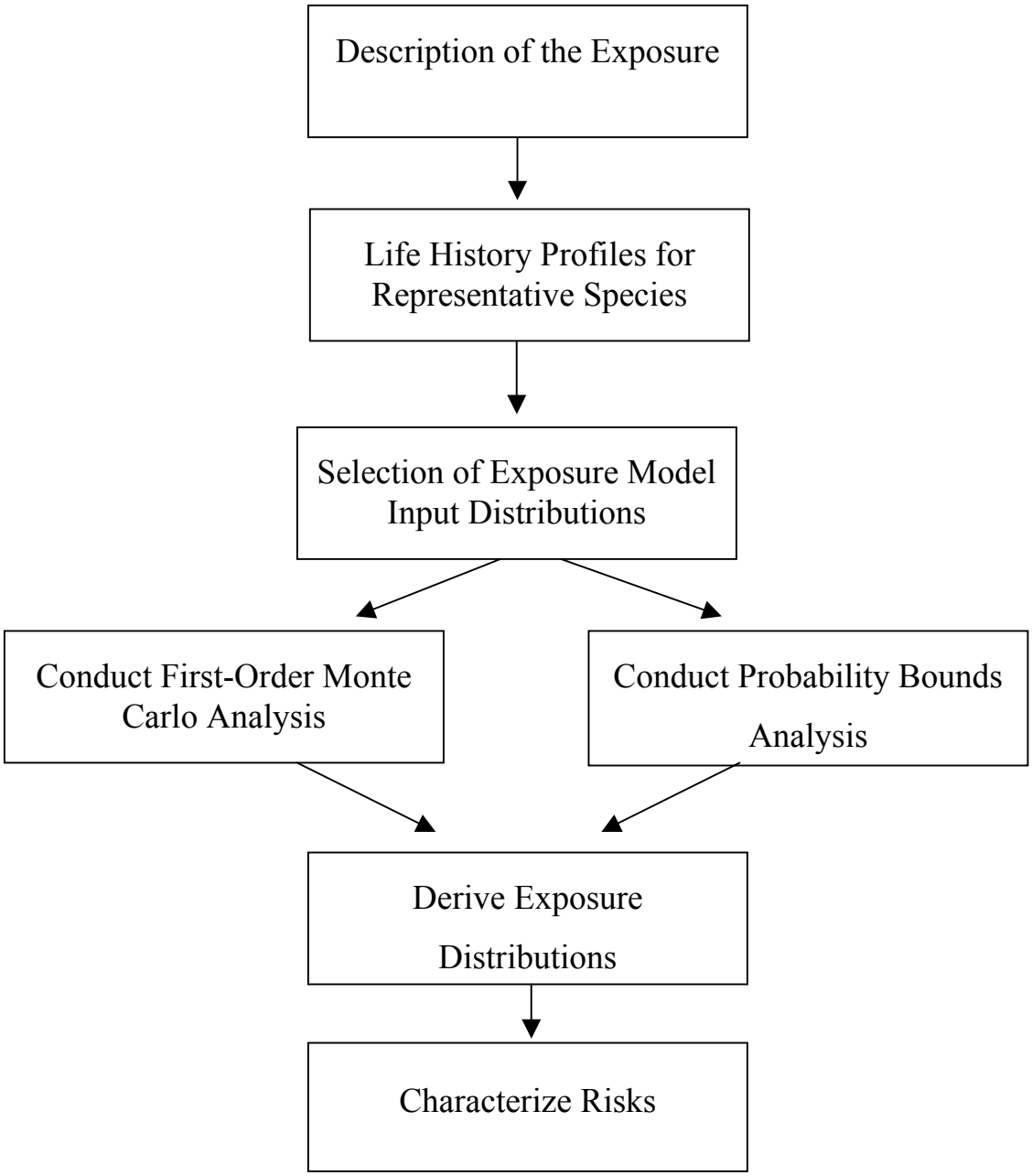


Figure J.1-2 Overview of Approach Used to Assess Modeled Exposure of Omnivorous and Carnivorous Mammals to Contaminants of Concern (COCs) in the Housatonic River PSA

EFFECTS

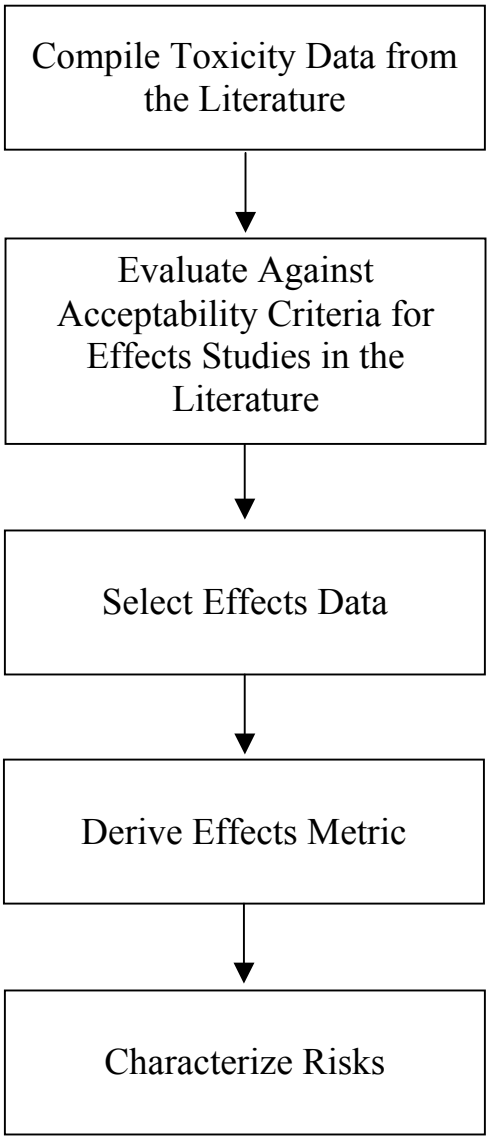


Figure J.1-3 Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Omnivorous and Carnivorous Mammals in the Housatonic River PSA

RISK CHARACTERIZATION

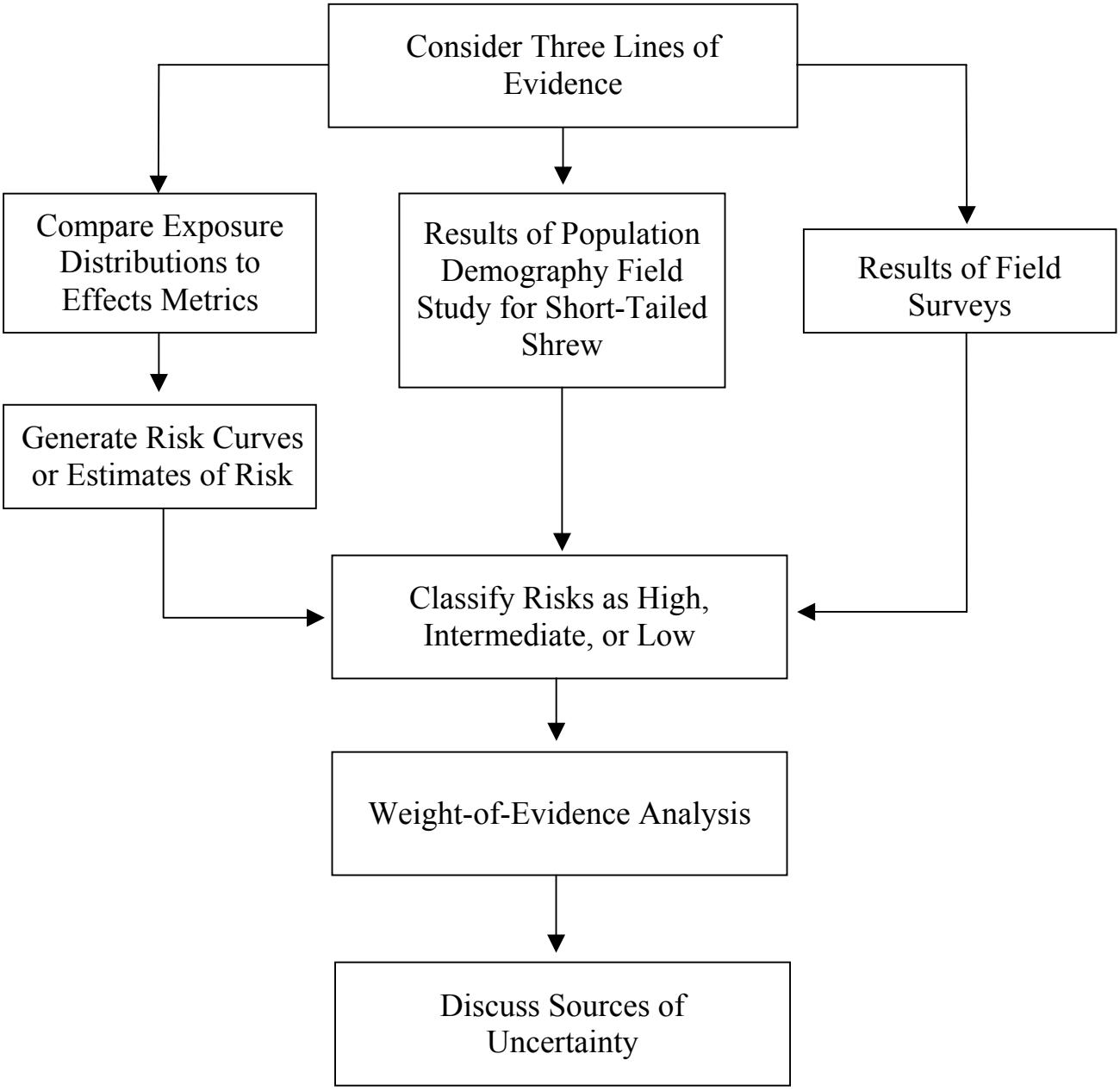
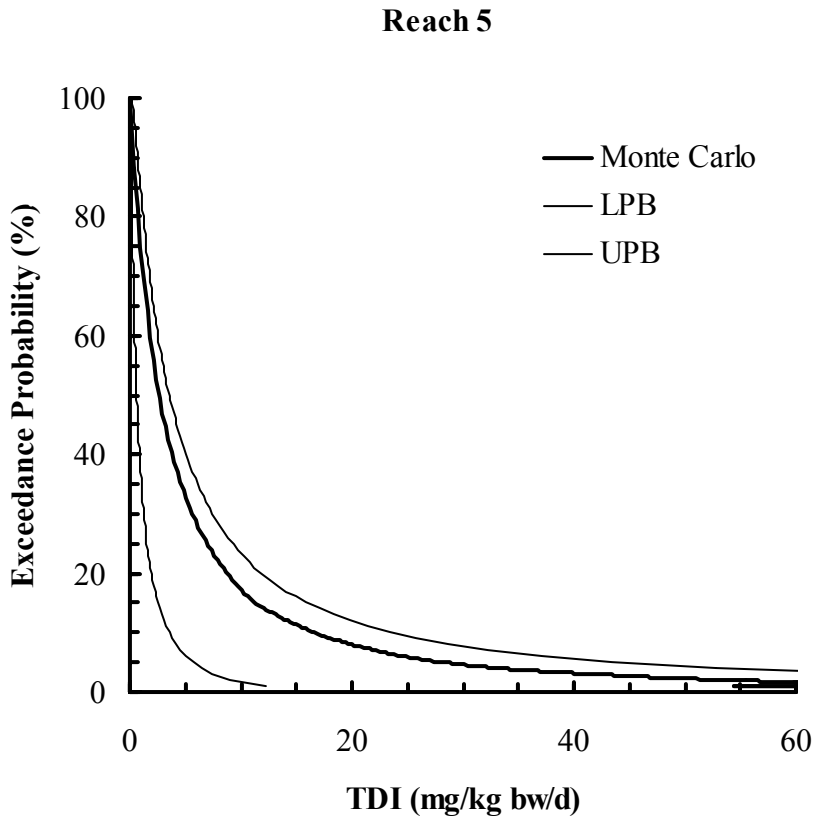


Figure J.1-4 Overview of Approach Used to Assess the Risks of Contaminants of Concern (COCs) to Omnivorous and Carnivorous Mammals in the Housatonic River PSA

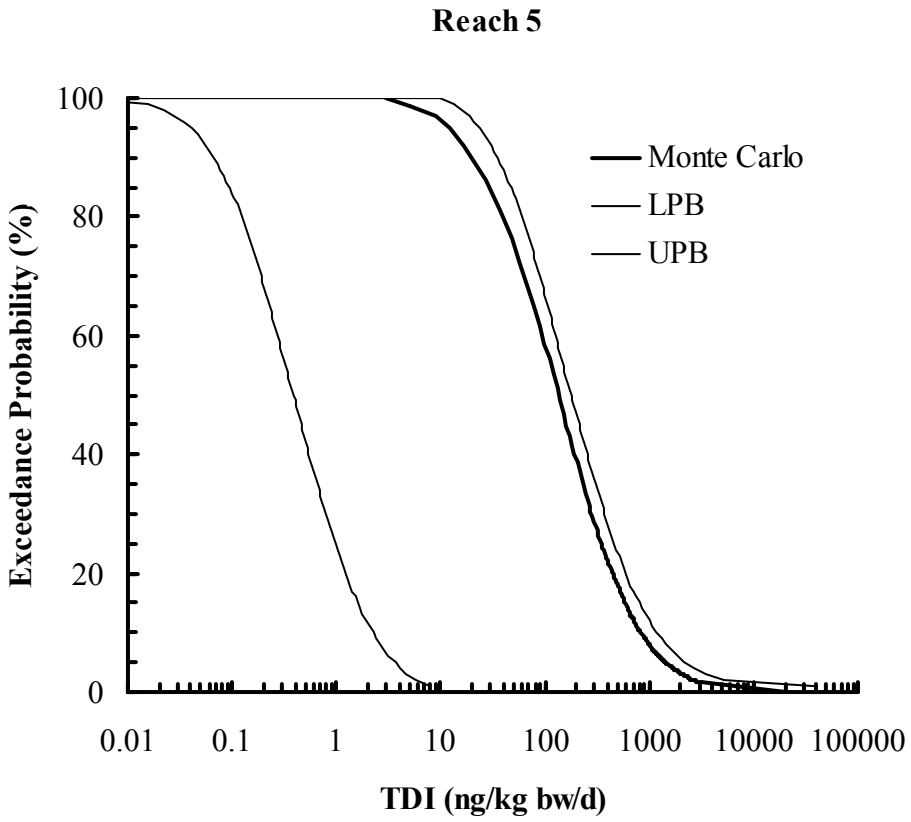


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-1 Exceedance Probability Distribution for Red Fox Exposed to tPCBs in Reach 5 of the PSA

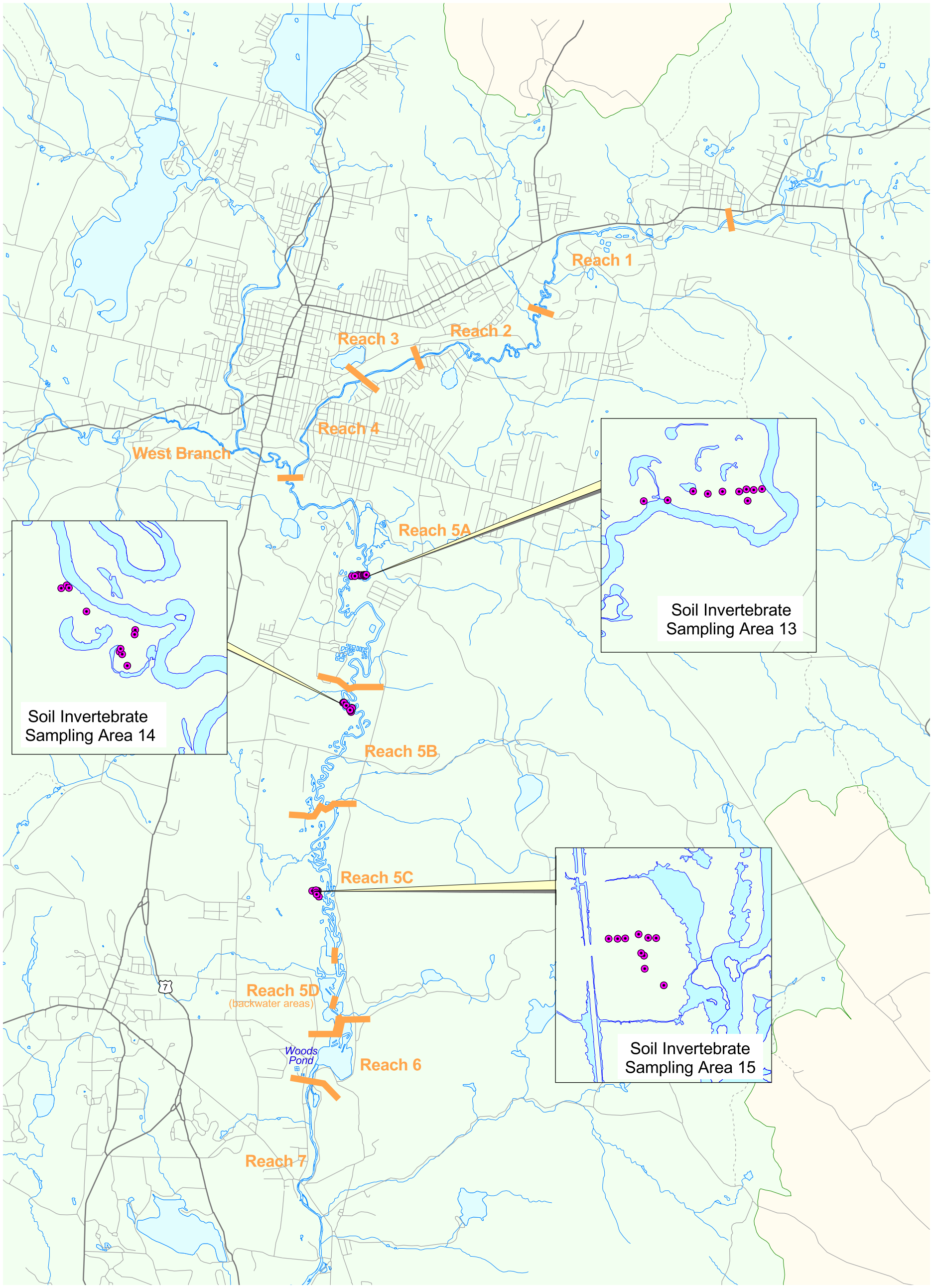


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-2 Exceedance Probability Distribution for Red Fox Exposed to TEQ in Reach 5 of the PSA



LEGEND:

- Soil Invertebrate Sample Location
- Reach Breaks
- Roads
- Hydrography
- Housatonic River Basin Boundary

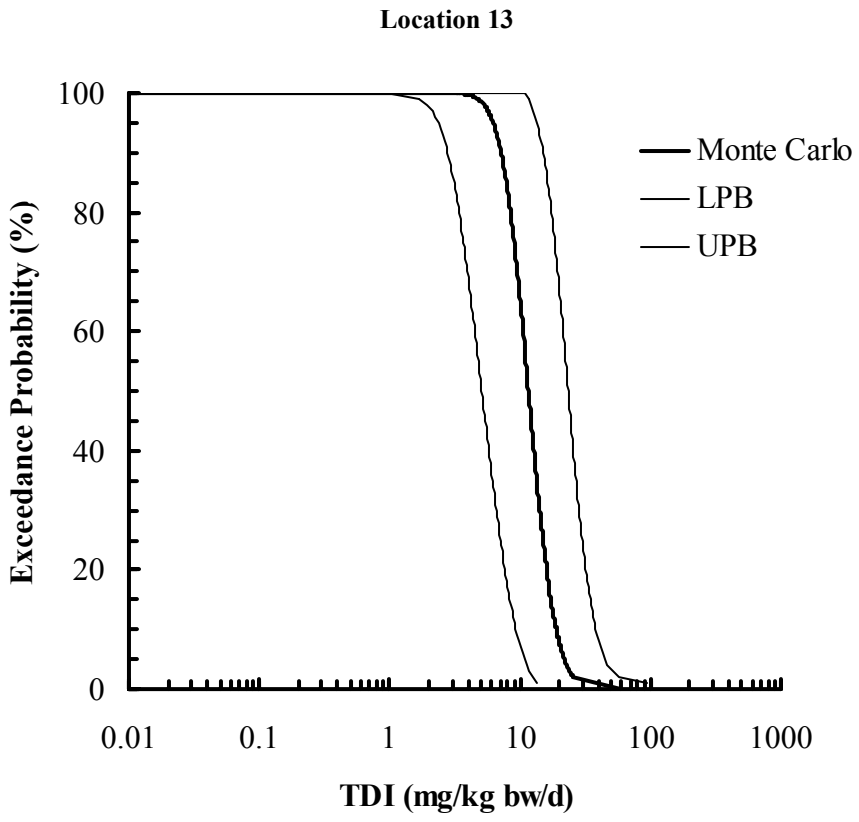
N W E S

2500 0 2500 5000 Feet

0.5 0 0.5 1 Miles

Ecological Risk Assessment
GE/Housatonic River Site
Rest of River

FIGURE J.2-3
SOIL INVERTEBRATE SAMPLE LOCATIONS IN THE HOUSATONIC RIVER PSA.

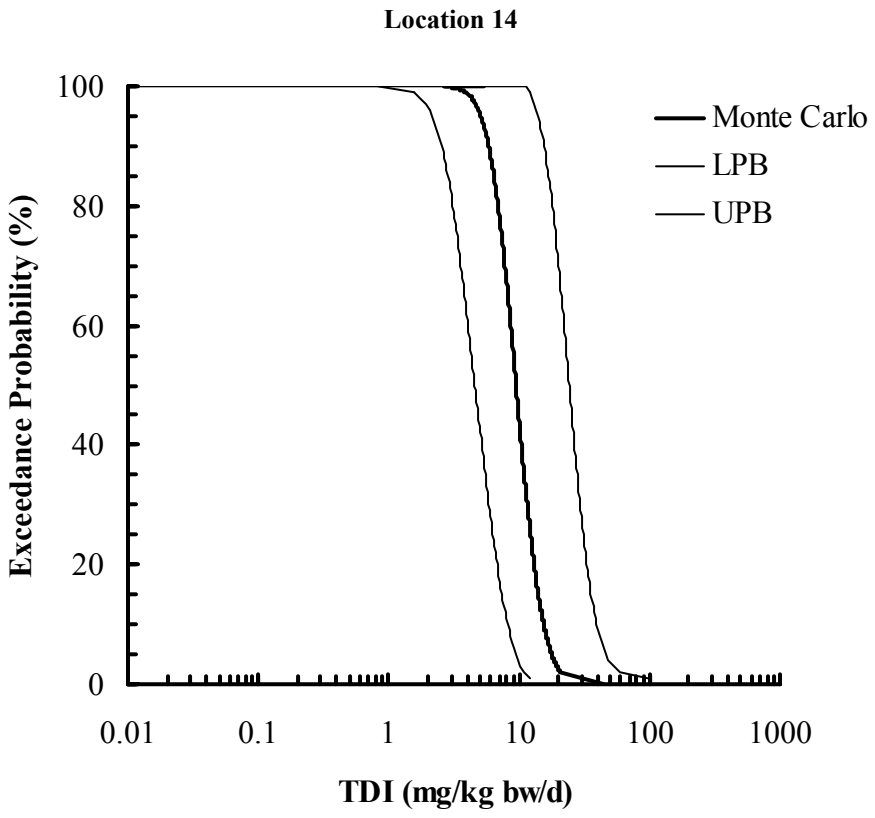


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-4 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 13 of the PSA

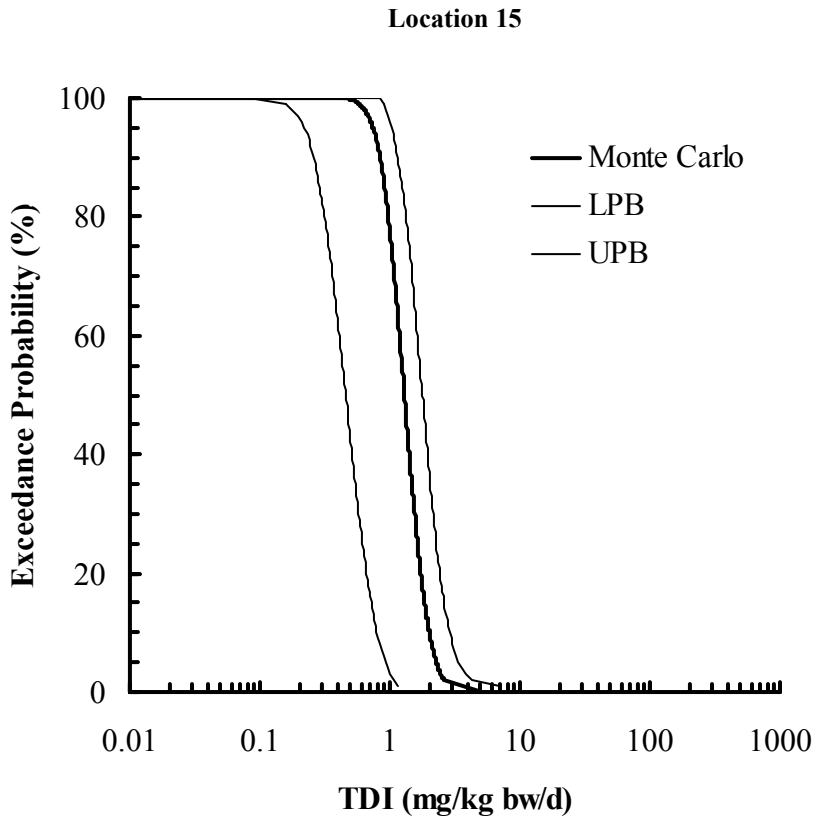


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-5 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 14 of the PSA

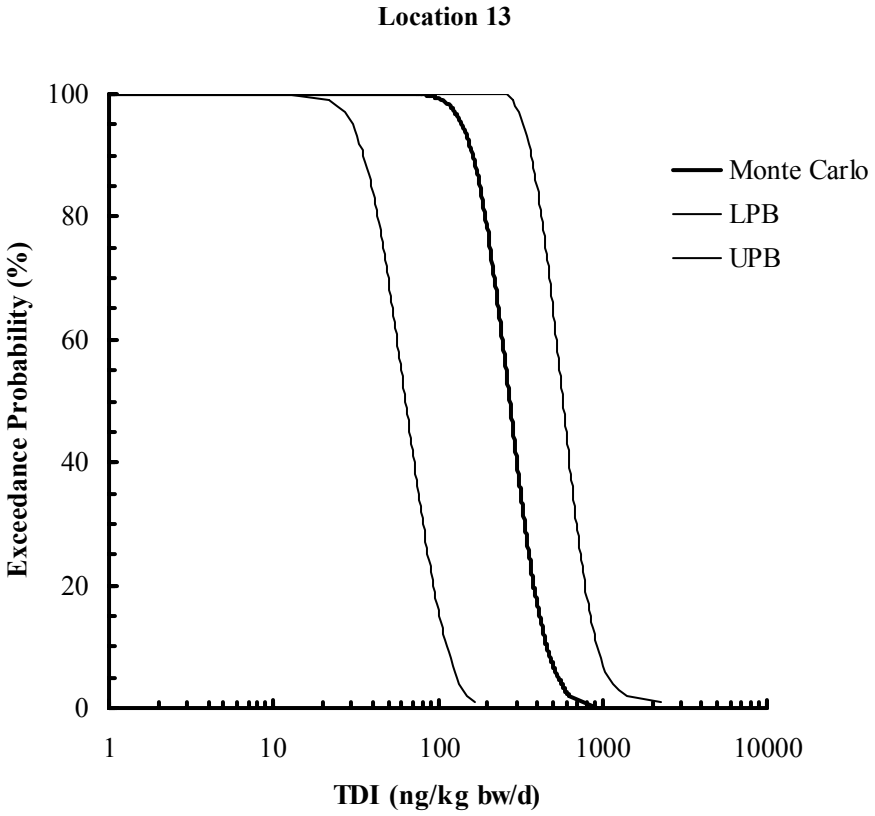


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-6 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 15 of the PSA

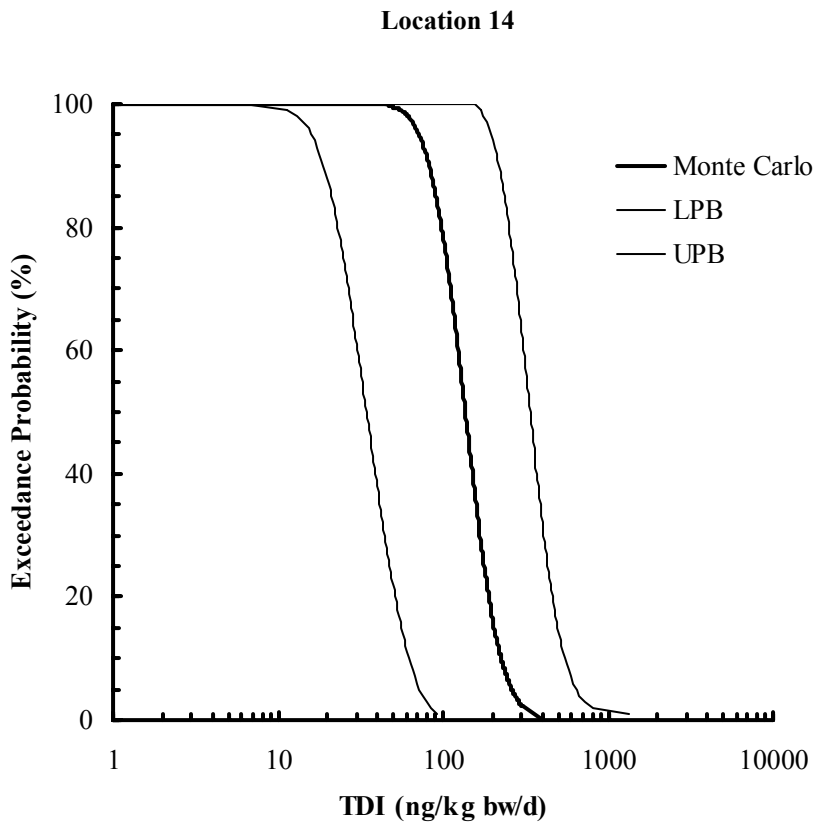


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-7 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 13 of the PSA

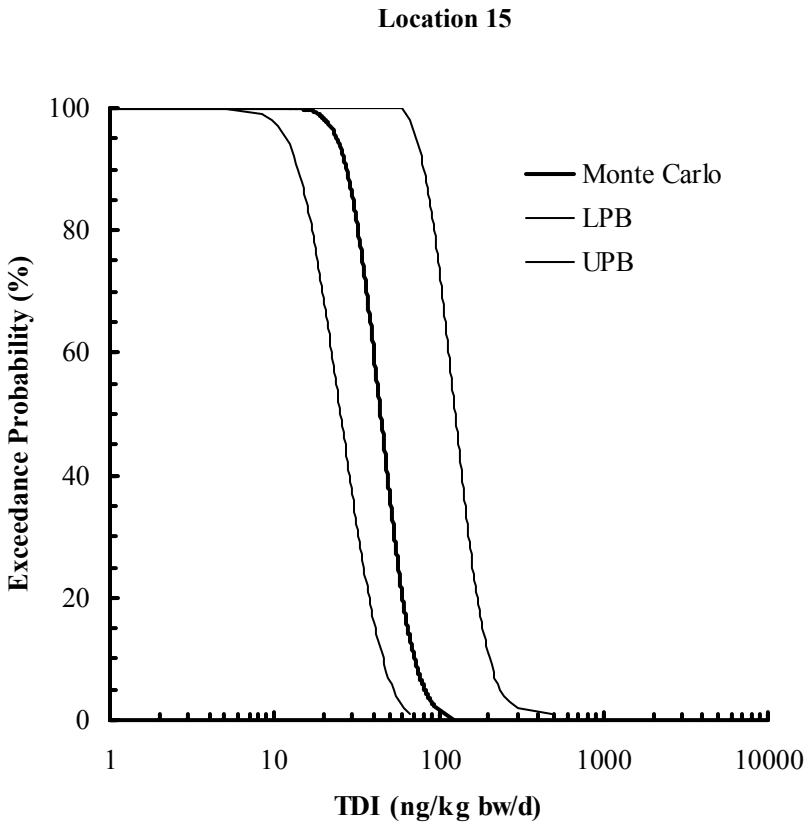


Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-8 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 14 of the PSA



Notes:

LPB = Lower Probability Bound

UPB = Upper Probability Bound

Figure J.2-9 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 15 of the PSA

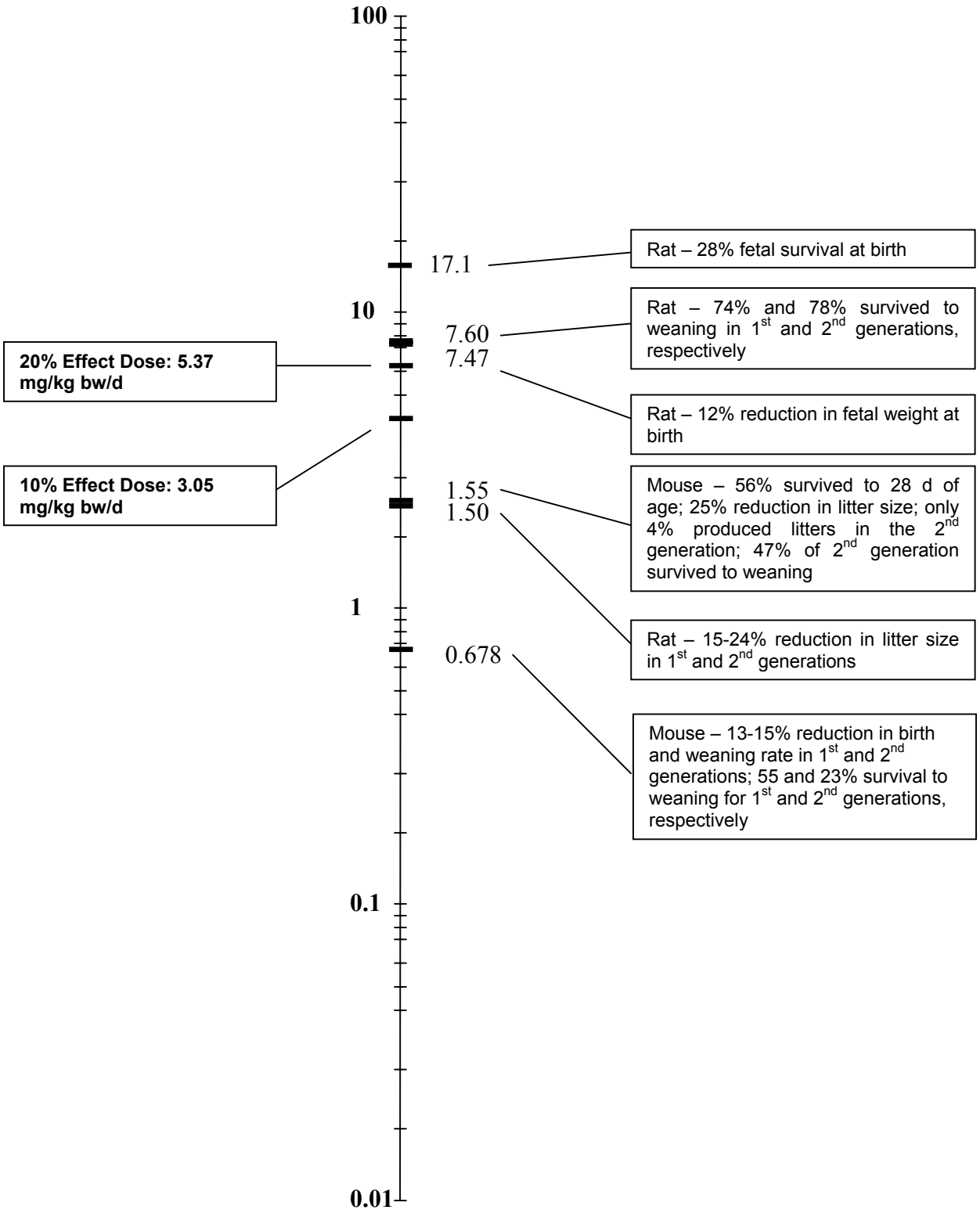


Figure J.3-1 Effects of Aroclor 1254 on Rats and Mice (mg/kg bw/d)

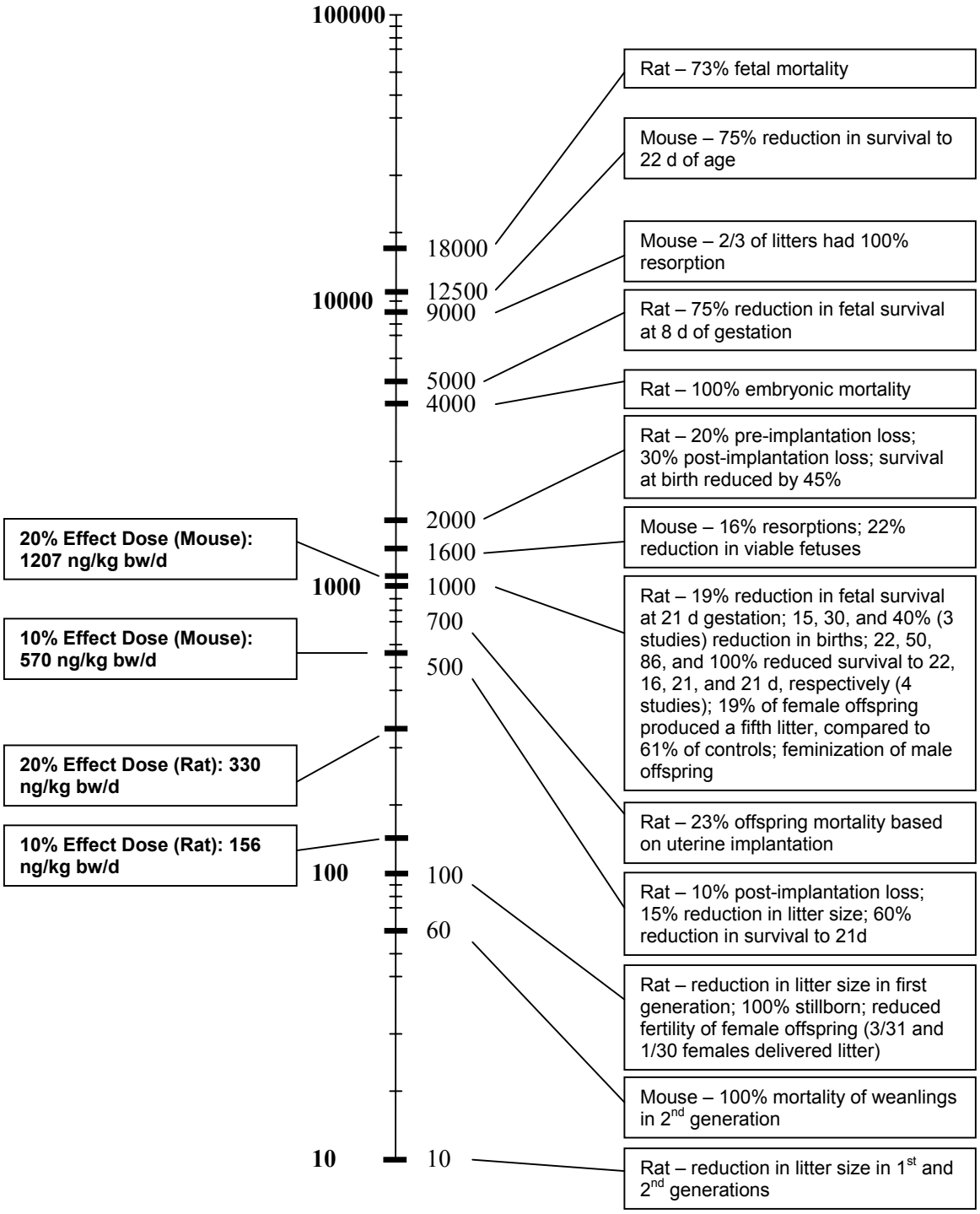
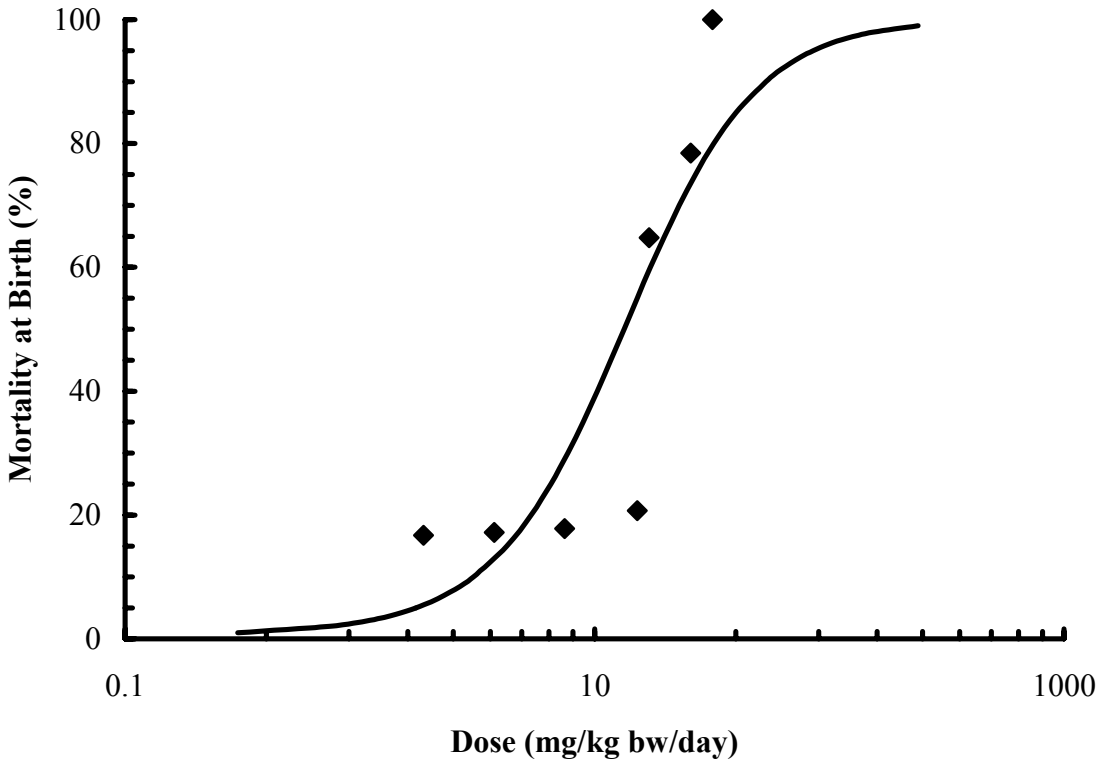


Figure J.3-2 Effects of TEQ on Rats and Mice (ng/kg bw/d)



Note: Symbols indicate raw data.

Figure J.3-3 Dose-Response Curve for Effects of tPCBs on Mortality at Birth of Rats

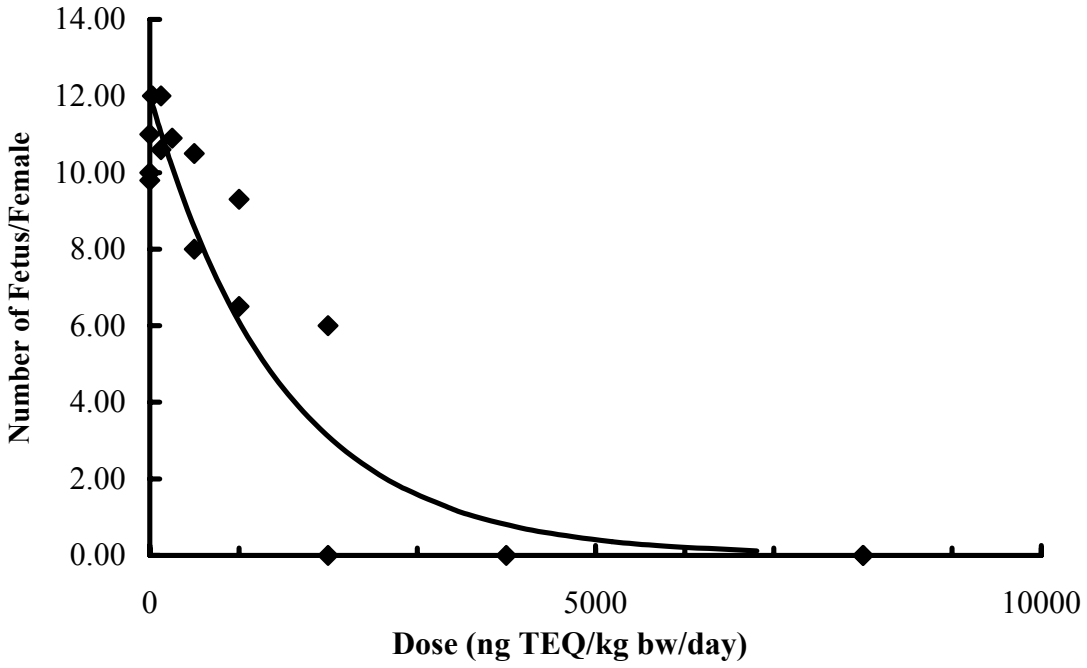
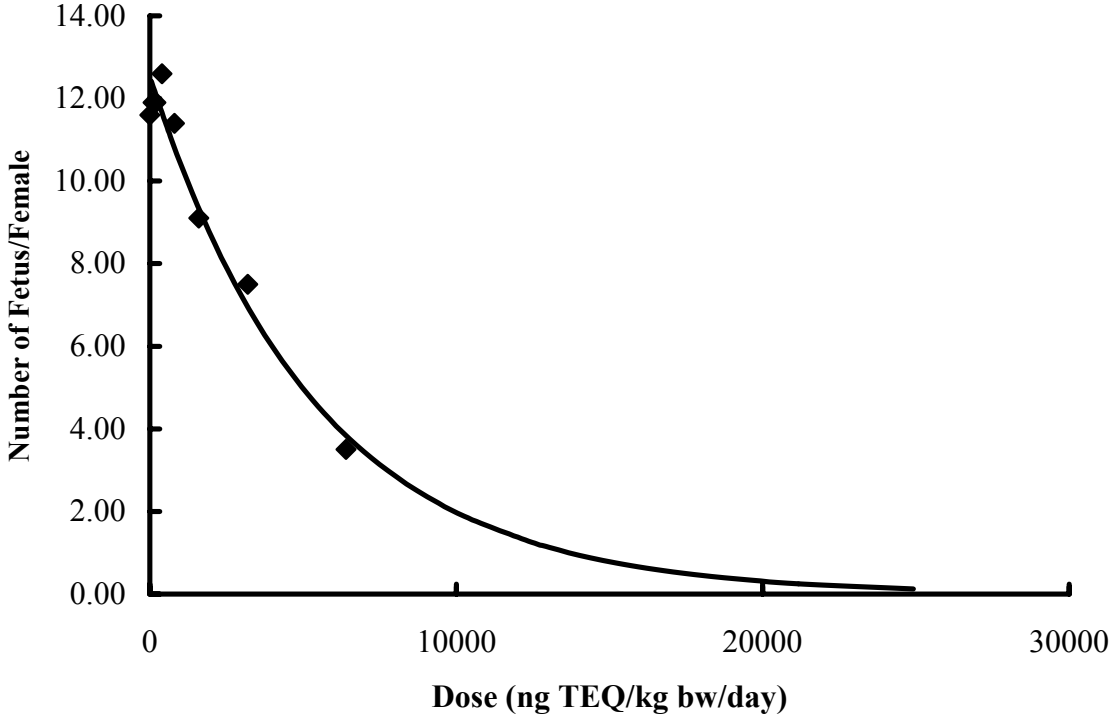


Figure J.3-4 Dose-Response Curve for Effects of TEQ on Reproductive Fecundity of Rats



Note: Symbols indicate raw data.

Figure J.3-5 Dose-Response Curve for Effects of TEQ on Reproductive Fecundity of Mice

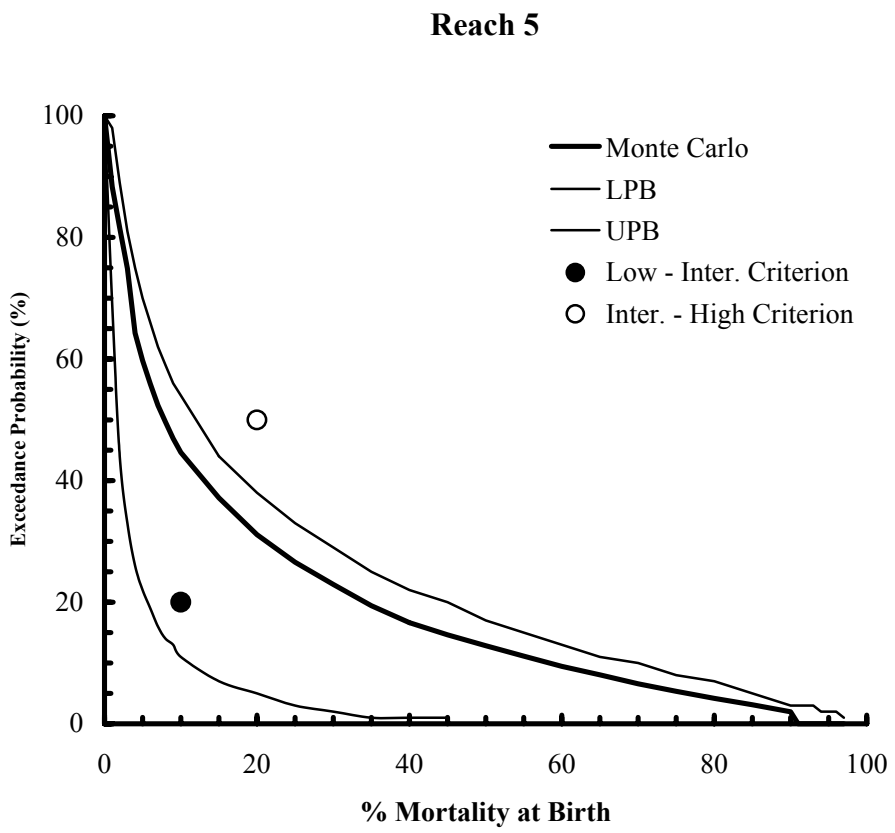


Figure J.4-1 Risk Function for Red Fox Exposed to tPCBs in Reach 5 of the Housatonic River

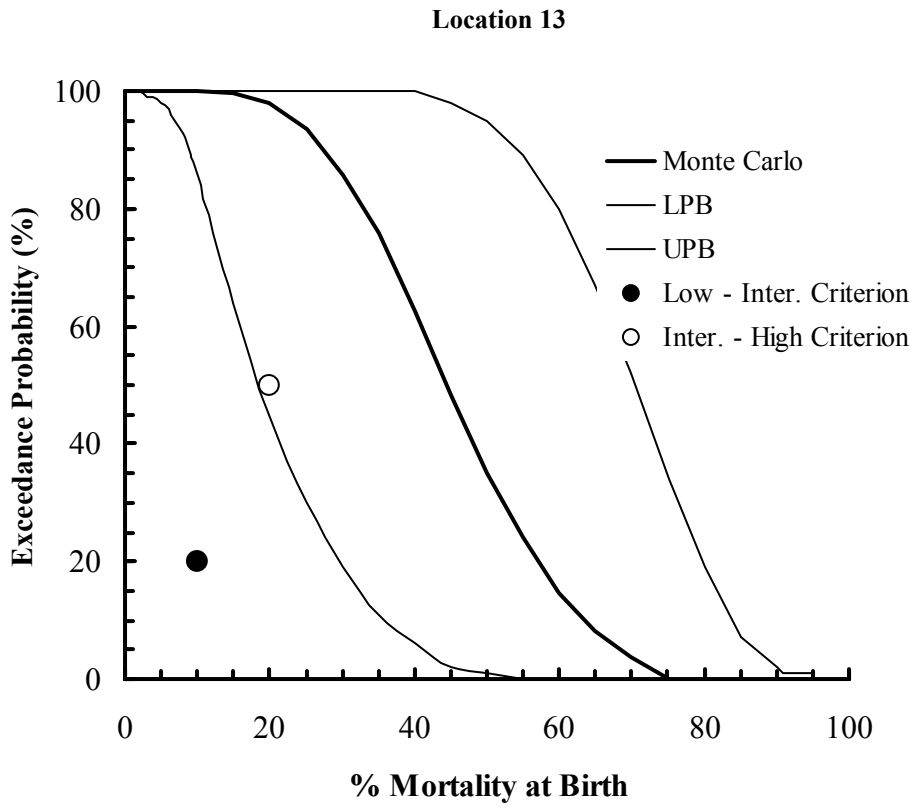


Figure J.4-2 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 13 of the PSA

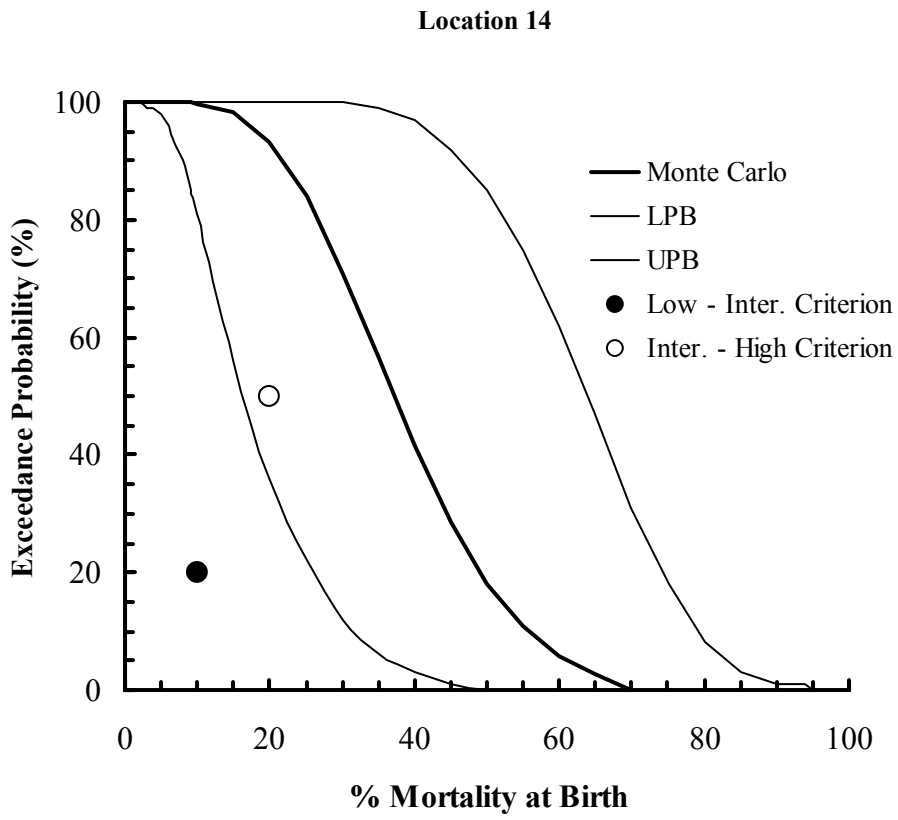


Figure J.4-3 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 14 of the PSA

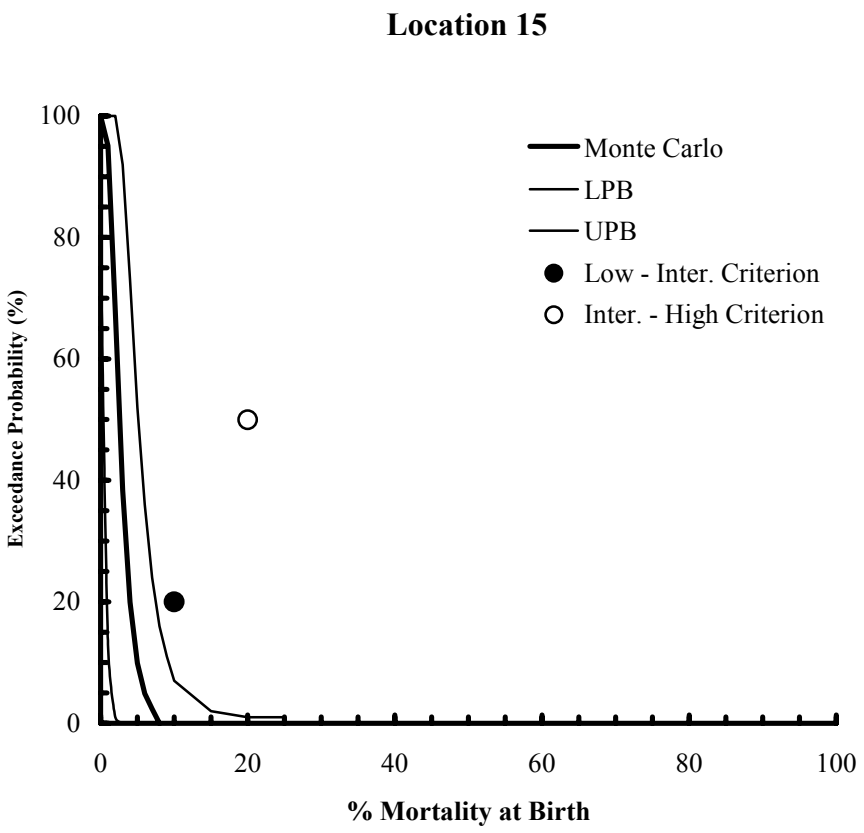
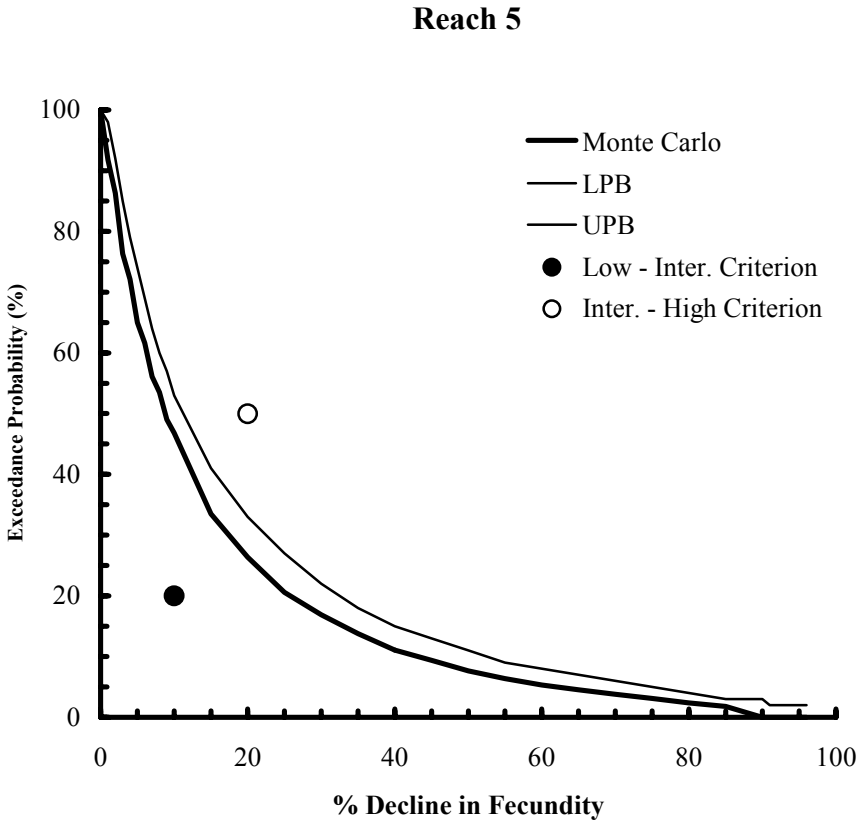


Figure J.4-4 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 15 of the PSA



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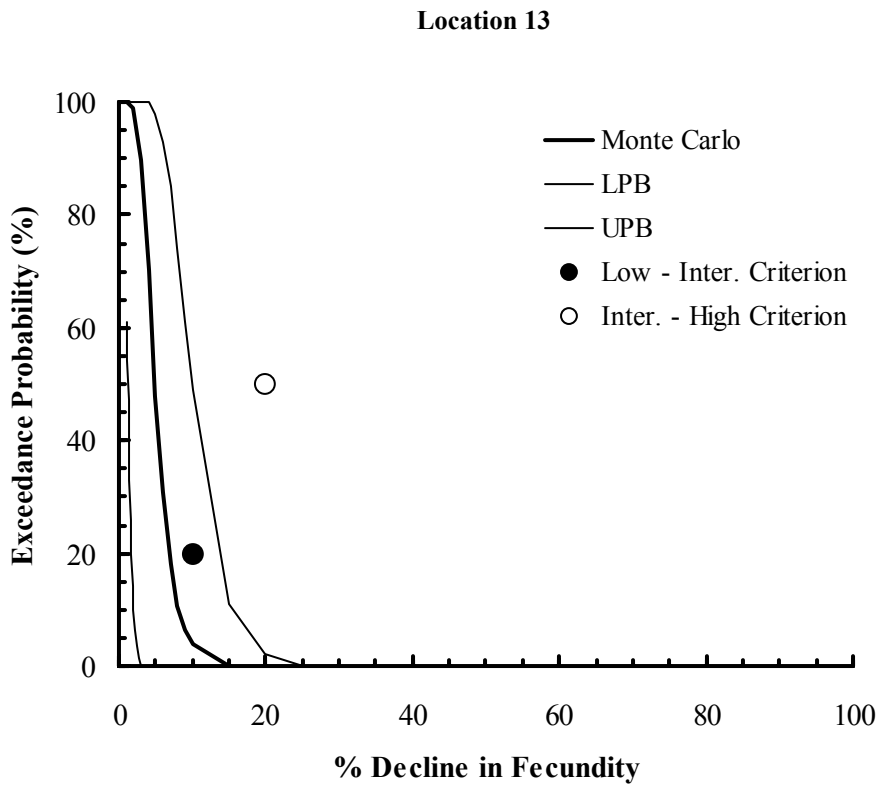
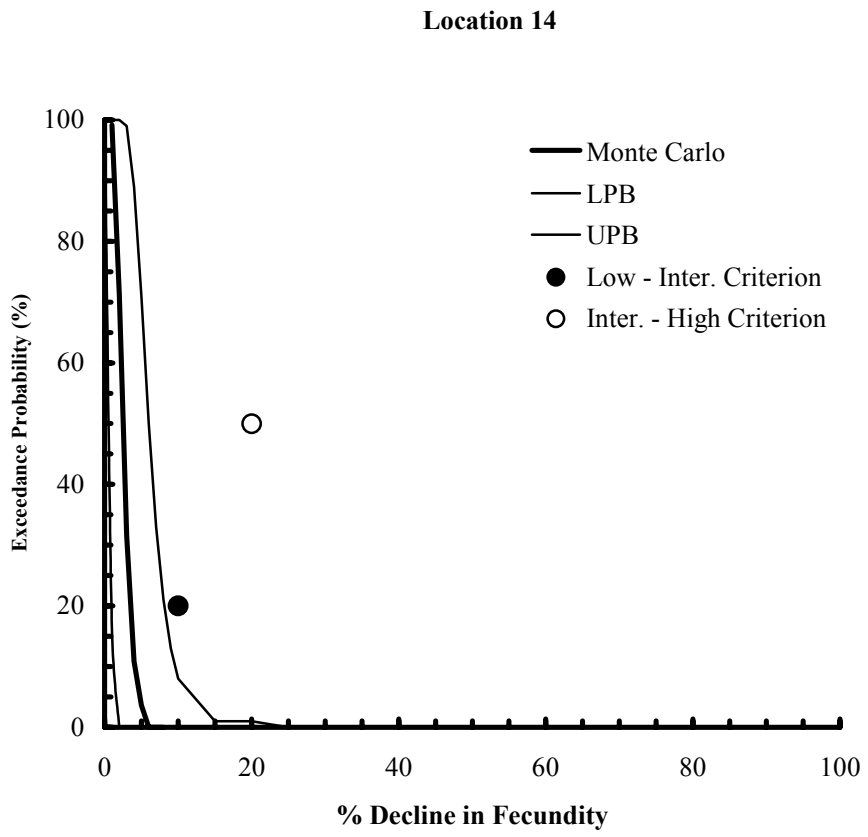


Figure J.4-6 Risk Function for Short-Tailed Shrew Exposed to TEQ at Location 13 of the PSA



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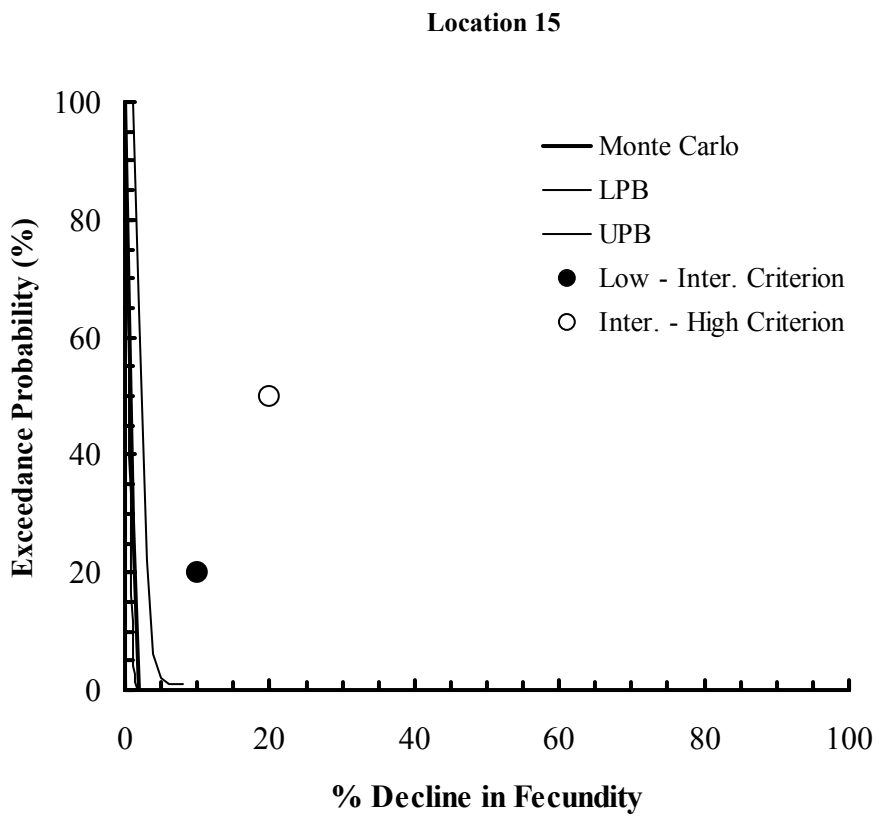

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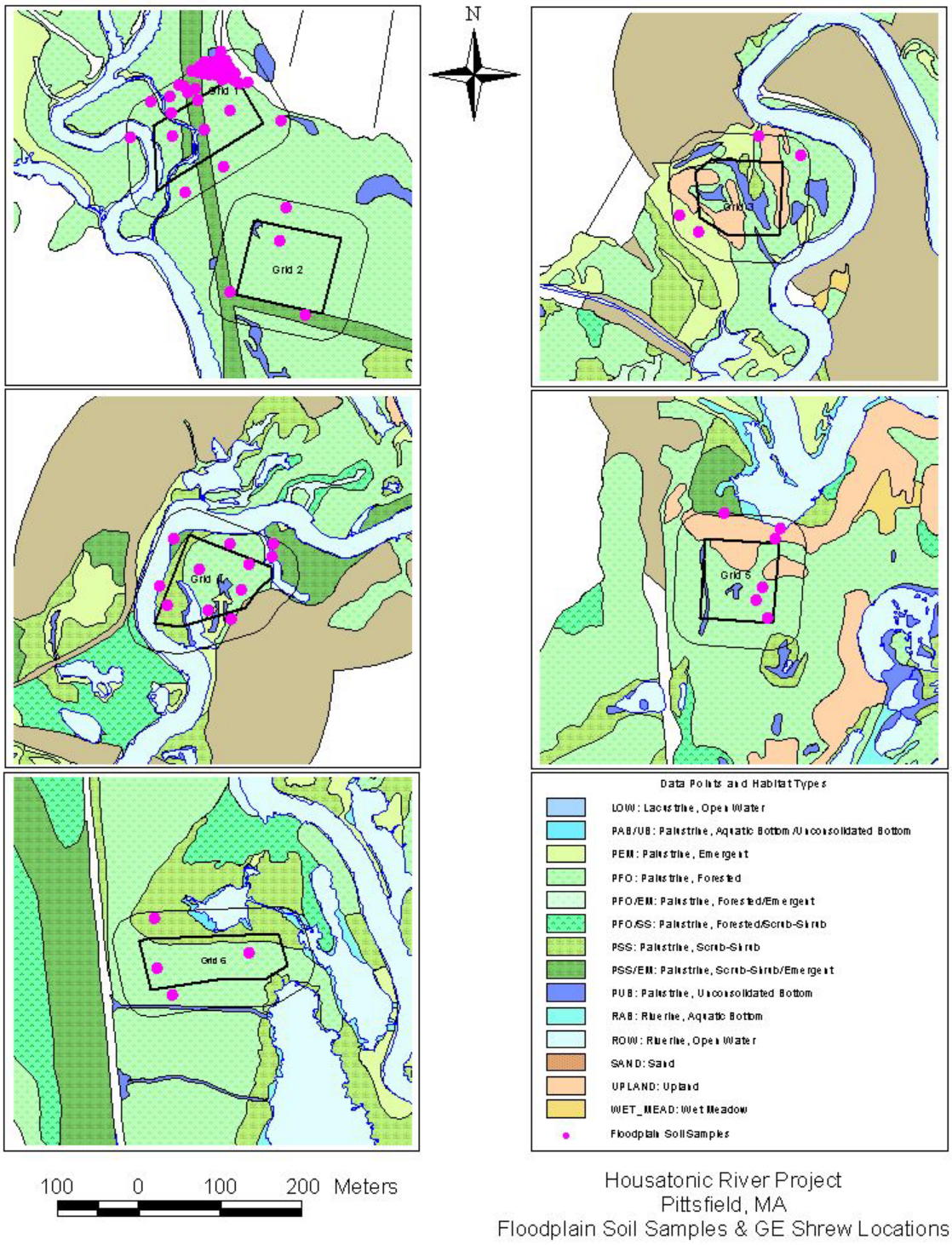

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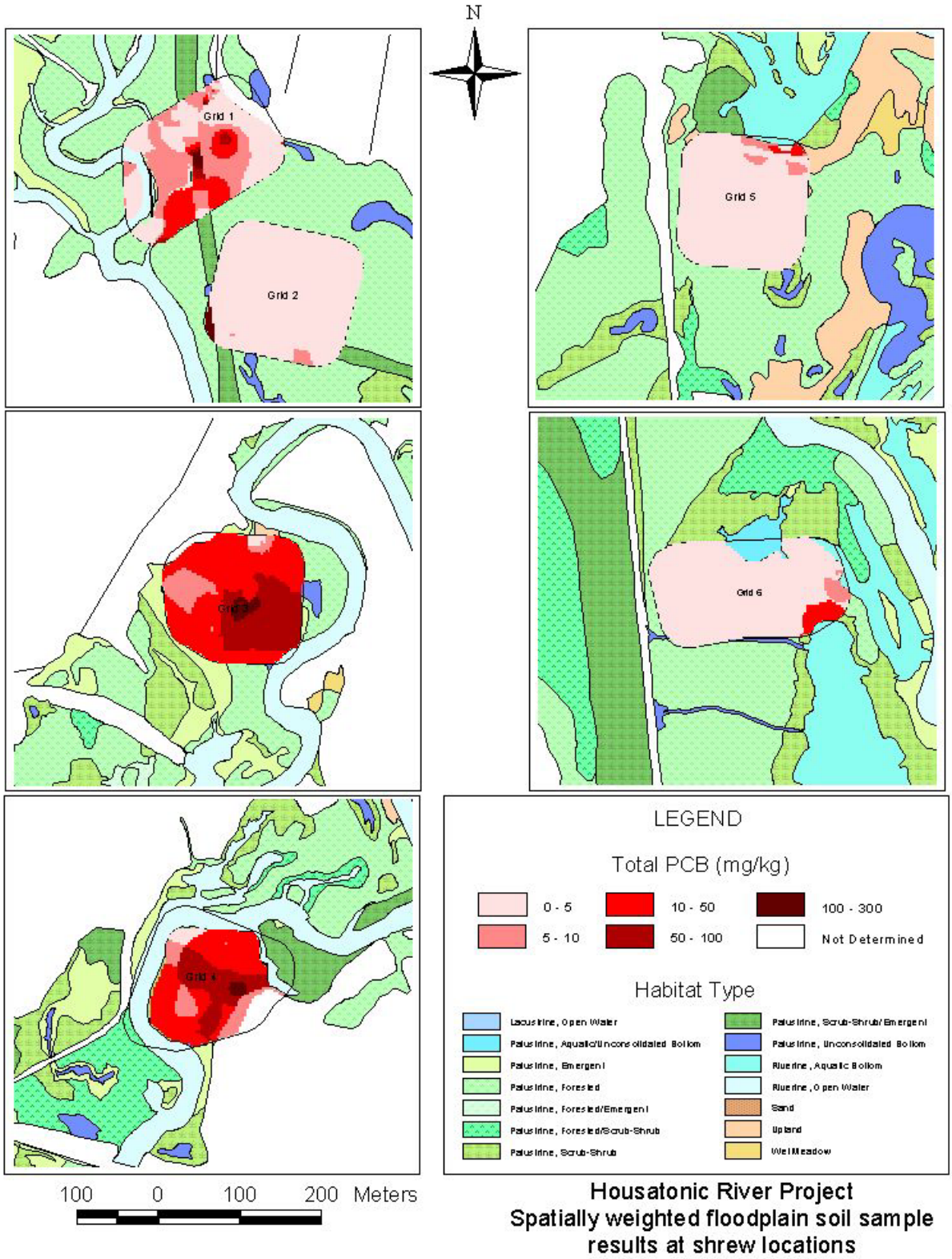

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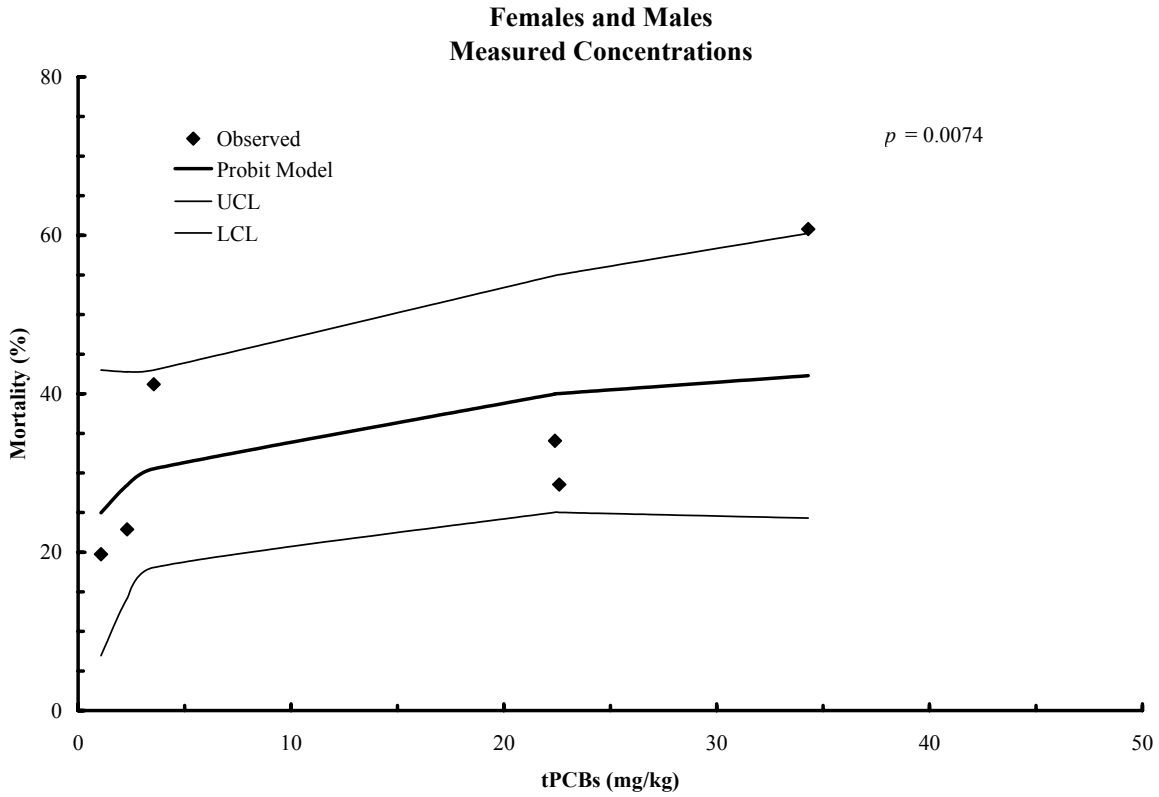
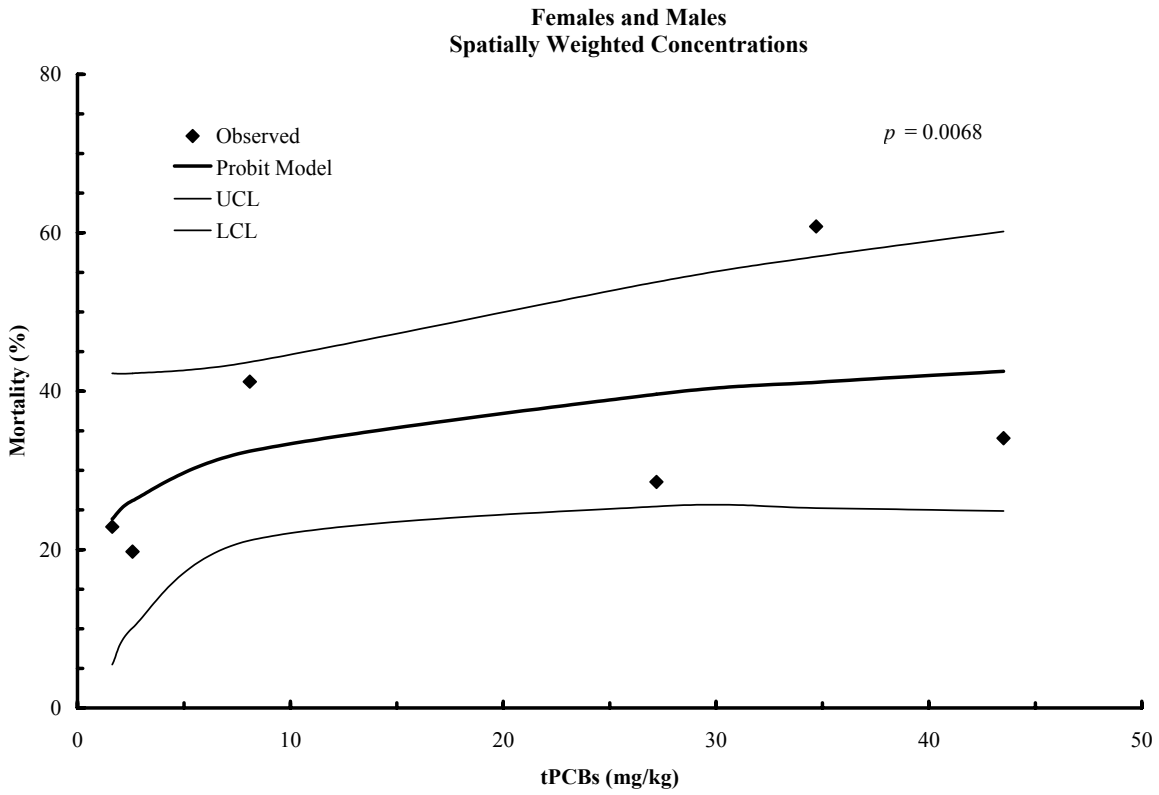


Figure J.4-11 Probit Model Curve for Effects of Measured tPCB Concentrations on Survival of Female and Male Short-Tailed Shrew



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APPENDIX K

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF THREATENED AND ENDANGERED SPECIES

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LIST OF ACRONYMS

2,3,7,8-TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
BBS	Breeding Bird Survey
BW	body weight
CAE	chemical absorption efficiency
COCs	contaminants of concern
COPCs	contaminants of potential concern
DQOs	data quality objectives
DL	detection limit
FIR	food intake rate
FMR	free metabolic rate
FT	foraging times
GE	General Electric Company
LCL	lower confidence limit
MATC	maximum acceptable threshold concentration
MNHESP	Massachusetts Natural Heritage and Endangered Species Program
PSA	Primary Study Area
T&E	rare, threatened, and endangered
TDI	total daily intake
TEF	toxic equivalency factor
TEQ	toxic equivalence
tPCBs	total polychlorinated biphenyls
UCL	upper confidence limit
WBC	whole body concentration
ww	wet weight

APPENDIX K

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF THREATENED AND ENDANGERED SPECIES

K.1 INTRODUCTION

The purpose of this appendix is to characterize and quantify the current and potential risks posed to rare, threatened, and endangered (T&E) species exposed to contaminants of concern (COCs) in the Housatonic River and floodplain, focusing on total polychlorinated biphenyls (tPCBs) and other COCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The Housatonic River watershed is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters of the watershed. The Primary Study Area (PSA) includes the river and 10-year floodplain from the confluence of the East and West Branches of the Housatonic River downstream of the GE facility to Woods Pond (Figure 1.1-2).

A pre-ERA was conducted to narrow the scope of the ecological risk assessment by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A three-tiered deterministic approach was used to screen contaminants of potential concern (COPCs). The deterministic assessments compared potential conservative estimates of exposure with conservative adverse effects benchmarks to identify what contaminants are of potential concern in the Housatonic River. A risk quotient (total daily intake/effect benchmark) greater than 1 in the Housatonic River area resulted in the COPC being screened through to the next tier assessment and to the probabilistic ecological risk assessment, if necessary.

The COPCs were then evaluated specifically for each assessment endpoint. The COCs that were retained in the probabilistic risk assessment for T&E species were tPCBs and 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) toxic equivalence (TEQ), hereafter referred to as contaminants of concern (COC). Total PCBs (tPCBs) detected in Housatonic River media samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which

are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6 of the ERA).

COCs for T&E Species

- tPCBs
- 2,3,7,8-TCDD TEQ

K.1.1 Overview of Approach

A step-wise approach was used to assess the risks of tPCBs and TEQ to T&E species in the Housatonic River watershed. The four main steps in this process include:

1. Derivation of a conceptual model (Figure K.1-1).
2. Assessment of exposure of T&E species to COCs (Figure K.1-2).
3. Assessment of the effects of COCs on T&E species (Figure K.1-3).
4. Characterization of risks to the T&E species community (Figure K.1-4).

K.1.1.1 Conceptual Model

The conceptual model presented in Figure K.1-1 illustrates the exposure pathways for T&E species exposed to tPCBs and TEQ in the PSA. Total PCBs and TEQ are persistent and highly lipophilic and hydrophobic. Therefore, they are bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001). Fish, amphibians, invertebrates, mammals, and birds comprise the major dietary items for T&E species discussed in this part of the ERA. In summary, T&E species that reside, or partially reside, within the PSA are exposed to tPCBs and TEQ principally through diet and trophic transfer. Other routes of exposure, considered to be less important, include inhalation, water consumption, and sediment ingestion (Moore et al. 1999).

Eighteen T&E species were observed in the PSA from 1998 to 2001. These include four invertebrates, four reptiles and amphibians, eight birds, and two mammals. Of these 18 species, the problem formulation (see Section 2 of the ERA) identified the bald eagle (*Haliaeetus leucocephalus*), American bittern (*Botaurus lentiginosus*), and small-footed myotis (*Myotis*

leibii) as the representative T&E species potentially exposed to tPCBs and TEQ from consumption of contaminated prey. Life history profiles for the bald eagle (Figure K.1-5), American bittern (Figure K.1-6), and small-footed myotis (Figure K.1-7) are summarized in text boxes. Additional life history information on these species is discussed in Sections K.2.1.5, K.2.1.6, and K.2.1.7, respectively.

Bald eagles nest downstream on the Housatonic River in Connecticut, have attempted to nest in the PSA, and have ample habitat available for nesting in the PSA. Similarly, because American bitterns have been observed during the breeding season in suitable nesting habitat, they were chosen for inclusion because of the potential for nesting. Small-footed myotis may occur in the PSA as well, because of their known range and the suitability of habitat.

Life History of Bald Eagle

The bald eagle is one of the largest and most conspicuous birds of prey in North America. Weights of adults and juveniles vary from 3.0 kg to over 7 kg. The bald eagle is currently federally listed as Threatened in the lower 48 states, but is more restrictively listed as Endangered (i.e., more at risk) by several New England states, including Massachusetts, Vermont, New Hampshire, and Connecticut.

- **Habitat** – Habitat use varies depending on the region, but proximity to large bodies of water with suitable foraging opportunities is critical, thus bald eagles are restricted to coastal areas, lakes, and rivers. Relatively open canopies, some type of habitat edge, and the availability of super-story trees with stout horizontal perching branches providing good access to nests are preferred habitat features for breeding pairs.
- **Home Range** – Large home ranges, minimum size of 1,730 acres (700 ha) and average size of 4,645 ± 2,224 acres (1,879.76 ± 900.02 ha), linear (riverine) foraging distances of 1.9 to 4.3 miles (3.1 to 6.9 km). Nesting eagles were reported to generally forage within 0.3 mile (0.5 km) of the nest, ranging up to 1.9 miles (3.0 km), and as far as 5.0 miles (8.0 km) from the nest.
- **Dietary Habits** – Feed primarily over water on aquatic prey, opportunistic feeders consuming a variety of live prey and scavenging carrion. Fish form the largest percentage of diet, taken primarily from shallow water. Fish consumption is 17.1% to 90.1%, depending on location, season, and prey availability. Birds, particularly waterfowl, can form large portions of the diet, more common during the winter and in coastal habitats. Mammal species average 4.9% of prey; reported as much as 11.7%, or as little as 0% of the diet.

Life History of American Bittern

The American bittern is a mid-sized, stocky heron of freshwater marshes. It is identified by its heavily streaked breast with vertical brown and white stripes below. American bittern populations have been declining since the 1960s, primarily as a result of habitat loss and wetland degradation. The Commonwealth of Massachusetts has included the American bittern on its list of Endangered species.

- **Habitat** – Use a wide range of freshwater wetlands, with diversity of vegetation classes (i.e., aquatic bed, emergent, and scrub-shrub) and high interspersion of open water and plant cover.
- **Home Range** – Vary with geographic area and availability of preferred habitat and prey species. Average home ranges 315 acres (127 ha) in Minnesota, with the birds using a 61-acre (25 ha) core area more than 50% of the time. In Massachusetts, breeding occurs with scattered localities in Berkshire County. Nests built in dense emergent vegetation over water with depths ranging from 5 to 20 cm (2 to 8 inches), consisting of a 15- to 25-cm (6- to 10-inch) high platform of reeds, sedges, or grasses bent down and lined with fine grasses.
- **Dietary Habits** – Prey upon insects, crayfish, amphibians, fish, and small mammals. Insect prey primarily adult and nymphal dragonflies, giant waterbugs, water scorpions, water beetles, and grasshoppers. Fish species vary with availability, and include eels, catfish, pickerel, sunfish, suckers, killifish, sticklebacks, and perch, typically from 10 to 100 mm in length.

Life History of Small-Footed Myotis

The small-footed myotis is a small bat. It is identified by its golden brown fur and black mask. The small-footed myotis is listed as a Species of Special Concern by the Massachusetts Natural Heritage and Endangered Species Program (MNHESP) (1984).

- **Habitat** – Use buildings, overhanging rocks, and caves as summer roosts and maternity sites. Females and young roost in small (typically less than 20 individuals) maternity colonies in rock crevices and crevice-like places on buildings; males are solitary. Hibernate hanging from walls or underneath fallen rock and rubble, from November to March, usually in the foothills of mountains up to 610 m (2,000 feet) in elevation, in coniferous woodlands.
- **Home Range** – Home range is unknown. It is assumed that home ranges are similar to other Myotis species (Indiana bat has a home range of 52 to 95 ha for pregnant and lactating females).
- **Dietary Habits** – Little is known about feeding habits; however, believed to be similar to other Myotis species. Flies, beetles, bugs, leafhoppers, and flying ants have been found in stomachs. Many species are opportunistic feeders, exploiting available food resources, fly low to the ground (1 to 3 m) when feeding, along forest openings, including waterways.

The assessment endpoint, which is the subject of this appendix, is the survival, growth, and reproduction of T&E species in the PSA. The two potential lines of evidence considered in the evaluation of the assessment endpoint included (1) comparisons of modeled exposure to doses of tPCBs and other COCs ingested in the diet reported in the literature to cause adverse effects to the survival, reproduction, or growth of bald eagle, American bittern, and small-footed myotis; and (2) conducting field surveys to determine the qualitative abundance of bald eagle, American bittern, and small-footed myotis in the Housatonic River floodplain.

Measurement Endpoint for T&E Species

- Determine, based on laboratory toxicity studies, the extent to which the concentrations of tPCBs and other COCs in the PSA may cause reductions in the survival, growth, or reproduction of T&E species.

K.1.1.2 Exposure Assessment

The exposure assessment estimates the exposure of T&E species to tPCBs and TEQ in the Housatonic River PSA (Figure K.1-2). It begins with a description of the exposure model. Input variables for the exposure model are determined using life history information on the representative species and concentrations of tPCBs and TEQ in prey collected in the PSA. Monte Carlo and probability bounds analyses are then conducted to estimate exposure for each COC.

K.1.1.3 Effects Assessment

The effects assessment provides an overview of the literature on the effects of tPCBs and TEQ to the survival, growth, and reproduction of representative wildlife species (Figure K.1-3). An extensive literature search is conducted to locate studies with data and information on the survival, growth, and reproduction of the representative wildlife species exposed to the COCs. Each of the studies is evaluated using appropriate acceptability criteria. Studies are then selected and used to derive the most appropriate effects metric.

K.1.1.4 Risk Characterization

The risk characterization evaluates the likelihood that adverse effects may occur as a result of wildlife exposure to tPCBs and TEQ (Figure K.1-4). Two potential lines of evidence were considered when characterizing risks to T&E species from exposure to tPCBs and TEQ:

- **Modeled Exposure and Effects** – The purpose is to determine the extent to which the concentrations of tPCBs and TEQ ingested in the diet will cause deleterious effects to the survival, reproduction, or growth of T&E species. A probabilistic food web model was used to estimate exposure of tPCBs and TEQ to bald eagle, American bittern, and small-footed myotis. The total daily intake (TDI) for bald eagle, American bittern, and small-footed myotis in the PSA were calculated using equations from the *Wildlife Exposure Factors Handbook* (EPA 1993) and related publications. These estimated exposures were compared to results of toxicological studies reported in the literature to determine if the T&E species are being exposed to tPCBs and TEQ at concentrations likely to induce adverse effects.

- **Field Surveys –** The purpose is to determine the abundance of T&E species in the Housatonic River floodplain.

A weight-of-evidence assessment was conducted for the modeled exposure and effects; the field surveys were qualitative and therefore not used in the weight-of-evidence assessment. The section concludes with a discussion of sources of uncertainty in the assessment of risks of COCs to T&E species, and the conclusions of the risk characterization.

K.1.2 Organization

This appendix is organized as follows:

- Section K.2 describes the exposure model, input parameters, and techniques to propagate uncertainty. Also presented in this section are the input data and exposure results for bald eagle, American bittern, and small-footed myotis.

- Section K.3 describes the effects to birds and mammals exposed to tPCBs and TEQ.

- The two potential lines of evidence are then discussed in the risk characterization section, Section K.4, as are the weight of evidence, sources of uncertainty regarding risk estimates, and the conclusions regarding risk for T&E species exposed to tPCBs and TEQ in the Housatonic River.

K.2 CHARACTERIZATION OF EXPOSURE

This exposure assessment for T&E species focuses on the PSA. Exposure assessments were not conducted at the reference locations because of a lack of prey tissue concentration data from these areas.

The species selected to represent T&E biota were the bald eagle, American bittern, and small-footed myotis. These T&E species occur in the PSA, potentially breed within the PSA, and feed on prey exposed directly to the COCs and through trophic transfer (see Appendix A). Exposure of these species to tPCBs and TEQ was estimated using a TDI model adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993). For a description of how TEQ was estimated, see Section 6.4 of the ERA.

This section begins with a description of the exposure model used for each of the representative species. Subsequent sections describe the inputs used in the exposure analysis for each representative species. The exposure assessment section concludes with a description of the Monte Carlo and probability bounds analyses conducted to estimate TDI of tPCBs and TEQ by each of the representative species in the Housatonic River PSA.

K.2.1 Exposure Model for T&E Species

The approach used to estimate exposure of T&E species to tPCBs and TEQ focused on ingestion of these contaminants through their diet. Other exposure routes (e.g., dermal, air) were not considered, because these routes were considered to be of negligible importance for tPCB and TEQ exposure to T&E species (Moore et al. 1999). The model used in the exposure analysis was:

$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i$$

where:

TDI = total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ)
FIR = normalized food intake rate (kcal/g bw/d)
FT = foraging time in the PSA (unitless)
P_i = proportion of *i*th food item in the diet (unitless)
C_i = concentration in *i*th food item (mg/kg tPCBs, ng/kg TEQ)

Each input variable is described for bald eagles, American bitterns, and small-footed myotis in the appropriate sections below.

K.2.1.1 Selecting Exposure Distributions

Input distributions were generally assigned as follows:

- Lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue).
- Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
- Point estimates for variables with low coefficients of variation.

In certain situations (e.g., poor fit to the data), other distributions were selected or other approaches were used. These latter situations are described in detail where they occurred in the following exposure analysis sections.

K.2.1.2 Incorporation of Spatial Averaging in Derivation of Estimated Prey Concentrations

Concentrations of tPCBs and TEQ vary spatially in prey. Many wildlife species, however, forage over distances ranging from tens of meters to greater than 10 km. An individual integrates the spatial variation in tissue concentrations of their prey. Therefore, estimates of the central tendency were used in the exposure model as an expression of the spatial and temporal averaging of concentrations of COCs in prey tissue. In the probabilistic exposure analyses, it was assumed that the spatially and temporally averaged exposure estimate did not vary between individuals foraging in the same area.

To account for uncertainty due to sample size, the measure of centrality in the Monte Carlo analysis was the minimum of (1) the 95% upper confidence limit (UCL) calculated using the Land H-statistic (as the data were lognormally distributed), or (2) the maximum concentration

measured. In the probability bounds analyses, however, the uncertainty regarding the arithmetic mean was accounted for with a different procedure. The procedure generally involved using the Land H-statistic to estimate the 95% lower confidence limit (LCL) and 95% UCL on the mean (Gilbert 1987), and then using the LCL and UCL to derive bounds on all possible distributions that exist within this range. The Land H-statistic has the underlying assumption that data are lognormally distributed. There were mechanistic reasons to assume lognormality for all prey concentration data sets; however, they were tested for lognormality using the Shapiro-Wilk test (see Section 6.3 of the ERA). This approach results in an expression of the uncertainty about the true value of the arithmetic mean that arises due to limited sample sizes. In cases where the 95% UCL could not be estimated or exceeded the data set maximum, other techniques were used to derive the bounds on the mean. These techniques and the criteria for their use are described in Section 6.5 of the ERA.

K.2.1.3 Techniques for Propagating Uncertainty

In this assessment, two types of probabilistic analysis were performed: Monte Carlo analysis and probability bounds analysis. The former is the most common probabilistic method employed in ecological risk assessments, and guidance for its use in Superfund assessments is available (EPA 1997). While Monte Carlo methods are appropriate for the determination of exposure risks when input distributions are known precisely, they do not adequately represent the effects of uncertainty around the input distributions (Ferson 1996). In many ecological risk assessments, the available data are quite limited and consequently the input distributions used to calculate risks are uncertain.

Probability bounds analysis is a tool for separating variability and uncertainty to obtain bounds on the result that explicitly account for the uncertainty about the input distributions. As in Monte Carlo analysis, the overall slopes of the bounds indicate how much variability exists in the system. The distance between the bounds, on the other hand, is an indication of the uncertainty that exists due to lack of knowledge. More detailed descriptions of Monte Carlo analysis and probability bounds analysis can be found in Appendix C.4.

For each Monte Carlo analysis, a sensitivity analysis was performed to determine the relative influence each input variable had on the output exposure distribution. This was done by

determining the correlation between the input values for a particular variable that were randomly chosen during the simulation and the corresponding output exposure estimates. Input variables that have a strong influence on the output exposure estimate tend to have high correlation coefficients, although this interpretation becomes problematic when there are dependencies between input variables. The results of the sensitivity analyses are included in this exposure assessment.

The exposure models for the ERA contain multiple variables, some of which may be correlated. The assumption of independence can be inappropriate, because even subtle dependencies can have substantial effects on the estimated exposure. If correlations are not accounted for, the variance and the tails of the exposure distribution may be poorly estimated. This assessment uses several approaches to address correlations between variables. These approaches include simulation of observed correlations, assumption of perfect covariance (e.g., when the diet consists of two prey items, the proportion of one item in the diet is equal to one minus the other item), or no assumptions at all about dependencies (all possible relationships between two variables can occur). The specific approach used depends on the type of data and the application. In cases where independence of variables seemed intuitively obvious (e.g., COC concentration in the prey item and proportion of that item in the diet), independence was assumed.

K.2.1.4 Treatment of Non-Detects

The approach for generating summary statistics or distributions when a data set includes samples with COC concentrations below the detection limit is described in detail in Appendix C.2. In summary, for data where contaminants were not detected (non-detect, ND), summary statistics were generated assuming that the contaminant concentration was zero (ND=0), and assuming that the contaminant concentration was equal to the detection limit (DL). If the ratio between the statistic of interest (e.g., mean) calculated assuming ND=DL and the statistic assuming ND=0 was less than or equal to 1.3, the analysis was performed assuming that all ND chemical concentrations were equal to half the detection limit (ND=1/2 DL)[1]. If the ratio was greater than

[1]This decision criterion supplements the procedures described in Appendix C.2.

1.3 and sample size and detection frequency were adequate (i.e., n >5, detection frequency >25%), the probability plot method described in Appendix C.2 was used to estimate concentrations for samples with COC concentrations below the detection limit. If sample size and detection frequency were not adequate, then the exposure calculations were performed assuming ND=0, ND=1/2 DL, and ND=DL. Figure K.2-1 summarizes the process for dealing with non-detects. A more detailed discussion of the treatment of NDs is presented in Section 6.4 of the ERA.

K.2.1.5 Bald Eagle

Bald eagles were chosen as one of the representative T&E species because the bald eagle is the only federally listed species that was observed in the PSA, habitat within the PSA is suitable for bald eagles, they have attempted to breed within the PSA and could do so in the future, and the prey they consume are directly exposed to COCs in the area. Therefore, bald eagles in the PSA could ingest tPCBs and TEQ through their diet. Typical dietary items of bald eagles are found in Table K.2-1. Suitable bald eagle habitat is found in the southern sections of the PSA below New Lenox Road (Reaches 5C, 5D, and 6), particularly the large open water areas of Woods Pond and its associated backwaters (Appendix A). Bald eagles were observed in the PSA foraging over these water bodies and roosting in large trees along the shoreline (Appendix A). A bald eagle pair attempted to nest in the PSA near Woods Pond in the mid-1990s; however, the nest was destroyed in an April snowstorm and the pair did not attempt to re-nest that year or subsequently (T. Gulo, MDFW, personal communication 2001).

Bald eagles have also nested along the Housatonic River in Connecticut. One bald eagle pair nested along the Housatonic River, just below Interstate 84. In 2001, one chick was reared. The pair returned in 2002 and displayed breeding activity (i.e., adding sticks to previous year's nest); however, no nesting was observed (J. Bictoria, CTDEP, personal communication 2002). Bald eagles are known to winter along the Housatonic River in southern Connecticut and feed in open water areas below Shepaug and Stevenson Dams (Veit and Petersen 1993; Bevier 1994; CTDEP 2000; Northeast Utilities 1998; Craig et al. 1988). Approximately 100 bald eagles winter in Connecticut, 25 of which are located along a 2-mile stretch of the Housatonic River, encompassing Shepaug Hydroelectric Station, George C. Waldo State Park, and the lower

portion of Lake Lillinonah (Northeast Utilities 1998; J. Bictoria, CTDEP, personal communication 2002).

K.2.1.5.1 Exposure Model Input Distributions

The parameterization of input variables used in the Monte Carlo and the probability bounds analyses to estimate exposure of bald eagles to tPCBs and TEQ are summarized in Tables K.2-2 and K.2-3. Additional information on each of the variables is provided below.

Body Weight (BW)

Body weight (BW) is not used in the model directly, but is a required variable in allometric models (e.g., Nagy 1987) to estimate food intake rates (FIRs) or free metabolic rates (FMRs). The typical weight of an adult bald eagle ranges from 3.0 kg to more than 7.0 kg. Adult males average 4.13 kg and adult females average 5.4 kg (Dunning 1992; EPA 1993; Buehler 2000; Canadian Wildlife Service 2000). For this risk assessment, the weight of female bald eagles was used because the effects endpoint is reproductive impairment and the female will have the greatest effect on survival of the young through transfer of PCBs to the egg.

In the Monte Carlo analysis, female body weight was assumed to be normally distributed with a mean of 5.35 kg and a standard deviation of 0.40 kg. The uncertainty in this variable is due to natural variability, rather than lack of knowledge or data (i.e., the variable is easily measured and many studies have been conducted which measured this variable). Accordingly, the same distribution was used in the probability bounds analysis for body weight of bald eagles.

Food Intake Rate (FIR)

In the EPA *Wildlife Exposure Factors Handbook* (EPA, 1993), three studies were reviewed for determining food intake rate. The first study (Stalmaster and Gessaman 1982) used captive eagles (obtained from a zoological garden), housed in 3 x 3 x 2.5-m chambers, in the feeding study. The second study (Stalmaster and Gessaman 1984) estimated feeding rates by remote observation of the amount of food consumed at feeding stations. Estimates were averages of the total estimated food consumed by the total number of eagles observed feeding, and assumed that the eagles fed exclusively at the stations, although the authors acknowledged that some birds fed

elsewhere. In the third study (Craig et al. 1988), Stalmaster and Gessaman's (1984) data were used to estimate prey consumption. Because of the problems associated with estimating food consumption in each of these studies, an FIR was derived from the estimated metabolic rate of free-living eagles using data from Nagy (1987) and Nagy et al. (1999).

The FIR developed from the Nagy studies was compared with those from EPA (1993). The measured FIRs reported in EPA (1993) are consistent with the FIR distribution estimated from the allometric equation. Measured values for free-flying eagles from Connecticut (Craig et al. 1988) were 0.12 to 0.14 g/g bw/d, whereas the median FIR from the allometric equation was 0.158 g/g bw/d.

Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic rate of free-living birds (*FMR*) in kilojoules per day using the following general equation:

$$FMR\ (kJ/d) = a \cdot BW(g)^b$$

For both the Monte Carlo and the probability bounds analyses, the FMR for bald eagles was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (*BW*, *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy et al. (1999), and further analyses of the data, assuming an underlying normal and lognormal distribution for each. Sufficient data were not available to generate an allometric equation for Falconiformes, of which bald eagles are members; therefore, the general bird equation was used. For these birds, log *a* had a mean of 1.02 and a standard error of 0.0393, and *b* had a mean of 0.681 and a standard error of 0.018 (Nagy et al. 1999). These values differ slightly from those presented in the paper, as two bird species were mistakenly not included in the calculations presented by Nagy et al. (1999) (K. Nagy, UCLA, personal communication 2002). The body weight distribution was described above. The results of the calculation were then converted to kcal/day.

FIR is derived from FMR using the following equation:

$$FIR = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$

where:

FMR = normalized free metabolic rate (kcal/kg bw/d)
AE_i = assimilation efficiency of ith food item (unitless)
GE_i = gross energy of ith food item (kcal/kg).

The gross energy of various components of wildlife diets is presented in the *Wildlife Exposure Factors Handbook* (EPA 1993). For bald eagles, the mean gross energy of fish is 1.2 kcal/g and the mean gross energy of both birds and mammals is 1.8 kcal/g (Table K.2-2).

The mean assimilation efficiency for fish consumed by birds is 79%. For the consumption of birds and mammals by birds, the mean assimilation efficiency is 78% (Table K.2-2). Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation (i.e., CV<10%).

Proportions of Dietary Items (P_i)

The proportions of prey items in bald eagle diets are listed in Table K.2-2. Studies of bald eagles in habitat similar to the PSA have found mean fish consumption to be 77.5% (range 71.0% to 90.1%) of the prey species taken during the breeding season. Bird species comprise on average 16.9% of the bald eagle diet in habitats similar to the PSA, but can be as little as 7.9% and as much as 26.1%. Consumption of mammals averages 4.8% and can range from 1.3% to 11.7%. Reptiles make up 0.24% of the diet and can range from 0% to 0.6%. Invertebrates, such as crayfish, crabs, and mussels, make up 0.12% of the diet on average and can range from 0% to 0.6% (Haywood and Ohmart 1986; Dunstan and Harper 1975; Todd et al. 1982; Watson et al. 1991; Stratus 1999). Reptiles and invertebrates were not included in the exposure analysis, however, because of their small contribution to the overall diet.

The proportion of prey items in the diet for the Monte Carlo analysis was parameterized to allow the diet to equal 1. This resulted in a diet of 50.6% bottom fish, 16.2% predatory fish, 11.8% forage fish, 16.4% birds, and 5.1% mammals (Table K.2-1). For the probability bounds analysis, the minimum, mean, and maximum values were used as a distribution-free statement in RiskCalc for each dietary item. The results are bounds that include all possible distributions, given the specified minimum, mean, and maximum values for the dietary items (Table K.2-3).

Stalmaster and Plettner (1992) studied wintering bald eagles along the North and South Platte Rivers. The study area was a 240-mile section of the river containing reservoirs, diversion and impoundment dams, supply canals, and hydroelectric and steam power plants. The majority of the water bodies freeze during the winter; however, several ice-free areas were available to bald eagles below hydroelectric dams and at a cooling pond of a power plant. Researchers observed foraging behavior at these areas and collected prey remains and pellets from below nearby perching and roosting trees. The habitat along the Housatonic River below Woods Pond is similar to this, with the majority of the river iced-over and open water areas occurring below dams. Therefore, the dietary ratios observed during the Stalmaster and Plettner (1992) study were used to calculate FIRs for bald eagles wintering along the Housatonic River. To account for observational bias between direct observations and examining prey remains, the data from both types of observations were averaged.

Foraging Times (FT)

All of the bald eagle sightings in the PSA occurred south of New Lenox Road and were primarily located at Woods Pond and the backwaters north of Woods Pond. Bald eagles would not be expected to regularly utilize the more shallow and narrow northern sections of the river. Therefore, the exposure area used for bald eagles was the southern portion of the PSA, from the more downstream portion of Reach 5B to Woods Pond. This entire area was assessed because individual bald eagles would likely forage throughout the area. Bald eagles nesting in the PSA would be expected to forage entirely within the PSA, as they generally forage within 0.3 mile (0.5 km) of the nest, with a maximum reported foraging distance of up to 5.0 miles (8.0 km) from their nest (Bowerman et al. 1995; Stratus 1999). As a result, for the purpose of modeling exposure, it was assumed that bald eagles would spend 100% of their time foraging in the PSA, based on the feeding habits of eagles and the availability of fish, waterfowl, and mammals in the PSA. The foraging time was specified as a point estimate.

Bald eagles also occur along the Housatonic River in southern Connecticut, where they have nested in Reach 15 (below Interstate 84), and also winter along the southern portions of the Housatonic River and feed in open water areas below Shepaug and Stevenson Dams (Northeast Utilities 1998). Exposures for bald eagles wintering in Reaches 14, 15, and 16 and nesting in Reach 15 were estimated.

K.2.1.5.2 Exposure Point Concentrations

Concentration of tPCBs in Fish (C_i) in Reach 5 and Woods Pond

Whole-body fish tissue concentrations of tPCBs and TEQ were used in this assessment. Concentrations were reconstituted as the weighted average of concentrations from fillet, offal, and ovary analyses for individual fish if they were not processed as whole fish. Fish tissue concentrations used for the bald eagle diet were derived from all of the fish samples collected in the lower portion of Reach 5 and Woods Pond, with the exception of composite samples of young-of-year or other very small fish (length <12 cm), too small to be consumed by eagles. Samples were divided into classes of fish: predatory, bottom feeder, and forage fish. Dietary studies show that bald eagles predominantly consume bottom feeders, such as suckers, bullheads, carp, and catfish. These species have the highest tPCB concentrations, whereas predatory fish have the intermediate concentrations and forage fish have the lowest. Therefore, evaluating the fish species according to dietary preference allows a more accurate representation of COC exposure.

In reconstituting fish that were analyzed as fillet and offal (the remainder of the fish after fillets were removed), there were five cases to consider. The cases and associated methods are described below.

1. Both fillets were analyzed by EPA as one sample.

   Whole body concentration (WBC) was estimated as:

$$\frac{(fwt * fconc) + (owt * oconc)}{(fwt + owt)}$$

   where:

   fwt = tissue weight of the fillet;
   fconc = concentration of the fillet;
   owt = tissue weight of the offal; and
   oconc = concentration of the offal.

2. Both fillets were analyzed by EPA, and one fillet was submitted as a duplicate sample for QA/QC purposes (fillets analyzed separately).

   WBC was estimated as:

$$\frac{(fwt * fconc) + (dupwt * dupconc) + (owt * oconc)}{(fwt + dupwt + owt)}$$

where:

dupwt = tissue weight for the duplicate fillet; and
dupconc = concentration for the duplicate fillet.

3. Ovaries and offal (including fillet) were analyzed separately by EPA.

   WBC was estimated as:

$$\frac{(2 * ovwt * ovconc) + (owt * oconc)}{(2 * ovwt + owt)}$$

where:

ovwt = ovary weight; and
ovconc = ovary concentration.

4. One fillet was analyzed by EPA, and one fillet was used as a split sample and submitted to GE. Although the weight of the GE fillet is known, there is no concentration available.

   WBC was estimated as:

$$\frac{(fwt * fconc) + (gewt * fconc) + (owt * oconc)}{(fwt + gewt + owt)}$$

where:

gewt = tissue weight of the GE fillet.

5. One fillet was analyzed by EPA, one fillet was used as a split sample and analyzed by GE, and data are available for the weight and concentration of the GE fillet.

   WBC was estimated as:

$$\frac{(fwt * fconc) + (gewt * geconc) + (owt * oconc)}{(fwt + gewt + owt)}$$

where:

geconc = concentration for the GE fillet.

The bottom feeder component of the bald eagle diet included 136 samples of brown bullhead (*Ictalurus nebulosus*), common carp (*Cyprinus carpio*), goldfish (*Carassius auratus*), and white

sucker (*Catostomus commersoni*). The predatory fish component included 134 samples of largemouth bass (*Micropterus salmoides*) and yellow perch (*Perca flavescens*). The forage fish component included 50 samples of pumpkinseed (*Lepomis gibbosus*). The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL was the lower of the two, with a value of 104 mg/kg for bottom fish, 88.6 mg/kg for predatory fish, and 43.4 mg/kg for forage fish (Table K.2-4).

For the probability bounds analyses, the 95% LCL and 95% UCL were used to parameterize a distribution-free statement in RiskCalc. The 95% LCL and 95% UCL were 78.4 and 104 mg/kg for bottom fish, 72.3 and 88.6 mg/kg for predatory fish, and 32.9 and 43.4 mg/kg for forage fish (Table K.2-4).

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Fish (C_i) in Reach 5 and Woods Pond

TEQ was measured in 60 bottom feeders, 66 predatory fish, and 30 forage fish. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean assuming ND = DL/2. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL value was the lower of the two. These values were 1,570 ng/kg for predatory fish, 1,480 ng/kg for bottom fish, and 777 ng/kg for forage fish (Table K.2-5).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum, assuming that ND = DL/2, was calculated using the greater of the 95% LCL and

the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set, the minimum and maximum values were used, representing ranges of 1,160 to 1,570 ng/kg for predatory fish; 1,040 to 1,480 ng/kg for bottom fish; and 548 to 777 ng/kg for forage fish (Table K.2-5).

Concentration of tPCBs in Fish (C_i) Downstream

Bald eagles winter downstream of the PSA in Reaches 14, 15, and 16. Twenty-one fish samples (from fish greater than 12 cm) were available from these reaches. The mean concentration was 8.09 mg/kg tPCBs, the median was 7.27, the minimum was 0.11 mg/kg , and the maximum was 29.4 mg/kg tPCBs.

Fish from Reach 15 were used to calculate the exposure for bald eagles nesting downstream. Six fish samples were available from this reach. The mean was 0.717, the median was 0.742, the minimum was 0.110, and the maximum was 1.70 mg/kg tPCBs.

Concentration of tPCBs in Waterfowl (C_i) in Reach 5 and Woods Pond

Waterfowl data were collected from trapping locations in Reaches 5C, 5D, and 6. Two species were collected, mallards (*Anas platyrhynchos*) and wood ducks (*Aix sponsa*). Data from all trapping locations and both species were averaged to derive concentrations for the diet. WBCs were reconstituted using the following equation:

$$WBC = \frac{(bwt * bconc) + (lwt * lconc) + (owt * oconc)}{(bwt + lwt + owt)}$$

where:

bwt = tissue weight of the breast
bconc = concentration for the breast
lwt = tissue weight for the liver
bconc = concentration for the liver
owt = tissue weight for the offal
oconc = estimated concentration for the offal

Only breast and liver tissue samples were analyzed; therefore, WBCs were estimated by calculating potential high, best estimate, and low concentrations. Low concentrations assumed

that the offal had COC concentrations of zero. The moderate concentrations assumed that the offal had COC concentrations equal to the corresponding breast concentrations. The high concentrations assumed that the offal had COC concentrations equal to the greater of the corresponding liver and breast concentrations.

Twenty-five duck tissue samples were collected from Reaches 5C, 5D, and 6. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean for the scenario assuming that the offal concentration was low (offal = zero concentration) and again for the scenario assuming that the offal concentration was high (offal = the higher of the liver and breast concentration). A triangular distribution was used in the Monte Carlo analysis to capture the uncertainty about the estimate of the measure of centrality. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean concentrations for the low, moderate, and high concentration estimates. The minimum value of the triangular distribution was calculated using the lower of the maximum value in the low (offal = zero) estimate and the 95% UCL. The best estimate of the triangular distribution was calculated using the lower of the maximum value in the moderate (offal = breast) estimate and the 95% UCL. The triangular distribution maximum was calculated using the lower of the maximum value for the high (offal = liver) estimate and the 95% UCL. The resulting triangular distribution parameters were: minimum = 2.03 mg/kg, best estimate = 9.90 mg/kg, and maximum = 19.9 mg/kg (Table K.2-4).

For the probability bounds analyses, the uncertainty regarding the tPCB concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum value was calculated using the higher of the minimum value in the low (offal = zero) estimate and the 95% LCL. The maximum value was calculated using the higher of the minimum value in the high (offal = liver) estimate and the 95% LCL. The resulting values range from a minimum of 1.22 to a maximum of 19.9 mg/kg.

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Waterfowl (Ci) in Reach 5 and Woods Pond

TEQ was measured in 25 waterfowl samples. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that

congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean for the scenario assuming that the offal concentration was low (offal = zero concentration) and again for the scenario assuming that the offal concentration was high (offal = the higher of the liver and breast concentration). A triangular distribution was used in the Monte Carlo analysis to capture the uncertainty about the estimate of the measure of centrality. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean concentrations for the low, moderate, and high concentration estimates. The minimum value of the triangular distribution was calculated using the lower of the maximum value in the low (offal = zero) estimate and the 95% UCL. The best estimate of the triangular distribution was calculated using the lower of the maximum value in the moderate (offal = breast) estimate and the 95% UCL. The triangular distribution maximum was calculated using the lower of the maximum value for the high (offal = liver) estimate and the 95% UCL. The resulting triangular distribution parameters were: minimum = 485 ng/kg, best estimate = 2,149 ng/kg, and maximum = 4,020 ng/kg (Table K.2-5).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum was calculated using the greater of the 95% LCL and the minimum value from the low (offal = 0) estimate. The maximum value was calculated using the lower of the maximum value and the 95% UCL from the high (offal = liver) estimate. In this data set, the minimum and maximum values were used representing ranges of 232 to 4,020 ng/kg (Table K.2-5).

Concentration of tPCBs and TEQ in Waterfowl (Ci) Downstream of Woods Pond

Waterfowl tissue concentrations for locations south of Woods Pond were not available. Waterfowl wintering along the southern Housatonic River are likely to include many individuals that migrated from other areas outside the Housatonic River drainage and would, therefore, be expected to have minimal concentrations of COCs related to the site. For this reason, waterfowl tissue concentrations for wintering bald eagles were assumed to be zero. For exposure to bald

eagles breeding downstream, tPCB concentrations for waterfowl were calculating in three ways to develop high, moderate, and low concentrations. High concentrations were calculated assuming that waterfowl from downstream would have tPCB concentrations equal to those used in the PSA assessment, 9.90 mg/kg tPCBs. Low concentrations were calculated assuming that waterfowl would have tPCB concentrations of zero. A moderate concentration was developed by determining fish to bird ratios based on concentrations in the PSA. Waterfowl tPCB concentrations averaged 15% of the total fish concentration. Therefore, moderate tPCB concentrations downstream were 0.108 mg/kg for birds.

Concentration of tPCBs in Small Mammals (C_i)

Small mammal data were available from three sampling locations in the PSA, one each in Reaches 5A, 5B, and 5C. However, the data were not representative of each reach because locations were chosen based on tPCB concentrations in the soil to achieve the study design objectives of sampling areas with low, moderate, and high concentrations. Location 14 in Reach 5A had moderate concentrations (1 to 30 mg/kg), Location 13 in Reach 5B had high concentrations (>30 mg/kg), and Location 15 in Reach 5C had low concentrations (<1 mg/kg). Therefore, small mammal data from all of the sites, which included 24 short-tailed shrews and 52 white-footed mice, were averaged to determine small mammal tPCB and TEQ tissue concentrations that would represent the variability in concentrations in the exposure area. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL was the lower of the two, with a value of 80.6 mg/kg. For the probability bounds analyses, the 95% LCL and 95% UCL, 23.2 and 80.6 mg/kg, respectively, were used to parameterize a distribution-free statement in RiskCalc (Table K.2-4).

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Small Mammals (C_i)

TEQ concentrations were measured in 12 small mammal samples collected from the three sampling locations. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet

concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=DL/2 data (value was equal to half the DL). The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the maximum value was the lower of the two, with a value of 3,980 ng/kg (Table K.2-5).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. The minimum, assuming that ND=DL/2, was calculated using the greater of the 95% LCL and the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set the minimum and maximum values were used, representing a range from 18.8 to 3,980 ng/kg (Table K.2-5).

Concentration of tPCBs in Mammals (Ci) Downstream of Woods Pond

Mammal tissue concentrations for locations south of Woods Pond were not available. When estimating exposure to bald eagles, tissue concentrations in mammals for downstream reaches were estimated three ways to derive high, moderate, and low concentrations. For the high estimate, mammals downstream were assumed to have the same concentration as those in the PSA: 80.66 mg/kg tPCB. For the moderate estimate, mammals were assumed to have a similar mammal-to-fish ratio of tPCBs as those in the PSA (mammals concentrations were 75% of fish concentrations). This resulted in a moderate estimated concentration of 0.538 mg/kg tPCBs. For the low estimate, mammals were assumed to have a tPCB concentration of zero. This may underestimate or overestimate the true exposure to eagles breeding downstream in Connecticut. Mammals were not included in the winter bald eagle diet.

K.2.1.5.3 Concentrations in Bald Eagle Eggs and Adult Females

The majority of toxicity studies conducted for bald eagles have focused on the COC concentration in eggs and effects on hatching and fledgling success. To compare potential

effects to bald eagles residing in the PSA to these studies, the female body burdens and associated egg concentrations were estimated. Toxicity thresholds established from laboratory and field studies of raptor species were compared in the risk characterization to estimated doses for bald eagles foraging and reproducing in the PSA.

The fate of COCs in birds was evaluated before estimating the body burden in eggs and adult females. The mechanisms of metabolism and excretion of PCBs are complex and vary with the number of chlorine substituent groups and their location, metabolism, and species. Avian species in general have little capacity to metabolize highly chlorinated PCBs (Nichols et al. 1995). Therefore, the assumption that avian species do not metabolize PCBs was made to simplify the estimated accumulation by bald eagles.

The mean chemical absorption efficiencies (CAE) for PCBs in avian species range from 0.80 to 0.97. The CAE for PCBs is similar to the CAE for lipids, which is 0.90 (Drouillard and Norstrom 2000). In ring doves (*Streptopelia risoria*), the CAE was 0.936 ± 0.003 and ranged from 0.86 to 0.97 for PCB153 (Drouillard and Norstrom 2000). Common terns (*Sterna hirundo*) were found to have CAEs of 0.8 to 0.96 for the coplanar PCBs, PCB-77 and PCB-169 (Bosveld 1995). Loss of PCB to feces through biliary excretion, intestinal content/feces portioning, sloughing off of intestinal cells during digestion, and loss through urine may contribute to underestimates of PCB CAEs; however, these losses are considered to be small for PCBs (Drouillard and Norstrom 2000).

Dahlgren et al. (1971) studied the retention of PCBs (Aroclor 1254) in pheasants. Pheasants injected with 2, 20, or 200 mg/kg PCBs and sacrificed after 24 hours retained the following proportions of the injected PCBs: 0.84 ± 0.06, 0.94 ± 0.05, and 1.03 ± 0.06, respectively. The average retention was 0.94 ± 0.07 for the three exposure doses. Pheasants given a single 50 mg dose of PCBs orally retained 40.5 mg (81%) after 28 days. Pheasants laid an average of 4 eggs over the 28-day period with average excretion through the eggs of 4.2 mg (8.4%) PCBs. An average of 4.0 mg PCBs was passed in the feces over the 28 days. The first week, 3.1 mg PCBs (6.2%) was excreted, 0.37 mg (0.7%) the second week, 0.33 mg (0.7%) the third, and 0.26 mg (0.5%) the fourth. Therefore, only 2.4% of the tPCBs dose was metabolized over a 28-day period.

Low, mean, and high concentrations of tPCBs and TEQ in bald eagle tissues (whole body) were estimated for an average (5,350 g) female bald eagle in the PSA. The low concentration was developed using the 10th percentile from the Monte Carlo analysis and assuming a low chemical absorption efficiency of 0.80. The mean concentration was developed using the mean TDI from the Monte Carlo analysis and assuming a mean chemical absorption efficiency of 0.89. The high concentration was developed using the 90th percentile from the Monte Carlo analysis and assuming a high chemical absorption efficiency of 0.97. These estimated tissue concentrations assumed that adult bald eagles arrived in the PSA on day one with no PCBs in their tissues. This likely underestimated the true PCB concentrations in adult eagles, as eagles arriving in the PSA would likely have some background concentrations of PCBs. Also, eagles return to the same breeding territory each year; therefore, they would likely have PCB burdens accumulated during previous years.

Transfer rates of maternal organochlorines to eggs vary depending upon species, reproductive strategy, egg size relative to maternal body size, chlorination, and concentration. Avian species have reported transfer rates of 1 to 20% of the maternal body burden (Bargar et al. 2001). Precocial species with relatively large eggs and few eggs in a clutch are likely to have the highest transfer rates, while altricial species with relatively small eggs and large clutch sizes will have lower transfer rates (Dahlgren et al. 1971; Lemmetyinen et al. 1982; Tanabe et al. 1986; Bargar et al. 2001; Drouillard and Norstrom 2001). Bald eagles are semialtricial with small clutch sizes and relatively small eggs compared to body size. Therefore, it was assumed that bald eagle transfer rates would fall in the middle of the known range of transfer rates (i.e., 10%).

Bald eagles migrate to the PSA in mid February to early March (Buehler 2000). Egg laying is likely to occur from March to May in the PSA (Veit and Petersen 1993). Assuming egg laying occurs on 1 April, the bald eagle will have been in the PSA approximately 30 days prior to egg laying.

K.2.1.5.4 Exposure Model Results

Exposure to tPCBs in the PSA

The Monte Carlo analysis indicated that exposure of bald eagles to tPCBs could range from a minimum of 6.23 mg/kg bw/d to a maximum of 25.4 mg/kg bw/d. The mean exposure was 13.2 mg/kg bw/d, and the median exposure was 13.0 mg/kg bw/d. Eighty percent of the exposure estimates were between 10.2 and 16.6 mg/kg bw/d. Figure K.2-2 depicts the cumulative distribution for bald eagle in the southern PSA (Table K.2-6).

Sensitivity analysis (Table K.2-7) revealed that the FMR power term used in the FMR equation was the most important variable (Pearson correlation coefficient (r_p) = 0.85) followed by the FMR slope term (r_p = 0.47), body weight (r_p = -0.13), waterfowl concentration (r_p = 0.02), proportion diet – bottom fish (r_p = 0.02), and proportion diet – birds (r_p = -0.01). Clarification of the significance of the FMR slope term and FMR power term is found in Section 6.

The probability bounds estimated for bald eagle foraging in the southern PSA are depicted in Figure K.2-2. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 3.73 and 13.4 mg/kg bw/d. The 50th percentile ranged between 5.41 and 17.9 mg/kg bw/d, and the 90th percentile ranged between 8.27 and 24.2 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 10.26, the 50th percentile was 13.0, and the 90th percentile was 16.6 mg/kg bw/d (Table K.2-6).

Exposure to 2,3,7,8-TCDD Toxic Equivalence (TEQ) in the PSA

The Monte Carlo analysis indicated that exposure of bald eagles to TEQ could range from a minimum of 117 ng/kg bw/d to a maximum of 514 ng/kg bw/d. The mean exposure was 256 ng/kg bw/d and the median exposure was 251 ng/kg bw/d. Eighty percent of the exposure estimates were between 197 and 321 ng/kg bw/d (Table K.2-8). Figure K.2-3 depicts the cumulative distribution of TEQ intake rates for bald eagle in the southern PSA.

Sensitivity analysis (Table K.2-7) revealed that the FMR power term used in the FMR equation was the most important variable (Pearson correlation coefficient (r_p) = 0.85) followed by the FMR slope term (r_p = 0.48), body weight (r_p = -0.14), waterfowl concentration (r_p = -0.03), proportion diet – bottom fish (r_p = 0.01), proportion diet – predatory fish (r_p = 0.01), and

proportion diet – forage fish (r_p = -0.01). Discussion of the significance of the FMR slope term and FMR power term is provided in Section 6.

The probability bounds estimated for TEQ exposure to bald eagle foraging in the southern PSA are depicted in Figure K.2-3. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 52.3 and 291 ng/kg bw/d. The 50th percentile ranged between 76.6 and 388 ng/kg bw/d, and the 90th percentile ranged between 115 and 526 ng/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 197, the 50th percentile was 251, and the 90th percentile was 321 ng/kg bw/d (Table K.2-8).

tPCBs Concentrations in Eggs in the PSA

Female bald eagles present in the PSA for 30 days prior to egg laying are estimated to have a mean tPCB egg concentration of 35.3 mg/kg, a low egg concentration of 23.0 mg/kg, and a high concentration of 51.5 mg/kg (Figure K.2-4).

TEQ Concentrations in Eggs in the PSA

Female bald eagles present in the PSA for 30 days prior to egg laying are estimated to lay eggs with a mean TEQ concentration of 683 ng/kg, a low concentration of 440 ng/kg, and a high concentration of 997 ng/kg (Figure K.2-5).

K.2.1.6 American Bittern

The American bittern was selected as a representative T&E species because American bitterns are listed as Endangered in Massachusetts and Connecticut (CTDEP 2000; MNHESP 2001), habitat within the PSA is suitable for American bitterns, they breed within the PSA, and the prey they consume are directly exposed to COCs in the area. Therefore, American bitterns in the PSA could ingest tPCBs and TEQ through their diet. Typical dietary items of American bitterns are found in Table K.2-9. American bitterns were documented in the PSA from 1998 to 2001, and in the October Mountain State Forest reference location in 2000 (Appendix A). Observations during the 3 years occurred from May to September, with most occurring in August and September. Bitterns were usually observed in wet meadow, shallow emergent marsh, and deep emergent marsh habitats throughout the southern portion of the PSA. However, suitable habitat

is available throughout the entire PSA and occasional observations of American bitterns occurred in northern sections. On two occasions (early July 1999 and May-June 2001), bitterns were heard calling from within the PSA, indicating intent to breed in the area (see Appendix A; E. Greenburg, R2 Consultants, Inc., personal communication 2001). Bitterns have also been known to breed in the backwaters north of Woods Pond (S. Melvin, MNHESP, personal communication 2002).

Small areas of American bittern breeding habitat occur along the Housatonic River downstream of Woods Pond in southern Berkshire County, Massachusetts, and in Litchfield County, Connecticut. No breeding events have been confirmed from the Housatonic floodplain south of Woods Pond in Massachusetts (S. Melvin, MNHESP, personal communication 2002). Several confirmed or probable breeding events have occurred in Connecticut in the last decade; however, these were outside of the Housatonic River floodplain (Veit and Petersen 1993; Bevier 1994; CTDEP 2000; MNHESP 2001).

K.2.1.6.1 Exposure Model Input Distributions

The input variable parameterizations used in the Monte Carlo and probability bounds analyses to estimate exposure of American bitterns to tPCBs and TEQ are summarized in Table K.2-10 and K.2-11. Additional information on each of the input variables is provided below.

Body Weight (BW)

The typical weight of an adult American bittern ranges from 370 g to >800 g (Gibbs et al. 1992; Dunning 1992). In the Monte Carlo analyses, body weight was assumed to be normally distributed, with a mean body weight of 707 g and a standard deviation of 183 (Dunning 1992). The uncertainty in this variable is due to variability, rather than lack of knowledge or data. Thus, the same distribution was used in the probability bounds analyses for this input variable.

Food Intake Rate (FIR)

The food intake rate of American bittern has not been measured. Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic rate of free-living birds using the following general equation:

$$FMR\ (kJ/d) = a \cdot BW(g)^b$$

For both the Monte Carlo and the probability bounds analyses, the FMR for American bittern was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (BW, *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy et al. (1999) and further analyses of the data, assuming an underlying normal distribution for each. The general bird equation was used for American bitterns. For this equation, log *a* had a mean of 1.02 and a standard error of 0.0393, and *b* had a mean of 0.681 and a standard error of 0.018 (Nagy et al. 1999). These values differ slightly from those presented in the paper, as two bird species were mistakenly not included in the calculations presented by Nagy et al. (1999) (K. Nagy, UCLA, personal communication 2002). The BW distribution was described above. The results of the calculation were then converted to kcal/day.

FIR is derived from FMR using the following equation:

$$FIR = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$

where:

FMR = normalized free metabolic rate (kcal/kg bw/d)
AE_i = assimilation efficiency of *i*th food item (unitless)
GE_i = gross energy of *i*th food item (kcal/kg)

The gross energy of various components of wildlife diets is presented in the *Wildlife Exposure Factors Handbook* (EPA 1993). The mean gross energy of both fish and amphibians is 1.2 kcal/g. The mean gross energy of mammals is 1.8 kcal/g and the gross energy of invertebrates is 1.6 kcal/g (Table K.2-10).

The mean assimilation efficiency for fish and amphibians consumed by birds is 79%. For the consumption of mammals by birds, the mean assimilation efficiency is 78%. For the consumption of invertebrates by birds, the mean assimilation efficiency is 72% (Table K.2-10).

Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation (i.e., CV<10%).

Proportion of Dietary Items (P_i)

An analysis of the stomach contents of 160 individuals reported that the American bittern diet consisted of invertebrates (23%), amphibians (21%), fish (21%), crayfish (19%), small mammals (10%), and snakes (5%) (Cottam and Uhler 1945, as cited in Gibbs et al.1992). It was assumed that American bitterns would consume the same proportion of prey items in each reach for this assessment. Reptiles were not included in the exposure analysis, however, because of the small contribution to the overall diet. The proportion in the diet for the Monte Carlo and probability bounds analysis was parameterized to allow the diet to equal 1. This resulted in a diet of 24.5% invertebrates, 20.2% macroinvertebrates (crayfish), 22.3% fish, 22.3% amphibians, and 10.6% small mammals (Table K.2-10).

Foraging Times (FT)

American bittern habitat occurs throughout the PSA, and bitterns have been observed from Canoe Meadows Wildlife Sanctuary south to Woods Pond (Appendix A). In addition, home range sizes and habitat requirements for this species are such that individuals forage predominantly within a reach. Therefore, for the analyses, four reaches were evaluated: Reach 5A, Reach 5B, Reach 5C, and Reaches 5D and 6 combined. For the TEQ analyses, samples from the PSA were combined in one analysis because the smaller sample sizes did not allow for statistically robust analyses to be conducted for each individual reach.

American bittern nesting in the PSA would be expected to forage entirely within the PSA and as discussed above, within the reaches, as they have territories averaging 315 acres (DeGraaf and Yamasaki 2001). As a result, for the purpose of modeling COC exposure, it was assumed that American bitterns would spend 100% of their time foraging in the PSA. The foraging time was specified as a point estimate.

K.2.1.6.2 Exposure Point Concentrations

Concentration of tPCBs in Fish (C_i)

Whole-body fish concentrations were reconstituted as described in Section K.2.1.5.2 from fillet, offal, and ovary data for individual fish. Reconstituted whole-body fish and composite fish samples consisting of fish that had lengths less than 10.0 cm were used. Fish species included largemouth bass, yellow perch, fallfish (*Semotilus corporalis*), bluegill (*Lepomis macrochirus*), pumpkinseed, golden shiner, and common carp. Twelve samples were collected in Reach 5A, 91 in Reach 5B, 14 in Reach 5C, and 82 in Reaches 5D and 6 (Table K.2-12). The Land H-statistic was used to determine the 95% LCL and 95% UCL of the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL was the lower of the two. For the probability bounds analyses, the 95% LCL and 95% UCL were used to parameterize a distribution-free statement in RiskCalc.

Reach	Monte Carlo UCL (mg/kg)	Probability Bounds LCL, UCL
Reach 5A	49.4	38.5, 49.4
Reach 5B	28.8	26.7, 28.8
Reach 5C	35.9	2.59, 35.9
Reach 5D and 6	32.5	27.5, 32.5

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Fish (C_i)

TEQ concentrations were measured in 48 fish samples collected from the PSA. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=DL/2 data (value was equal to half the DL). The lower of the maximum measured concentration and the 95% UCL was used as a

point estimate in Monte Carlo analysis. In this analysis, the 95% UCL value was the lower of the two, with a value of 622 ng/kg (Table K.2-13).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RickCalc. The minimum, assuming that ND=DL/2, was calculated using the greater of the 95% LCL and the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set, the minimum and maximum values were used representing a range from 492 to 622 ng/kg (Table K.2-13).

Concentration of tPCBs in Amphibians (C_i)

Amphibian data from two studies were used to develop amphibian tissue concentrations for use in the exposure model. Bullfrog tissue data were available from Woods Pond and the associated backwaters. The bullfrog tissue data were reconstituted from leg muscle and offal tissue concentrations in a manner analogous to that described previously for fish samples. Leopard frog tissue data were available from several sites located throughout the PSA. The leopard frog data were grouped according to the subreach from which they were collected. Amphibian tissue concentrations were estimated from the two frog species combined. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL was the lower of the two. For the probability bounds analyses, the 95% LCL and 95% UCL were used to parameterize a distribution-free statement in RiskCalc.

Reach	Monte Carlo UCL (mg/kg)	Probability Bounds LCL, UCL
Reach 5A	5.39	1.21, 5.39
Reach 5B	6.33	0.55, 6.33
Reach 5C	7.73	0.16, 7.73
Reach 5D and 6	9.22	0.03, 9.22

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Amphibians (C_i)

TEQ concentrations were measured in five amphibian samples collected from the PSA. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=DL/2 data (value was equal to half the DL). The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the maximum value was the lower of the two, with a value of 81.5 ng/kg (Table K.2-13).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RickCalc. The minimum, assuming that ND=DL/2, was calculated using the greater of the 95% LCL and the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set, the minimum and maximum values were used representing a range from 36.6 to 81.5 ng/kg (Table K.2-13).

Concentration of tPCBs and TEQ in Small Mammals (C_i)

Small mammal data were averaged (as described for bald eagles) across the PSA for both tPCBs and TEQ. In the Monte Carlo analysis, the tPCB concentration was 80.7 mg/kg, and the corresponding value for TEQ was 3,980 ng/kg. The 95% LCL and 95% UCL were 23.2 and 80.7 mg/kg tPCBs and 18.8 and 3,980 ng/kg TEQ.

Concentration of tPCBs in Insects (C_i)

Invertebrate tPCB concentrations were available from composite tissue samples collected from the benthic invertebrate sampling program. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the

95% UCL was used as a point estimate in Monte Carlo analysis. For the probability bounds analyses, the 95% LCL and 95% UCL were used to parameterize a distribution-free statement in RiskCalc.

Reach	Monte Carlo UCL (mg/kg)	Probability Bounds LCL, UCL
Reach 5A	18.2	2.23, 18.2
Reach 5B	32.5	28.6, 32.5
Reach 5C	47.6	6.38, 47.6
Reach 5D and 6	14.9	2.29, 14.9

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Insects (C_i)

TEQ concentrations were measured in six invertebrate samples collected from the PSA. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=DL/2 data (value was equal to half the DL). The lower of the maximum measured concentration and the maximum was used as a point estimate in Monte Carlo analysis. In this analysis, the maximum value was the lower of the two, with a value of 2,670 ng/kg (Table K.2-13).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RickCalc. The minimum, assuming that ND=DL/2, was calculated using the greater of the 95% LCL and the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set, the minimum and maximum values were used representing a range from 43.9 to 4,610 ng/kg (Table K.2-13).

Concentration of tPCBs in Crayfish (Ci)

Crayfish (macroinvertebrate) samples were available from Reaches 5A, 5B, 5C, and 5D. No crayfish data were collected in Reach 6. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. For the probability bounds analyses, the 95% LCL and 95% UCL were used to parameterize a distribution-free statement in RiskCalc.

Reach	Monte Carlo UCL (mg/kg)	Probability Bounds LCL, UCL
Reach 5A	36.2	15.8, 36.2
Reach 5B	19.9	8.97, 19.9
Reach 5C	11.8	6.55, 11.8
Reach 5D and 6	8.90	5.48, 8.90

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Crayfish (C_i)

TEQ concentrations were measured in 12 crayfish samples collected from the PSA. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation that co-eluted with others compromised 100% of the doublet and triplet concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The assumption of how to treat ND congeners in the TEQ calculations also did not have an important influence on the estimated TEQ concentration. Therefore, the Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=DL/2 data (value was equal to half the DL). The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. In this analysis, the 95% UCL value was the lower of the two, with a value of 818 ng/kg (Table K.2-13).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RickCalc. The minimum, assuming that ND=DL/2, was calculated using the greater of the 95% LCL and

the minimum value. The maximum value was calculated using the lower of the maximum value and the 95% UCL. In this data set, the minimum and maximum values were used representing a range from 224 to 818 ng/kg (Table K.2-13).

K.2.1.6.3 COC Concentrations in American Bittern Eggs

As with bald eagles, the majority of the toxicity literature for heron species reported toxicity based on egg concentrations. Therefore, egg concentrations of American bitterns breeding in the PSA were estimated and compared to literature toxicity thresholds. High, median, and low accumulation of tPCBs and TEQ by American bitterns was estimated for each reach using the methods described above for bald eagles. American bitterns migrate to the PSA in mid March. Egg laying typically occurs from 1 May to 13 June in Massachusetts (Veit and Petersen 1993). Therefore, an adult American bittern is likely to forage in the PSA between 45 and 90 days before egg laying begins.

K.2.1.6.4 Exposure Model Results

Exposure to tPCBs in Reach 5A

The Monte Carlo analysis indicated that exposure of American bittern to tPCBs could range from a minimum of 4.70 to a maximum of 18.6 mg/kg bw/d. The mean exposure was 9.24 mg/kg bw/d, and the median exposure was 9.07 mg/kg bw/d (Table K.2-14). Eighty percent of the exposure estimates were between 7.30 and 11.4 mg/kg bw/d. Figure K.2-6 depicts the cumulative distribution for American bittern in Reach 5A of the PSA.

Sensitivity analysis (Table K.2-15) revealed that the FMR power term used in the FMR equation was the most important variable (Pearson correlation coefficient (r_p) = 0.66), followed by the FMR slope term (r_p = 0.50) and the body weight (r_p = -0.48). Discussion of the significance of the FMR slope term and FMR power term is provided in Section 6.

The probability bounds estimated for American bittern foraging in Reach 5A of the PSA are depicted in Figure K.2-6. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 3.53 and 7.44 mg/kg bw/d. The 50th percentile ranged between 4.42 and 9.19 mg/kg bw/d, and the 90th percentile ranged between 5.49 and 11.7 mg/kg

bw/d. In comparison, the 10th percentile of the Monte Carlo output was 7.30, the 50th percentile was 9.07, and the 90th percentile was 11.4 mg/kg bw/d (Table K.2-14).

Other Exposure Scenarios

Exposures of American bittern to tPCBs in Reaches 5B, 5C, and 5D and 6 were similar or lower than in Reach 5A, having a mean TDI of 7.84, 9.03, and 6.53 mg/kg bw/d, respectively (Table K.2-14). Thus, exposure of American bittern to tPCBs is similar for all reaches of the PSA. The uncertainty of these exposure estimates, as illustrated by the probability bounds distributions, indicates a similar degree of uncertainty for all reaches in the PSA (Figures K.2-6 to K.2-9).

Mean exposure of American bittern to TEQ was 372 for the PSA (Table K.2-16). Figure K.2-10 depicts the cumulative distribution for TEQ intake, as well as the probability bounds.

For each exposure scenario, sensitivity analysis revealed that the FMR slope was the most important variable in the Monte Carlo analysis (Table K.2-15).

tPCBs Concentrations in Eggs in Reaches 5A, 5B, 5C, and 5D and 6

Estimated tPCB egg concentrations for American bitterns over time are shown in Figures K.2-11 through K.2-14. The lowest egg concentrations after 45 days in the PSA were 24.7, 21.0, 24.2, and 17.5 mg/kg for Reaches 5A, 5B, 5C, and 5D and 6, respectively. Mean egg concentrations in the PSA after 45 days were 37.0, 31.4, 36.2, and 26.2 mg/kg for Reaches 5A, 5B, 5C, and 5D and 6, respectively. High egg concentrations after 45 days in the PSA were 53.0, 44.9, 51.8, and 37.4 mg/kg for Reaches 5A, 5B, 5C, and 6, respectively.

2,3,7,8-TCDD Toxic Equivalence (TEQ) Concentrations in Eggs in Reaches 5 and 6

The estimated TEQ egg concentration for American bitterns over time is shown in Figure K.2-15. The lowest egg concentration after 45 days in the PSA was 898 ng/kg, the mean concentration was 1,490 ng/kg, and the high concentration was 2,290 ng/kg.

K.2.1.7 Small-Footed Myotis

The small-footed myotis was chosen as one of the representative T&E species because it is a Species of Special Concern in Massachusetts; there is suitable small-footed myotis habitat within the PSA; myotis could potentially breed in the PSA, and the prey they consume are directly exposed to COCs in the area. Therefore, small-footed myotis in the PSA could ingest tPCBs and TEQ through their diet. Typical dietary items for small-footed myotis are found in Table K.2-17. Suitable summer habitat for small-footed myotis is present in and adjacent to the PSA, and it is believed that the small-footed myotis occurs there. Small-footed myotis have been recorded in western Massachusetts and have been documented in adjacent Hampden County, Massachusetts (MNHESP 1984; Godin 1977), making their presence in the PSA likely. Other studies conducted in the region reported small-footed myotis observations (Zimmerman and Glanz 2000; Krusic et al. 1996). Small-footed myotis are unlikely to occur in southern Berkshire County because little suitable habitat is found there and small-footed myotis are believed extirpated from Connecticut (CTDEP 2000).

K.2.1.7.1 Exposure Model Input Distributions

The input variable parameterizations used in the Monte Carlo and probability bounds analyses to estimate exposure of small-footed myotis to tPCBs and TEQ are summarized in Table K.2-18 and K.2-19. Additional information on each of the input variables is provided below.

Body Weight (BW)

Body weight is not used in the model directly, but is a required variable in allometric models (e.g., Nagy 1987) to estimate FIRs or FMRs. Small-footed myotis typically weigh 5 to 7 g, although their weights can range from 3 to 8 g (Kurta 1995). In the Monte Carlo analysis a mean BW of 6 g with a standard deviation of 0.7 g was used for small-footed myotis. Body weight was assumed to be normally distributed. The uncertainty in this variable was likely due to natural variability, rather than lack of knowledge or data (i.e., the variable is easily measured and many studies have been conducted which measured this variable). Thus, the same distribution was used in the probability bounds analysis for this the BW of small-footed myotis.

Food Intake Rate (FIR)

The food intake rate of small-footed myotis has not been measured. Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic rate of free-living mammals using the following general equation:

$$FMR\ (kJ/d) = a \cdot BW(g)^b$$

For both the Monte Carlo and the probability bounds analyses, the FMR for small-footed myotis was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (BW, *a*, *b*) and combined according to the above equation. The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy et al. (1999), assuming an underlying normal and lognormal distribution, respectively. A lognormal distribution was used for the power term (*b*) because the standard error was large and would have generated negative FMR values had a normal distribution been assumed. For non-herbivores, log *a* had a reported mean of 0.812 and a standard error of 0.196, and *b* had a reported mean of 0.681 and a standard error of 0.162 (Nagy et al. 1999). The BW distribution was described above.

FIR is derived from FMR using the following equation:

$$FIR = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$

where:

FMR	=	normalized free metabolic rate (kcal/kg bw/d)
AE_i	=	assimilation efficiency of *i*th food item (unitless)
GE_i	=	gross energy of *i*th food item (kcal/kg).

The gross energies of various wildlife food sources are summarized in the *Wildlife Exposure Factors Handbook* (EPA 1993). The mean gross energy of invertebrates is 1.6 kcal/g. The assimilation efficiency for invertebrates consumed by mammals is 72%. Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation.

Proportion of Dietary Items (P_i)

Along the Housatonic River, the small-footed myotis likely forages on small emergent aquatic insects, as does the little brown bat. Adult little brown bats in New York were found to consume Chironomidae (76.4% of food volume), Trichoptera (18.2%), Lepidoptera (4.2%), and Coleoptera (1.2%) (Belwood and Fenton 1976). Other studies conducted in the Northeast found these to be commonly consumed species along with other Diptera, such as Tipulidae, Culicidae, Homoptera, Hymenoptera, Neuroptera, Psocoptera, and Ephemeroptera (Griffith and Gates 1985; Anthony and Kunz 1977).

For this exposure assessment, the proportion of invertebrates in the diet was assumed to be a point estimate, with invertebrates accounting for 100% of the small-footed myotis diet.

Foraging Time (FT)

Little is known about home range size of the small-footed myotis. The Indiana bat (*Myotis sodalis*), a similar *Myotis* species, has a range averaging 128 acres (52 ha), but this range may be as large as 232 acres (94 ha) for lactating female bats (Kurta 1995; DeGraaf and Yamasaki 2001). Small-footed myotis feed predominantly over water on emergent insects; therefore, it was assumed that small-footed myotis would forage 100% of the time in the PSA.

K.2.1.7.2 Exposure Point Concentrations

Concentration of tPCBs in Prey Items (C_i)

Small-footed myotis prey items were not directly sampled in the PSA. Concentrations of tPCBs in invertebrates were obtained from samples of tree swallow (*Tachycineta bicolor*) gut contents (Custer 2002). Tree swallow gut content samples were used for small-footed myotis prey because these samples contain prey species that are more representative of actual myotis prey items than are the benthic invertebrate samples (Table K.2-17). Small-footed myotis and tree swallows are both aerial insectivores that forage primarily over open water and consume similar types of invertebrates; therefore, gut contents of tree swallows are likely to be similar to that of the small-footed myotis. Total PCB and TEQ exposure estimates were determined for reaches from which tissue concentrations for prey species were available (Reaches 5A, 5B, and 5C) and

combined for this analysis, because the small sample sizes did not allow for statistically robust exposure estimates for each reach.

Concentrations of tPCBs were measured in 12 tree swallow gut samples collected from the PSA. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean. The lower of the maximum measured concentration and the 95% UCL was used as a point estimate in Monte Carlo analysis. For this analysis, the upper 95% confidence was the lower of the two, with a value of 22.0 mg/kg tPCBs. For the probability bounds analyses, the 95% LCL and 95% UCL, 6.40 and 22.0 mg/kg bw/d, were used to parameterize a distribution-free statement in RiskCalc (Table K.2-20).

Concentration of 2,3,7,8-TCDD Toxic Equivalence (TEQ) in Prey Items (Ci)

TEQ concentrations were measured in six tree swallow gut samples collected in the PSA. Using the decision criteria outlined in Section 6.6, the assumption of how to treat co-eluted congeners in the TEQ calculation did not have an important influence on the estimated TEQ concentration. Therefore, it was assumed that congeners used in the TEQ calculation (i.e., PCB-123 and PCB-157) and which co-eluted with others (i.e., PCB-149/PCB-123; PCB-201/PCB-157/PCB-173) comprised 100% of the doublet (PCB-149/PCB-123) and triplet (PCB-201/PCB-157/PCB-173) concentrations. This data set was used to calculate the distribution parameters for the Monte Carlo and probability bounds analyses. The Land H-statistic was used to determine the 95% LCL and 95% UCL on the mean of ND=0, ND=1/2DL, and ND=DL, creating a triangular distribution. However, in all cases the maximum was <UCL, so distributions were estimated using maximum values. In this analysis, the value of 1,500 ng/kg was used (Table K.2-21).

In the probability bounds analysis, the uncertainty regarding the TEQ concentration was addressed by specifying the range of possible values in a distribution-free statement in RiskCalc. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the minimum from ND=0 and maximum concentrations from ND=DL were used in the probability bounds analysis. In this data set, the minimum and maximum values were used, and the resulting values ranged from 59.3 to 2,820 ng/kg (Table K.2-21).

K.2.1.7.3 Exposure Model Results

Exposure to tPCBs in the PSA

The Monte Carlo analysis indicated that exposure of small-footed myotis to tPCBs could range from a minimum of 2.05 to a maximum of 96.0 mg/kg bw/d. The mean exposure was 16.7 mg/kg bw/d, and the median exposure was 14.5 mg/kg bw/d (Table K.2-22). Eighty percent of the exposure estimates were between 7.17 and 28.8 mg/kg bw/d. Figure K.2-16 depicts the cumulative distribution for small-footed myotis in the PSA.

Sensitivity analysis (Table K.2-23) revealed that the FMR slope term used in the FMR equation was the most important variable (Pearson correlation coefficient (r_p) = 0.83) followed by the FMR power term (r_p = 0.53) and the body weight (r_p = -0.06). Discussion of the significance of the FMR slope term and FMR power term is provided in Section 6.

The probability bounds estimated for small-footed myotis foraging in the PSA are depicted in Figure K.2-16. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 1.96 and 7.67 mg/kg bw/d. The 50th percentile ranged between 4.05 and 15.0 mg/kg bw/d, and the 90th percentile ranged between 8.09 and 32.3 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 7.17, the 50th percentile was 14.5, and the 90th percentile was 28.8 mg/kg bw/d (Table K.2-22).

Exposure to 2,3,7,8-TCDD Toxic Equivalence (TEQ) in the PSA

The Monte Carlo analysis indicated that exposure of small-footed myotis to tPCBs could range from a minimum of 61.4 to a maximum of 7,020 ng/kg bw/d. The mean exposure was 1,130 mg/kg bw/d, and the median exposure was 936 ng/kg bw/d. Eighty percent of the exposure estimates were between 381 and 2,120 ng/kg bw/d. Figure K.2-17 depicts the cumulative distribution for small-footed myotis in the PSA (Table K.2-24).

Sensitivity analysis (Table K.2-23) revealed that the FMR slope term used in the FMR equation was the most important variable (Pearson correlation coefficient (r_p) = 0.82) followed by the FMR power term (r_p = 0.52), body weight (r_p = -0.04) and dietary concentration (r_p = -0.01). Clarification of the significance of the FMR slope term and FMR power term is found in Section 6.

The probability bounds estimated for small-footed myotis foraging in the PSA are depicted in Figure K.2-17. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 18.8 and 985 ng/kg bw/d. The 50th percentile ranged between 38.1 and 1,910 ng/kg bw/d, and the 90th percentile ranged between 74.8 and 3,900 ng/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 381, the 50th percentile was 936, and the 90th percentile was 2,120 ng/kg bw/d (Table K.2-24).

K.3 EFFECTS ASSESSMENT

This section examines the literature with respect to dietary and in ovo exposures of PCBs and TEQ to representative T&E species or their surrogates. The objective of this review was to identify the relevant studies to be used to characterize effects to T&E species (i.e., bald eagle, American bittern, and small-footed myotis). The studies were selected based on their overall quality and their relevance to the selected assessment endpoint. A summary of these toxicity studies is presented in Table K.3-1.

Studies were considered in the development of the effects metrics if sufficient information was provided to permit evaluation of study design, study execution, chemical analysis methods, statistical analyses, and other key aspects of the study. Those studies with major issues (e.g., incomplete explanation of statistical analyses) were included in the review of the effects literature that follows, but were not considered in the derivation of the effects metrics used in the assessment.

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to concentration- or dose-response curves for the functional group of interest (e.g., benthic invertebrates, mammalian piscivores). In this ERA, the effects characterization preferentially relies on concentration- or dose-response curves, but may default to benchmarks or other estimates of effect (e.g., no observed adverse effect level [NOAEL], lowest observed adverse effect level [LOAEL]) when insufficient data are available to derive dose-response curves. Effects associated with growth, survival, and reproduction are generally the preferred measures of effect. The details of the decision criteria used are provided in Section 6 of the ERA.

The decision criteria were applied to each COC-receptor combination. For PCBs and dioxins/furans, two measures of concentration were considered: tPCBs and TEQ. For tPCBs, the focus was on studies using mixtures with a higher degree of chlorination (i.e., 54 to 60%) because these mixtures most closely resemble the mixtures occurring in the PSA. For TEQ, the toxicity caused by coplanar PCBs, dioxins, and furans was considered together using the approach described by Van den Berg et al. (1998).

For some COC-receptor combinations, several effects metrics were developed to account for different routes of exposure. In all cases, the effects metrics were consistent with the metrics used in the exposure analysis.

Dose-response relationships are combined with the corresponding exposure distribution in risk characterization to derive risk curves that characterize the relationship between probability and magnitude of effect.

K.3.1 Bald Eagle

K.3.1.1 PCB Mixtures

Laboratory studies on the toxicity of PCBs to bald eagles have not been conducted. However, studies using other avian species were available. Appendix H, Piscivorous Birds, provides detailed descriptions of dietary and in ovo exposures of PCBs and TEQ to surrogate bird species.

Laboratory studies on raptor species demonstrated that PCBs cause adverse effects. American kestrels (*Falco sparverius*) dosed in ovo to produce a mean PCB tissue concentration of 34.1 mg/kg on a whole egg wet weight (ww) basis (PCBs were a 1:1:1 mixture of Aroclors 1248:1254:1260) had decreased reproductive success, including suppression of egg laying, delays in clutch initiation, smaller clutch sizes, and reduced fledgling survival (Fernie et al. 2001a). Twenty-five percent of exposed females failed to lay any eggs compared to 9% of the control females. PCB-exposed females also had lower fledgling success, 55% compared to 93.3% in the control group. Males exposed to PCBs in ovo also showed reduced reproductive success with 63.5% of their broods experiencing complete mortality compared to 0% complete mortality in the control group (Fernie et al. 2001a).

Numerous field studies have found that organochlorine compounds negatively impact the reproductive success of raptors and piscivorous birds (see overview in Donaldson et al. 1999). Toxicological effects include reduced hatching success, malformation, edema, and reduced organ and body weight (Elliott et al. 1996). Concentrations of PCBs accumulated by bald eagles in the wild and the toxicological effects of PCBs to bald eagles have been documented. Wiemeyer et al. (1984) found that bald eagle reproductive success was inversely correlated with PCB

concentrations in eggs. From 1969 to 1979, bald eagle eggs were collected from breeding areas in 14 states across the United States and tested for organochlorine pesticides, PCBs, and mercury. PCBs, DDE, and other organochlorine pesticides were found to be highly intercorrelated and, therefore, a principal component analysis was used to obtain independent regressor variables for each analysis. Populations with mean PCB residue in eggs of less than 4.5 mg/kg (ww) had a mean 5-year reproductive rate (successfully fledged) of 0.834 young/nest. Breeding areas with 4.5 to 10 mg/kg, 10 to 19 mg/kg, 19 to 33 mg/kg, and greater than 33 mg/kg PCBs (ww in eggs) had reproductive rates of 0.601, 0.538, 0.275, and 0.20 young/nest, respectively. A reproductive rate of 0.70 young/nest or greater is needed to maintain a stable population (Sprunt et al. 1973).

The Wiemeyer study also found that mean PCB residues in eggs from successful nests (one or more young fledged) were lower (7.2 mg/kg wet weight) than in eggs from unsuccessful nests (13.0 mg/kg). These populations also contained DDE. DDE was found to have a greater effect on the mean 5-year reproductive rate than PCB residues. DDE and the mean 5-year reproductive rate had a correlation coefficient of –0.565 ($p \leqq 0.001$), whereas PCBs had a correlation coefficient of -0.436 ($p \leqq 0.01$). Sampling of bald eagle eggs continued from 1980 to 1984, and further analyses were conducted during this time on the relative effects of PCBs and DDEs (Wiemeyer et al. 1993).

Most populations of bald eagles that have been sampled have bioaccumulated PCBs and DDE (Sprunt et al. 1973; Frenzel and Anthony 1989; Donaldson et al. 1999); therefore, it is difficult to determine whether PCBs, DDE, or their combined toxicity is responsible for the reported effects. PCB concentrations were reported to account for 16% in the variation of young production compared with 30% for DDE. No evidence was found that PCBs magnify or added to the impact of DDE on eggshell thinning or young production (Wiemeyer et al. 1993). However, studies on piscivorous birds in Europe, where much lower DDE to PCB ratios are found, also show adverse reproductive effects not easily explained by the concentrations of DDE alone (Voogt et al. 2001).

Three bald eagle nests from the Delaware River in New Jersey with a mean PCB concentration in eggs of 40 mg/kg failed to produce young (Clark et al. 1998). PCB concentrations reported for prey fish species were 0.62, 0.85, and 1.73 mg/kg in brown bullhead, white perch (*Morone*

americana), and common carp, respectively. As with other studies, high concentrations of DDE (ranging from 6.3 to 18 mg/kg) also occurred in this population. Two of the breeding pairs' failure to breed successfully occurred after several years of prior successful nesting at this location, suggesting that reproductive failure may occur after contaminants bioaccumulate and exceed an effect threshold (Clark et al. 1998).

Wiemeyer et al. (1984) reported mean 5-year reproduction to be normal in areas where eggs contained <3.0 mg/kg DDE. Reproduction decreased significantly at concentrations exceeding 5.1 mg/kg DDE, and concentrations of >15.0 mg/kg DDE caused almost complete reproductive failure. DDE was also inversely correlated with eggshell thickness (Wiemeyer et al. 1984).

Bald eagle eggs collected from sites in Green Bay, Wisconsin, had PCB concentrations ranging from 13 to 120 mg/kg (ww), with an average concentration of 46.1 mg/kg. Eggs collected from uncontaminated inland sites in Michigan and Wisconsin had average PCB concentrations of less than 5 mg/kg (Stratus 1999). PCB concentrations in the blood plasma of nestling bald eagles from Green Bay and the lower Fox River, Wisconsin, averaged 0.285 and 0.3 mg/kg, respectively, while concentrations from inland nestlings averaged 0.024 mg/kg. PCB concentration (mg/kg ww) in whole body tissues of bald eagle prey items from Green Bay averaged 2.6 in suckers, 2.1 in bullheads, 10.5 in pike, and 4.0 in carp. Productivity from nests on Green Bay was reduced compared to inland nests. Fifty-three percent of Green Bay nests produced no fledglings, whereas only 37% of both inland Michigan and Wisconsin nests produced no young. Twenty percent of Green Bay nests produced one chick, and 27% produced two or three chicks. Twenty-five and 31% of inland nests produced one chick, and 41% and 34% of inland nests produced two or more chicks (Stratus 1999). PCB concentrations >3 mg/kg in eggs may have had adverse effects on fledging success, and major impacts (reductions of 50% or greater) can occur at egg concentrations as low as 13 mg/kg (Stratus 1999).

Malformations, primarily bill deformities, in fish-eating birds have been linked to non-orthosubstituted (planar) PCB congeners (Hoffman et al. 1987; Gilbertson et al. 1991; Bowerman et al. 1994). Such defects have been recorded in several Great Lakes bald eagle nestlings with a mean PCB concentration in the blood plasma of 0.183 mg/kg (Bowerman et al. 1994). Bill deformities have also been recorded in white-tailed eagles (*Haliaeetus albicilla*)

from the Baltic exposed to high concentrations of PCBs and to domestic chicken embryos in PCB-contaminated eggs (Bowerman et al. 1994). A tPCB egg concentration of 50 mg/kg has been suggested as a critical level, above which embryotoxic effects may occur in raptors (Nobel et al. 1993).

Wiemeyer et al. (1993) reported a significant reproductive decline in bald eagles with egg concentrations greater than 13 mg/kg. However, PCB concentrations were highly correlated with DDE concentrations, thus this threshold must be considered with caution. A threshold value of 20 mg/kg in bald eagle eggs was suggested in the recent assessment of the Fox River/Green Bay system (Stratus 1999). That value is consistent with other raptor studies that suggest tPCBs have higher egg thresholds for reproductive effects than does DDE (Helander et al. 1982; Peakall et al. 1990; Nobel and Elliott 1990). Therefore, the field-based threshold selected for tPCB in bald eagle eggs was 20 mg/kg.

No suitable field data were available to develop toxicity thresholds for tPCBs exposure to bald eagles. American kestrels were considered a surrogate species for bald eagles when evaluating toxicity studies. A daily intake rate of 7 mg/kg bw/d was shown to cause an increase in laying lag, and a decrease in the number of fledglings per breeding pair (Fernie et al. 2001a and 2001b). These birds had a long exposure period (100 days) and the study covered a sensitive life stage. A chronic NOAEL was estimated by applying a factor of 10 to the LOAEL, resulting in a NOAEL of 0.7 mg/kg bw/d. Therefore, this dose was considered to be the toxicity threshold for tPCBs.

K.3.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

Several researchers estimated NOAEL and LOAEL values for TEQ for bald eagles (Giesy et al. 1995; Bowerman et al. 1995; Elliott et al. 1996). Giesy et al. (1995) and Bowerman et al. (1995) sampled the impact of contaminated prey fish on bald eagles in the Great Lakes region and derived a NOAEL for bald eagle eggs of 7 ng/kg TEQ. This value is based on toxicity studies conducted using other avian species, including the chicken, wood duck, and American kestrel. The bald eagle is less sensitive to TEQ compared to chickens, ducks, and other gallinaceous species; therefore, this value may be low (Elliott et al. 1996; Elliott and Harris in press). Elliott et al. (1996) reported a NOAEL of 135 ng TEQ/kg egg and a LOAEL of 400 ng TEQ/kg egg

based on studies of incubated bald eagle eggs taken from nests in British Columbia. Studies conducted on ospreys (*Pandion haliaetus*) found a similar NOAEL on hatching success of 136 ng/kg (Elliott et al. 2001) and Woodford et al. (1998) found 162 ng/kg had no effect on productivity, but may have been influencing growth of young. Using the Elliott et al. (1996) study, the NOAEL selected was 135 ng/kg TEQ in eggs, and the corresponding LOAEL is 400 ng/kg TEQ in eggs.

No suitable field data were available to develop toxicity thresholds for TEQ exposure to bald eagles. A dose of 5,000 ng/kg bw/d TEQ did not produce any adverse effects on American kestrel chicks and, therefore, was selected as the toxicity threshold for exposure to TEQ by bald eagles.

K.3.2 American Bittern

K.3.2.1 PCB Mixtures

No studies have addressed the effects of PCBs on American bitterns. Studies have been conducted on similar heron species, such as the great blue heron and black-crowned night heron, which also inhabit shallow wetlands and feed on a variety of fish, amphibians, reptiles, crustaceans, and insects (see Appendix A).

Hoffman et al. (1986) found a negative correlation between embryonic weight and tPCB residues in eggs of black-crowned night herons nesting in the San Francisco Bay. Heron eggs had a mean PCB concentration of 4.1 mg/kg (ww). PCB-contaminated embryos, with the yolk sac removed, had a significantly lower (15%) weight than embryos from clean sites. Concentrations of other organochlorines were low (mean DDE concentration of 1.7 mg/kg). Other effects associated with PCB exposure in black-crowned night herons included reduced femur to body weight ratio, increased edema, and increased hepatic aryl hydrocarbon hydroxylase activity (Hoffman et al. 1993); some of these effects may have been related to the presence of other contaminants, although the authors state that concentrations of these other contaminants were not high enough to account for the observed effects.

Laporte (1982) reported that mean tPCB concentrations of 15 mg/kg in eggs negatively impacted great blue heron reproductive success in Quebec.

Eggshell thinning has been commonly reported in herons exposed to organochlorines. However, eggshell thickness is most closely correlated with DDE and is only loosely correlated with PCBs (Ohlendorf et al. 1979; Henny et al. 1984; Ohlendorf et al. 1988; Fitzner et al. 1988; Elliott et al. 1989). Fitzner et al. (1988) found that PCB concentrations as high as 13 mg/kg in eggs accounted for only 3% of the variability in eggshell thickness.

Great blue herons in Indiana showed no observable effects at 4.9 mg/kg tPCBs in their eggs (predominantly PCB congeners 118/106, 105, and 156) (Custer et al. 1998). Great blue herons in Texas had a mean of 6.2 mg/kg PCBs in their eggs and fledged 1.6 young per nest, a value within the range of stable populations (Mitchell et al. 1981). Total egg PCB concentrations of 1 mg/kg were found to have no effect on great blue heron productivity (Elliott et al. 1989).

This review indicates that the threshold for toxic effects to herons is in the range of 4 to >6 mg/kg PCBs in eggs. American bitterns can be reasonably represented by black-crowned night and great blue herons. For this assessment, a threshold of 4.9 mg/kg PCBs in eggs was selected for American bitterns.

There were insufficient data available to develop a field-based threshold for American bitterns exposed to tPCBs. In the absence of such data, a range of toxic effects was estimated to span the range for sensitive to tolerant avian species, as was done for piscivorous birds (Appendix H). The most sensitive species was the white leghorn chicken (*Gallus domesticus*), which had a NOAEL of 0.12 mg/kg bw/d. The tolerant species was the American kestrel, which had a NOAEL of 0.7 mg/kg bw/d.

K.3.2.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

Black-crowned night heron pipping embryos had TEQ concentrations of 30, 622, and 272 ng/kg (Rattner et al. 2000). BROD and EROD activity were elevated at the 272 and 622 ng/kg TEQ concentration. Elliott et al. (1989) found a TEQ of 230 ng/kg in the eggs of great blue herons to cause reduced reproductive success. The same study found TEQ concentrations of 11, 14, 34,

64, and 79 ng/kg in eggs to have no effect on hatching success. The NOAEL selected for TEQ in eggs was 79 ng/kg, and the LOAEL was 230 ng/kg TEQ in eggs.

Toxicological thresholds for effects of TEQ on American bitterns were not available. However, studies conducted using other species can be extrapolated to American bitterns. Therefore, the NOAEL of 79 ng/kg for black-crowned night herons was used as the threshold for TEQ exposure to American bittern eggs.

There were insufficient data to develop a field-based threshold for American bitterns exposed to TEQ. In the absence of such data, a range of toxic effects was estimated to span the range for sensitive to tolerant avian species, as was done for piscivorous birds (Appendix H). The sensitive species was the ring-necked pheasant (*Phasianus colchicus*), which had a NOAEL of 14 ng/kg bw/d. The tolerant species was the American kestrel, which had a NOAEL of 5,000 ng/kg bw/d.

K.3.3 Small-Footed Myotis

K.3.3.1 PCB Mixtures

No PCB toxicology studies have been conducted on small-footed myotis; however, studies have been conducted for little brown bats and big brown bats. The little brown bat is a closely related species that has similar habitat, behavior, diet, and size. The big brown bat also shares many traits with the small-footed myotis (see Appendix A).

Studies have shown that bats accumulate PCBs from their diet (Clark and Lamont 1976a; Clark and Lamont 1976b; Clark and Prouty 1976; Clark 1978; Clark and Stafford 1981). Clark and Prouty (1976) found that little brown bats accumulated higher concentrations of organochlorine compounds (including PCBs, DDT, and DDE) than did other bat species at the same location. Little brown bats also accumulated brain PCB concentrations that were higher than brain concentrations of other bat species, which may make them more susceptible to PCB poisoning (Clark and Prouty 1976). Like the little brown bat, small-footed myotis may also be more susceptible to PCBs because they have similar physiology, diet, and habitat.

Adult little brown bats fed mealworms with 15 mg/kg PCBs (Aroclor 1260) for 40 days accumulated a mean PCB concentration of 92 mg/kg (carcass ww) (Clark and Stafford 1981). In the same study, five little brown bats were fed on a diet of mealworms containing 1,000 mg/kg PCBs; four of the bats died before 40 days. These four bats had a mean PCB concentration of 3,300 mg/kg (ww). The one surviving bat had a PCB concentration of 940 mg/kg (Clark and Stafford 1981).

Residues of PCBs in bat brains are a linear function of the amount of fat and residues in the carcass (Clark and Prouty 1977; Clarke et al. 1978; Clark and Stafford 1981). During hibernation, the percent of lipids in the body decreases, but the lipid percentage in the brain does not change, which results in elevated concentrations of PCBs in the brain (Clark and Prouty 1977; Clark and Stafford 1981). Elevated concentrations of PCBs in the brain may lead to tremoring, a common symptom of organochlorine poisoning (Clark and Stafford 1981). Bats in hibernation have energy stores that are closely balanced against needs, and any disturbance, such as tremoring, that increases metabolic rates can cause mortality through starvation (Clark and Stafford 1981).

PCBs are also known to have adverse reproductive effects on bats (Clark and Lamont 1976a; Clark and Lamont 1976b; Clark 1978). Female bats transfer PCBs to their young through the placenta (Clark et al. 1975; Clark 1978). Clark and Lamont (1976a) found that neonates contain 16.8% to 31.8% as much PCBs as their parents. Organochlorine contaminants are also passed to the young through the mother's milk (Clark et al. 1975; Clark and Lamont 1976a). Milk collected from the stomachs of young big brown bats contained a PCB (Aroclor 1260) concentration of 13 mg/kg. The young had a mean PCB concentration of 0.7 mg/kg (Clark and Lamont 1976a). Wild captured female big brown bats that produced dead young contained significantly higher concentrations of PCBs (1.99 mg/kg ww) than those that produced live young (0.56 mg/kg ww) (Clark and Lamont 1976b).

There were insufficient data to develop a field-based threshold for small-footed myotis exposed to tPCBs. In the absence of such data, the dose-response curve for rats used in the estimate for omnivorous and carnivorous mammals (see Appendix J) was adopted. Rats are omnivorous animals for which toxicological dosing and effects data were available. Although rats are

dissimilar to small-footed myotis in several ways, they were considered the best available surrogate.

K.3.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

No studies evaluating TEQ for small-footed myotis have been conducted. In the absence of such data, the dose-response curve for rats used in the estimate for omnivorous and carnivorous mammals (see Appendix J) was adopted. Rats are omnivorous animals for which toxicological dosing and effects data were available. Although rats are dissimilar to small-footed myotis in several ways, they were considered the best available surrogate.

K.3.3.3 Effects Metrics for Characterizing Risk

The results of controlled investigations that treated representative species with at least five dose levels and measured for effects on growth, survival, or reproduction are preferred for assessing the effects of tPCBs and TEQ to species in the Housatonic River. Controlled toxicity studies on these endpoints for small-footed myotis were not available for PCBs or TEQ. As a result, surrogate mammals for these representative species were used to assess toxicity. For the small-footed myotis, the rat was used as a surrogate mammal. Rats are small omnivorous animals for which toxicological dosing and effects data were available. Although rats are dissimilar to bats in several ways, they were the best surrogates available.

K.3.3.3.1 tPCBs

The dose-response curve for tPCBs (Figure K.3-1) was generated using the Spencer (1982) study. This study investigated effects on reproduction of rats by administering TCDD on gestation days 6 through 15. The study investigated effects on mortality at birth using eight treatment levels. The curve fitting of dose-response data to a GLiM was performed using SAS® (SAS Institute, Cary, NC). Mortality data required a logit link function and a binomial error distribution was assumed (Bailer and Oris 1997). Figure K.3-1 presents the dose-response curve for mortality at birth of rats. In this analysis, Abbott's formula was used to correct for control mortality (Newman 1995). Because mean responses were used in the analysis (the raw data were not available), fiducial limits and goodness of fit were not estimated. The model parameters

were β_0 = -15.4, β_1 = 3.71, seβ_0 = 1.19, seβ_1 = 0.29, and corr$\beta_0\beta_1$ = -0.997. The dose-response model was significant at $p<0.0007$ (F value = 30.8, 6 degrees of freedom).

K.3.3.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

The dose-response curve (Figure K.3-2) for TEQ was derived using the results of Khera and Ruddick (1973) and Sparschu et al. (1971). Khera and Ruddick (1973) treated pregnant Wistar rats with several doses of TEQ on gestation days 6 to 15. Animals were sacrificed on day 22 of gestation. A dose-related decrease in live fetuses was observed; 100% embryonic lethality was reported when animals were exposed to a dose of 4,000 ng TEQ/kg bw/d. Sparschu et al. (1971) made similar observations in Sprague Dawley rats fed several doses of TCDD on days 6 to 15 of gestation. The number of viable fetuses decreased and the total number of resorptions increased in a dose-dependent manner, starting at 125 ng TEQ/kg bw/d. These two studies were combined to generate an effects curve used to characterize risk. The dose-response curve developed indicated that 10% and 20% declines in reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d TEQ, respectively.

K.4 RISK CHARACTERIZATION

This section characterizes risk to T&E species exposed to PCBs and 2,3,7,8-TCDD TEQ in the PSA of the Housatonic River. The risk characterization includes the application of the weight-of-evidence approach developed by MDEP (Menzie et al. 1996), which is described in detail in Section 2.9 of the ERA.

The risk characterization for T&E species in the PSA considers two potential lines of evidence: (1) field surveys, and (2) probabilistic exposure and effects modeling. However, the population and community-level surveys were not performed using a quantitative study design; therefore, they are not included in the weight-of-evidence assessment.

Section K.4.1 provides a brief overview of the methodology, results, and interpretation of the field surveys pertaining to T&E species. A more detailed presentation of this information can be found in Appendix A. Section K.4.2 compares the quantitative probabilistic exposure estimates calculated for T&E species (Section K.2) with the relevant ecological effect doses for tPCBs and TEQ presented in Section K.3. Section K.4.3, Weight-of-Evidence Analysis, summarizes the findings, provides an analysis of the relative significance of the evaluation, and discusses the overall findings of the risk assessment.

The purpose of this risk characterization is to address the following risk questions:

- Are the concentrations of tPCBs and TEQ in the prey of T&E species sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?
- If so, how severe are the risks and what are their potential consequences?

K.4.1 Field Surveys

K.4.1.1 Introduction

Potential T&E species were characterized by identifying all species that could occur in the PSA, their preferred habitats, and when they would use these habitats. This work included a review of relevant literature on wildlife populations in western Massachusetts and Connecticut. Local and regional references on T&E species in the study area were used to identify the species whose

range encompassed the study area. General and technical references on habitat requirements and use, seasonality of occurrence, and relative abundance in the region were then used to refine the list and build a matrix that included those species whose preferred habitats are within the PSA (Appendix A). As part of this effort, local and regional experts were consulted to obtain unpublished records regarding the historical occurrence of species in the area. For example, the MNHESP, the Massachusetts Division of Fisheries and Wildlife (MDFW), and the U.S. Fish and Wildlife Service were contacted to identify historical occurrences and to review recent records from the area. Information received from these agencies, organizations, and individuals was then incorporated into the species matrix (see Attachment C in Appendix A).

Based on a review of habitat in the PSA and the distribution and home range information, 18 T&E animal species could potentially occur in the PSA. Of these, 14 were recorded during field surveys (Appendix A).

K.4.1.2 Methods

K.4.1.2.1 Field Surveys for T&E Species

T&E species in the Housatonic River study area were surveyed from 1998 to 2001. Field data were collected using methods targeted at specific species or family groups, as well as more general, reconnaissance-level investigations of species' presence, relative abundance, and habitat use. Surveys for T&E species were conducted as part of broader survey efforts. Throughout this period, any observations were recorded along with notes on habitat use, breeding signs, and behavior.

K.4.1.2.2 Bird Community Surveys

The avian community in the PSA was studied over a 4-year period, from 1998 to 2001. Surveys were conducted to record presence, abundance, and habitat usage for each major group of birds. These surveys included wading and marsh bird surveys, hawk and owl surveys, and forest bird surveys. Additional studies were conducted to sample animal tissues (i.e., waterfowl sampling, tree swallow study). Observations recorded in the field were used to refine the matrix to depict

habitat use and seasonality of occurrence for bird species expected to occur in the primary and reference study areas.

Marsh and wading bird surveys were conducted in 1998 using playback point counts to identify species using the PSA wetlands and reference locations (Appendix A). Playbacks were broadcast at each station for Virginia rail (*Rallus limicola*), sora (*Porzana carolina*), green heron (*Butorides virescens*), American bittern, least bittern (*Ixobrychus exilis*), American coot (*Fulica americana*), common moorhen, pied-billed grebe (*Podilymbus podiceps*), and king rail (*Rallus elegans*). All marsh and wading birds seen or heard during the surveys were recorded, along with information on location, habitat use, and weather conditions. Additional observation data for T&E wading and marsh birds were recorded from 1998 to 2001 during other surveys. Each time a T&E species was observed, the location, habitat, behavior, and date were recorded.

Playback point counts were also used in 1999 to survey hawks and owls (raptors) in the PSA and in three reference locations. In the PSA, raptor transects were positioned along the Housatonic River from the confluence of the East and West Branches to Woods Pond (Appendix A). Calls were broadcast for eight species of raptors expected to occur in the study area. Approximately 10 minutes were spent at each point, during which time hawks and owls observed or heard were identified and recorded. Additional data recorded included type of observation (e.g., call, visualization) and behavior.

In addition, data from the North American Breeding Bird Survey (BBS) were used to determine the occurrence of species in the vicinity of the study area. The BBS is a large-scale avian survey program initiated in 1966 and coordinated by the U.S. Geological Survey. The BBS is a roadside survey program, with more than 4,100 24.5-mile-long survey routes that are surveyed annually. Three survey routes occur in Berkshire County: in Pittsfield, Cheshire, and Sheffield.

K.4.1.2.3 Mammal Community Surveys

The mammalian community in the PSA was studied from 1998 to 2001. Field data included methods targeted at specific species, as well as more general, reconnaissance-level investigations of species presence, relative abundance, and habitat use.

Bat surveys were conducted to determine their presence in the PSA by recording their echolocation calls. Three transects were established along the river in the northern, central, and southern sections of the PSA (Appendix A). There is a large amount of overlap between the call characteristics of the little brown bat, small-footed myotis, and Indiana bat, which makes it difficult to distinguish between these *Myotis* species using echolocation. When recording the results, these three species were all labeled as *Myotis* sp. The majority of these calls were likely little brown bat; however, a small number of the calls had parameters that suggested small-footed myotis rather than little brown bats or Indiana bats. Small-footed myotis cannot be confirmed without having animals in hand for visual identification.

K.4.1.3 Results

K.4.1.3.1 Bald Eagle

Bald eagles were observed in the PSA and reference locations eight times during the course of field investigations, although some of those observations may have been of the same individuals. Most observations occurred near Woods Pond and the adjacent backwaters, and many of the eagles observed were apparently hunting over these shallow water habitats. The observations occurred primarily in the spring and fall, suggesting that they were migrating individuals. No evidence of permanent residence or breeding activity was observed between 1998 and 2001. Bald eagles are closely associated with aquatic habitats, usually nesting in large trees along shorelines and feeding on fish. The study area provides suitable nesting and foraging habitat for bald eagles. In the mid 1990s, a pair of bald eagles constructed a nest at Woods Pond (T. Gulo, MDFW, personal communication 2001). The nest was reportedly destroyed during an April snowstorm and the pair did not attempt to re-nest.

The bald eagle historically nested throughout the United States and Canada in deciduous and coniferous forests, especially along bodies of water. Eagle populations experienced significant declines, due to eggshell thinning from DDT poisoning, habitat loss, and shooting, to the point that it was formerly listed as Endangered nationwide. Bald eagle populations are currently recovering because of habitat protection and other recovery efforts such as hacking. Hacking at artificial nest platforms on Quabbin Reservoir, for example, led to the first successful

Massachusetts breeding record in more than 80 years, when two pairs produced a total of three young (Veit and Petersen 1993). Since this success in 1989, numbers of breeding pairs of bald eagles have been increasing in the state. In 2000, nine pairs of bald eagles were known to have nested in Massachusetts. These nests were located along the Connecticut River and Quabbin Reservoir. Since 1966, observers on the three BBS routes in Berkshire County have documented bald eagles only once (Sauer et al. 2000). Bald eagles are also known to nest in Connecticut at the Barkhamsted Reservoir and on the upper Connecticut River. In 2001, a bald eagle pair nested along the Housatonic River below Interstate 84 and raised one chick. The pair returned in 2002 and displayed breeding activity (i.e., adding sticks to previous year's nest); however, no nesting was observed (J. Bictoria, CTDEP, personal communication 2002).

K.4.1.3.2 American Bittern

Eight American bittern sightings were documented from 1998 to 2001 in the PSA, and two in the October Mountain State Forest reference location in 2000. Observations during these years ranged from May to September. The majority of these observations occurred in August and September, and these American bitterns were likely migrating individuals. Several observations, however, did occur during the breeding season, and one individual was heard calling, indicating intent to breed in the area. These observations were incidental observations, occurring while researchers were on-site for other surveys. No bitterns responded to marsh bird playback calls in 1998. Bitterns were usually observed in wet meadow, shallow emergent marsh, and deep emergent marsh habitats. The majority of the sightings occurred in the southern half of the PSA, although sightings also occurred in the extensive shallow emergent marsh near the confluence of the East and West Branches and in the Canoe Meadows Wildlife Sanctuary.

American bitterns breed in eastern North America, from Newfoundland to North Carolina, west to central Oklahoma and Manitoba, and winter in coastal marshes from Massachusetts to the Gulf Coast (Gibbs et al. 1992). In Massachusetts, breeding populations of American bitterns have been declining since the 1960s. From 1976 to 1980, the Massachusetts Breeding Bird Atlas project confirmed only 17 breeding pairs in the state. Breeding occurs primarily in northeastern Essex County and along the Sudbury River, with scattered localities in Plymouth and Berkshire

Counties (Veit and Petersen 1993). The decline in breeding populations of bitterns is due in a large part to the disappearance of extensive cattail marshes, their primary breeding habitat.

K.4.1.3.3 Small-Footed Myotis

Suitable summer habitat for small-footed myotis is present in and adjacent to the study area, and it is likely that the species occurs there. The small-footed myotis has been recorded in western Massachusetts and has been documented twice since 1978 in Hampden County, Massachusetts (MNHESP 1984; Godin 1977), making their presence in the study area possible. It is believed that this species was recorded during bat surveys; however, as previously mentioned, limitations of echolocation technology prevent this species from being definitively identified. Other studies conducted in the region have reported small-footed myotis observations (Zimmerman and Glanz 2000; Krusic et al. 1996).

The small-footed myotis ranges from Ontario and southern Quebec, down the Appalachian Mountains to northern Georgia, and west into Arkansas and Oklahoma. These bats usually occur in mountainous regions. Small-footed myotis use buildings, overhanging rocks, and caves as summer roost and maternity sites. Suitable summer habitat is present in and adjacent to the study area, and it is likely that the small-footed myotis occurs there. They hibernate from November to March in caves and mines usually in the foothills of mountains, up to 2,000 feet (615 m) in elevation, in coniferous woodlands (DeGraaf and Yamasaki 2001).

K.4.1.4 Conclusions

Bald eagles were not observed during raptor surveys in the PSA or any of the three reference locations. However, incidental bald eagle observations were made in the PSA (primarily in the vicinity of Woods Pond) and at the Threemile Pond reference location. American bitterns were not observed during marsh bird surveys in the PSA, and no marsh bird surveys were conducted outside of the PSA. Incidental observations of American bitterns occurred in the PSA and at Washington Mountain Lake, including breeding calls in the southern PSA. During timed bird surveys (i.e., playback surveys), the results in the PSA were the same for reference locations; no bald eagles or American bitterns were observed in either location. Small-footed myotis

observations in the PSA have not been confirmed, and no bat surveys were conducted in reference locations.

Any differences in population structure between the PSA and the reference locations cannot be evaluated due to the overall low population size of T&E species, the limited number of sightings, and the qualitative study design. The number of observations for these species by definition is expected to be low, and the lack of observations in one location does not necessarily reflect the suitability of the habitat or the absence of an individual.

K.4.2 Comparison of Estimated Exposures to Laboratory-Derived Effect Doses

For bald eagles, exposure was assessed in the southern portion (Reach 5C to Woods Pond) of the PSA. Exposure of American bitterns in the PSA was assessed in four areas: Reaches 5A, 5B, 5C, and 5D and 6 combined. Exposure of small-footed myotis was assessed for all of the PSA combined.

After a review of the potential exposure pathways and their relative contribution to overall exposure to PCBs and TEQ, it was determined that the ingestion of food items was the only pathway of concern for these species. A detailed presentation of the exposure analyses for representative T&E species is provided in Section K.2.

The effects characterization for T&E species provides a detailed overview of the current toxicological literature on the potential effects of PCBs and TEQ to bald eagles, American bitterns, small-footed myotis, and similar surrogate species. Because few studies in the literature directly quantified the effects of tPCBs and TEQ to bald eagles and no studies directly quantified the effects of tPCBs and TEQ to American bittern and small-footed myotis, surrogate species were used to characterize the effects for T&E species.

The following discussion presents an integration of the exposure distributions and effects thresholds developed for bald eagles and American bitterns. The toxicity thresholds for these species were based on the NOAELs. This results in a more stringent threshold for T&E species, which is appropriate because any effects to T&E species could potentially have a major impact on the already reduced population.

- For bald eagles, the categories of low, intermediate, and high risk were derived using the following criteria: If the probability of exceeding the toxicity threshold was less than 20%, the risk to T&E species was considered to be low.
- If the probability of exceeding the toxicity threshold was greater than 50%, the risk to T&E species was considered to be high.
- All other outcomes were considered to have intermediate risk.

For American bittern, the categories of low, intermediate, and high risk were derived using the following criteria:

- If the probability of exceeding the lower toxicity threshold was less than 20%, the risk to T&E species was considered to be low.
- If the probability of exceeding the upper toxicity threshold was greater than 20%, the risk to T&E species was considered to be high.
- All other outcomes were considered to have intermediate risk.

This exercise was done separately for the results of the Monte Carlo analysis, and the lower and upper bounds from the probability bounds analyses. The "risk category" refers to the level of risk based on the results of the Monte Carlo analysis. The "risk range" refers to the levels of risk based on the results of the probability bounds analyses.

For small-footed myotis integration of the exposure distribution and dose/response curve, the categories of low, intermediate, and high risk were derived using the following criteria:

- If the probability of 10% or greater effect was less than 20%, then the risk to small-footed myotis was considered to be low.
- If the probability of 20% or greater effect was greater than 50%, then the risk to small-footed myotis was considered to be high.
- Other outcomes were considered to have intermediate risk.

K.4.2.1 Bald Eagle Risk Characterization

K.4.2.1.1 tPCBs

The toxicity threshold for bald eagles is 0.7 mg/kg bw/d based on American kestrel dietary studies (Fernie et al. 2001a and 2001b), the best available information; however, eagles and

kestrels have different body weights and feeding guilds. Therefore, the use of the threshold derived from toxicity to kestrels has some uncertainty. The Monte Carlo predictions for TDIs of tPCBs by bald eagles in the PSA indicated that there was a 100% probability of exceeding the threshold (Figure K.4-1, Table K.4-1). The probability bounds analysis indicated that the probability of exceeding the toxicity threshold is 100%. Therefore, bald eagles feeding and reproducing in the Housatonic River PSA are at a high risk from exposure to tPCBs.

K.4.2.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

The TEQ threshold for the bald eagle is 5,000 ng/kg bw/d. The Monte Carlo predictions for TDIs of TEQ by bald eagles indicated that there was a 0% probability of exceeding the NOAEL and 0% probability of exceeding the threshold. (Table K.4-1, Figure K.4-2). The threshold selected for bald eagles is also the threshold for the most tolerant avian species studied, which may underestimate risk.

K.4.2.1.3 Egg tPCBs

The toxicity threshold for bald eagle eggs was 20 mg/kg. The best estimate indicates that bald eagle eggs will exceed the toxicity threshold after the female has spent 17 days foraging in the PSA. Egg concentrations using the lower bound would not have exceeded this threshold until after the female had been in the PSA for 27 days, while the upper bound shows eggs exceeding the threshold after 12 days (Figure K.4-3). While assuming bald eagles are likely to spend 30 days in the summer territory prior to breeding, they may spend up to 90 days in the summer territory prior to breeding (Veit and Petersen 1993; Buehler 2000). Therefore, it is likely that if bald eagles nested and laid eggs in the PSA, the eggs/young would be adversely affected by tPCBs.

K.4.2.1.4 Egg 2,3,7,8-TCDD Toxic Equivalence (TEQ)

Elliott et al. (1996) estimated the NOAEL for bald eagle eggs to be 135 ng/kg and the LOAEL to be 400 ng/kg. The toxicity threshold selected was the NOAEL. TEQ concentrations in eggs of bald eagles breeding in the PSA are estimated to exceed the toxicity threshold. A female bald eagle would need to forage for 5, 6, or 10 days (high, moderate, and low values respectively) in

the PSA prior to breeding for the eggs to exceed the toxicity threshold (Figure K.4-4). Therefore, it is likely that if bald eagles nested and laid eggs in the PSA, the eggs/young would be adversely affected by TEQ. The lack of concordance between the risk estimates using estimated egg concentrations and the TDI model suggest that bald eagles feeding and reproducing in the Housatonic River PSA are at an intermediate risk of toxicity from exposure to TEQ.

K.4.2.1.5 Risk Estimates Downstream of Woods Pond

Risks to T&E species due to contaminants in the river and associated floodplains below Woods Pond were also assessed. Total PCBs measured in sediment, soil, and fish tissue samples from the river segments above and below Woods Pond are presented in Appendix H. The PCB concentration data indicates that contamination levels in soil, sediment, and fish tissues decline substantially after Woods Pond Dam.

Risk for Bald Eagles Wintering Downstream of Woods Pond

The risk for bald eagles associated with exposure to tPCBs downstream of Woods Pond was assessed by comparing concentrations of tPCBs in prey fish in Reaches 7 to 16 to a maximum acceptable threshold concentration (MATC) developed specifically for bald eagles. The MATC of 30.41 mg/kg tPCBs in fish (whole body, wet weight) was developed as the concentration at which bald eagle TDI would exceed the toxicity threshold for eggs. The TDI was calculated assuming that eagles wintering downstream of Woods Pond would consume 83.4% fish and 16.1% waterfowl (Stalmaster and Plettner 1992). The waterfowl concentration was assumed to be zero, as waterfowl wintering on the Housatonic are likely to have migrated there from northern locations outside the study area. Fish tissue data were obtained from sampling and analysis conducted since 1998. The results of the analysis, which indicated that bald eagles would be at risk only in Reach 8 (Rising Pond), are presented in Figure K.4-5. This result is conservative in that it assumes bald eagles would consume fish only from Rising Pond, which is unlikely given that the pond is considerably smaller than the typical bald eagle foraging area.

Risk for Bald Eagles Breeding Downstream of Woods Pond

Figure K.4-5 presents an assessment of risk to bald eagles exposed to tPCBs downstream of Woods Pond. Bald eagles are known to breed downstream of Woods Pond. In particular, one bald eagle pair nested in Reach 15, just south of Interstate 84, in 2001. The pair raised one chick. In 2002, the pair returned to the nest and displayed some breeding activity but did not nest (J. Bictoria, CTDEP, personal communication 2002).

Risk from exposure to tPCBs was estimated for bald eagles nesting at this location. Bald eagles have a linear (riverine) foraging distance of 1.9 to 4.3 miles (3.1 to 6.9 km) (Craig et al. 1988). Therefore, bald eagles nesting near Interstate 84 could potentially be foraging in Reach 15 and the southern section of Reach 14 (Lake Lillinonah).

Total PCB concentrations for prey items from Reaches 14 and 15 were available only for fish. Because of the small number of fish samples for Reaches 14 and 15, all fish were combined instead of separating them into classes (i.e., predatory fish, bottom feeder, forage fish). Fish in Reaches 14 and 15 had an average concentration of 0.717 mg/kg. Bald eagles on average consume a summer diet consisting of 78.6% fish, 16.8% birds, and 5.1% mammals (see Section K.2.1.5). Mammal and bird tPCB concentrations were not available for downstream reaches. Total PCB concentrations for these prey items were estimated in three ways to give high, moderate, and low concentrations. High concentrations assumed that waterfowl and mammals from downstream would have tPCB concentrations equal to those in the PSA. Low concentrations assumed that waterfowl and mammals from downstream would have tPCB concentrations of zero. A moderate concentration was developed by determining fish-to-mammal and fish-to-bird ratios based on concentrations in the PSA. Mammal tPCB concentrations in the PSA are on average 75% of the total fish concentration, and waterfowl tPCB concentrations averaged 15% of the total fish concentration. Therefore, moderate tPCB concentrations downstream were 0.538 mg/kg for mammals and 0.108 mg/kg for birds.

The low, moderate, and high tPCB intake rates averaged 0.022 mg/kg bw/d, 0.025 mg/kg bw/d, and 0.243 mg/kg bw/d, respectively. These values fall below the lower toxicity threshold of 0.7 mg/kg bw/d.

Risks from TEQ to bald eagles in the PSA were low to intermediate; therefore, it was assumed that risk from TEQ to bald eagles breeding downstream would be low.

K.4.2.2 American Bittern Risk Characterization

K.4.2.2.1 tPCBs

Toxicity thresholds based on TDI for American bitterns were estimated from studies conducted on other avian species. The threshold range for American bitterns exposed to tPCBs is 0.12 to 0.7 mg/kg bw/d based on reproductive studies conducted on white leghorn chickens and American kestrels, respectively. The Monte Carlo predictions for TDIs of tPCBs by American bitterns indicated that there was a 100% probability of exceeding the lower toxicity threshold in Reaches 5A, 5B, 5C, and 6. The corresponding probabilities for exceeding the upper threshold estimated by the Monte Carlo analysis were also 100% for these reaches.

The probability bounds analysis indicated that the probability of exceeding the lower toxicity threshold was 100% for Reaches 5A, 5B, 5C, and 6. The probability bounds analysis indicated that the corresponding probability of exceeding the upper threshold was also 100% (Figures K.4-6 through K.4-9). Therefore, American bitterns feeding and reproducing in the Housatonic River PSA are at a high risk of toxicity from exposure to tPCBs in these reaches (Table K.4-2).

K.4.2.2.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

The threshold range for avian species exposed to TEQ is 14 to 5000 ng/kg bw/d, based on studies of pheasants and American kestrels, respectively. The Monte Carlo predictions for TDIs of TEQ by American bitterns indicated that there was a 100% probability of exceeding the lower toxicity threshold and a 100% probability of exceeding the upper threshold in the PSA.

The probability bounds analysis indicated that the probability of exceeding the lower toxicity was 100%. The corresponding probability of exceeding the upper threshold was 0% for the PSA. Therefore, American bitterns feeding and reproducing in the Housatonic River PSA are at intermediate risk of toxicity from exposure to TEQ (Table K.4-2, Figure K.4-10).

K.4.2.2.3 Egg tPCBs

A field-based toxicity threshold of 4 mg/kg was developed based on studies of heron species. The concentrations of tPCBs estimated in eggs for all reaches exceeded the toxicity threshold of 4.0 mg/kg. An American bittern would need to forage in Reaches 5A, 5B, 5C, and 5D and 6 for 6, 7, 7, and 9 days, respectively, prior to breeding for the eggs to exceed the toxicity threshold, based on median egg concentrations (Figures K.4-11 through K.4-14). American bitterns nesting in the PSA are exposed to concentrations of tPCBs high enough to cause adverse effects.

K.4.2.2.4 Egg 2,3,7,8-TCDD Toxic Equivalence (TEQ)

A field based toxicity threshold of 79 ng/kg was developed based on studies of heron species. The concentrations of TEQ in eggs all exceeded the toxicity threshold of 79 ng/kg (Figure K.4-15). An American bittern would need to forage in the PSA a mean of 3 days and a range of 2 to 5 days prior to breeding for her eggs to exceed the toxicity threshold, based on the median egg concentrations. American bitterns nesting in the PSA are exposed to concentrations of TEQ high enough to cause adverse effects.

K.4.2.3 Small-Footed Myotis

K.4.2.3.1 tPCBs

The effects metric for tPCBs was derived using the results of Spencer (1982). A dose-response curve was developed for mammals based on this laboratory study with rats. The dose-response curve developed from this study indicated that 10% and 20% declines in reproductive fecundity would be expected at tPCB doses of 3.48 and 5.37 mg/kg bw/d, respectively. The Monte Carlo predictions for TDI of tPCBs by small-footed myotis in Reach 5 indicated that there was a 99.1% probability of exceeding the 10% effect dose, and a 96.3% probability of exceeding the 20% dose (Figure K.4-16).

The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 69 to 100%. The corresponding probability of exceeding the 20% effect dose ranges from 30 to 98% (Table K.4-1). Thus, small-footed myotis feeding and reproducing in Reach 5 of the Housatonic River are at a high risk of toxicity from exposure to tPCBs (Table K.4-2).

K.4.2.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

A dose-response curve was developed for mammals based on two laboratory studies with rats. In these studies, reproductive effects of TCDD on Sprague Dawley rats fed treated diets on days 6 through 15 of gestation were determined (Khera and Ruddick 1973; Sparschu et al. 1971). The dose-response curve developed from this study indicated that 10% and 20% declines in reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d, respectively. The Monte Carlo predictions for TDI of TEQ by small-footed myotis in Reach 5 indicated that there was a 99.2% probability of exceeding the 10% effect dose, and a 92.9% probability of exceeding the 20% dose (Figure K.4-17).

The probability bounds analysis indicated that the probability of exceeding the 10% effect dose ranges from 0 to 100%. The corresponding probability of exceeding the 20% effect dose could range from 0 to 100% (Table K.4-1). Thus, small-footed myotis are potentially at a high risk of toxicity from exposure to TEQ (Table K.4-2). There is, however, considerable uncertainty about the risk estimates for small-footed myotis exposed to TEQ in Reach 5.

K.4.2.3.3 Conclusions

Small-footed myotis feeding and reproducing in Reach 5 of the Housatonic River are at high risk of toxicity from exposure to tPCBs and may also be at risk from exposure to TEQ. Life history traits of small-footed myotis may compound this risk. Small-footed myotis are a long-lived species, with a maximum longevity of 12 years (Kurta 1995). Therefore, small-footed myotis have the potential to bioaccumulate large amounts of tPCBs and TEQ over their lifespan. Hibernation can affect the way bats accumulate organochlorines and their effects. During hibernation as body lipids are metabolized, PCBs can accumulate in the brain, since brain lipids are not metabolized. This may lead to neurological poisoning or sublethal neurological effects such as tremoring (Clark and Prouty 1977; Clark and Stafford 1981). During hibernation, energy stores are closely balanced against need, and any disturbances that may increase the metabolic rate can cause mortality through starvation. Therefore, bats that have accumulated high concentrations of PCBs may experience stressors that limit their ability to survive hibernation.

K.4.3 Weight-of-Evidence Analysis

A weight-of-evidence analysis was used to assess the measurement endpoints and to determine whether significant risk of harm from PCBs was posed to the T&E species in the Housatonic River. This analysis of risk to T&E species has uncertainty from a few factors, including limited feeding rate data for wild populations; few observations of bald eagles, American bitterns, and small-footed myotis (however, this is expected for T&E species, particularly with the contamination present in the study area); little or no toxicity data in the literature for the representative species; and no site-specific toxicity tests. The analysis follows the methodology developed by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996). Details on this weight-of-evidence approach are provided in Section 2.9 of the ERA. The analyses were conducted separately for tPCBs and TEQ. As noted previously, the field surveys were qualitative and therefore not used in this analysis.

K.4.3.1 Evaluating Measurement Endpoints

Measurement endpoints were assigned relative weights for nine attributes listed in Menzie et al. (1996) that account for strength of association between the assessment and measurement endpoints and study design and execution. For this weight-of-evidence evaluation, it was assumed that the relative importance of each attribute was equal; therefore, endpoint weighting was simply a matter of qualitatively evaluating each attribute and subsequently determining an overall weight for that endpoint. This approach is consistent with the guidance provided by the Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996). The measurement endpoint values are summarized in Table K.4-3 for tPCBs and TEQ. The measurement endpoint values for each attribute are discussed in more detail in the following sections.

K.4.3.1.1 Relationship Between Measurement and Assessment Endpoints

Degree of Association

This attribute quantifies the degree of biological association between the measurement and assessment endpoints. The assessment endpoint is the survival, growth, and reproduction of T&E species.

The measurement endpoint for the bald eagles was high for tPCBs and moderate/high for TEQ. American bitterns and small-footed myotis were rated as moderate/high for both tPCBs and TEQ. The exposure models were species-specific, but studies on surrogate species were used to derive the effects matrix for American bitterns and small-footed myotis, hence, the moderate/high value.

Stressor/Response

This attribute measures the ability to correlate the magnitude of response with the degree of exposure. The modeled exposure was species and stressor-specific. Effects metrics used a toxicity threshold for bald eagles exposed to tPCBs, resulting in a value of moderate/high as the kestrel threshold was used. Surrogate raptor species were used for TEQ, resulting in a moderate value. A toxicity threshold range based on surrogate species was used for American bitterns. This made it difficult to determine the relationship between the magnitude of response and the degree of exposure for American bitterns, thus the value of moderate for tPCBs and TEQ. A dose-response curve based on surrogate mammal species was used as the effects metric for small-footed myotis. This quantitatively correlates toxicity with magnitude of exposure, but a surrogate animal was used; therefore, a value of moderate/high was assigned.

Utility of Measure

This attribute reflects the quality of the accepted criteria, standards, or performance-based measures used to judge environmental harm. For tPCBs and TEQ, the values for bald eagles, American bitterns, and small-footed myotis were moderate/high. The techniques used to model exposure and effects are well established and accepted by the scientific community. These measurement endpoints are sensitive and should be capable of detecting effects to the maintenance of local populations of T&E species. The effects metrics used in the risk characterization for T&E species included toxicity threshold ranges, field-based toxicity thresholds, and dose-response curves. The toxicity threshold range used for American bitterns is expected to bracket the thresholds of this species; however, the broad range limits its utility for judging environmental harm. The field-based metric used for bald eagles and American bittern eggs was based on estimates of chemical absorption efficiency and maternal transfer to eggs.

The dose-response curve used for small-footed myotis provides more certainty in the effects metric; however, it is based on surrogate species.

Data Quality

This attribute refers to data quality objectives (DQOs), the extent to which they are comprehensive and rigorous, and the extent to which they are met. DQOs should evaluate the appropriateness of data collection and analysis with respect to the intended use of the data. When DQOs are not met, the reasons why and the potential impact on the assessment should be documented.

The modeled exposure and effects data for bald eagle, American bitterns, and small-footed myotis received a high score for this attribute because DQOs for the sampling analysis and tissue samples were met for the tissue contaminant data used in the exposure analysis for both tPCBs and TEQ.

K.4.3.1.2 Study Design

Site Specificity

This attribute referred to the extent to which data, media, species, environmental conditions, and habitat types used in the study design reflected the site of interest. For this attribute, the values for tPCBs and TEQ for all three T&E species was moderate. The biological tissue data used in exposure models was site-specific and exposure parameters reflected site conditions. The effects studies used to develop the threshold ranges, however, were not specific to the Housatonic River area.

Sensitivity

This attribute refers to the ability of the measurement endpoint to detect changes due to a stressor against a background of natural variability and other sources of uncertainty. The modeled exposure and effects assessed the exposure-response relationship. For bald eagles, the tPCB value was moderate/high and the TEQ value was high. Effects data for tPCBs were field-based and contained multiple stressors. American bitterns were given a value of high for tPCBs and

TEQ. Small-footed myotis was given a value of high for tPCBs and TEQ, as a dose-response curve was used for this species.

Spatial Representativeness

This attribute reflects the degree of spatial overlap of study area, measurement locations, locations of stressors, locations of representative species, and points of potential exposure to those stressors. Bald eagles and small-footed myotis had a value of high for tPCBs and TEQ. American bitterns had a moderate/high value for tPCBs and TEQ. Modeled exposures relied on tissue data collected in the measurement locations and habitat of representative species. The American bittern exposure used small mammal tissue data from mammals within the PSA, but that these tissue samples were not collected in optimum bittern foraging habitat.

Temporal Representativeness

This attribute related to the temporal comparability between the data collection for the measurement endpoint and the period during which effects of concern were likely to occur. A value of moderate/high was given for both tPCBs and TEQ for bald eagles, American bitterns, and small-footed myotis. The exposure model was designed to span the reproductive cycle of T&E species. Prey tissue samples used in measurement endpoints were collected during the time when effects would be expected to be manifested (the breeding season); however, for many species, only one sampling event was conducted.

Quantitative Measure

This attribute indicates the degree to which the response can be quantified by a given measurement endpoint. This attribute also referred to the objectivity of the results, whether results were sufficient to test for statistical significance, and whether biological significance could be inferred from statistical significance. All three T&E species were given a value of high for tPCBs and TEQ. The probabilistic exposure and effects modeling were highly quantitative and propagated uncertainty associated with modeling procedures.

Standard Method

This attribute quantified the extent to which measurement endpoint studies adhered to recognized scientific protocols. All three T&E species were given a value of moderate/high for tPCBs and TEQ. Generally accepted exposure and effects modeling procedures were followed, but probability bounds analysis is a relatively new technique for propagating uncertainty.

K.4.3.1.3 Total Value

The total value assigned to each measurement endpoint is a weighted average of the values from the attributes (Table K.4-3). The overall value for bald eagles, American bitterns, and small-footed myotis was moderate/high for both tPCBs and TEQ.

K.4.3.2 Magnitude of Response in Measurement Endpoints

The measurement endpoint values were used together with the magnitude of response in the measurement endpoints to judge the weight-of-evidence for adverse effects on representative species. The magnitude of response can be presented as qualitative or quantitative measures. Qualitative measures were used for this assessment. The weighting values, evidence of harm, and magnitudes of responses for each measurement endpoint are presented Table K.4-4 for tPCBs and Table K.4-5 for TEQ.

The results from the modeled exposure and effects line of evidence indicated that there was risk from exposure to tPCBs for bald eagles and American bitterns. The risks to bald eagles and American bittern were high, whereas the tPCB risk to small-footed myotis was undetermined. There was no risk from exposure to TEQ for adult bald eagles but high risk for bald eagle eggs. Risk was undetermined for American bittern and small-footed myotis.

K.4.3.3 Concurrence Among Measurement Endpoints

The final component in the weight-of-evidence approach examines the agreement, logical connections, interdependencies, and correlations among the measurement endpoints as they relate to each assessment endpoint. The methodology for detecting concurrence involves the use of a graphical method where measurement endpoints are plotted on a matrix that also includes

the weight of each endpoint and degree of response. Tables K.4-6 and K.4-7 depict the outcome for T&E species for tPCBs and TEQ, respectively.

K.4.4 Sources of Uncertainty

The assessment of risk to T&E species contains uncertainties. Each source of uncertainty can influence the estimates of risk; therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to T&E species are described below.

- In this assessment, it was assumed that dietary exposure represents the most important pathway of exposure for T&E species exposed to COCs. Although unlikely to provide a major contribution to the risk, other pathways could increase the exposure and perhaps increase risk slightly (Moore et al. 1999). When including drinking water and vegetation as part of the diet for T&E species, the exposure model showed negligible increases in exposure.

- The Monte Carlo sensitivity analyses suggest that the FMR slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. However, no measurements of FMRs were available for the representative wildlife species. Similarly, measured FIRs were not available for bald eagles, American bitterns, small-footed myotis, or reasonable surrogate species. Therefore, FMRs were estimated using allometric equations. The use of allometric equations introduces some uncertainty into the exposure estimates because they have model-fitting error and are based on species different from the representative species used in this assessment. For example, the small-footed myotis analysis used the insectivore model of Nagy et al. (1999) as the most appropriate allometric model to estimate FMRs. Although the insectivore model was developed using bat species, other insectivores, including shrew, mole, and mouse species, were also used to derive the model. Given the lack of data on representative species used in the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as input for the allometric slope and power terms.

- The free-living metabolic rate equations require body weight (BW) as an input variable. Data on the BW of bald eagle, American bittern, and small-footed myotis living in the PSA are not available. As a result, BWs of these species were estimated from the literature. The potential magnitude and direction of the uncertainty associated with lack of site-specific information on BWs are unknown, but are likely small.

- Because no stomach contents or other dietary analyses are available for the bald eagle, American bittern, and small-footed myotis in the PSA, dietary compositions were derived from those reported in the literature for birds and mammals collected from other geographical locations. The potential magnitude and direction of the uncertainty associated with lack of information on diet are unknown. The uncertainty due to lack of knowledge on bald eagle, American bittern, and small-footed myotis in the PSA was partially addressed by using distributions to represent variability in diets at other similar sites.

- When estimating exposure to bald eagles downstream of Woods Pond, concentrations of tPCBs in birds and mammals were assumed to be zero. This may be the case for birds consumed by wintering eagles, as these are likely waterfowl that have migrated from areas outside the Housatonic River floodplain. However, this may underestimate the true exposure to bald eagles occurring downstream.

- Sample sizes were limited for the analyses of COC concentrations in some prey items. Only two to four invertebrate samples for Reaches 5B, 5C, and 6 were available to estimate exposure of American bittern to PCBs. Uncertainty due to sample size was explicitly addressed in the uncertainty analyses. In the Monte Carlo analysis, sample size uncertainty was addressed by use of the 95% UCL on the mean. Use of the UCL addressed uncertainty, but is biased towards overestimating exposure. In the probability bounds analysis, uncertainty was addressed by specifying concentration variables as a range from the 95% LCL to the UCL. This treatment of uncertainty is unbiased.

- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). This source of uncertainty was addressed in the uncertainty analyses by estimating prey concentrations assuming concentrations of PCB-123 and PCB-157 were equal to zero, and assuming that concentrations of PCB-123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. The resulting TEQ estimates were then compared. If the ratio of the upper to lower bound TEQ estimate was less than 1.3, this source of uncertainty was deemed unimportant and disregarded. If the ratio exceeded 1.3, the uncertainty due to the co-elution was propagated through the uncertainty analyses.

- The foraging times of bald eagles, American bitterns, and small-footed myotis in the PSA were assumed to be 100%. This assumption is valid for many bitterns and myotis as they have small foraging ranges. However, the foraging radius of bald eagles can range up to 5 miles from the nest. The availability of prey fish and ample suitable foraging area within the PSA suggest that bald eagles in the area could forage exclusively within the PSA. These birds may, however, also forage beyond the PSA, thus reducing exposure to COCs.

- The adult body burden and associated egg concentration was estimated assuming that avian species do not metabolize PCBs, to simplify the estimated accumulation by bald eagles. This assumption may result in an overestimate of the amount of PCBs accumulated.

- The adult body burden was estimated assuming that a breeding adult would arrive in the PSA with no tPCBs in the body. New breeding pairs colonizing the PSA would be expected to have low concentrations of tPCBs in their tissue. However, eagles return to the same breeding area, and often the same nest, each year. Bald eagles returning to the PSA during subsequent years would have COC body burdens accumulated during previous breeding seasons. Therefore, body burdens and egg concentrations are likely underestimated for eagles that have previously bred in the PSA.

- The largest source of uncertainty in the effects assessment was associated with the lack of toxicity studies involving the representative species. There were no toxicity studies available for American bittern and small-footed myotis exposed to tPCBs or TEQ. As a result, laboratory studies involving surrogate species were used to estimate effects to these species. These extrapolations introduced uncertainty in the effects assessment because of the variations in physiological and biochemical differences between the species, such as uptake, metabolism, and disposition, that can alter the potential toxicity of a contaminant. The sensitivity of wildlife to an environmental contaminant may differ from that of a laboratory or domestic species due to behavioral and ecological parameters, including stress factors (e.g., competition, seasonal changes in temperature or food availability), disease, and exposure to other contaminants. Inbred laboratory animal strains may also have an unusual sensitivity or resistance to a tested substance.

- For small-footed myotis exposed to TEQ, two toxicity studies, Khera and Ruddick (1973) and Sparschu et al. (1971), were combined to derive a dose-response curve. Both studies investigated reproduction effects in rats by administering TCDD on gestation days 6 to 15. The studies used similar protocols, and when combined, yielded a data set with 17 treatment levels. Although these studies were similar in terms of endpoints, duration of exposure, quality, and acceptability of protocols, there is some uncertainty in the data set generated from combining the studies.

- The effects metrics used to estimate risk to T&E species were derived for Aroclor 1254 mixtures. Some uncertainty is inherent in extrapolating from those mixtures to the specific congener patterns observed in the PSA of the Housatonic River. The potential magnitude and direction of the uncertainty associated with this extrapolation are unknown for T&E species.

- For T&E species, data for two potential lines of evidence were available. For these assessments, toxicity studies performed in situ in the PSA of the Housatonic River or feeding studies involving prey and food items from the PSA would have improved the weight-of-evidence assessment. Such studies would have accounted directly for the specific characteristics of the Housatonic River ecosystem and the toxicity of the PCB mixture found on site.

K.4.5 Conclusions

The weight-of-evidence analysis indicates that T&E species such as bald eagles, American bitterns, and small-footed myotis are at some risk in the PSA as a result of exposure to tPCBs and TEQ. For bald eagles and American bitterns, exposure and effects for both tPCBs and TEQ were estimated for body burden and eggs separately. In the weight-of-evidence analysis, the risks for both of these factors were combined and presented together because ecologically if there is risk to either life stage, there is risk to the organism. The risk estimates for bald eagles and American bittern exposed to tPCBs are high. The risk estimates for bald eagle exposed to TEQ are intermediate, and for American bittern exposed to TEQ, risks are undetermined. The risk estimates for small-footed myotis exposed to tPCBs and TEQ are undetermined.

The bald eagle, American bittern, and small-footed myotis were chosen to represent T&E species inhabiting the Housatonic River PSA. Other T&E species that occur in the area include one mussel (triangle floater); three dragonflies (riffle snaketail, zebra clubtail, and arrow clubtail); a turtle (wood turtle); three salamanders (Jefferson salamander, four-toed salamander, and northern spring salamander); three hawks (northern harrier, sharp-shinned hawk, and Cooper's hawk); two warblers (northern parula and blackpoll warbler); a wading bird (common moorhen); and a shrew (northern water shrew). Some of these species will be assessed in other appendices and compared to the risks to other, more appropriate assessment endpoints (i.e., amphibians for salamanders).

The purpose of this section is to qualitatively compare exposure to tPCBs and TEQ for the representative T&E species to other similar species. The major factors that influence exposure to tPCBs and TEQ include:

1. Foraging behavior and dietary composition.
2. Foraging and home range.
3. Species body weight and other life history characteristics.

The comparison highlights similarities and differences, and their potential to influence exposure and risks from tPCBs and TEQ (see Table K.4-8).

Bald Eagle to Other Species

Because similar species of piscivorous birds, such as osprey, are addressed in Appendix H, they are not discussed here.

American Bittern to Other Species

As noted earlier, effects studies on American bittern were not available. Similarly, effects data were not available for other carnivorous bird species living in the Housatonic River area. As a result, the same surrogate effects data used to estimate effects to American bittern would be used for other piscivorous bird species.

Least Bittern

The least bittern's life history, foraging behavior, habitat preferences, and feeding habits are similar to the American bittern. The least bittern is much smaller than the American bittern; therefore, it has a higher metabolism and its ability to accumulate contaminants is greater, leading to increased exposure to tPCBs and TEQ. The habitat and feeding habits of the two bitterns are very similar. Therefore, the least bittern is expected to have a higher level of risk from exposure to tPCBs and TEQ than the American bittern.

Green Heron

The green heron's life history, foraging behavior, habitat preferences, and feeding habits are similar to the American bittern. The green heron is slightly smaller in size. Thus it has a higher metabolism and its ability to accumulate contaminants will be greater, leading to increased exposure to tPCBs and TEQ. Both species have similar foraging habitat and diet. Therefore, the green heron is expected to have a higher level of risk from exposure to tPCBs and TEQ than the American bittern.

Great Blue Heron

The great blue heron has similar life history, foraging behavior, habitat preferences, and feeding habits as the American bittern, although great blue heron consume a greater proportion of fish. Fish contain higher tPCBs than do other prey groups (i.e., amphibians, crayfish); therefore, great

blue herons would be exposed to greater amounts of tPCBs than bittern. The great blue heron is larger than the bittern. Therefore, it has a lower metabolism and is exposed to less COCs. Overall, great blue heron that have foraging territories within the PSA are expected to have similar levels of risk from exposure to tPCBs as do American bittern.

King Rail

The king rail's life history, foraging behavior, habitat preferences, and feeding habits are similar to the American bittern. The king rail however, predominantly consumes large crustaceans and aquatic insects along with some amphibians, mollusks, seeds of marsh plants. The king rail is smaller that the American bittern and thus has a higher metabolism and its ability to accumulate contaminants will be greater, leading to increased exposure to tPCBs and TEQ. Therefore, the king rail is expected to have a similar level of risk from exposure to tPCBs and TEQ than the American bittern.

Virginia Rail

The Virginia rail's life history, foraging behavior, habitat preferences, and feeding habits are similar to the American bittern. The Virginia rail is much smaller that the American bittern and thus has a higher metabolism and its ability to accumulate contaminants will be greater, leading to increased exposure to tPCBs and TEQ. Both species have similar foraging habitat and diet. Therefore, the Virginia rail is expected to have a higher level of risk from exposure to tPCBs and TEQ than the American bittern.

Pied-Billed Grebe

The pied-billed grebe's life history, foraging behavior, habitat preferences, and feeding habits are similar to the American bittern. The pied-billed grebe is smaller that the American bittern and thus has a higher metabolism and its ability to accumulate contaminants will be greater, leading to increased exposure to tPCBs and TEQ. Both species have similar foraging habitat and diet. Therefore, the pied-billed grebe is expected to have a higher level of risk from exposure to tPCBs and TEQ than the American bittern.

Sora

The sora's life history, foraging behavior, and habitat preferences are similar to the American bittern. The sora however, consumes a large proportion of vegetable matter and fish and amphibians lacking in its diet. Therefore, the sora is expected to have a lower level of risk from exposure to tPCBs and TEQ than the American bittern.

Small-Footed Myotis to Other Species

Myotis Species

The life history, foraging behavior, habitat preferences, and feeding habits of the Indiana bat, northern myotis, and little brown bat are similar to the small-footed myotis. These species all belong to the genus *Myotis*; therefore, they are expected to have similar risk from exposure to tPCBS and TEQ. When data were not available for small-footed myotis, data from the Indiana bat and little brown bat were used in the analysis.

Risk Summary

The weight-of-evidence analysis indicates that the bald eagle and American bittern are at risk in the PSA as a result of exposure to tPCBs. Risk to the small-footed myotis is undetermined for both tPCB and TEQ exposure in the PSA. Bald eagles are at intermediate risk from TEQ exposure in the PSA, whereas American bittern risk as a result of TEQ exposure in the PSA is undetermined.

Other piscivorous wading bird species that could occur in the PSA include the least bittern, green heron, great blue heron, king rail, least rail, sora, and pied-billed grebe. A qualitative analysis of risk to these species indicates that the great blue heron and king rail are expected to have a similar level of risk compared to the American bittern. The wading birds that have similar diets but are smaller and have higher metabolisms—such as least bittern, green heron, Virginia rail, and pied-billed grebe—are expected to have a higher level of risk than the American bittern. Wading birds that consume plant material, such as the sora, are expected to have low levels of risk.

Other bat species, especially those in the myotis family (little brown bat, Indiana bat, and northern myotis) are expected to have a similar level of risk as the small-footed myotis.

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APPENDIX K

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF THREATENED AND ENDANGERED SPECIES

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Table K.2-1

Dietary Studies for the Bald Eagle

Dietary Composition	Haywood and Ohmart 1986[a]	Dunstan and Harper 1975[b]	Todd et al. 1982[c]	Watson et al. 1991[d]	Stratus Consulting 1999[e]
	% of Prey Items	% of Prey Items	% of Prey Items	% of Prey Items	% of Prey Items
Fish					
Predatory Fish					
Salmon	--	--	0.5	8.6	--
Pike/Pickerel	--	13.9	20.1	--	16.8
Largemouth Bass	--	5	--	--	--
Yellow Bass	1	--	--	--	--
Bass sp.	1.2	--	3.8	--	1.8
Other Centrarchids	--	--		1.6	3.6
Perch	--	--	3.6	1.5	
Walleye	--	--	--	--	1.2
Total Predatory Fish	2.2	18.9	28	11.7	23.4
Forage Fish					
Fallfish	--	--	0.5	--	--
Cyprinids	--	--	--	14	--
Creek Chub	--	--	0.5	--	--
Rock Bass	--	4	--	--	--
Black Crappie	--	--	--	1	--
Herring	--	--	1.7	--	--
Smelt	--	--	0.5	--	--
Alewife	--	--	--	--	0.6
Bowfin	--	--	--	--	6.6
Shad	--	--	--	13	2.4
Total Forage Fish	--	4	3.2	28	9.6
Bottom Feeders					
Catfish	42	--	--	--	--
Bullheads	1.7	35.1	24.8	--	18

Table K.2-1

Dietary Studies for the Bald Eagle (Continued)

Dietary Composition	Haywood and Ohmart 1986[a]	Dunstan and Harper 1975[b]	Todd et al. 1982[c]	Watson et al. 1991[d]	Stratus Consulting 1999[e]
	% of Prey Items	% of Prey Items	% of Prey Items	% of Prey Items	% of Prey Items
Suckers	17.7	29.1	19.5	17.3	13.8
Carp	12.3	--	--	10.8	8.4
Eel	--	--	0.5	--	--
Total Bottom Feeders	73.7	64.2	44.8	28.1	40.2
Other/Unidentified Fish	0.6	3	0.5	3.2	--
Total Fish	76.5	90.1	76.5	71	73.2
Birds					
Heron	1.2	--	1.5	--	0.6
Gulls	0.0	2.0	4.0	2.7	9.0
Waterfowl	5.6	4.6	5.5	16	3.6
Other/Unidentified Birds	4.2	1.3	5.8	7.4	9.6
Total Birds	11	7.9	16.8	26.1	22.8
Mammals					
Rabbit and Hares	6.4	--	2.3	1	--
Rodents	2.6	1.3	1	--	1.2
Other/Unidentified Mammals	2.7	--	3.8	1	1.2
Total Mammals	11.7	1.3	6.8	2	2.4
Reptiles					
Total Reptiles (Snake, Turtle, Lizard)	0.6	--	--	--	0.6
Invertebrates					
Total Invertebrates (Clam, Crayfish)	--	0.6	--	--	--

[a] Location = Central Arizona; Habitat = Interior River; Season = Breeding

[b] Location = North-central Minnesota; Habitat = Interior; Season = Breeding

[c] Location = Maine; Habitat = Interior; Season = Year round

[d] Location = Oregon; Habitat = Coastal and Interior; Season = Year round

[e] Location = Lake Michigan; Habitat = Interior; Season = Breeding

Table K.2-2

Input Variables Used in the Monte Carlo Exposure Analyses for Total Daily Intake of tPCBs and TEQ by Bald Eagles Inhabiting the Housatonic River

Variable	Distribution	Bald Eagle Parameters
Body Weight (g)	Normal	x = 5350 s = 400
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 1.02 s = 0.039
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.018
Gross Energy (kcal/g)		
Fish	Point Estimate	1.2
Birds	Point Estimate	1.8
Mammals	Point Estimate	1.8
Assimilation Efficiency (unitless)		
Fish	Point Estimate	0.790
Birds (Waterfowl)	Point Estimate	0.780
Mammals	Point Estimate	0.780
Proportion Diet (unitless)		
Bottom Fish	Beta	α = 22.0; β = 22.0; scale = 1.01
Predatory Fish	Beta	α = 12.0; β = 25.0; scale = 0.50
Forage Fish	Beta	α = 5.0; β = 15.0; scale = 0.47
Birds (Waterfowl)	Beta	α = 13.0; β = 15.0; scale = 0.35
Mammals	Beta	α = 6.3; β = 25.0; scale = 0.25
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-3

Input Variables Used in the RiskCalc Probability Bounds Analyses for Total Daily Intake of tPCBs and TEQ by Bald Eagles Inhabiting the Housatonic River

Variable	Distribution	Bald Eagle Parameters
Body Weight (g)	Normal	x = 5350 s = 400
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 1.02 s = 0.039
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.018
Gross Energy (kcal/g)		
Fish	Point Estimate	1.2
Birds	Point Estimate	1.8
Mammals	Point Estimate	1.8
Assimilation Efficiency (unitless)		
Fish	Point Estimate	0.790
Birds (Waterfowl)	Point Estimate	0.780
Mammals	Point Estimate	0.780
Proportion Diet (unitless)		
Predatory Fish	Minmaxmean	0.022, 0.28, 0.168
Bottom Fish	Minmaxmean	0.281, 0.737, 0.502
Forage Fish	Minmaxmean	0, 0.28, 0.090
Birds (Waterfowl)	Minmaxmean	0.079, 0.261, 0.169
Mammals	Minmaxmean	0.013, 0.117, 0.048
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-4

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in the Prey of Bald Eagles

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs (mg/kg)	Probability Bounds Inputs (mg/kg)
			Min	25th	50th	75th	Max			
Bottom Feeders	Southern PSA	136	7.19	32.3	59.1	123	447	UCL > Max: No	Point Estimate UCL = 104	Distribution-Free Range LCL = 78.4 UCL = 104
Predatory Fish	Southern PSA	134	8.26	47.1	64.8	95.1	424	UCL > Max: No	Point Estimate UCL = 88.6	Distribution-Free Range LCL = 72.3 UCL = 88.6
Forage Fish	Southern PSA	50	7.82	23.0	34.5	44.6	82.1	UCL > Max: No	Point Estimate UCL = 43.4	Distribution-Free Range LCL = 32.9 UCL = 43.4
Birds	Southern PSA	25	1.04	4.79	6.09	8.70	19.0	UCL > Max: No Reconstitution Issue[c]: Yes	Triangular Distribution Min = 2.03 Best Estimate = 9.90 Max = 19.9	Distribution-Free Range Min = 1.22 Max = 19.9
Mammals	Southern PSA	76	0.15	1.78	4.98	29.3	148	UCL > Max: No	Point Estimate UCL = 80.6	Distribution-Free Range LCL = 23.2 UCL = 80.6

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations. For birds, statistics are calculated as for other prey species and the reported values are for the moderate (offal = breast) estimate.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis, and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis.

[c] For Monte Carlo Inputs, the minimum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for the low (offal = 0) estimate. The best estimate of the triangular distribution was the lower of maximum value and the upper 95% CL for the moderate (offal = breast) estimate. The maximum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for high (offal = liver) estimate. For the probability bounds, the minimum value was the higher of the minimum value and the lower 95% CL from the low (offal = 0) estimate. The maximum value was the lower of the maximum values and the upper 95% CL from the high (offal = liver) estimate.

Table K.2-5

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in the Prey of Bald Eagles

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Bottom Feeders	Southern PSA	60	171	588	958	1,560	5,370	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 1,480	Distribution-Free Range LCL = 1,040 UCL = 1,480
Predatory Fish	Southern PSA	66	261	663	1,090	1,880	3,270	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 1,570	Distribution-Free Range LCL = 1,160 UCL = 1,570
Forage Fish	Southern PSA	30	254	376	584	769	2,330	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 777	Distribution-Free Range LCL = 548 UCL = 777
Birds	Southern PSA	25	128	423	702	1,440	6,690	Co-elution issue: No Detection limit issue: No Reconstitution issue[c]: Yes UCL > Max: NA	Triangular Distribution Min = 485; Best Estimate = 2,149; Max = 4,020	Distribution-Free Range Min = 232 Max = 4,020
Mammals	PSA	12	18.8	168	321	747	3,980	Co-elution issue: No Detection limit issue: No UCL > Max: Yes	Point Estimate Max = 3,980	Distribution-Free Range Min = 18.8 Max = 3,980

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations. For birds, statistics are calculated as for other prey species and the reported values are for the moderate (offal = breast) estimate.

[b] If assumptions about concentrations for samples below the DL or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL and UCL were used in the probability bounds analysis.

[c] For Monte Carlo Inputs, the minimum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for the low (offal = 0) estimate. The best estimate of the triangular distribution was the lower of maximum value and the upper 95% CL for the moderate (offal = breast) estimate. The maximum value of the triangular distribution was the lower of the maximum value and the upper 95% CL for high (offal = liver) estimate. For the probability bounds, the minimum value was the higher of the minimum value and the lower 95% CL from the low (offal = 0) estimate. The maximum value was the lower of the maximum values and the upper 95% CL from the high (offal = liver) estimate.

Table K.2-6

Exposure Model Results for Bald Eagles Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Lower PSA	6.23	25.4	13.2	13.0	10.2	13.0	16.6	3.73	5.41	8.27	13.4	17.9	24.2

Table K.2-7

Sensitivity Results From Monte Carlo Analysis of Bald Eagles Exposed to tPCBs and TEQ

tPCBs		TEQ	
Variable	**Sensitivity**	**Variable**	**Sensitivity**
FMR – Power Term	0.85	FMR – Power Term	0.85
FMR – Log_{10} Slope	0.47	FMR – Log_{10} Slope	0.48
Body Weight (g)	-0.13	Body Weight (g)	-0.14
Waterfowl Concentration (mg/kg)	0.02	Waterfowl Concentration (mg/kg)	-0.03
Proportion Diet – Bottom Fish	0.02	Proportion Diet – Bottom Fish	0.01
Proportion Diet – Birds	-0.01	Proportion Diet – Birds	-0.00
Proportion Diet – Predatory Fish	0.00	Proportion Diet – Predatory Fish	0.01
Proportion Diet – Mammals	0.00	Proportion Diet – Mammals	-0.00
Proportion Diet – Forage Fish	-0.00	Proportion Diet – Forage Fish	-0.01

Note: Sensitivity determined by absolute value.

Table K.2-8

Exposure Model Results for Bald Eagles Exposed to TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Lower PSA	117	514	256	251	197	251	321	52.3	76.6	115	291	388	526

Table K.2-9

Dietary Studies for the American Bittern

Dietary Composition	Gibbs et al. 1992
	% of Prey Items
Invertebrates (dragonflies and nymphs, water beetles, giant waterbugs, water scorpions, grasshoppers)	23.0
Fish (sunfish, perch, pickerel, killifish, catfish, suckers, eels)	21.0
Amphibians	21.0
Small Mammals	10.0
Reptiles (snakes)	5.0
Crayfish	19.0

Table K.2-10

Input Variables Used in the Monte Carlo Exposure Analyses for Total Daily Intake of tPCBs and TEQ by American Bittern Inhabiting the Housatonic River

Variable	Distribution	American Bittern Parameters
Body Weight (g)	Normal	x = 707 s =183
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 1.02 s = 0.039
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.018
Gross Energy (kcal/g)		
Invertebrates	Point Estimate	1.6
Fish	Point Estimate	1.2
Amphibians	Point Estimate	1.2
Mammals	Point Estimate	1.8
Assimilation Efficiency (kcal/g)		
Invertebrates	Point Estimate	0.720
Fish	Point Estimate	0.790
Amphibians	Point Estimate	0.790
Mammals	Point Estimate	0.780
Proportion Diet (unitless)		
Invertebrates	Point Estimate	0.245
Macroinvertebrates	Point Estimate	0.202
Fish	Point Estimate	0.223
Amphibians	Point Estimate	0.223
Mammals	Point Estimate	0.106
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-11

Input Variables Used in the RiskCalc Probability Bounds Analyses for Total Daily Intake of tPCBs and TEQ by American Bittern Inhabiting the Housatonic River

Variable	Distribution	American Bittern Parameters
Body Weight (g)	Normal	x = 707 s =183
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 1.02 s = 0.039
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.018
Gross Energy (kcal/g)		
Invertebrates	Point Estimate	1.6
Fish	Point Estimate	1.2
Amphibians	Point Estimate	1.2
Mammals	Point Estimate	1.8
Assimilation Efficiency (unitless)		
Invertebrates	Point Estimate	0.720
Fish	Point Estimate	0.790
Amphibians	Point Estimate	0.790
Mammals	Point Estimate	0.780
Proportion Diet (unitless)		
Invertebrates	Point Estimate	0.245
Macroinvertebrates	Point Estimate	0.202
Fish	Point Estimate	0.223
Amphibians	Point Estimate	0.223
Mammals	Point Estimate	0.106
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in the Prey of American Bittern Inhabiting the Housatonic River

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Fish	Reach 5A	12	24.2	40.1	45.1	48.5	54.3	UCL > Max: No	Point Estimate UCL = 49.4	Distribution-Free Range LCL = 38.5 UCL = 49.4
	Reach 5B	91	15.0	24.0	27.0	31.5	41.0	UCL > Max: No	Point Estimate UCL = 28.8	Distribution-Free Range LCL = 26.7 UCL = 28.8
	Reach 5C	14	2.60	22.3	24.9	26.5	35.9	UCL > Max: Yes	Point Estimate Max = 35.9	Distribution-Free Range Min = 2.59 Max = 35.9
	Reach 5D & 6	82	8.80	22.0	26.2	31.3	120	UCL > Max: No	Point Estimate UCL = 32.5	Distribution-Free Range LCL = 27.5 UCL = 32.5
Invertebrates	Reach 5A	6	2.23	5.93	10.5	15.5	18.2	UCL > Max: Yes	Point Estimate Max = 18.2	Distribution-Free Range Min = 2.23 Max = 18.2
	Reach 5B	2	28.6	29.6	30.6	31.5	32.5	UCL > Max: N/A[c]	Point Estimate Max = 32.5	Distribution-Free Range Min = 28.6 Max = 32.5
	Reach 5C	3	6.38	8.26	10.2	28.9	47.60	UCL > Max: N/A[c]	Point Estimate Max = 47.6	Distribution-Free Range Min = 6.38 Max = 47.6
	Reach 5D & 6	4	2.29	4.23	9.59	14.4	14.9	UCL > Max: N/A[c]	Point Estimate Max = 14.9	Distribution-Free Range Min = 2.29 Max =14.9

Table K.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in the Prey of American Bittern Inhabiting the Housatonic River (Continued)

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Macro-invertebrates	Reach 5A	10	8.07	10.7	18.0	24.8	52.1	UCL > Max: No	Point Estimate UCL = 36.2	Distribution-Free Range LCL = 15.8 UCL = 36.2
	Reach 5B	10	4.35	6.96	8.57	14.6	31.6	UCL > Max: No	Point Estimate UCL = 19.9	Distribution-Free Range LCL = 8.97 UCL = 19.9
	Reach 5C	10	2.59	6.92	7.80	8.54	14.7	UCL > Max: No	Point Estimate UCL = 11.8	Distribution-Free Range LCL = 6.55 UCL = 11.8
	Reach 5D & 6	10	3.95	4.84	5.81	6.71	15.8	UCL > Max: No	Point Estimate UCL = 8.90	Distribution-Free Range LCL = 5.48 UCL = 8.90
Amphibians	Reach 5A	5	1.21	1.23	2.45	3.59	5.39	UCL > Max: Yes	Point Estimate Max = 5.39	Distribution-Free Range Min = 1.21 Max = 5.39
	Reach 5B	5	0.55	1.13	1.76	2.11	6.33	UCL > Max: Yes	Point Estimate Max = 6.33	Distribution-Free Range Min = 0.55 Max = 6.33
	Reach 5C	7	0.16	0.53	1.80	3.68	7.73	UCL > Max: Yes	Point Estimate Max = 7.73	Distribution-Free Range Min = 0.16 Max = 7.73
	Reach 5D & 6	22	0.03	2.90	4.42	5.52	9.22	UCL > Max: Yes	Point Estimate Max = 9.22	Distribution-Free Range Min = 0.03 Max = 9.22

Table K.2-12

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in the Prey of American Bittern Inhabiting the Housatonic River (Continued)

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Mammals	PSA	76	0.15	1.78	4.98	29.3	148	UCL > Max: No	Point Estimate UCL = 80.7	Distribution-Free Range LCL = 23.2 UCL = 80.7

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis. When the number of samples was below 5, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis.

[c] The maximum measured concentration was used as a point estimate in the Monte Carlo analysis because the sample size was too small to calculate confidence limits using the Land H-statistic.

Table K.2-13

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in the Prey of American Bittern

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs (ng/kg)	Probability Bounds Inputs (ng/kg)
			Min	25th	50th	75th	Max			
Fish	PSA	48	257	336	475	635	1,860	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 622	Distribution-Free Range LCL = 492 UCL = 622
Invertebrates	PSA	6	125	247	357	470	495	Co-elution issue: No Detection limit issue: No UCL > Max: Yes	Point Estimate Max = 2,670	Distribution-Free Range LCL = 43.9 UCL = 4,610
Macro-invertebrates	PSA	12	65.3	129	188	591	1,070	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 818	Distribution-Free Range Min = 224 Max = 818
Amphibians	PSA	5	36.6	38.2	60.9	71.0	81.4	Co-elution issue: No Detection limit issue: No UCL > Max: Yes	Point Estimate Max = 81.5	Distribution-Free Range Min = 36.6 Max = 81.5
Mammals	PSA	12	18.8	168	321	747	3,980	Co-elution issue: No Detection limit issue: No UCL > Max: No	Point Estimate UCL = 3,980	Distribution-Free Range LCL = 18.8 UCL = 3,980

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] If assumptions about concentrations for samples below the DL or concentrations of co-eluted congeners did not affect calculated arithmetic mean (i.e., maximum possible mean: minimum possible mean ≤ 1.3), uncertainty due to assumptions was ignored. Otherwise, uncertainty was explicitly incorporated in Monte Carlo and probability bounds analysis. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis, and the LCL and UCL were used in the probability bounds analysis.

Table K.2-14

Exposure Model Results for American Bittern Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Reach 5A	4.70	18.6	9.24	9.07	7.30	9.07	11.4	3.53	4.42	5.49	7.44	9.19	11.7
Reach 5B	3.99	15.8	7.84	7.70	6.19	7.70	9.64	3.89	4.88	6.06	6.32	7.80	9.90
Reach 5C	4.60	18.2	9.03	8.87	7.13	8.87	11.1	1.21	1.51	1.88	7.28	8.99	11.4
Reach 6	3.33	13.2	6.53	6.41	5.16	6.41	8.03	2.38	2.98	3.70	5.26	6.49	8.25

Table K.2-15

Sensitivity Analysis Results for American Bittern Exposed to PCBs and TEQ

tPCBs		TEQ	
Variable	**Sensitivity**	**Variable**	**Sensitivity**
FMR – Power Term	0.66	FMR – Power Term	0.66
FMR – Log10 Slope	0.50	FMR – Log10 Slope	0.50
Body Weight (g)	-0.48	Body Weight (g)	-0.47
		Amphibian Concentration - PSA	-0.2
		Invertebrate Concentration - PSA	-0.1

Table K.2-16

Exposure Model Results for American Bittern Exposed to TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	**Maximum**	**Mean**	**Median**	**10th Percentile**	**50th Percentile**	**90th Percentile**	**10th Percentile**	**50th Percentile**	**90th Percentile**	**10th Percentile**	**50th Percentile**	**90th Percentile**
PSA	145	867	372	363	271	363	485	40.9	51.17	63.6	409	505	642

Table K.2-17

Dietary Studies for the Small-Footed Myotis

Dietary Composition		Myotis Diet	Tree Swallow Diet
		Belwood and Fenton 1976; Kurta 1995	Blancher et al. 1987; Blancher and McNicol 1991; Nichols et al. 1995
Order	**Common Name**	**% of Prey Items**	**% of Prey Items**
Diptera	flies, midges, mosquitoes	76.4	45.9 – 58.2
Homoptera	cicadas, hoppers, aphids	trace	8.7 – 26
Ephemeroptera	mayflies	trace	<5 – 15.6
Odonata	dragonflies and damselflies	--	5.0 – 14.2
Hemiptera	bugs	trace	<1 – 17.9
Trichoptera	caddisflies	18.2	--
Lepidoptera	butterflies and moths	4.2	--
Coleoptera	beetles	1.2	4
Hymenoptera	sawflies, ants, wasps	trace	1

Table K.2-18

Input Variables Used in the Monte Carlo Exposure Analyses for Total Daily Intake of tPCBs and TEQ by Small-Footed Myotis Inhabiting the Housatonic River

Variable	Distribution	Small-Footed Myotis Parameters
Body Weight (g)	Normal	x = 6 s = 0.7
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 0.812 s = 0.196
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.162
Gross Energy (kcal/g)		
Invertebrates	Point Estimate	1.6
Assimilation Efficiency (unitless)		
Invertebrates	Point Estimate	0.720
Proportion Diet (unitless)		
Invertebrates	Point Estimate	1.0
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-19

Input Variables Used in the RiskCalc Probability Bounds Analyses for Total Daily Intake of tPCBs and TEQ by Small-Footed Myotis Inhabiting the Housatonic River

Variable	Distribution	Small-Footed Myotis Parameters
Body Weight (g)	Normal	x = 6 s = 0.7
Free Metabolic Rate (FMR; kcal/g bw/d)	FMR = a BW^b	
a = FMR - Log_{10} Slope	Normal	x = 0.812 s = 0.196
b = FMR - Power Term	Lognormal	x = 0.681 s = 0.162
Gross Energy (kcal/g)		
Invertebrates	Point Estimate	1.6
Assimilation Efficiency (unitless)		
Invertebrates	Point Estimate	0.720
Proportion Diet (unitless)		
Invertebrates	Point Estimate	1.0
Proportion Foraging Time in PSA (unitless)	Point Estimate	1.0

Table K.2-20

Summary Statistics and Exposure Analysis Input Values for Concentrations of tPCBs in Prey of Small-Footed Myotis

Organism	Location	Sample Size	Percentile[a] (mg/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs (mg/kg bw/d)
			Min	25th	50th	75th	Max			
Invertebrates (Tree swallow gut contents)	PSA	12	1.54	3.47	7.10	11.57	31.9	UCL > Max: No	Point Estimate UCL = 22.0	Distribution-Free Range LCL = 6.40 UCL = 22.0

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming non-detected values were equal to half the detection limit and co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] Co-elution of congeners was not an issue when estimating concentrations of tPCBs in prey tissues. Also, all tPCBs concentrations in prey tissues were above the detection limit. When the maximum concentration was less than the UCL calculated using the Land H-statistic, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis. Otherwise, the UCL was used in the Monte Carlo analysis and the LCL and UCL were used in the probability bounds analysis. When the number of samples is below 5, the maximum concentration was used in the Monte Carlo analysis and the minimum and maximum concentrations were used in the probability bounds analysis.

Table K.2-21

Summary Statistics and Exposure Analysis Input Values for Concentrations of TEQ in Prey of Small-Footed Myotis

Organism	Location	Sample Size	Percentile[a] (ng/kg)					Decision Criteria[b]	Monte Carlo Inputs	Probability Bounds Inputs
			Min	25th	50th	75th	Max			
Invertebrates (Tree swallow gut contents)	PSA	6	234	314	564	800	1,440	UCL > Max: Yes	Point Estimate Max = 1,500	Distribution-Free Range Min = 59.3 Max = 2,820

Min=minimum; Max=maximum; LCL=lower confidence limit on the mean; UCL=upper confidence limit on the mean; ND=not detected (i.e., concentration below the detection limit); DL=detection limit; N/A=not applicable

[a] Calculated assuming co-eluted congener concentrations were equal to doublet or triplet concentrations.

[b] When the maximum concentration was less than the UCL calculated using the Land H-statistic, the minimum from non-detected values equal to zero and maximum concentrations from non-detected values equal to the DL were used in the probability bounds analysis. In the Monte Carlo analysis the value was based on the maximum values in dataset assuming ND=0, ND=1/2DL, and ND=DL.

Table K.2-22

Exposure Model Results for Small-Footed Myotis Exposed to tPCBs (mg/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Reach 5	2.05	96.0	16.7	14.5	7.17	14.5	28.8	1.96	4.05	8.09	7.67	15.0	32.3

Table K.2-23

Sensitivity Analysis Results for Small-Footed Myotis Exposed to PCBs

tPCBs		TEQ	
Variable	Sensitivity	Variable	Sensitivity
FMR – Log10 Slope	0.83	FMR – Log10 Slope	0.82
FMR – Power Term	0.53	FMR – Power Term	0.52
Body Weight (g)	- 0.06	Body Weight (g)	-0.04
		Dietary Concentration – (ng/g)	-0.01

Table K.2-24

Exposure Model Results for Small-Footed Myotis Exposed to TEQ (ng/kg bw/d)

Location	Monte Carlo Analysis							Probability Bounds Analysis					
								Lower Probability Bound			Upper Probability Bound		
	Minimum	Maximum	Mean	Median	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile	10th Percentile	50th Percentile	90th Percentile
Reach 5	61.4	7,020	1,130	936	381	936	2,120	18.8	38.1	74.8	985	1,910	3,900

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Aroclor 1248									
Screech owl	Adult	3	420 d (minus 60 d)		7.1			NOAEL: No significant effects on number of eggs laid, number of eggs hatched, or number of young fledged	McLane and Hughes 1980
American kestrel	Adult	3						5% reduction in eggshell thickness	Lowe and Stendell 1991
PCB 77									
American kestrel	Embryo		Single inj	0.688 (egg)		0.05		Calculated LD_{50}	Hoffman et al. 1996
American kestrel	Embryo		Single inj	0.100 (egg)		0.05		Increased mortality (23%), decreased hatching success (38%)	Hoffman et al. 1998
American kestrel	Embryo		18 d		0.316 (egg)	0.05	0.016 (egg)	LD_{50} Embryo mortality	Hoffman et al. 1998
American kestrel	Embryo		20 d		0.100 (egg)	0.05	0.005 (egg)	LOAEL: Embryo mortality	Hoffman et al. 1998
American kestrel	Embryo				0.68			LD_{50} Embryo mortality	Hoffman et al. 1995, 1998
PCB 126									
American kestrel	1 d/M-F	0.25	10 d	25		0.1	2.5	Significant decrease in bone length, decreased spleen wt., decrease in bw, but not significant	Hoffman et al. 1996
American kestrel	1 d/M-F	0.05	10 d	0.500		0.1	0.05	Bone growth	Hoffman et al. 1996

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
American kestrel	Embryo		Single inj	0.065		0.1	0.0065	Calculated LD_{50}	Hoffman et al. 1996
American kestrel	Embryo		20 d		0.065 (egg)	0.1	0.0007 (egg)	LD_{50} Embryo mortality	Hoffman et al. 1998
American kestrel	Embryo		20 d		0.233 (egg)	0.1	0.023 (egg)	LOAEL: Embryo mortality	Hoffman et al. 1998
American kestrel	Embryo		20 d		0.023 (egg)	0.1	0.002 (egg)	NOAEL: Embryo mortality	Hoffman et al. 1998
Common tern	Embryo		Single inj	0.104		0.1	0.0104	Calculated LD_{50}	Hoffman et al. 1998
Common tern	Embryo		Single inj	0.044		0.1	0.0044	Reduced hatching success (60%)	Hoffman et al. 1998
Common tern	Embryo		18 d		0.104 (egg)	0.1	0.010 (egg)	LD_{50} Embryo mortality	Hoffman et al. 1998
Common tern	Embryo		18 d		0.044 (egg)	0.1	0.004 (egg)	LOAEL: Embryo mortality	Hoffman et al. 1998
Double-crested cormorant	Embryo		Single inj	0.158 (egg)		0.1	0.0158 (egg)	Calculated LD_{50}	Powell et al. 1997
Double-crested cormorant	Embryo		Single inj	0.177 (egg)		0.1	0.0177 (egg)	Calculated LD_{50}	Powell et al. 1998
Double-crested cormorant	Embryo		21 d		0.158 (egg)	0.1	0.016 (egg)	Calculated LD_{50}	Powell et al. 1998
Double-crested cormorant	Embryo		21 d		0.400 (egg)	0.1	0.040 (egg)	LOAEL: Embryo mortality	Powell et al. 1997
Double-crested cormorant	Embryo		21 d		0.200 (egg)	0.1	0.020 (egg)	NOAEL: Embryo mortality	Powell et al. 1997

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
TCDD									
Heron	Embryo		Single inj	0.000021 (egg)				ED_{50} Brain deformities	Henshel 1998
Cormorant	Embryo		Single inj	0.000034 (egg)				ED_{50} Brain deformities	Henshel 1998
Cormorant	Embryo		Single inj	0.00004 (egg)				Increased mortality (50%)	Powell et al. 1997
Double-crested Cormorant	Embryo		21 d		0.004 (egg)	1	0.004 (egg)	LOAEL: Embryo mortality	Powell et al. 1997
Double-crested Cormorant	Embryo		21 d		0.001 (egg)	1	0.001 (egg)	NOAEL: Embryo mortality	Powell et al. 1997
Great blue heron	Embryo		Embryonic day 9 to hatch		0.002 (egg)	1	0.002 (egg)	No effect on growth rate of chicks	Janz and Bellward 1996
tPCBs									
Cormorant	Adult/F	21 (egg)	Trophic					Decreased egg hatchability compared to 10 mg/kg egg site	Dirksen et al. 1995
Heron	Adult/M-F	0.00021 (TCDD egg)	Trophic					Complete colony hatching failure; 0.000009-0.000066 mg/kg had no effects	Elliott et al. 1989

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Bald eagle	Embryo		Trophic		45			Reduced reproductive success	Clark et al. 1998
Bald eagle	Embryo		Trophic		8.7			LOAEL: 10% reduction in reproductive success	Wiemeyer et al. 1984[a], 1993
Bald eagle	Embryo		Trophic		5.5			NOAEL: Reproductive success	Wiemeyer et al. 1984[a], 1993
Bald eagle	Embryo		Trophic		33.2 - 64 (in yolk sac)			No effect on hatching success	Elliott et al. 1996
Bald eagle	Embryo		Trophic		26.4			No effect on reproductive output	Donaldson et al. 1999
Double-crested cormorant	Embryo		Trophic		23.8			Reduced hatching and fledgling success	Weseloh et al. 1983
Caspian tern	Embryo		Trophic		4.2 - 18			Increasing rate of embryo deformities	Yamashita et al. 1993
Foster's tern	Embryo	0.176 (estimated)	Trophic					Wasting syndrome	Harris et al. 1993
Foster's tern	Embryo		Trophic		22.2			LOAEL: Hatching success, growth	Kubiak et al. 1989; Hoffman et al. 1987
Foster's tern	Embryo		Trophic		7.3			NOAEL: Hatching and fledgling success	Harris et al. 1993
Foster's tern	Embryo		Trophic		4.5			NOAEL: Hatching success, growth	Kubiak et al. 1989; Hoffman et al. 1987

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Common tern	Embryo		Trophic		7.6			LOAEL: Hatching success	Hoffman et al. 1993
Common tern	Embryo		Trophic		7.6			LOAEL: Hatching success	Becker et al. 1993
Common tern	Embryo		Trophic		6.3			NOAEL: Hatching success	Becker et al. 1993
Common tern	Embryo		Trophic		4.7			NOAEL: Hatching success	Hoffman et al. 1993
Black-crowned night heron	Embryo		Trophic		4.1			Adverse effect on growth	Hoffman et al. 1986
Red-breasted merganser	Embryo		Trophic		19.3			No effect on hatching success, duckling production	Heinz et al. 1983
Great blue heron	Embryo		Trophic		2.01			No effect on reproductive success	Halbrook et al. 1999
Bald eagle	Embryo		Trophic		< 4.5			Normal 5-year reproductive rate - 0.83	Wiemeyer et al. 1984[a]
Bald eagle	Embryo		Trophic		4.5 - 10			Reduced 5-year reproductive rate - 0.60	Wiemeyer et al. 1984[a]
Bald eagle	Embryo		Trophic		10 - 19			Reduced 5-year reproductive rate - 0.54	Wiemeyer et al. 1984[a]
Bald eagle	Embryo		Trophic		19 - 33			Reduced 5-year reproductive rate - 0.28	Wiemeyer et al. 1984[a]

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Bald eagle	Embryo		Trophic		> 33			Reduced 5-year reproductive rate - 0.20	Wiemeyer et al. 1984[a]
Bald eagle	Embryo		Trophic		1.3			NOAEL: Reproductive success	Wiemeyer et al. 1984[a]
Bald eagle	Embryo		Trophic		7.2			LOAEL: Reproductive success	Wiemeyer et al. 1984[a]
Bald eagle	Embryo				13			LOAEL: Reproductive success	Bosveld and Van den Berg 1994
Bald eagle	Embryo				45.0		0.000705[b]	0.14 reproductive rate over 9 nesting attempts 12.9% eggshell thinning[c]	Clark et al. 1998
Bald eagle	Adult	0.62 - 1.95 (in prey fish species)						0.14 reproductive rate over 9 nesting attempts 12.9% eggshell thinning	Clark et al. 1998
Bald eagle	Nestling	0.183 (in blood plasma)						Malformations (primarily bill deformities)	Bowerman et al. 1994
Bald eagle	Embryo				4			LOAEL: Reproductive success	Ludwig et al. 1993
Black-crowned night heron	Embryo				4.1			Lower embryotic weight (15% lower than control)	Hoffman et al. 1993
Great blue heron	Embryo				15			Decreased reproductive success	Laporte 1982

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Great blue heron	Embryo				4.9			No observable effects	Custer et al. 1998
Aroclor 1248:1254:1260 mix									
American kestrel	Adult/M-F	7	100 d	34 (egg)				2nd generation increased brood mortality; decreased female clutch size	Fernie et al. 2001a
American kestrel	Adult/M-F	7	100 d	34 (egg)				Decreased number of fertile eggs, hatchlings and fledglings per breeding pair	Fernie et al. 2001b
American kestrel	Adult	7			34.1			Significant delay in egg laying, smaller clutch size, greater number of infertile eggs, reduced fledgling success (55% in dosed and 93.3% in control)	Fernie et al. 2001b
PCB-126, -153, others									
Common tern	21d/M-F	0.00125 (TEQ in diet)						EROD induction 10 times that of controls	Bosveld et al. 2000
TEQ									
Bald eagle	Embryo				0.0002			NOAEL: Reproductive success	Elliott et al. 1996

Table K.3-1

Toxicity of tPCBs and TEQ in T&E Species (Continued)

Species	Age/Sex	Dose mg/kg bw/d	Exposure (d)	Total Dose mg/kg bw	Egg Concentration mg/kg egg	TEF	Dose Dioxin Equivalents (TEQ) mg/kg bw/d	Endpoint	Reference
Osprey	Embryo				0.00014			NOAEL: Reproductive success	Woodford et al. 1998
Great blue heron	Embryo				0.00002			NOAEL: Chick growth and deformities	Hart et al. 1991
Great blue heron	Embryo				0.000245			LOAEL: Chick growth and deformities	Hart et al. 1991

LOAEL = lowest observed adverse effect level; NOAEL = no observed adverse effect level; TEF = toxic equivalency factor.

[a]PCBs correlated with DDE, and it is difficult to separate effects of PCBs from DDE and other COCs.

[b]Calculated using WHO TEFs for birds (van Leeuwen 1997) and does not include dioxins or furans.

[c]Below threshold associated with population decline (>15 - 18%) (Blus 1996).

Table K.4-1

Summary of Exceedance Probabilities for T&E Species Total Daily Intakes from the Housatonic River Study Area

Location	Probability of Threshold Exceedance (%)											
	tPCBs						TEQ					
	LPB		*FOMC*		*UPB*		*LPB*		*FOMC*		*UPB*	
	LD	UD	LD	UD	LD	UD	LD	UD	LD	UD	LD	UD
Bald Eagle												
Southern PSA	100%	--	100%	--	100%	--	0%	--	0%	--	0%	--
American Bittern												
Reach 5A	100%	100%	100%	100%	100%	100%						
Reach 5B	100%	100%	100%	100%	100%	100%						
Reach 5C	100%	100%	100%	100%	100%	100%						
Reach 5D&6	100%	100%	100%	100%	100%	100%						
Entire PSA							100%	0%	100%	0%	100%	0%
Small-Footed Myotis												
Entire PSA	69%	30%	99.1%	96.3%	100%	98%	0%	0%	99.2%	92.9%	100%	100%

LPB = Lower Probability Bound
FOMC = First Order Monte Carlo
UPB = Upper Probability Bound
LD = Lower Toxicity Dose (10% effect)
UD = Upper Toxicity Dose (20% effect)

Table K.4-2

Summary of Qualitative Risk Statements for T&E Species from the Housatonic River Study Area

Bird / Location	Qualitative Risk Statements			
	tPCBs		TEQ	
	Risk Category[a]	Risk Range[b]	Risk Category[a]	Risk Range[b]
Bald Eagle				
Southern PSA	High	High	Low	Low
American Bittern				
Reach 5A	High	High	Intermediate	Intermediate
Reach 5B	High	High	Intermediate	Intermediate
Reach 5C	High	High	Intermediate	Intermediate
Reach 5D and 6	High	High	Intermediate	Intermediate
Small-Footed Myotis				
Reaches 5 and 6	High	Intermediate - High	High	Low - High

[a]Risk category is the risk level based on First Order Monte Carlo (FOMC).

[b]Risk range is the range of risk encompassed by the upper and lower probability bounds (UPB and LPB).

Table K.4-3

Weighting of Measurement Endpoints for T&E Species Weight-of-Evidence Evaluation

Attributes	Modeled Exposure and Effects for Bald Eagles Exposed to tPCBs and TEQ	Modeled Exposure and Effects for American Bitterns Exposed to tPCBs and TEQ	Modeled Exposure and Effects for Small-Footed Myotis Exposed to tPCBs and TEQ	Rationale
I. Relationship Between Measurement and Assessment Endpoints				
1. Degree of Association	H (tPCBs) M/H (TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Exposure models were species-specific, but effects metrics for bald eagle (body burden), American bitterns, and small-footed myotis were derived from studies of surrogate species. Effects metrics for bald eagle eggs were species-specific for tPCBs.
2. Stressor/Response	M/H (tPCBs) M (TEQ)	M (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Exposure modeling was species- and stressor-specific. Effects metrics for representative species were available only for bald eagle eggs exposed to tPCBs. A dose-response curve was used for small-footed myotis, rather than thresholds.
3. Utility of Measure	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Modeled exposure and effects procedures used were standardized and widely accepted; the primary limitation was lack of species-specific effects data, except for bald eagle eggs exposed to tPCBs.
II. Data Quality				
4. Data Quality	H (tPCBs and TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	The field surveys were performed according to well-defined and documented protocols. The low numbers of individuals observed, and the inability to confirm identification of the Myotis without handling, limited the ability to observe site-specific effects. The DQOs for the sampling analysis and tissue samples were met for the tissue residue data used in the exposure analysis for both tPCBs and TEQ.

Table K.4-3

Weighting of Measurement Endpoints for T&E Species Weight-of-Evidence Evaluation (Continued)

Attributes	Modeled Exposure and Effects for Bald Eagles Exposed to tPCBs and TEQ	Modeled Exposure and Effects for American Bitterns Exposed to tPCBs and TEQ	Modeled Exposure and Effects for Small-Footed Myotis Exposed to tPCBs and TEQ	Rationale
III. Study Design				
5. Site Specificity	M (tPCBs and TEQ)	M (tPCBs and TEQ)	M (tPCBs and TEQ)	Biological tissue data used in exposure models were site specific, and other exposure parameters were representative of site conditions. However, effects measures were not site specific.
6. Sensitivity	M/H (tPCBs) H (TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	Modeled exposure and effects directly assessed exposure-response relationship. Laboratory studies from which effects data were derived were stressor-specific.
7. Spatial Representativeness	H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	H (tPCBs and TEQ)	Modeled exposures relied on tissue data collected throughout the study area and areas of actual exposure. American bittern exposure based on small mammals trapped in unfavorable foraging area.
8. Temporal Representativeness	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Modeled exposure and effects lines of evidence spanned critical life stages and, in general, tissue data used were collected when exposure was expected to be high.
9. Quantitative Measure	H (tPCBs and TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	Probabilistic exposure and effects modeling were highly quantitative and propagated uncertainty associated with modeling procedures.
10. Standard Method	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Generally accepted exposure and effects modeling procedures were followed, but probability bounds analysis is a relatively new technique for propagating uncertainty.
Overall Endpoint Value	**M/H (tPCBs and TEQ)**	**M/H (tPCBs and TEQ)**	**M/H (tPCBs and TEQ)**	**---**

M = Moderate
H = High

Table K.4-4

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	High
Modeled exposure and effects, American Bittern	Moderate/High	Yes	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High

Table K.4-5

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	Intermediate
Modeled exposure and effects, American Bittern	Moderate/High	Undetermined	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High

Table K.4-6

Risk Analysis Summary for T&E Species Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of T&E species

Harm/Magnitude	Weighting Factors (increasing confidence of weight)				
	Low	**Low/ Moderate**	**Moderate**	**Moderate/ High**	**High**
Yes/High				BE, AB	
Yes/Intermediate					
Yes/Low					
Undetermined/High				SFM	
Undetermined/Intermediate					
Undetermined/Low					
No/Low					
No/Intermediate					
No/High					

BE = Bald eagle

AB = American bittern

SFM = Small-footed myotis

Table K.4-7

Risk Analysis Summary for T&E Species Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of T&E species

Harm/Magnitude	Weighting Factors (increasing confidence of weight)				
	Low	**Low/Moderate**	**Moderate**	**Moderate/High**	**High**
Yes/High					
Yes/Intermediate				BE	
Yes/Low					
Undetermined/High				AB, SFM	
Undetermined/Intermediate					
Undetermined/Low					
No/Low					
No/Intermediate					
No/High					

BE = Bald eagle

AB = American bittern

SFM = Small-footed myotis

Table K.4-8

Comparison of Representative and Other T&E Species in the Housatonic River PSA

Representative Species	Other Species	Size	Residency	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
Bald Eagle		5350 g	Breeding season	Forage 0.5 to 8 km from nest	Predominantly fish, with birds, mammals, reptiles, and crustaceans occasionally taken.	Occurs in proximity of large bodies of water.	DeGraaf and Yamasaki 2001; Bowerman et al. 1995
	Osprey	1600 g	7 months	Forage 1.7 to 15 km from nest	Predominantly fish, with small mammals, reptiles, and crustaceans occasionally taken.	Habitat preferences and feeding habits similar to bald eagle.	DeGraaf and Yamasaki 2001; Ehrlich et al. 1992
American Bittern		707 g	Breeding season	315 acres	Amphibians, crustaceans, insects, small fish, small snakes,	Similarities in habitat and diet to American bittern.	DeGraaf and Yamasaki 2001; Dechant et al. 2001;
	Least Bittern	80g	Breeding season	Not territorial	Small fishes, crustaceans (mainly crayfish), frogs, snakes, salamanders, leeches, slugs, occasionally small mammals (shrews & mice).	Similarities in habitat and diet to American bittern.	DeGraaf and Yamasaki 2001
	King rail	360 g	Breeding season	Unknown	Large crustaceans and aquatic insects, also amphibians, mollusks, seeds of marsh plants.	Similarities in habitat and diet to American bittern, though it consumes less fish. Threatened in Massachusetts and Endangered in Connecticut.	DeGraaf and Yamasaki 2001

Table K.4-8

Comparison of Representative and Other T&E Species in the Housatonic River PSA (Continued)

Representative Species	Other Species	Size	Residency	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
	Virginia rail	85 g	Breeding season		Invertebrates, worms, slugs, snails, crustaceans, small fish, seeds of marsh plants, berries and occasionally frogs and small snakes.	Similar in habitat and diet to American bittern.	DeGraaf and Yamasaki, 2001
American Bittern (cont.)	Green Heron	210g	Breeding season		Small fishes, crustaceans, mollusks, terrestrial and aquatic insects, reptiles, amphibians, spiders, leeches.	Similar foraging habitat and diet to American bittern	DeGraaf and Yamasaki 2001
	Great blue heron	2400g	Breeding season	Forage up to16 km from nest	Primarily fish, but also aquatic and terrestrial insects, amphibians, reptiles, crustaceans and occasionally small birds and mammals in shallow marshes, wet meadows and upland fields.	Similar habitat and some similarities in diet to American bittern, but fish make up a greater portion of diet.	DeGraaf and Yamasaki 2001
	Sora	75g	Breeding season		Seeds of wetland plants, aquatic insects, mollusks, and crustaceans. In fall consumes wild and cultivated grains.	Similar habitat and some similarities in diet to American bittern, but seeds and grains important in diet and fish and amphibians lacking.	DeGraaf and Yamasaki 2001
	Pied-billed grebe	450g	Breeding season	Home range approx. 300-ft radius around nest.	Aquatic insects, small fish, snails, aquatic worms, crayfish, amphibians, leeches, minor amounts of aquatic vegetation.	Similar habitat and diet to American bittern.	DeGraaf and Yamasaki 2001

Table K.4-8

Comparison of Representative and Other T&E Species in the Housatonic River PSA (Continued)

Representative Species	Other Species	Size	Residency	Foraging/ Home Range (ha)	Diet	Life History/ Miscellaneous	References
Small-Footed Myotis		6 g	Year round	Unknown	Midges, flies, beetles, caddisflies, mayflies, bugs, leafhoppers, moths		DeGraaf and Yamasaki 2001; Kurta 1995
	Indiana bat	6 – 11 g	Year round	52 – 95 ha	Leafhoppers, beetles, flies, caddisflies, mayflies, moths, ichneumons	Similar in habitat and diet to small-footed myotis.	DeGraaf and Yamasaki 2001; Kurta 1995
Small-Footed Myotis (cont.)	Little brown bat	6 – 12 g	Year round	Unknown	Midges, mayflies, mosquitoes, moths, beetles, bugs, caddisflies, stoneflies and other insects.	Similar in habitat and diet to small-footed myotis.	DeGraaf and Yamasaki 2001; Kurta 1995
	Northern myotis	5 – 8 g	Year round	Unknown	Bugs, moths, flies, leafhoppers, stoneflies, caddisflies, weevils, bark lice	Similarities in habitat and diet to small-footed myotis, but northern myotis feeds mostly in forests.	DeGraaf and Yamasaki 2001; Kurta 1995

APPENDIX K

ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF THREATENED AND ENDANGERED SPECIES

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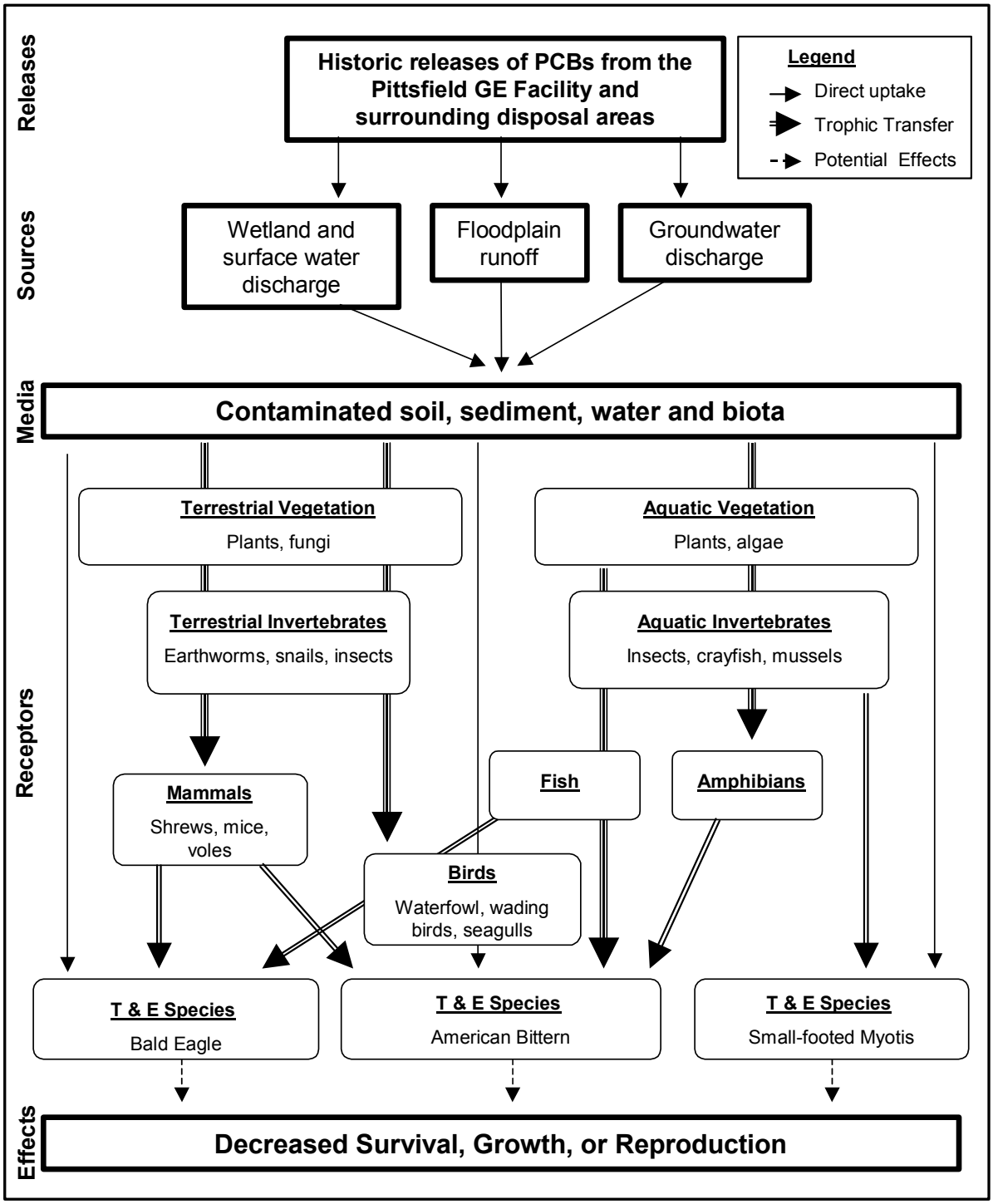

Figure K.1-1 Conceptual Model Diagram: Exposure Pathways for Threatened and Endangered Species Exposed to COCs in the Housatonic Primary Study Area

Exposure

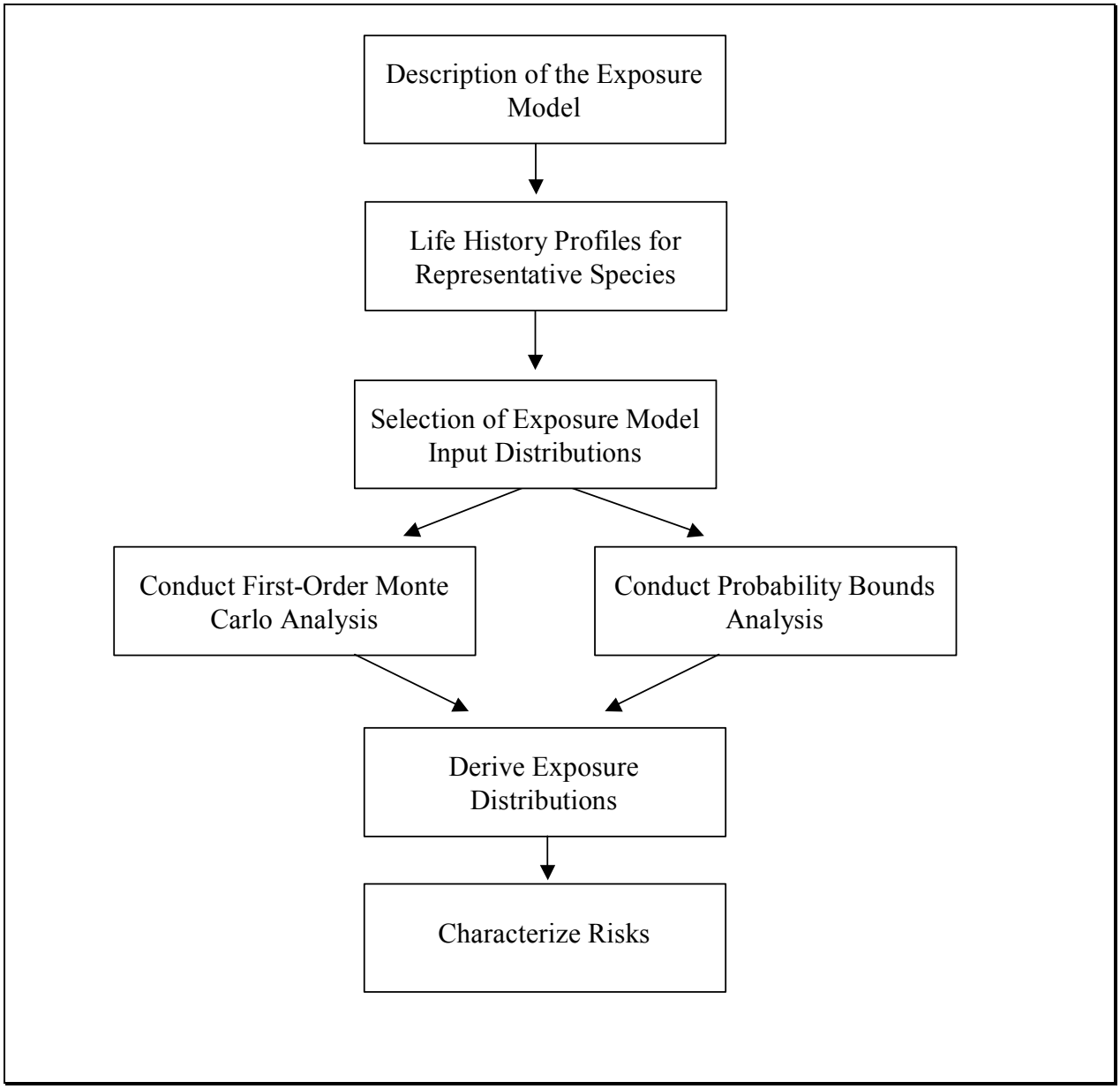

Figure K.1-2 Overview of Approach Used to Assess Modeled Exposure of Threatened and Endangered Species to COCs in the Housatonic River Primary Study Area

Effects

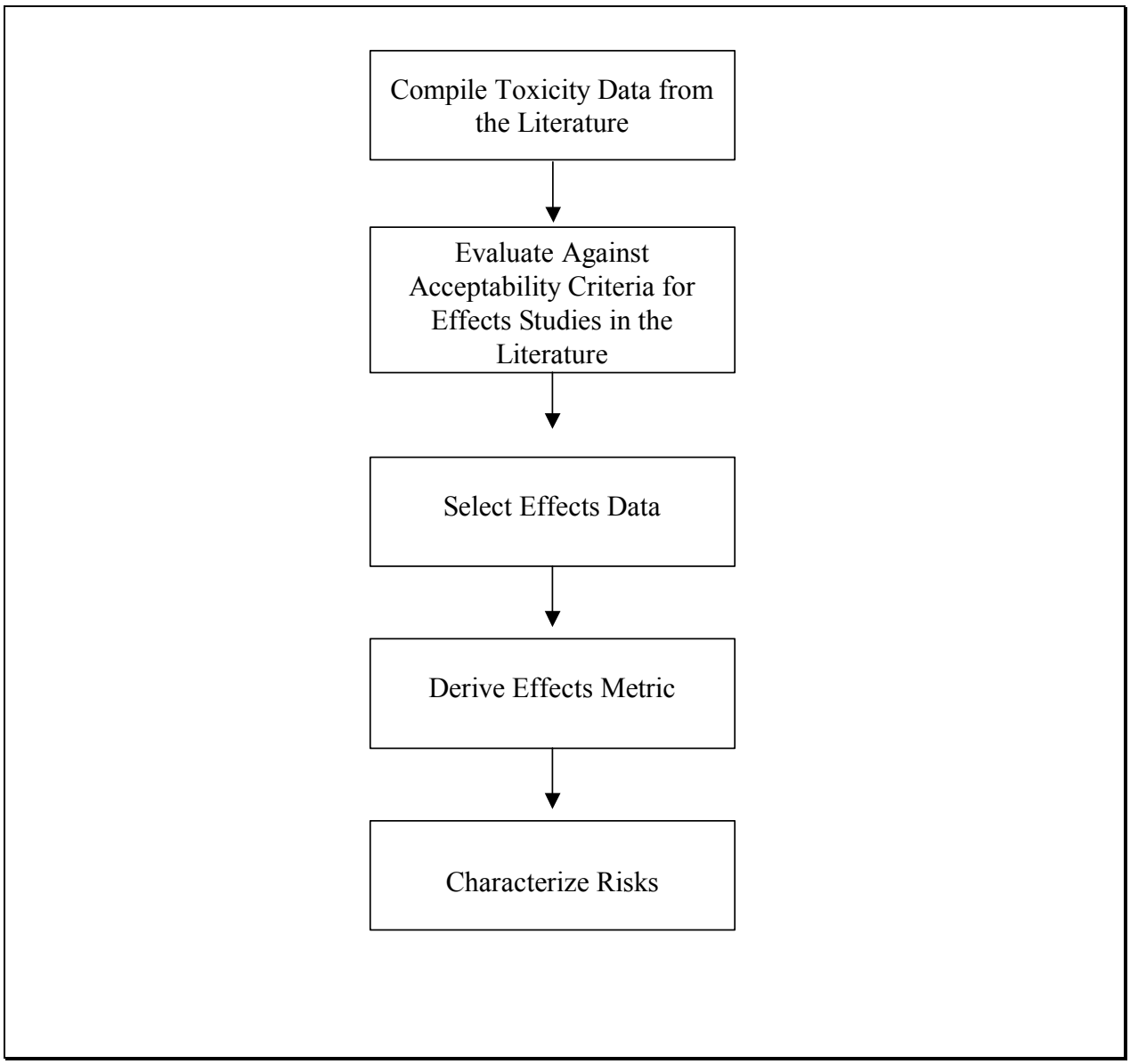

Figure K.1-3 Overview of Approach Used to Assess the Modeled Effects of COCs to Threatened and Endangered Species in the Housatonic River Primary Study Area

Risk Characterization

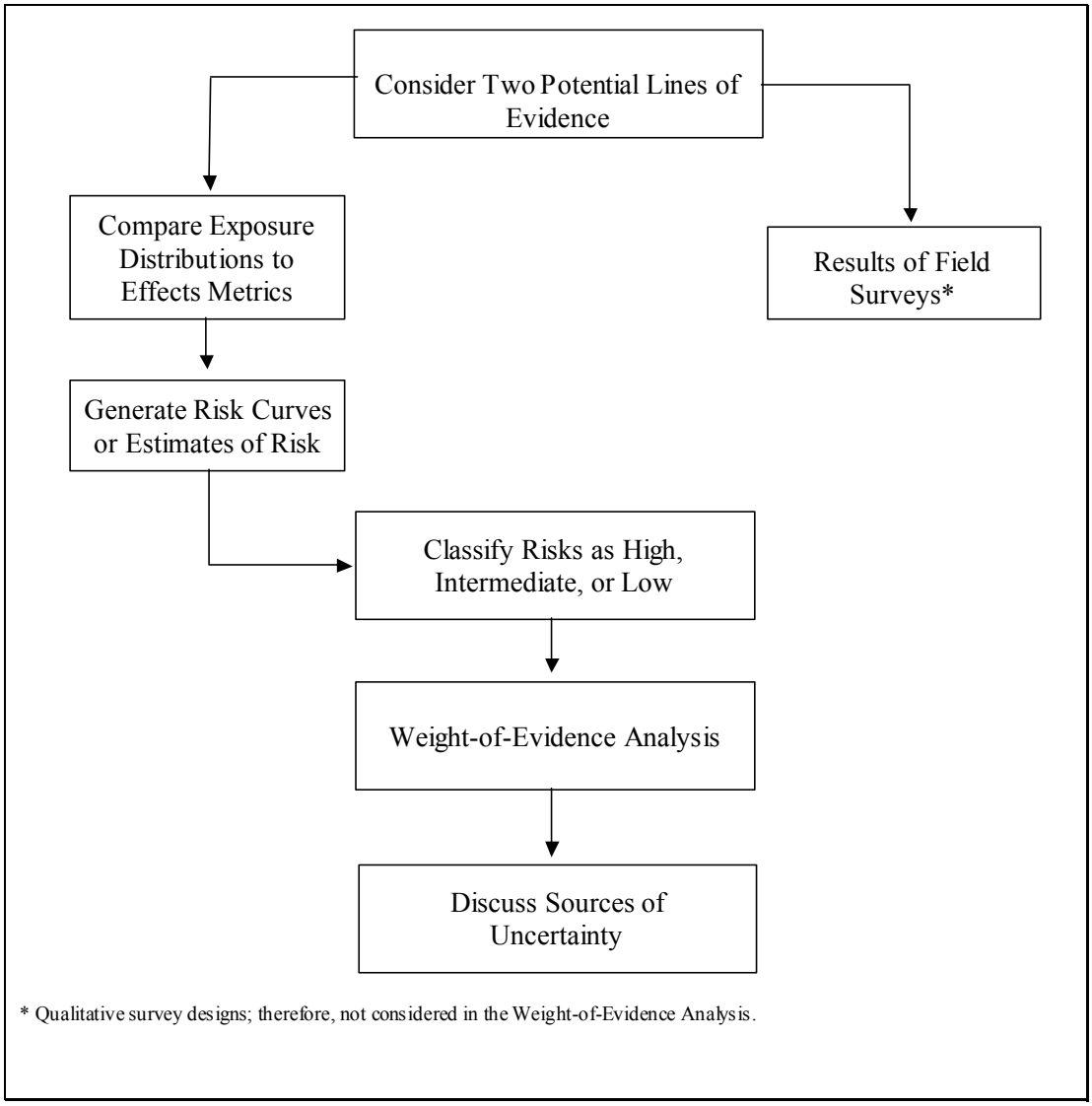

Figure K.1-4 Approach Used to Characterize the Risks of COCs to Threatened and Endangered Species in the Housatonic River Primary Study Area

Photo by: Karol Worden

Figure K.1-5 Bald Eagle (*Haliaeetus leucocephalus*)

Photo by: Scott Robinson

Figure K.1-6 American Bittern (*Botaurus lentiginosus*)

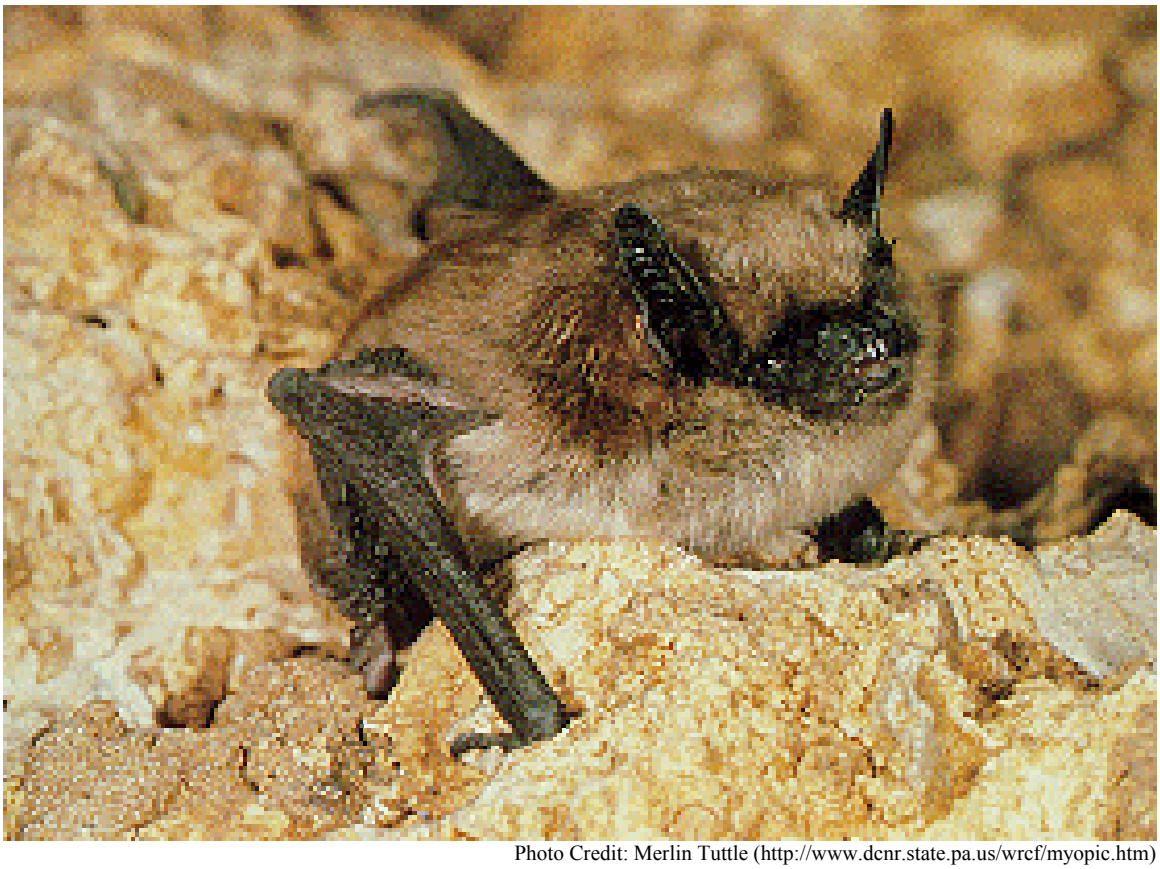

Photo Credit: Merlin Tuttle (http://www.dcnr.state.pa.us/wrcf/myopic.htm)

Figure K.1-7 Small-Footed Myotis (*Myotis leibii*)

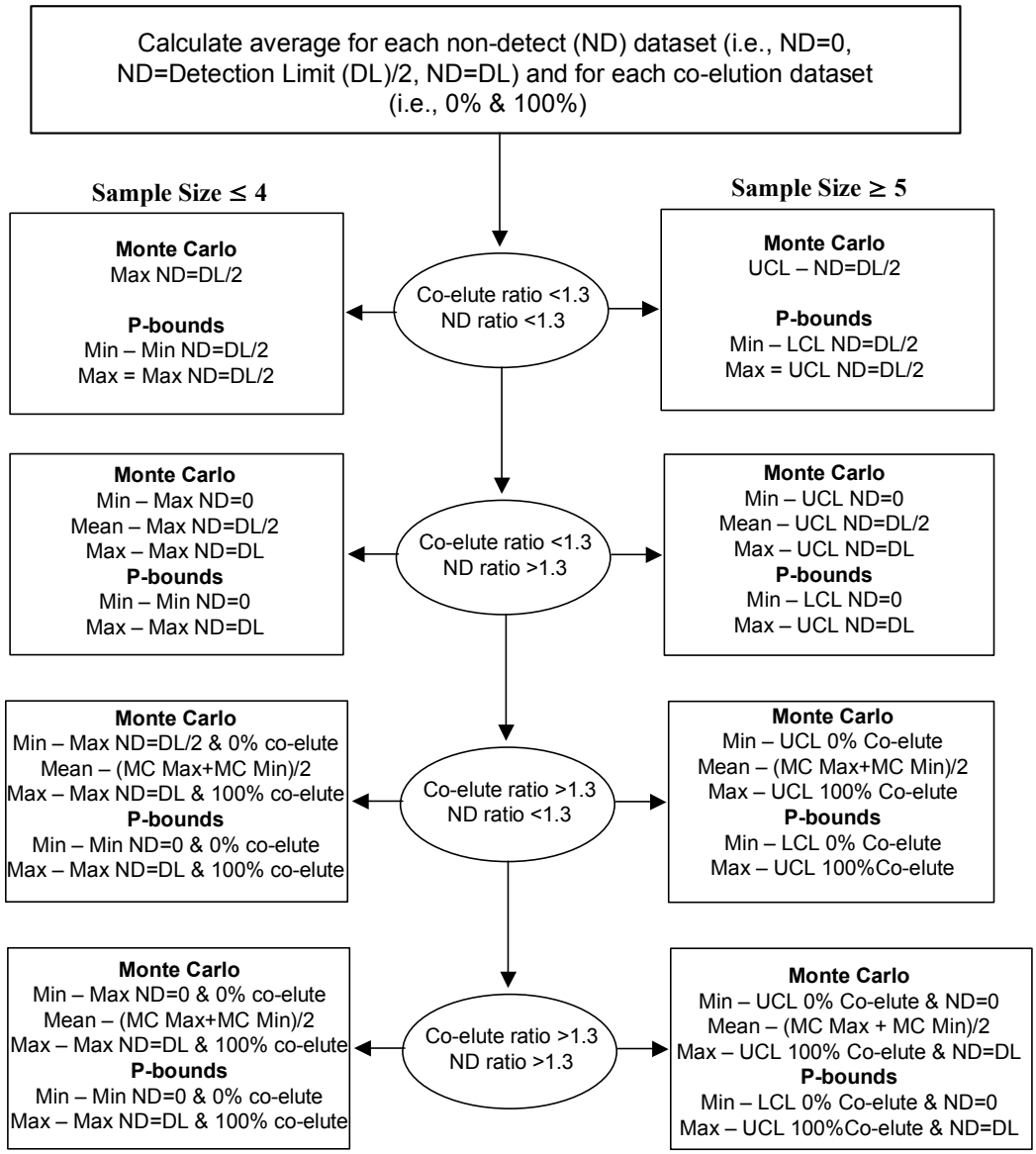

Figure K.2-1 Non-Detect Co-Elution Decision Tree

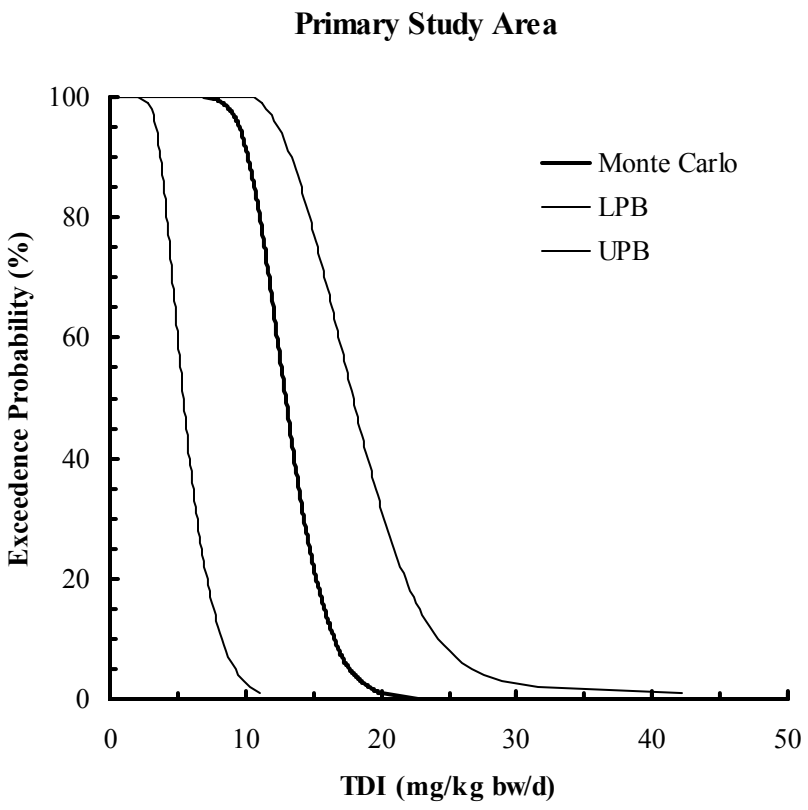

Figure K.2-2 Total Daily Intake (TDI) of tPCBs by Bald Eagles in the Housatonic River Primary Study Area

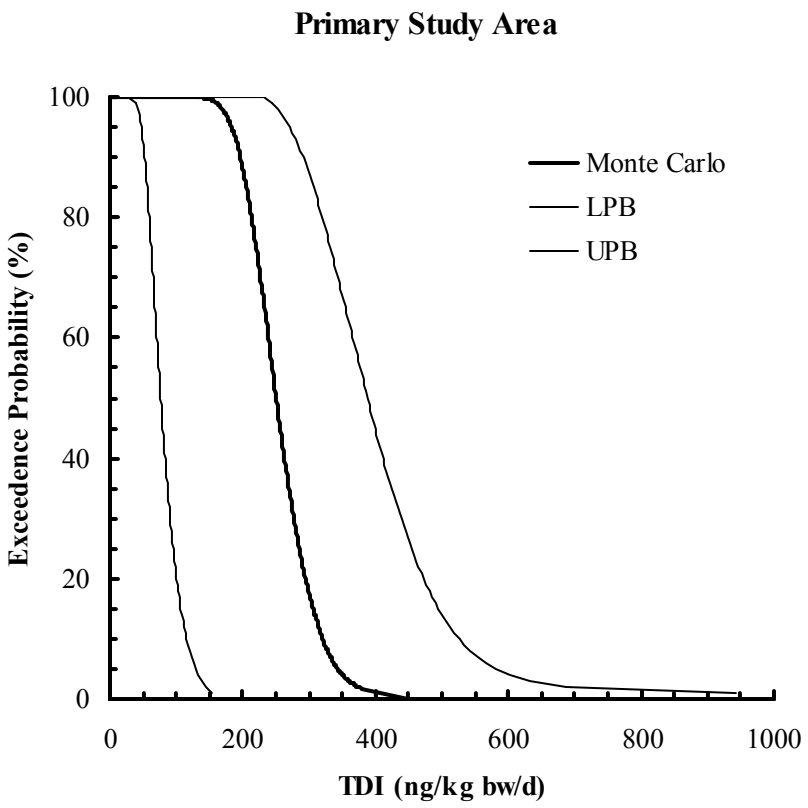

Figure K.2-3 Total Daily Intake (TDI) of TEQ by Bald Eagles in the Housatonic River Primary Study Area

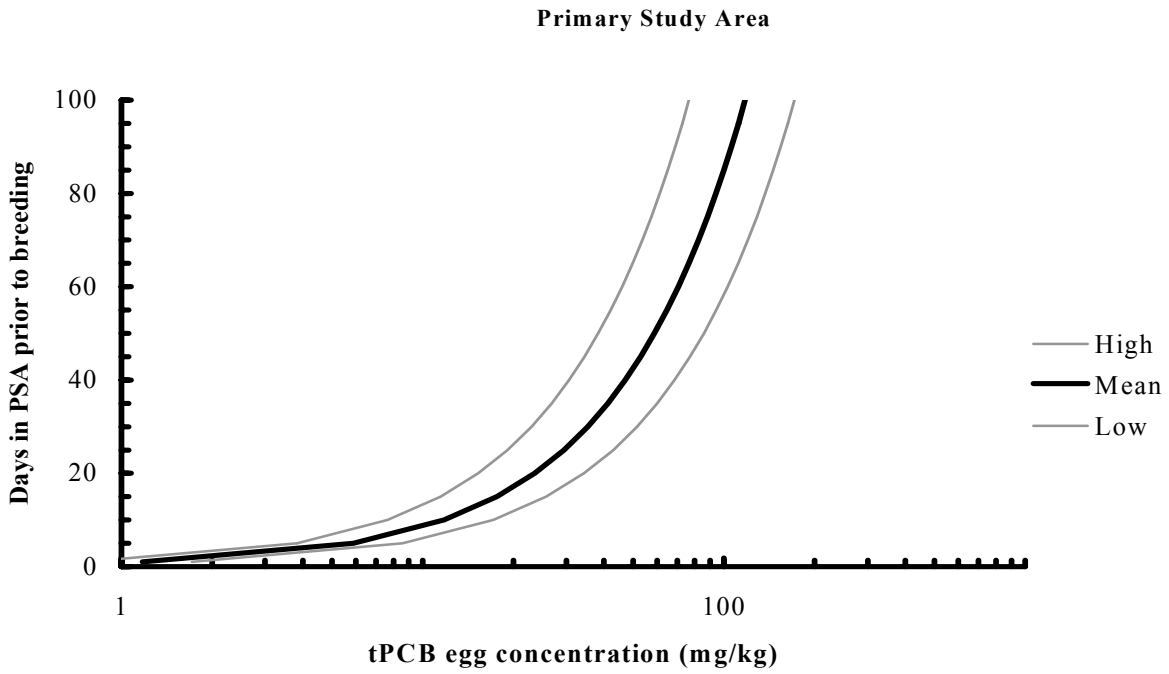

Figure K.2-4 Bald Eagle Egg Exposure to PCBs in the Housatonic River Primary Study Area

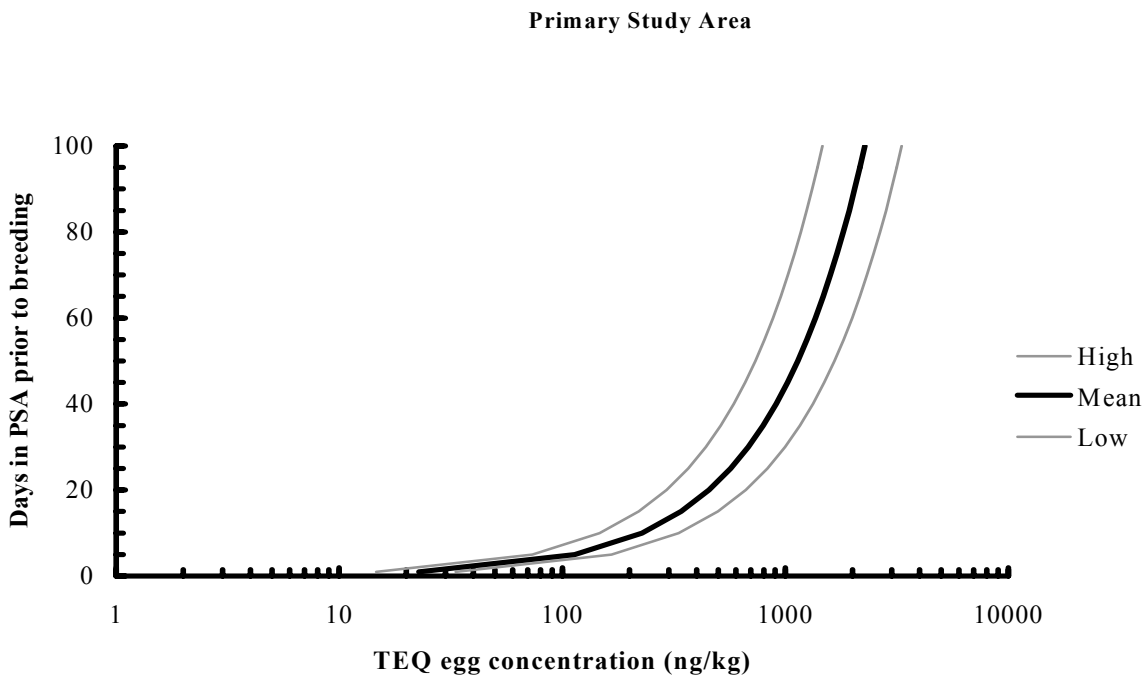

Figure K.2-5 Bald Eagle Egg Exposure to TEQ in the Housatonic River Primary Study Area

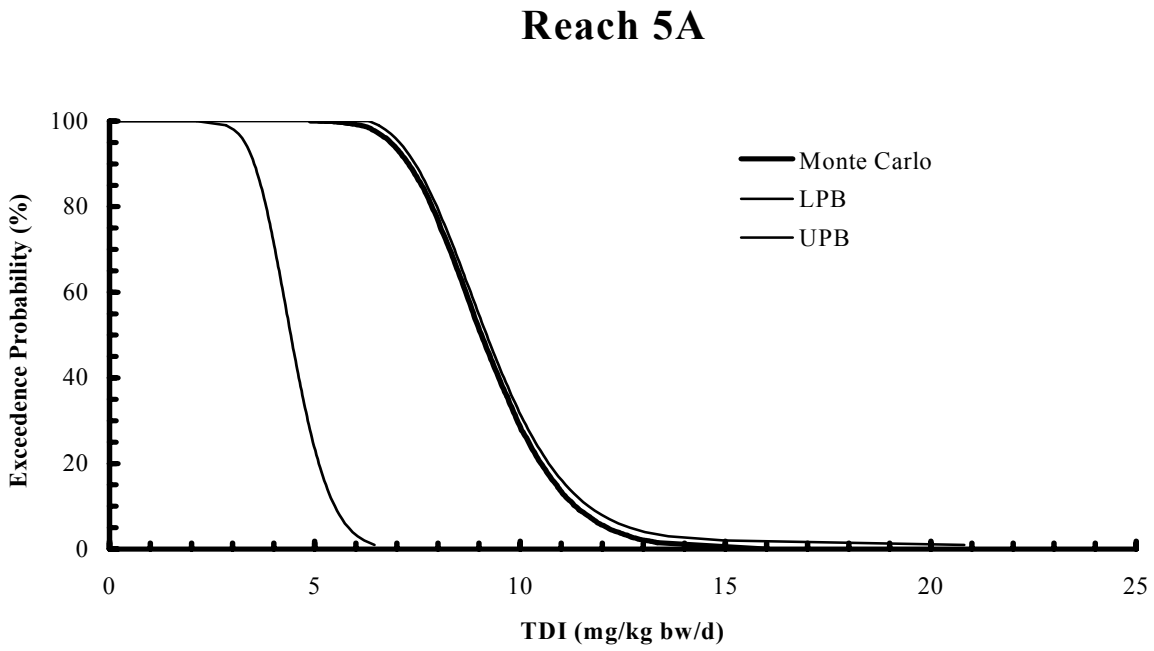

Figure K.2-6 Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5A of the Housatonic River Primary Study Area

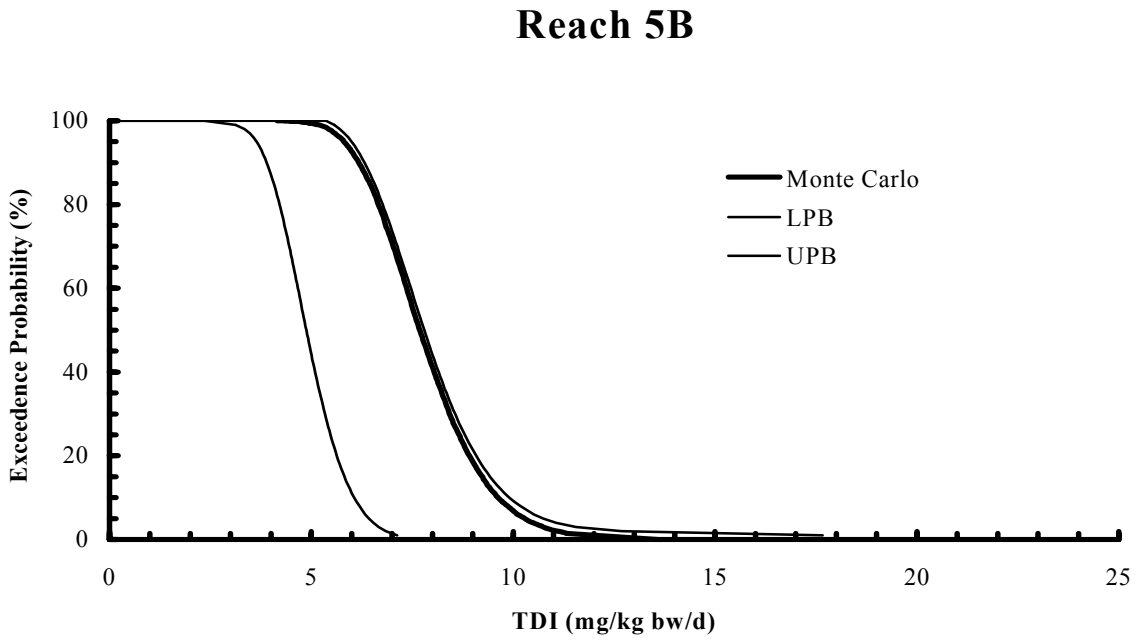

Figure K.2-7 Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5B of the Housatonic River Primary Study Area

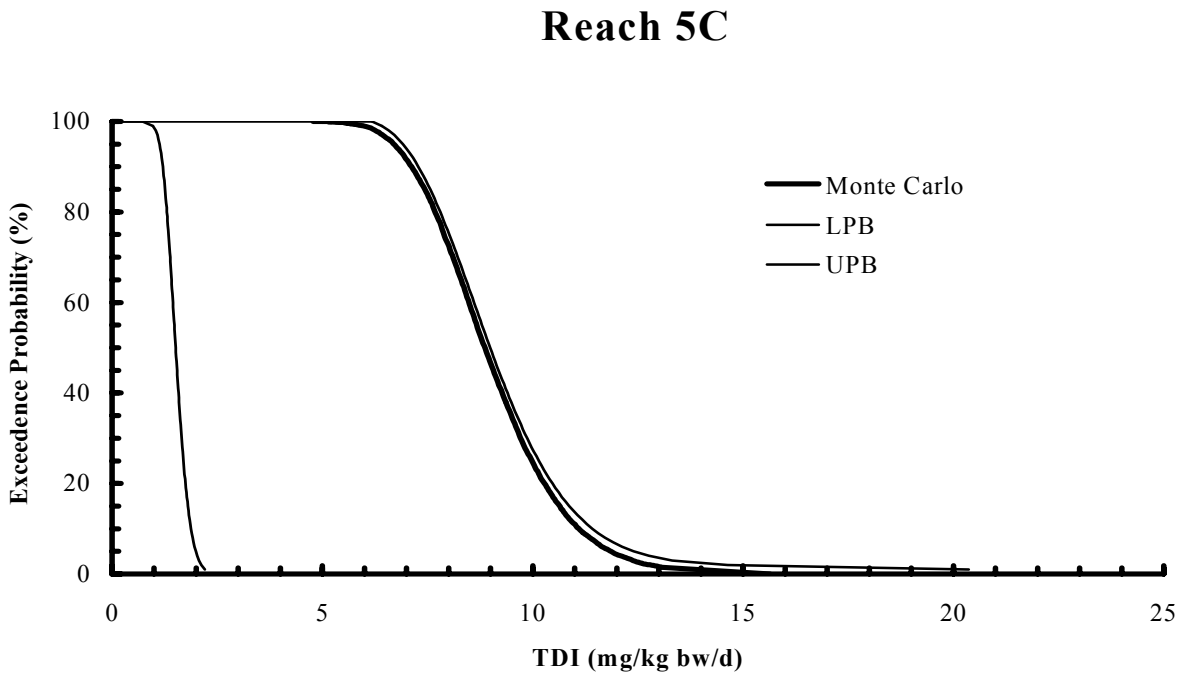

Figure K.2-8 Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5C of the Housatonic River Primary Study Area

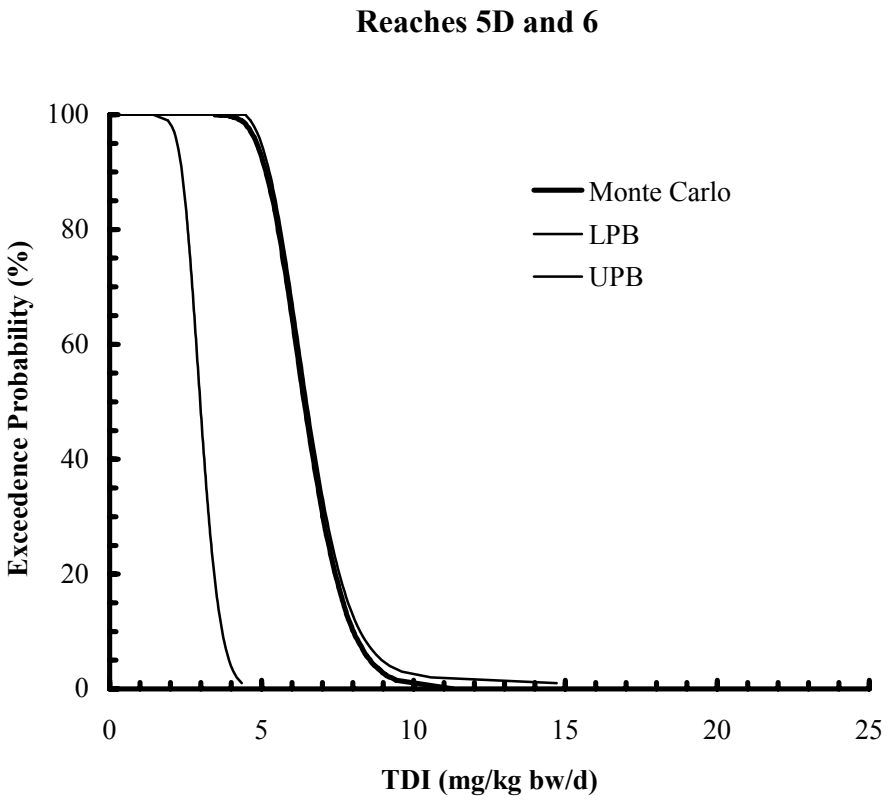

Figure K.2-9 Total Daily Intake (TDI) of tPCBs by American Bittern in Reaches 5D and 6 of the Housatonic River Primary Study Area

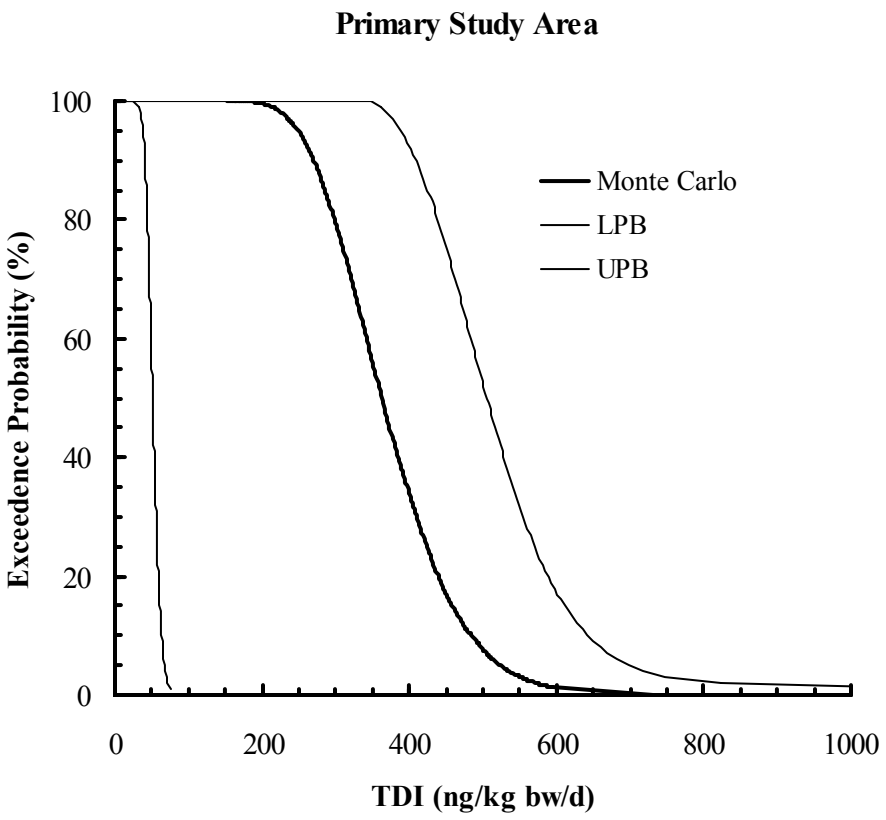

Figure K.2-10 Total Daily Intake (TDI) of TEQ by American Bittern in the Housatonic River Primary Study Area

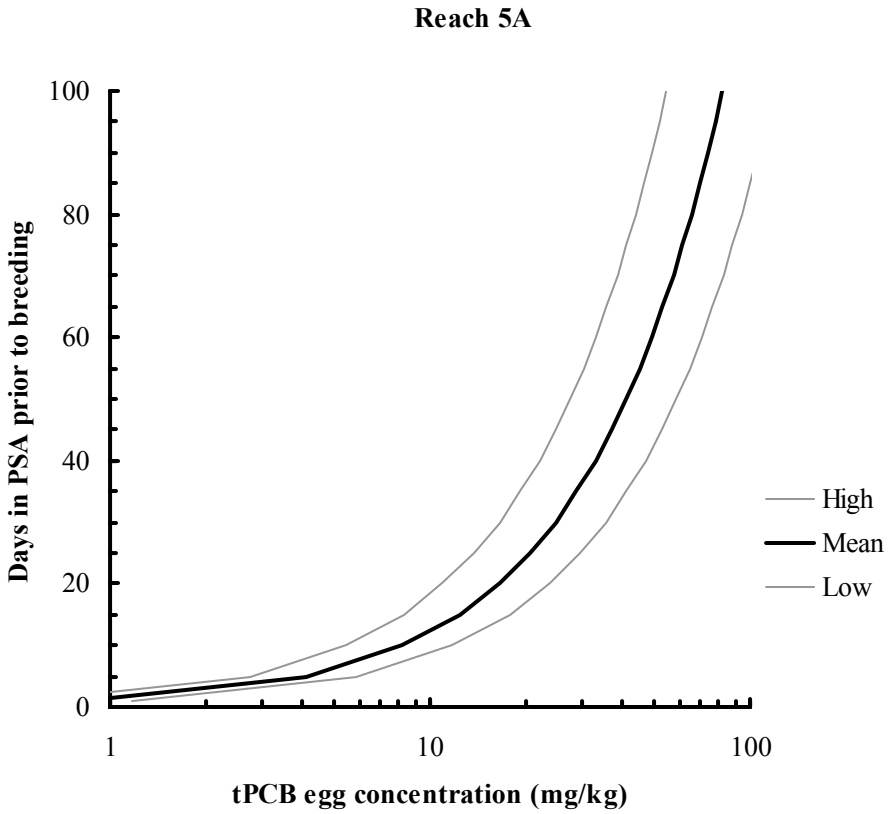

Figure K.2-11 American Bittern Egg Exposure to tPCBs in Reach 5A of the Housatonic River Primary Study Area

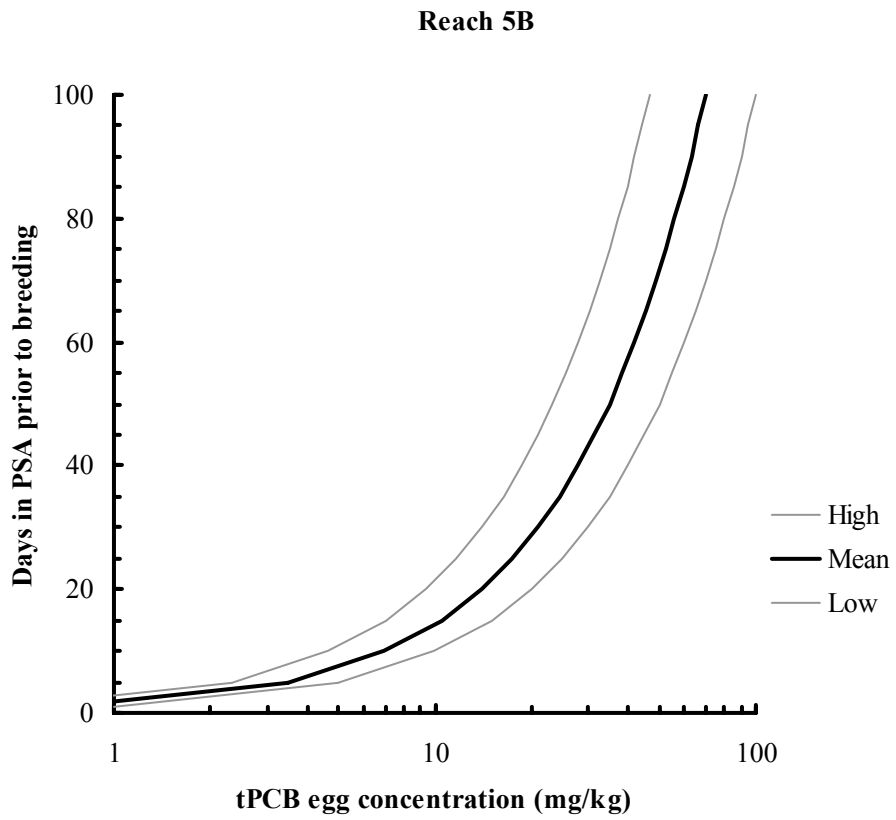

Figure K.2-12 American Bittern Egg Exposure to tPCBs in Reach 5B of the Housatonic River Primary Study Area

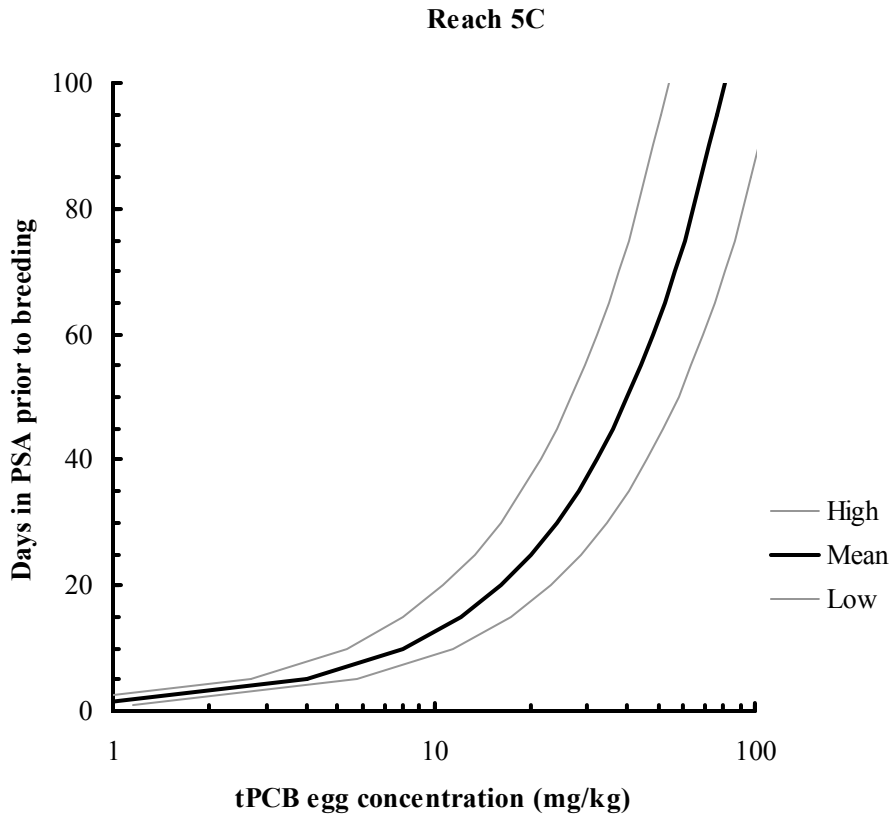

Figure K.2-13 American Bittern Egg Exposure to tPCBs in Reach 5C of the Housatonic River Primary Study Area

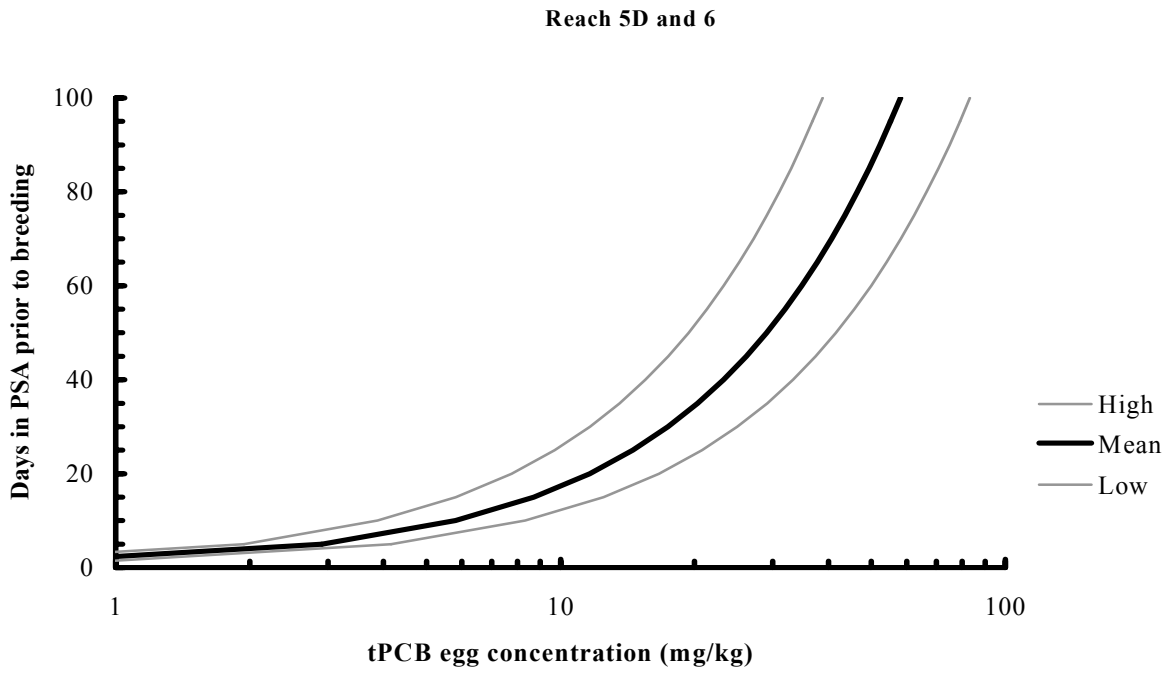

Figure K.2-14 American Bittern Egg Exposure to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area

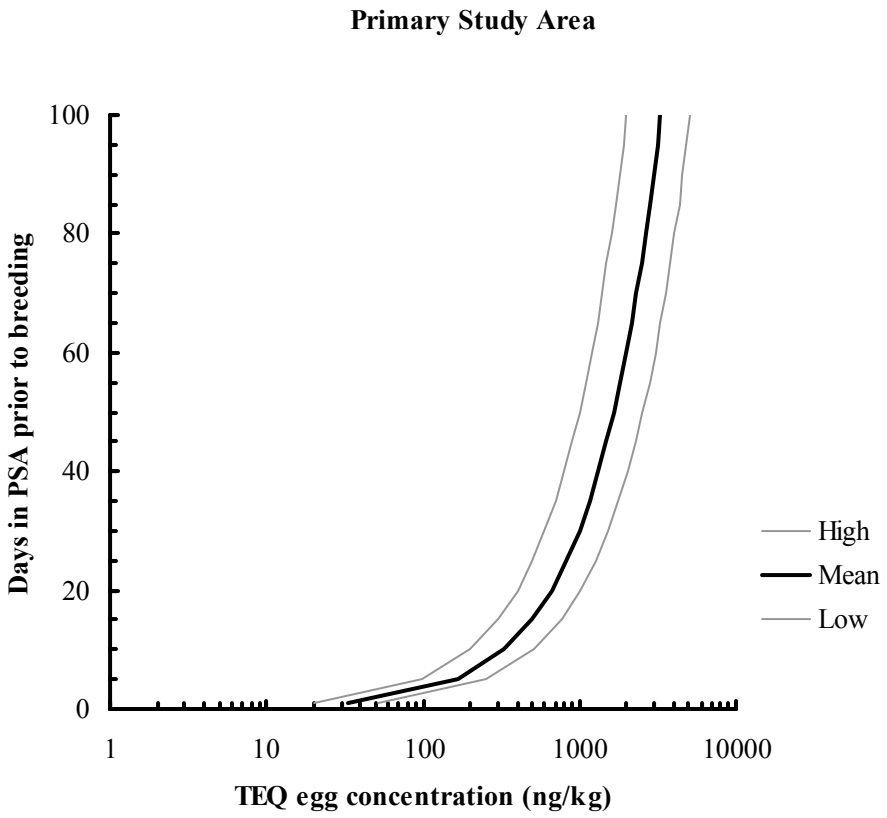

Figure K.2-15 American Bittern Egg Exposure to TEQ in Reaches 5 and 6 of the Housatonic River Primary Study Area

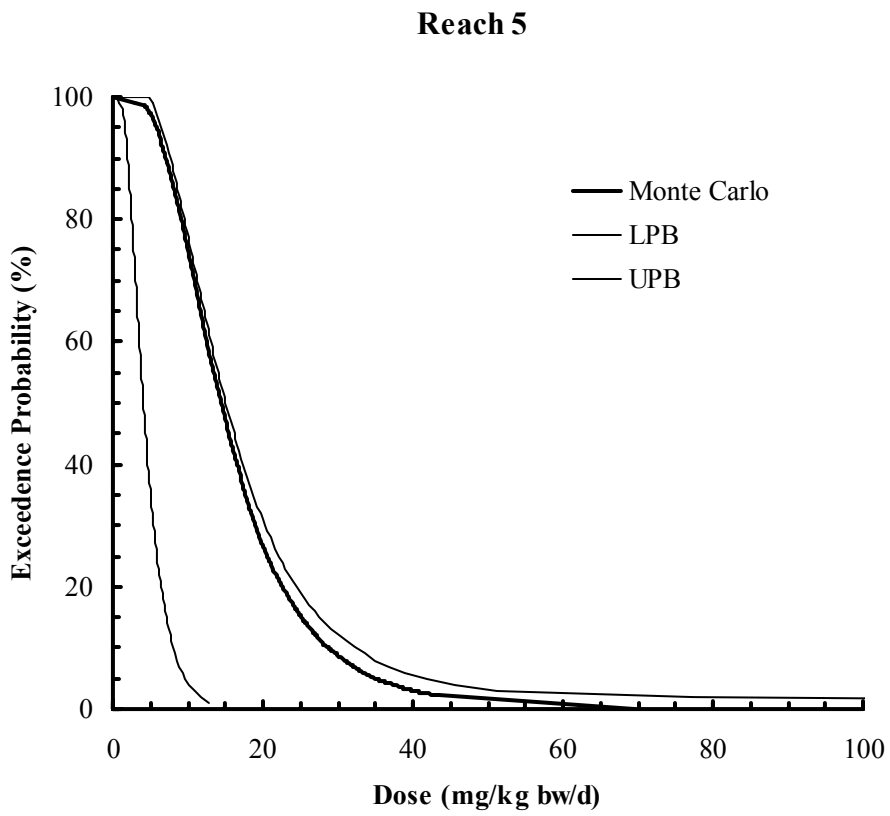

Figure K.2-16 Total Daily Intake (TDI) of tPCBs by Small-Footed Myotis in Reach 5 of the Housatonic River Primary Study Area

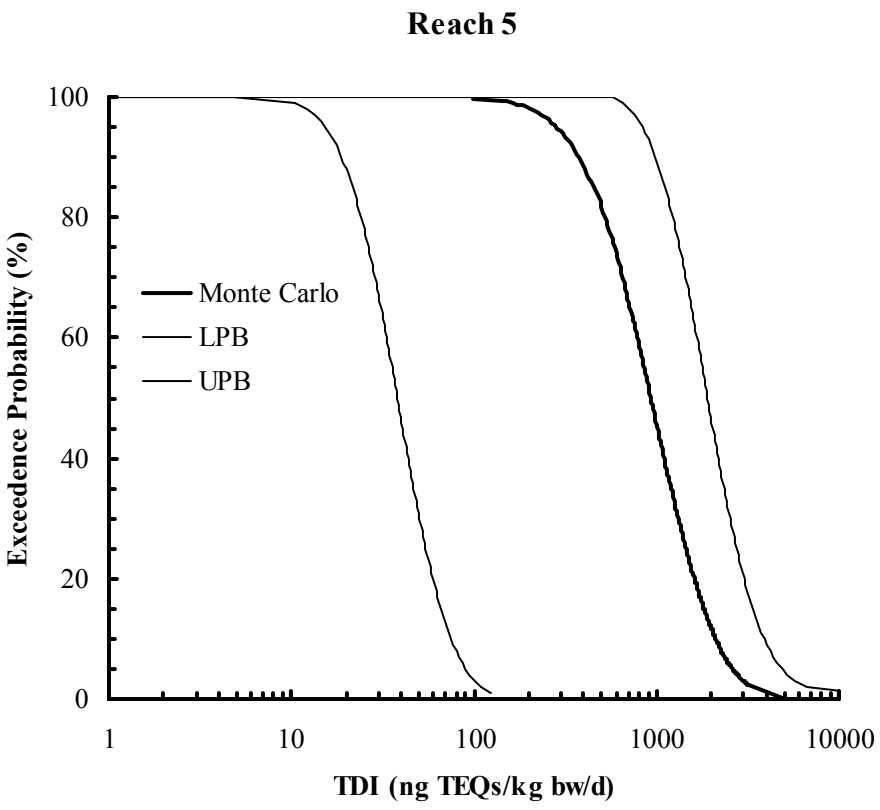

Figure K.2-17 Total Daily Intake (TDI) of TEQ by Small-Footed Myotis in Reach 5 of the Housatonic River Primary Study Area

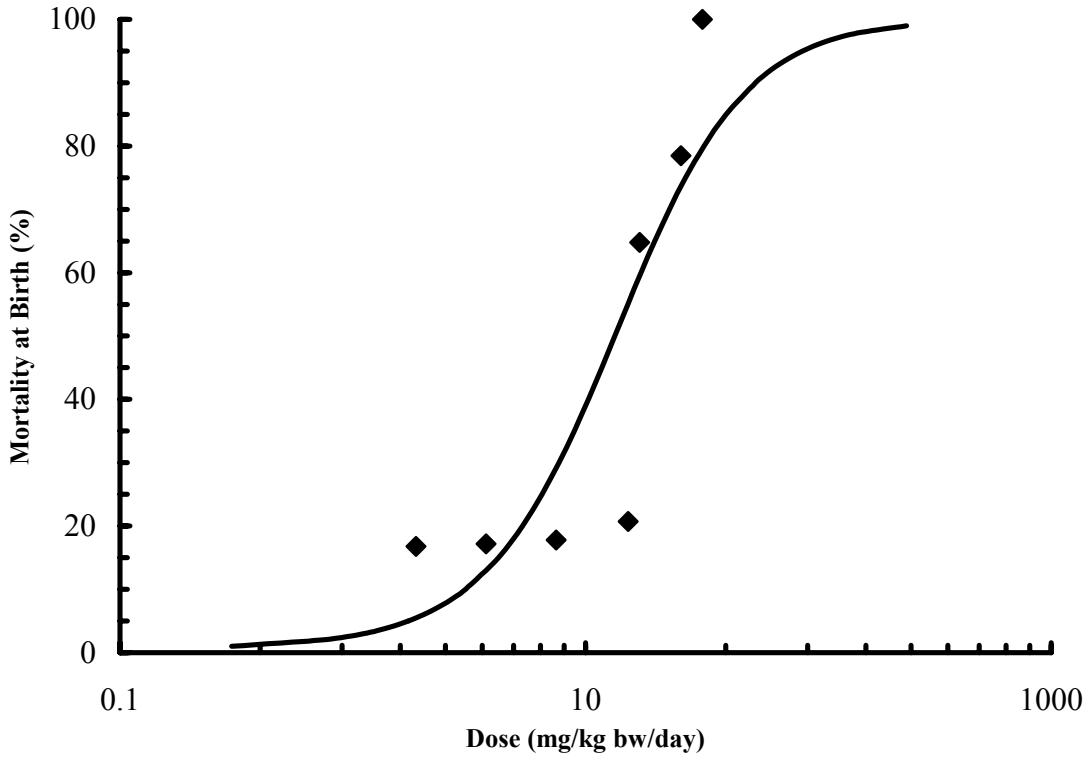

Figure K.3-1 Dose-Response Curve for Effects of tPCBs on Mortality at Birth of Rats

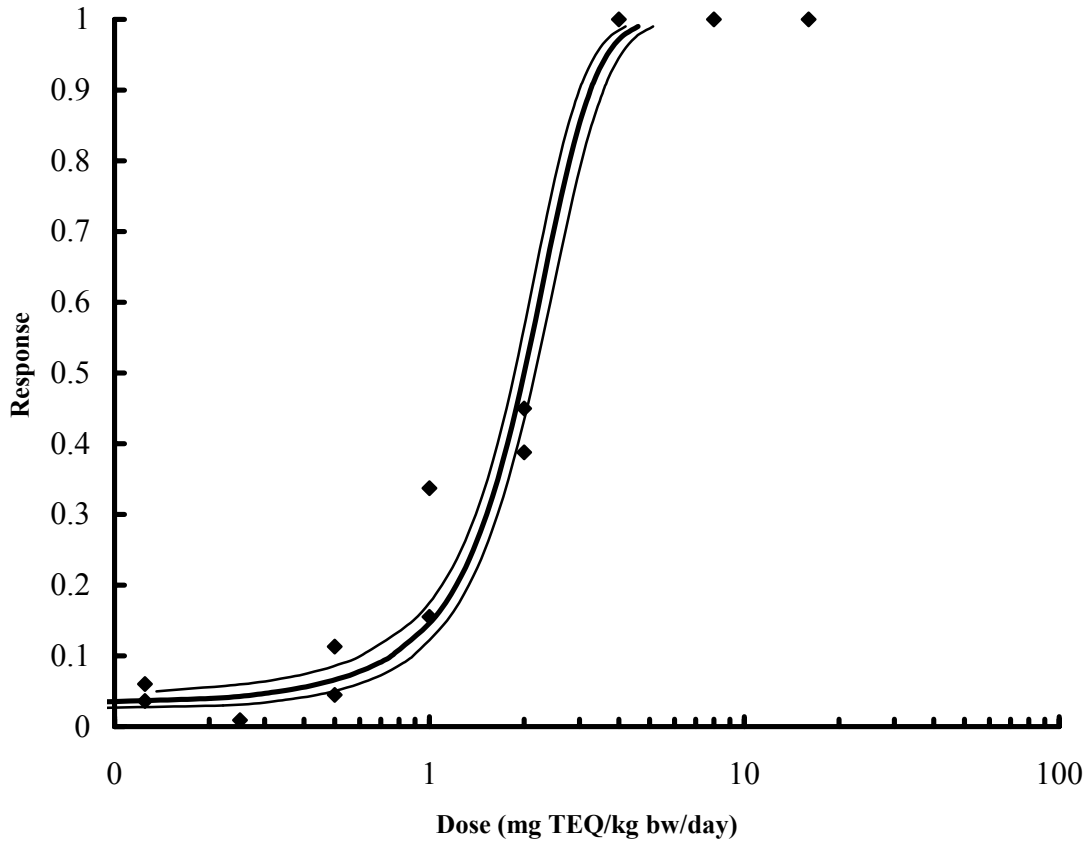

Figure K.3-2 Dose-Response Curve for Effects of TEQ on Mortality at Birth of Rats

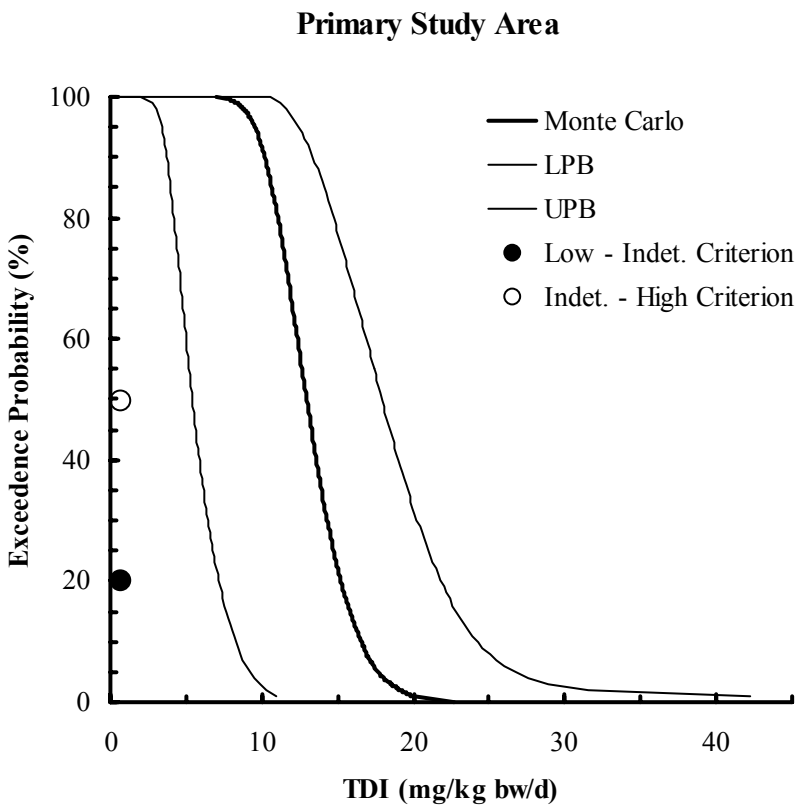

Figure K.4-1 Risk Curves for Bald Eagles Exposed to tPCBs in the Housatonic River Primary Study Area

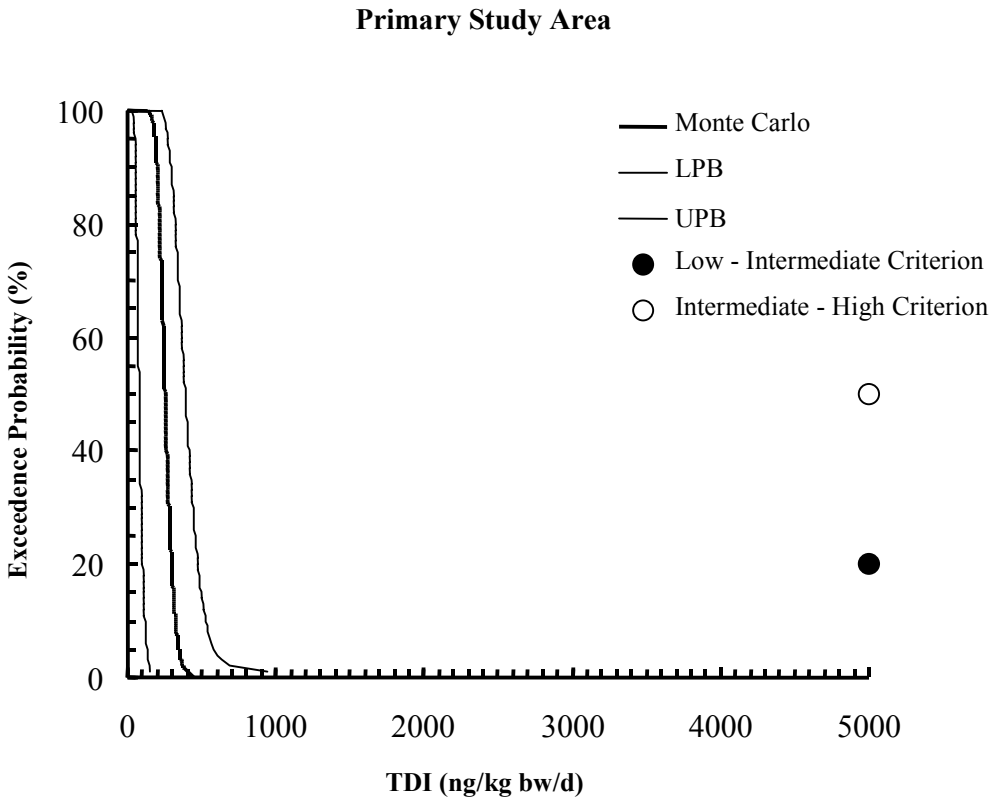

Figure K.4-2 Risk Curves for Bald Eagles Exposed to TEQ in the Housatonic River Primary Study Area

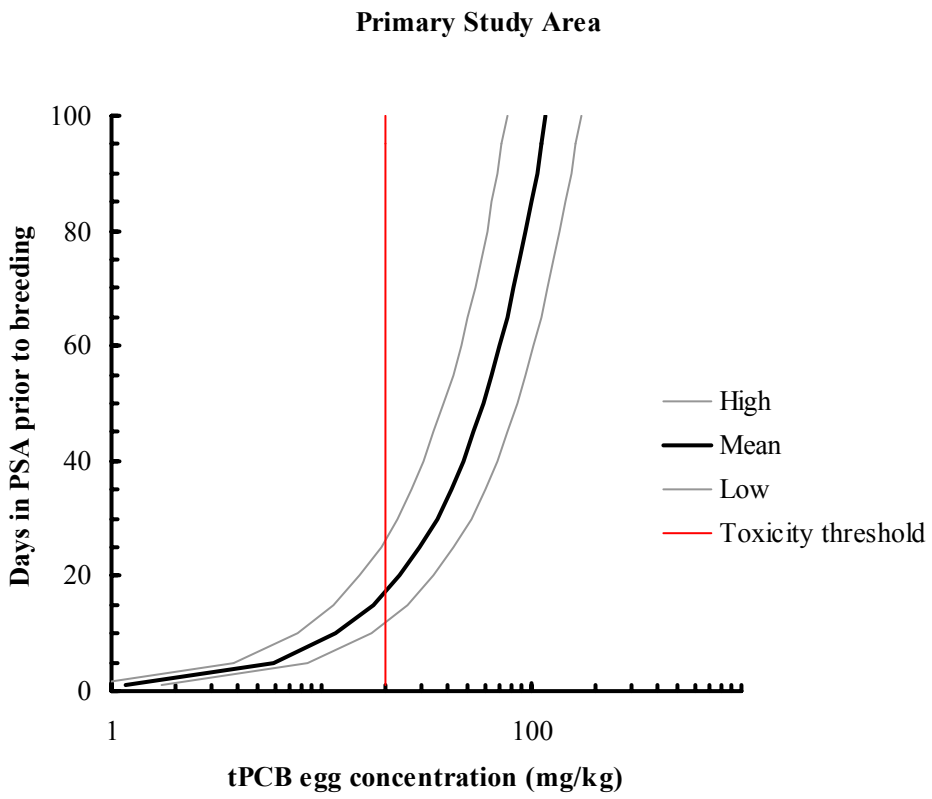

Figure K.4-3 Risk for Bald Eagle Eggs Exposed to tPCBs in the Housatonic River Primary Study Area

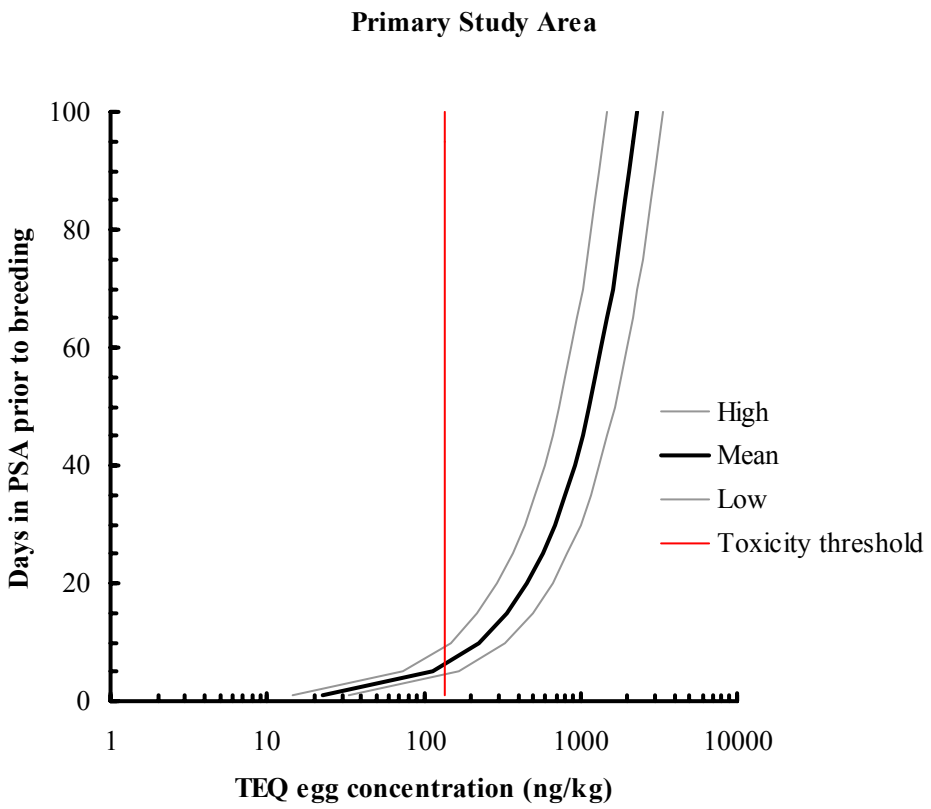

Figure K.4-4 Risk for Bald Eagle Eggs Exposed to TEQ in the Housatonic River Primary Study Area

NOTES:
Risk to eagles is based on a maximum acceptable threshold concentration (MATC) of 30.4 mg/kg total PCB (tPCB) concentration (wet weight) in whole body fish tissue greater than or equal to 12 cm.
* Only fish collected in 1998 to the present (2000) were included.
* Young-of-year bass composites were scaled by a factor of 3.5.
* Young-of-year perch composites were scaled by a factor of 2.5.

LEGEND:

EAGLE RISK
- < 30.4 mg/kg
- >= 30.4 mg/kg

- Town
- Reach Break
- Roads
- Housatonic River Basin Hydrology
- State Boundary

N W E S

2 0 2 4 6 8 Miles

Ecological Risk Assessment
GE/Housatonic River Site
Rest of River

FIGURE K.4-5
ASSESSMENT OF RISK TO BALD EAGLE EXPOSED TO tPCBs DOWNSTREAM OF WOODS POND

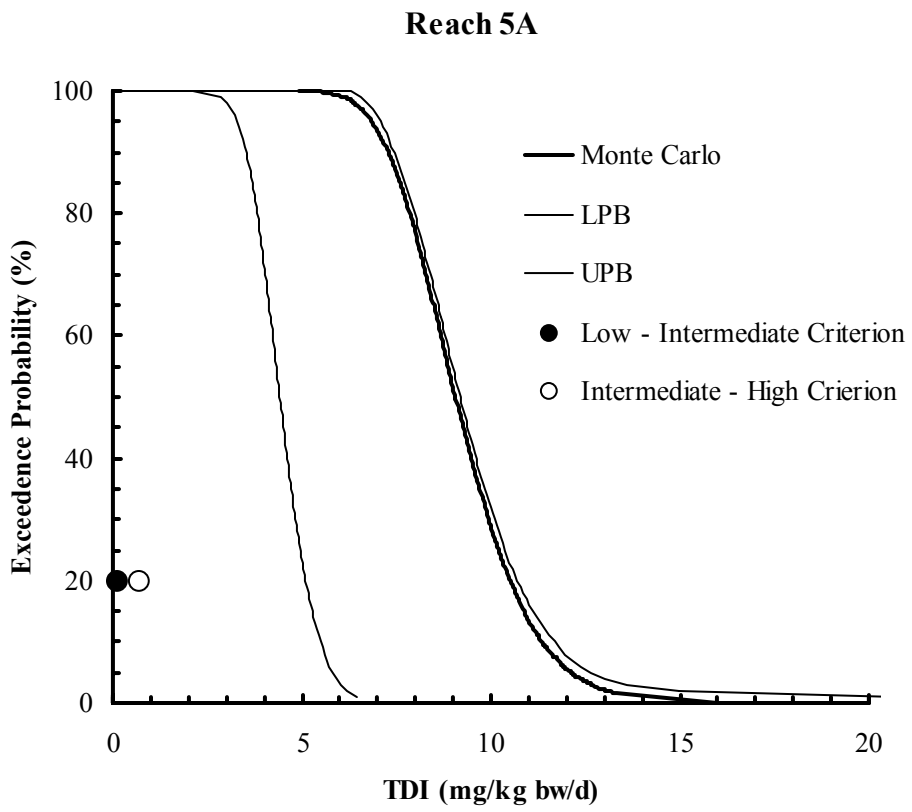

Figure K.4-6 Risk Curves for American Bittern Exposed to tPCBs in Reach 5A of the Housatonic River Primary Study Area

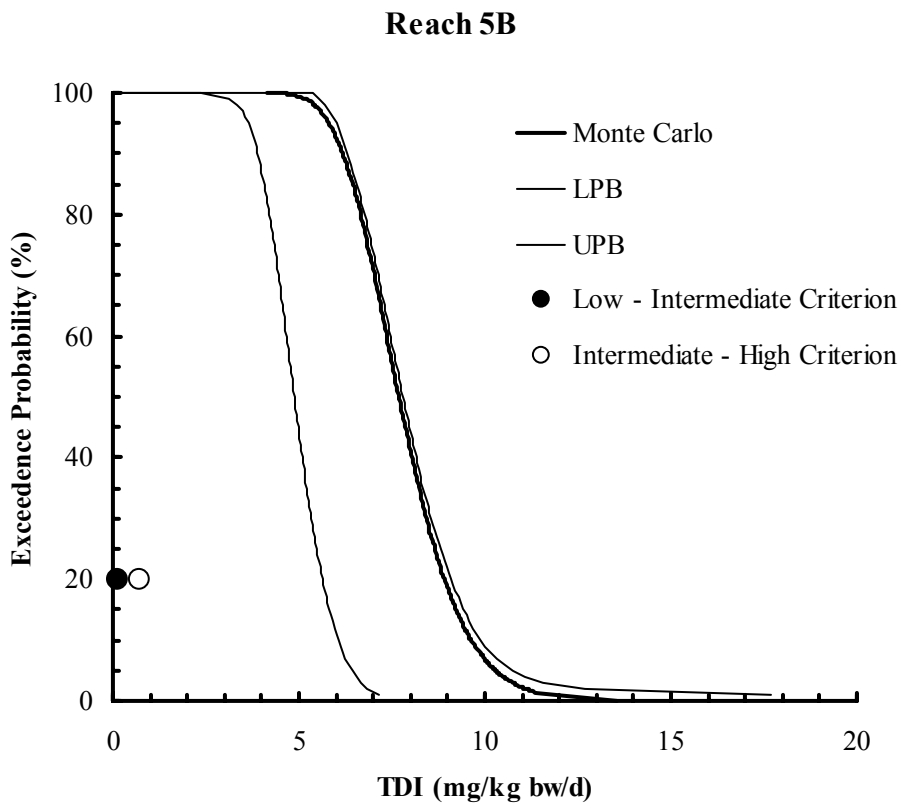

Figure K.4-7 Risk Curves for American Bittern Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area

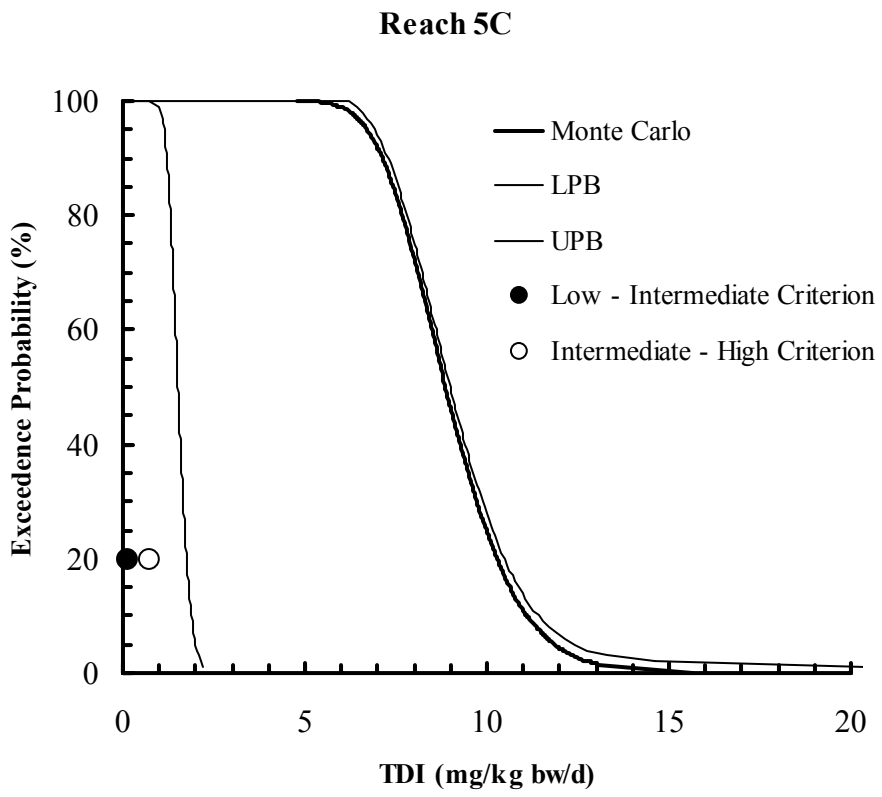

Figure K.4-8 Risk Curves for American Bittern Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area

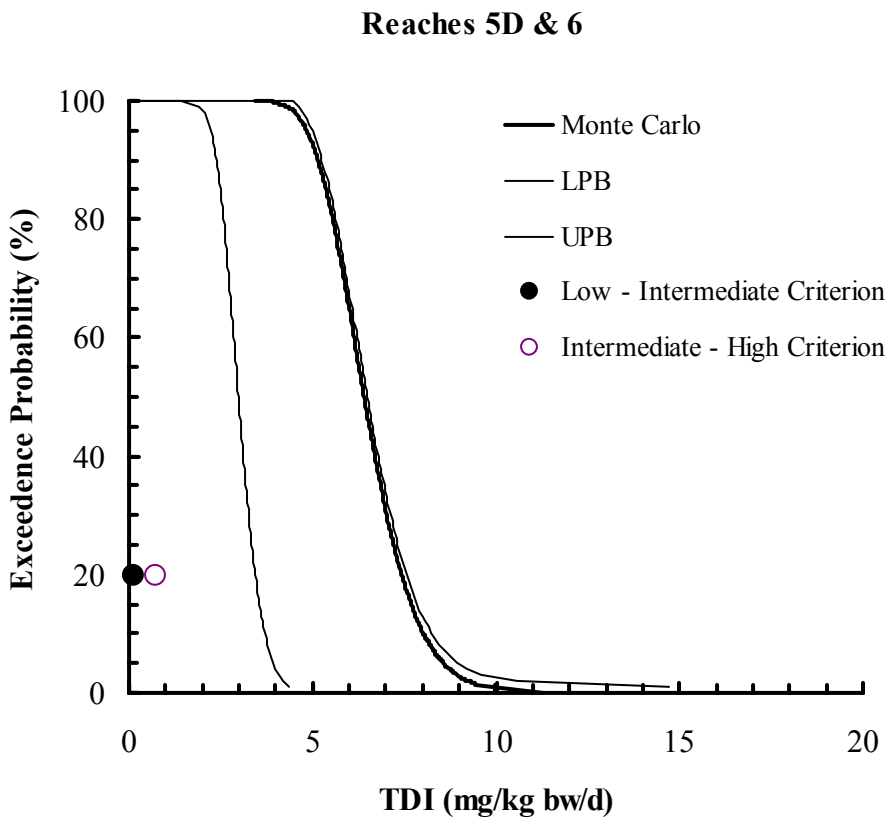

Figure K.4-9 Risk Curves for American Bittern Exposed to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area

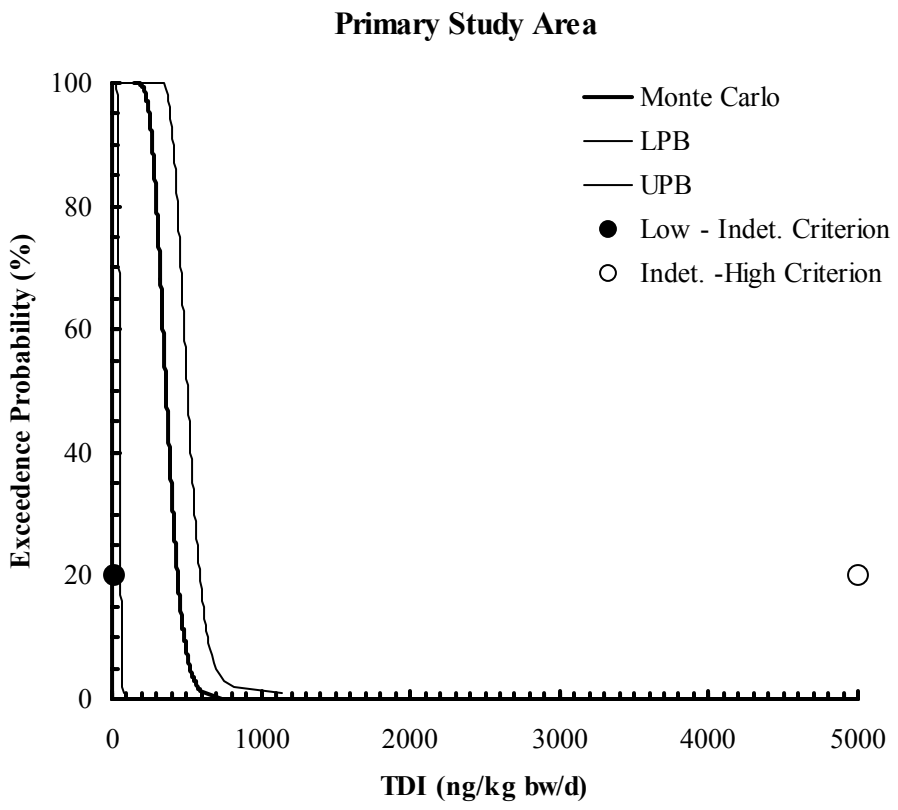

Figure K.4-10 Risk Curves for American Bittern Exposed to TEQ in the Housatonic River Primary Study Area

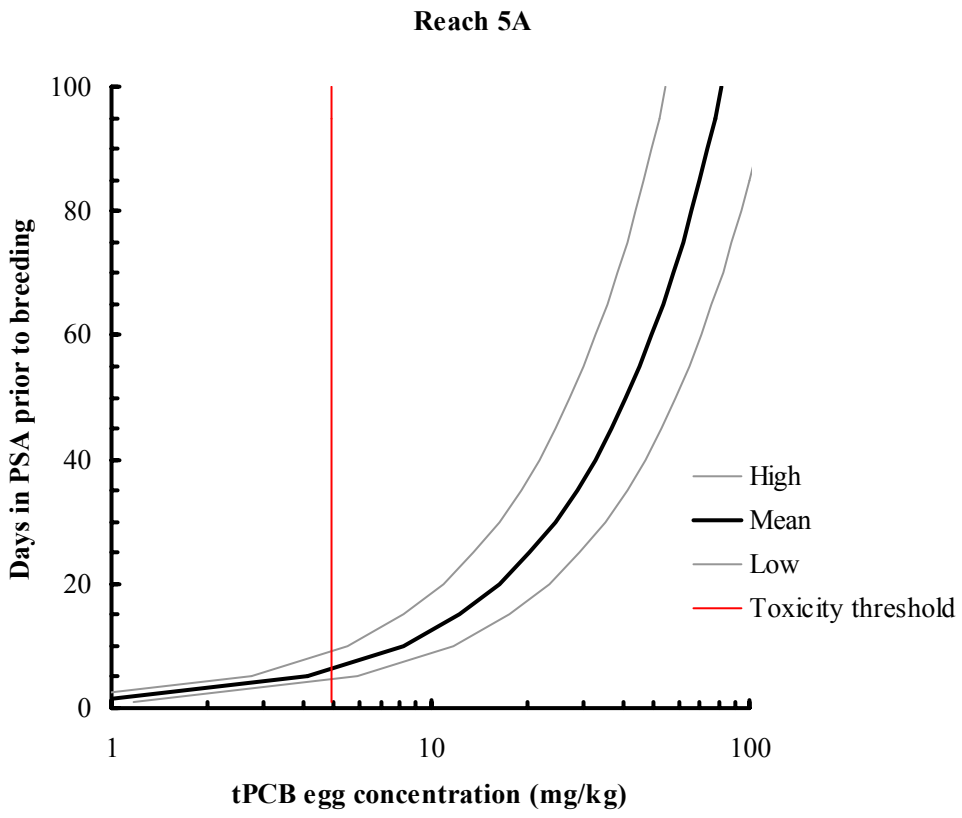

Figure K.4-11 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5A of the Housatonic River Primary Study Area

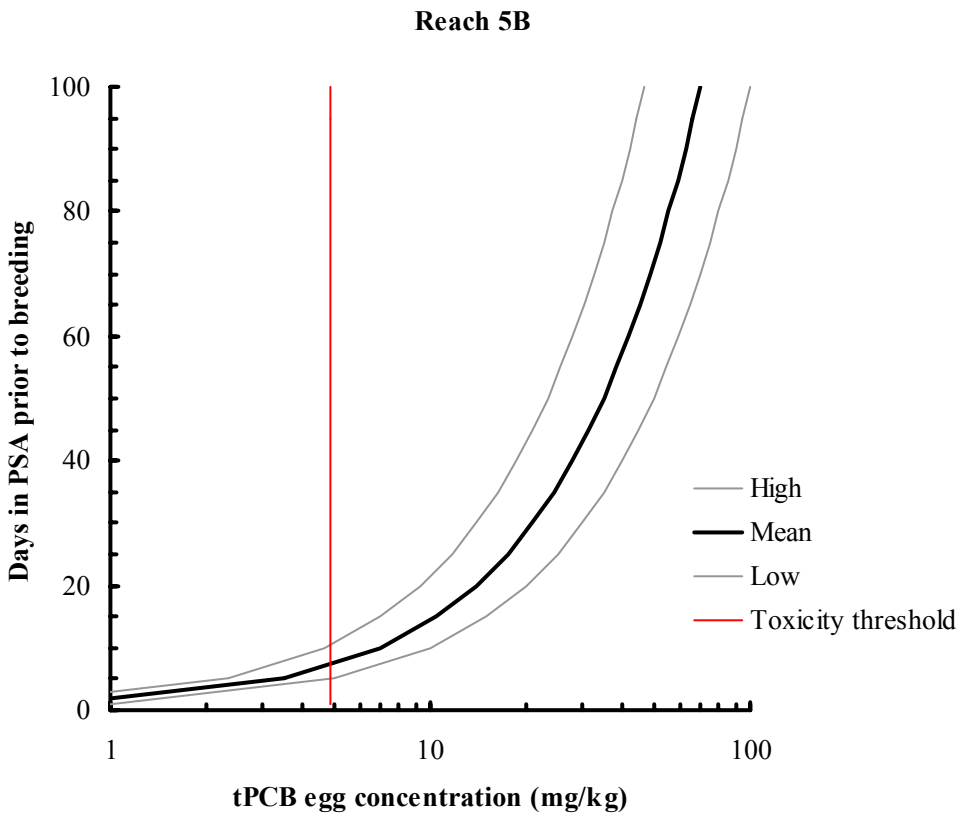

Figure K.4-12 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area

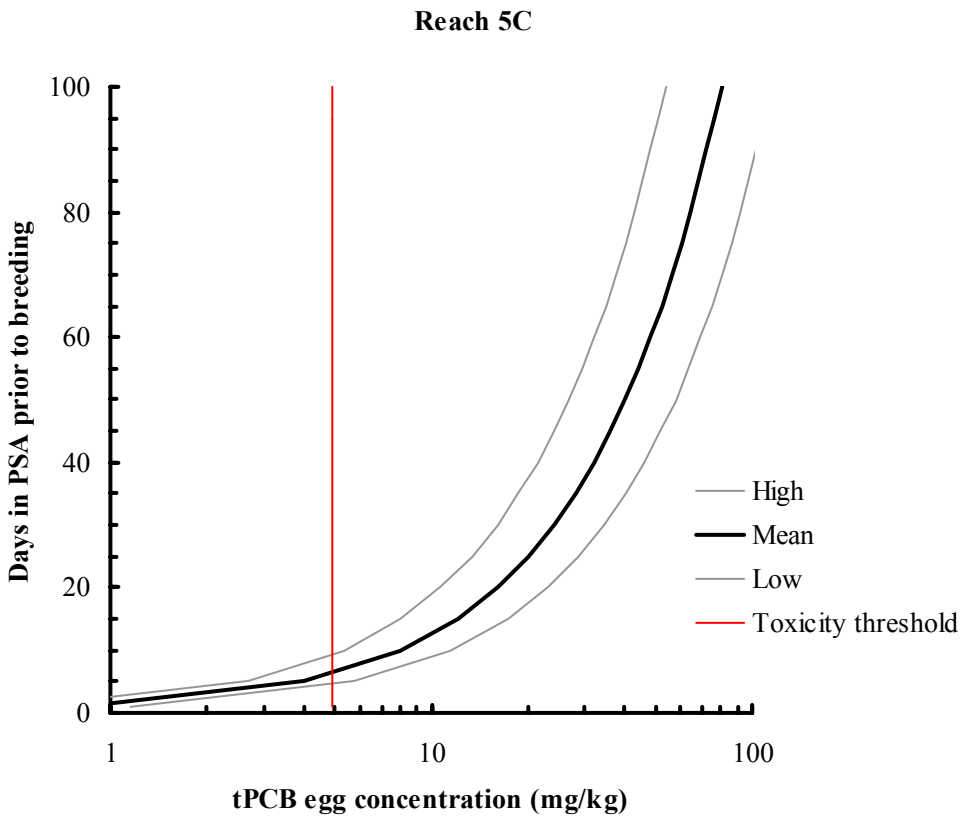

Figure K.4-13 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area

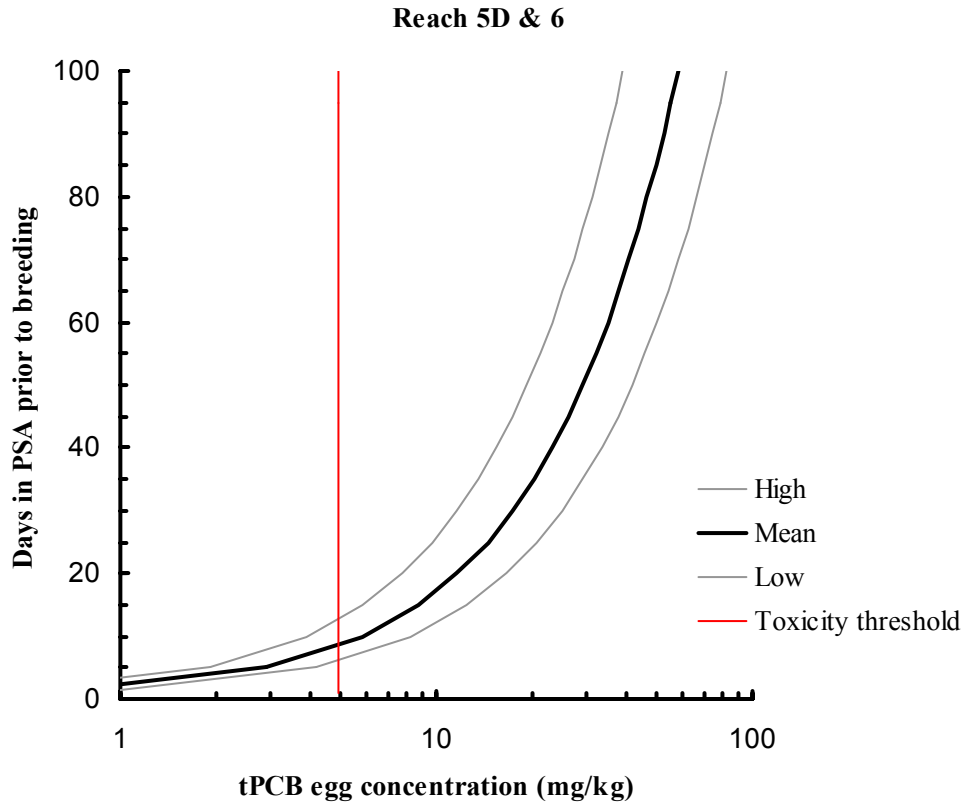

Figure K.4-14 Risk for American Bittern Eggs Exposed to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area

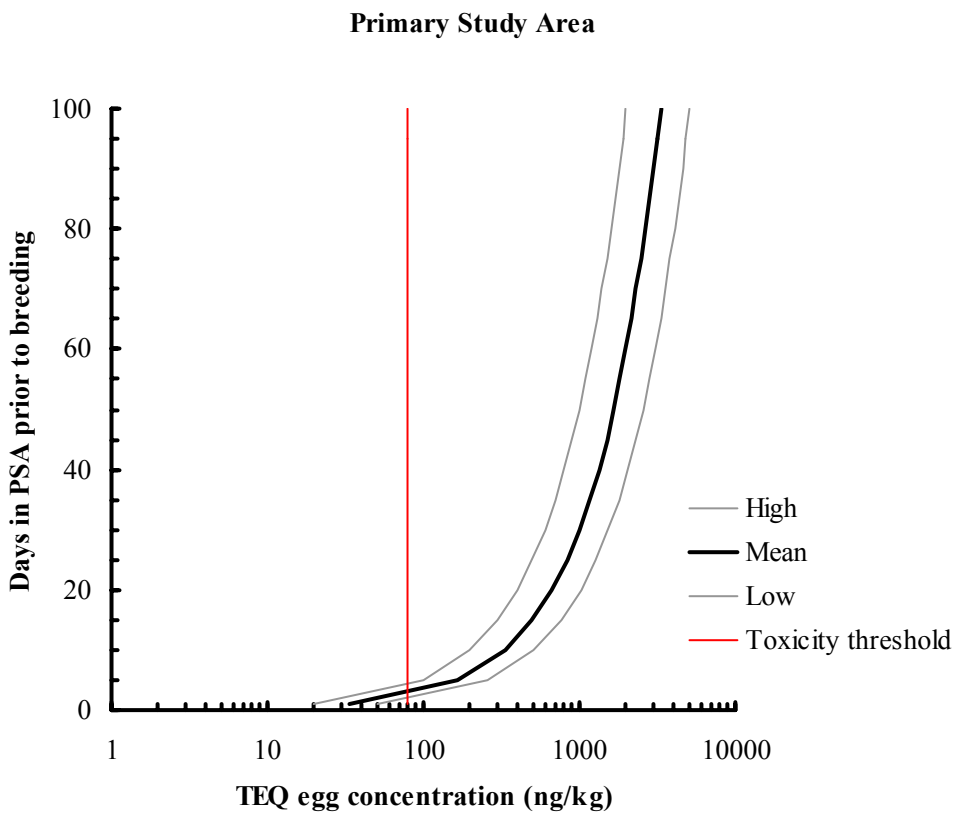

Figure K.4-15 Risk for American Bittern Eggs Exposed to TEQ in the Housatonic River Primary Study Area

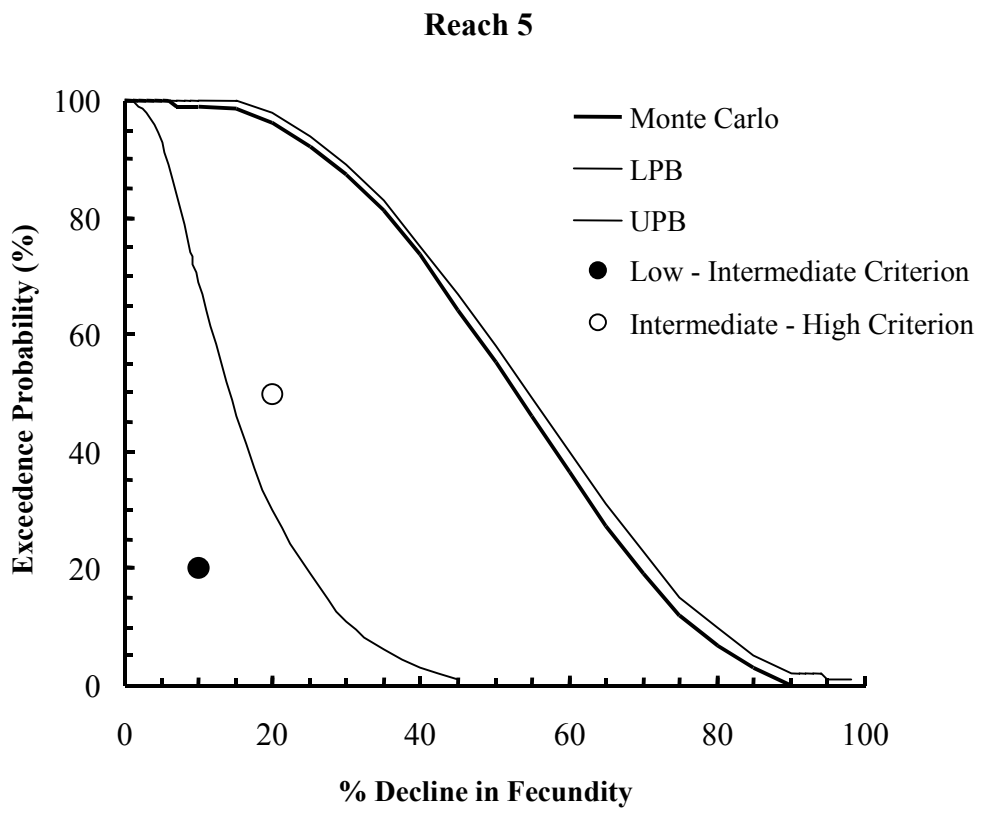

Figure K.4-16 Risk Curves for Small-Footed Myotis Exposed to tPCBs in Reach 5 of the Housatonic River Primary Study Area

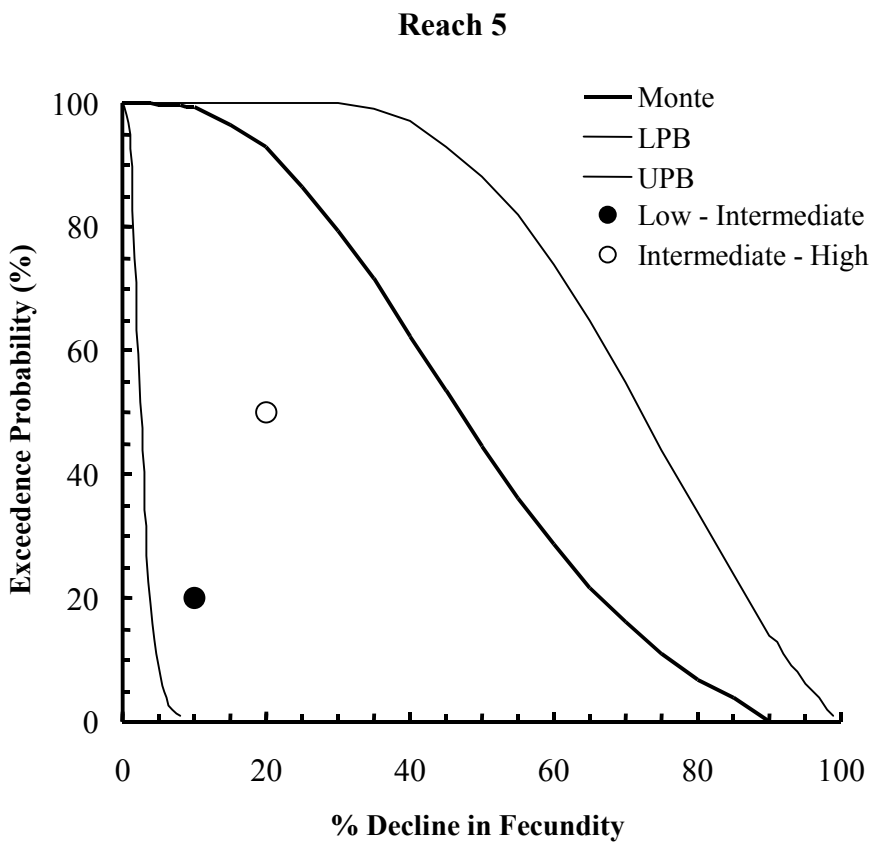

Figure K.4-17 Risk Curves for Small-Footed Myotis Exposed to TEQ in Reach 5 of the Housatonic River Primary Study Area