

**Focused Ecological Risk Assessment
PCBs and Mammalian and Avian Piscivores
in Conard's Branch and Richland Creek**

Neal's Landfill Site, Bloomington, Monroe County, Indiana



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Acronyms and Units

A1248 - Aroclor (number specifies particular Aroclor) [commercial PCB product]
 Ah - genetic locus in higher organisms that governs biological responses to certain aromatic hydrocarbons
 AhR - aryl hydrocarbon receptor [molecule in cells that binds with dioxin-like chemicals and initiates a sequence of signals affecting gene activity]
 aka - also known as
 AUF - area use factor [fraction of diet an animal consumes from a particular area]
 AWQC - ambient water quality criteria
 BCC - bioaccumulative chemical of concern [persistent chemicals that accumulate through foodchains]
 BMF - biomagnification factor [concentration in animal tissue divided by concentration in diet]
 BRH - black redhorse [fish]
 BW - body weight
 CB - Conard's Branch
 C_c - PCB concentration or TEC in crayfish
 C_{CB} - PCB concentration or TEC in Conard's Branch fish
 C_f - PCB concentration or TEC in fish
 $C_{f1\&2}$ - PCB concentration or TEC in fish consumed from Reach 1 (Conard's Branch) and Reach 2 (Richland Creek)
 CFR - Code of Federal Regulations
 cfs - cubic feet [of water] per second
 CI - 95 % confidence interval [the upper and lower range of mean values that would be obtained with repeated sampling or testing]
 Cl - chlorine
 conc. - concentration
 CP - common carp [fish]
 CRC - creek chub [fish]
 C_{RC} - PCB concentration or TEC in Richland Creek (Reach 2) fish
 CV - coefficient of variation [= SD / mean]
 CYP1A1 - cytochrome enzyme that catalyzes monooxygenation of lipophilic substances such as TCDD and dioxin-like PCB congeners (aka P450IA1)
CYP1A1 - gene that codes for the cytochrome enzyme that catalyzes monooxygenation of lipophilic substances
 d - day
 DNA - deoxyribonucleic acid [the key molecule encoding genetic information]
 ECOD - ethoxycoumarin-O-deethylase, one of the cytochrome P450IA1 subfamily isozymes (CYP1A1 isozymes)
 EC_x - effective concentration [dietary chemical concentration associated with x % decrease in a measured response compared to the control response; or dietary chemical concentration associated with adverse effects in x % of test animals]
 ED_x - effective dose [chemical dose associated with x % decrease in a measured response compared to the control response]
 e.g. - for example
 ERA - ecological risk assessment
 EROD - ethoxyresorufin-O-deethylase, one of the cytochrome P450IA1 subfamily (CYP1A1 isozymes)
 est. - estimated
 FD_{ingest} - food ingestion rate
 FERA - focused ecological risk assessment [restricted to specific chemicals, receptors, and/or areas]
 ft - foot
 fw - fresh weight [moist weight, not dried]
 g - gram

gal - gallon
 GE - General Electric
 GLI - Great Lakes Initiative
 GS - green sunfish
 H4IIE - rat hepatoma cell line [used for a bioassay in one approach for determining TEC]
 HC_x - hazard concentration for x % of species, e.g., HC₅ represents the TEC in fish that is expected to result in adverse effects in 5 % of bird species that prey on fish
 HI - hazard index [sum of HQs of chemicals with similar toxicological modes of action and effects]
 HQ - hazard quotient [ratio of concentration or dose of a chemical divided by the TRV for that chemical]
 ID - sample identification number
 IDEM - Indiana Department of Environmental Management
 IN - Indiana
 Inc. - incorporated
 I-TEQ - dioxin toxic equivalent calculated with Ahlborg, et al. (1992;1994) TEFs
 IUPAC - International Union of Pure and Applied Chemistry
 kg - kilogram [1000 grams]
 kg_{BW} - kilogram of bodyweight
 L - liter
 lb - pound
 LC_n - lethal concentration of a chemical to n % of exposed animals
 LOAEC - lowest observed adverse effect concentration [lowest chemical concentration showing an adverse effect]
 LOAEL - lowest observed adverse effect level [lowest dose (chemical amount per bodyweight per day) showing an adverse effect]
 LS - longear sunfish
 MA - Massachusetts
 MI - Michigan
 MQL - method quantitation limit [lowest concentration reliably reported by an analytical procedure]
 N or n - number of samples
 NA or na - not available or cannot be calculated
 NCP - National Contingency Plan
 ng - nanogram [10⁻⁹ (one-billionth) gram]
 NOAA - National Oceanic and Atmospheric Administration
 NOAEC - no observed adverse effect concentration [highest chemical concentration showing no adverse effect]
 NOAED - no observed adverse effect dose [highest intake (chemical amount per animal per day) showing no adverse effect]
 NOAEL - no observed adverse effect level [highest dose (chemical amount per bodyweight per day) showing no adverse effect]
 NY - New York
 P4501A or P450IA1 - cytochrome enzyme that catalyzes monooxygenation of lipophilic substances such as TCDD and dioxin-like PCB congeners (aka CYP1A or CYP1A1)
 PAC - percent allowable consumption [the percent diet an animal can take from a stream reach without adverse effects]
 PADI - percent allowable daily intake [percent of food ingestion per day an animal can take from a contaminated area without adverse effects]
 PCB - polychlorinated biphenyl
 PCDF - polychlorinated dibenzofuran
 PCH - polychlorinated hydrocarbon
 p-PCH - planar or coplanar PCH congeners
 PCN - polychlorinated naphthalene

pers. comm. - personal communication
 pg - picogram [10^{-12} (one-trillionth) gram]
 ppb - part per billion [$\mu\text{g}/\text{kg}$]
 ppm - part per million [mg/kg]
 ppt - part per trillion [pg/g]
 RC - Richland Creek
 RCRA - Resource Conservation and Recovery Act
 SD - standard deviation
 SRH - shorthead redhorse [fish]
 S-TEQ - dioxin toxic equivalent calculated with Safe (1993) TEFs
 TEC - dioxin toxic equivalent concentration [the concentration of TCDD expected to have same toxicological effect as a mixture of several chemicals with the same mode of action as dioxin]
 TEF - dioxin toxic equivalency factor [relative toxicity of a chemical compared to TCDD]
 TEQ - dioxin toxic equivalent [the quantity of TCDD expected to have same toxicological effect as a mixture of several chemicals with the same mode of action as dioxin]
 TCDD - 2,3,7,8-tetrachlorinated dibenzo-*p*-dioxin [the most toxic dioxin congener]
 tPCB - total PCBs
 TRV - toxicity reference value [no effect or lowest effect value based on published studies]
 U - not detected
 UCL - 95 % upper confidence limit of the mean [an upper estimate of the mean (average), specifically, the highest value of the mean that would occur out of 95 % of the times sampling was repeated in the same area]
 μg - microgram [10^{-6} (one-millionth) gram]
 μL - microliter [10^{-6} (one-millionth) liter]
 USACE - United States Army Corps of Engineers
 USEPA - United States Environmental Protection Agency
 USFWS - United States Fish and Wildlife Service
 WQC - water quality criteria
 WHO - World Health Organization
 WHO-TEQ - dioxin toxic equivalent calculated with Van den Berg, et al. (1998) TEFs
 WI - Wisconsin
 wk - week
 WS - white sucker [fish]
 yd - yard
 yr - year

Summary

All of the receptors evaluated in the risk assessment (mink, kingfisher, and fish) show a similar pattern of risk—elevated risk to at least 2 miles downstream from Neal’s Landfill, low risk beyond 13 miles downstream, and possible, but sparsely documented, risk 3 to 6 miles downstream.

Mammals and birds feeding on a mixed diet of fish and crayfish from Conard’s Branch and the upper portion of Richland Creek downstream of the confluence with Conard’s Branch are potentially at risk of reproductive impairment due to PCB exposure. Risk decreases with downstream distance from Neal’s Landfill consistent with discharge of elevated concentrations of PCBs from Neal’s Landfill. Elevated risk is well documented for 2 miles downstream of Neal’s Landfill, and low to no likelihood of risk is well documented 13 to 35 miles downstream. Risk is sparsely characterized between 3 and 6 miles downstream of the site. Based on sparse whole-fish data between 3 and 6 downstream miles, mink or kingfisher exposures appear to be in the gray area—likely higher than no effect levels, but lower or just equal to adverse effect levels. The risk estimates are made specifically for mink and kingfisher, but are protective for other fish-eating animals such as otter, raccoon, egrets, or herons; and are expected to be protective for direct effects to fish or other aquatic organisms.

Two additional lines of evidence support the risk findings. The measured PCB concentration and dioxin toxic equivalent concentration (TEC) in the liver of a road-killed mink collected from the Highway 48 bridge over Richland Creek are consistent with modeled uptake by mink foraging 3 to 13 miles downstream in Richland Creek, but not further downstream or upstream, and exceed published mink liver thresholds for impaired reproduction (appears to be in the gray area between reported no and adverse effect levels), vitamin A deficiency (possible effects include impaired disease resistance, reproduction, development, and vision), and jaw lesions (which can result in loss of teeth). The road-killed mink is a single sample, so it does not provide definitive proof regarding exposure or risk by itself, but the liver data support the procedures used in the risk assessment for modeling mink exposures and evaluating risks (or, at the least, do not contradict the exposure and risk estimates of the risk assessment).

The second additional line of evidence is the potential risk of PCB exposure to adult fish based on fish tissue TEC, which shows elevated risk of growth suppression and behavioral impairment in fish in Conard’s Branch and the upper portion of Richland Creek. The downstream extent of elevated risk to fish is similar to that for elevated risk to mink or kingfisher, after adjusting the fish risk estimates for reported differences among fish species in dioxin sensitivity.

Mink and kingfisher risks are estimated for both total PCBs and dioxin toxic equivalent concentration (TEC). Mink are a well-studied subject of PCB research. Kingfisher are assessed with a range of toxicity reference values (TRVs) intended to represent higher and middle sensitivities to PCBs since the sensitivity of kingfisher is unknown. Also, kingfisher risk is characterized by multiple approaches—ingestion dose modeling to adult birds (compared to dose TRVs), and modeling PCB accumulation in eggs (compared to egg TRVs). This means that the overall kingfisher risk estimates varied more than the mink estimates within the same sample location. Tissue (egg) modeling tended to produce higher risk estimates than ingestion dose modeling. The kingfisher “consensus” risk estimates, defined as the overlap or gap between the ingestion dose and egg risk estimate ranges, are generally consistent with the mink risk estimates.

Preliminary goals are calculated for PCBs and TEC in fish expected to be protective for mink and kingfisher foraging along Richland Creek. The protective fish goals for mink are 0.7 to 0.9 mg/kg PCB (no to low effect) and 7 to 26 pg/g TEC (mammalian) (no to low effect). The fish goals can be relaxed in Conard's Branch under the assumption that mink would only partially forage in Conard's Branch. The Conard's Branch protective fish goals for mink are 2 mg/kg PCB (no or low effect) and 17 to 68 pg/g TEC (mammalian). Note that the goals apply to the overall mix of fish taken from a given reach.

The preliminary fish goals protective for kingfisher have wider ranges than for mink, reflecting uncertainty over the relative sensitivity of kingfisher to PCBs compared to the bird species that have been studied. However, the kingfisher fish goals bracket the less uncertain mink goals.

1 Introduction

1.1 Site History

The Neal's Landfill site occupies nearly 18 acres of property surrounded by farms and woodlands approximately 4.5 miles west of Bloomington, IN. The landfill accepted industrial and municipal wastes from 1950 until 1972. Westinghouse Electric Corporation disposed of waste electrical equipment in the landfill, including electrical capacitors containing PCBs and other PCB-contaminated material. Westinghouse was later acquired by CBS Corporation, then Viacom Inc., and subsequently reverted to CBS¹. Neal's landfill is located over a karst formation². Several springs at the foot of the landfill feed Conard's Branch, a tributary to Richland Creek. Elevated PCB concentrations were detected in surface soil, spring water, sediments, and fish, and the site was placed on the National Priorities List in 1981.

Interim measures implemented by Westinghouse in 1987 included removal of visible capacitors and stained soil, installation of a 2-ft thick clay cap over the main landfill, fencing, removal of sediments and banks along Conard's Branch (4300 ft distance) and part of Richland Creek (230 ft), and installation of a collection and treatment system for the spring water discharges. A planned incinerator for treating the PCB-contaminated materials from Neal's Landfill and other locations was blocked by legislation.

In 1999, CBS Corporation excavated nearly 42,000 tons of contaminated material for off-site disposal, and over 4000 capacitors for off-site incineration. Approximately 90,000 cubic yd of landfill material were consolidated to reduce the landfill area from 18 to 10 acres, which was covered with a RCRA Subtitle C cap.

The remedial actions reduced PCB concentrations in sediment, which were as high as 2500 ppm in Conard's Branch and over 400 ppm in the landfill springs, to less than 50 ppm in spring sediments, and to maximum detected concentrations of 21 (1988), 4 (1998), and 0.3 ppm (2001) in Conard's Branch, and 0.1 - 2 ppm in

¹ The draft versions of this risk assessment were written when Viacom was the parent corporation, and the name "Viacom" is retained throughout the document as reflecting corporate ownership at the time of the investigations performed for the risk assessment. The current parent corporation is, however, CBS, not Viacom.

² Karst is "a limestone plateau or region characterized by underground drainage, sinkholes, rolling surfaces, and caverns" (Barnhart 1986).

Richland Creek. Similarly, maximum concentrations of PCBs in fish declined from over 200 ppm in the early 1980's to 25 ppm in Conard's Branch and 2 ppm in Richland Creek in 1998 (Tetra Tech 2002).

In 1982 and 1983, PCB concentrations were as high as 7 ppb in spring water, and 2.7 ppb in Conard's Branch surface water. Following the remedial action, PCB concentrations have ranged from 0.2 to 2.4 ppb in two springs (South and North) that feed Conard's Branch. However, the water treatment system capacity of 1 cfs is insufficient for storm events, for example, the discharge flow rate in Conard's Branch may be as high as 2.3 cfs. This means that discharge from the landfill bypasses the treatment system during storms releasing untreated PCB-contaminated water to Conard's Branch and Richland Creek. Spring water PCB concentrations peaked at 4.5 to 10 ppb during storm events before the remedial action, and 3 to 12 ppb following remediation, with one event peaking at >30 ppb (4/00) (Tetra Tech 2002). In comparison, the federal aquatic life ambient water quality criterion for PCBs is 0.014 ppb (USEPA 1999).

The purpose of the present focused ecological risk assessment (FERA) is to evaluate the protectiveness of the Neal's Landfill remedial actions on wildlife utilizing Conard's Branch and Richland Creek. The risk assessment focuses on risks due to PCBs to assess whether the remedial actions take to date are adequately protective for wildlife.

1.2 Ecological Risk Assessment

Two important goals of ecological risk assessments are: 1) to determine whether harmful effects are likely for wild animals or plants, and 2) if there is risk, to calculate protective cleanup levels that would reduce the risks to wild animals or plants. Human health risks are separately assessed, and are not discussed in ERAs.

According to the National Contingency Plan (NCP) ³, both present and future risks should be assessed. "[T]he lead agency shall conduct a site-specific baseline risk assessment to characterize the current and potential threats to human health and the environment that may be posed by contaminants migrating to ground water or surface water, releasing to air, leaching through soil, remaining in the soil, and bioaccumulating in the food chain" (40 CFR Part 300.430(d)(4)). Similarly, the remedies should provide for present and future protection. "The national goal of the remedy selection process is to select remedies that are protective of human health and the environment, [and] that maintain protection over time ..." (40 CFR Part 300.430(a)(1)(i)). Similar statements are made in 40 CFR Part 300.415(b)(2)(i, ii, and iv).

The process for performing an ERA is described in the Superfund guidance for ecological risk assessment (USEPA 1997a). The main steps of an ERA are outlined below.

An initial step of an ERA is to decide which components of an ecosystem ⁴ should be protected, that is, which species should be the focus of the ERA. This is different from human health risk assessments in which the species

³ The NCP describes the organizational structure and procedures for responding to releases of hazardous substances. It was published in the Federal Register vol. 55 no. 46, March 8, 1990, pp. 8666-8865 (reprinted in USEPA 1992a), and may be accessed on-line at: <http://www.epa.gov/epahome/cfr40.htm>, select "Chapter 1", "Subchapter J", and "Part 300".

⁴ An ecosystem is the sum of all the living organisms and the physical factors in a particular area (life, geology, and climate).

is predetermined (humans). The decisions of what to protect and how to measure it are made in the Problem Formulation step of the ERA.

Problem formulation begins with development of a conceptual model, which is a representation of how the contaminants at a site are expected to behave in the environment. The conceptual model is based on fate (does a contaminant degrade to less toxic forms, transform into other toxic substances, or persist in the environment?) and transport (how does a contaminant move through the environment, and in what components will it likely occur?). In risk assessment language, the species potentially exposed to a contaminant are called “receptors”, and the contaminant is a “stressor”—something that could adversely affect receptors ⁵. The various components of the environment a chemical stressor moves through from the place it was released until it is absorbed by a receptor is called the “exposure pathway”. An exposure pathway is described as “complete” when a contaminant can move from a source through the environment to a receptor, and “incomplete” if contaminant movement is blocked at some stage so the receptor is not exposed. The conceptual model is used to narrow attention to the animals or plants likely to be exposed to the contaminants at the site, or, in formal terms, the receptors likely to be part of complete exposure pathways.

It is not possible to evaluate every species that is potentially at risk at a site. In the Great Lakes region there are some 75 species of amphibians and reptiles, 80 species of mammals, over 200 species of breeding birds (and a roughly equal number of nonbreeding and accidental species), a couple of hundred species of fish, several thousand species of terrestrial plants, tens of thousands of species of insects, and so forth. The purpose of the problem formulation is to focus attention on a few species or groups of species that are appropriate for answering the question of whether an ecological risk exists at the site. An additional consideration is the availability of toxicological studies in the same or, more often, a related species for the chemicals in question, because risk cannot be estimated without information on toxicity to wildlife or plants.

Different terms are used to refer to what should be protected (assessment endpoint) and what will be studied (measurement endpoint). Assessment endpoints are explicit expressions of the environmental value that is to be protected (USEPA 1997a), that is, specific entities, processes, or functions (e.g., reproduction in predaceous birds, fish productivity, plant diversity, and so forth). Measurement endpoints are measurable ecological characteristics that are related to the assessment endpoints, and may include measures of effects (caused by a contaminant) or measures of exposure (to a contaminant) (USEPA 1997a). In other words, what will actually be investigated to determine the level of risk.

Assessment and measurement endpoints may be one and the same, or different but related to each other. For example, fish production could be an assessment endpoint. A possible measurement endpoint would be to perform a field study of fish productivity at the site (measurement and assessment endpoints are the same). Another approach would be to measure the impact of contaminants on benthic invertebrates ⁶ (measurement endpoint), which is related to fish productivity (assessment endpoint) because benthic invertebrates form the base of the food

⁵ Besides chemical stressors, the other broad categories are physical stressors and biological stressors.

⁶ Benthic invertebrates are the insects, worms, and other small creatures that live on and in the bottoms of streams and other bodies of water.

chain that supports freshwater fish populations. In this example, effects on benthic invertebrates are assessed for the ERA, but the reason for doing so is concern over potential impacts on fish.

An ERA should focus on specific risk questions that state the effects, responses, or changes that are considered important. This serves two purposes. First, similar to the rationale for endpoint selection, it is not possible to investigate every imaginable outcome and interaction. Ideally, resources should be focused on assessment of likely major effects, and not squandered on unlikely, minor, or purely speculative effects (a selective approach may not be feasible for contaminants for which the potential adverse effects are poorly understood). Second, explicitly stating the risk questions provides objectives by which the adequacy of the investigations can be judged (e.g., appropriate detection limits for chemical analyses, level of effort needed for field studies, statistical power).

The next step is the Site Evaluation and Analysis Phase, which consists of two parallel investigations—Characterization of Exposure and Characterization of Ecological Effects.

Characterization of Exposure summarizes what is known of the extent of contamination at the site, bioavailability, complete exposure pathways, and the measured or modeled uptake of contaminants by the ecological receptors. Exposure should be characterized in two ways: 1) central tendency, which is based on mean (average) contaminant concentrations, and 2) an upper estimate of exposure (reasonable maximum), which is based on either the 95 percent upper confidence limit of the mean (UCL) or maximum values measured at the site. The reason for including an upper estimate of exposure is to account for uncertainty in calculating mean contaminant concentrations from limited site data. The UCL is an estimate of how high the mean concentration could be if the same sample design was repeated multiple times ⁷, specifically, the highest value of the mean that would occur out of 95 % of the times the sampling was repeated in the same area (USEPA 1992b).

For Characterization of Ecological Effects, the potential adverse effects of the contaminants are described as they relate to the measurement and assessment endpoints and risk questions. The information is taken from literature of field and laboratory studies performed for the particular contaminant, and, if available, from investigations of ecological impacts at the site. An important objective is to calculate the contaminant concentration or dose that is associated with adverse effects, that is, how much contaminant exposure is necessary to potentially cause an adverse effect?

The two lines of investigation are combined the next step, Characterization of Risk, in which the amount of exposure of the ecological receptors is compared with the concentration or dose associated with adverse effects to determine whether the contamination at the site presents a potentially significant risk. If risk is indicated for the site, back-calculations are performed to determine ecologically protective cleanup goals, such that exposures would be reduced below levels of concern.

⁷ Since a mean concentration is calculated from a limited number of samples that never exactly represent the distribution of concentrations at a site, the calculated mean will differ each time sampling is repeated at the same site. The successive means may differ slightly (if the site data are reasonably uniform and an adequate sample design is used) or differ greatly (if the site data are highly variable and/or the sample design is inadequate).

An Uncertainty section describes the uncertainties associated with assumptions, data, extrapolations, and models, and the possible effects of these uncertainties on the outcome of the risk assessment. Uncertainty refers to gaps in knowledge that could be reduced by further studies. The natural (true) variability of some measured quantity is not itself an uncertainty, because the inherent variability would not change with additional studies. However, not knowing what the inherent variability is could be an important uncertainty (in this case, more research could provide a more accurate estimate of natural variability).

The results of the ERA are summarized and recommendations are made regarding risk and, if appropriate, preliminary remedial goals.

2 Problem Formulation

The ecological risk assessment focuses solely on the PCB-related risks to wildlife utilizing Conard's Branch and Richland Creek downstream of Neal's Landfill. A focused ecological risk assessment (FERA) approach is used to specifically investigate the ecological protectiveness of the previous remedial actions taken at the Neal's Landfill site. PCBs are the sole class of contaminant considered because the remedial actions were based on concerns over PCBs. Since sediments were remediated in a previous action, the primary source of PCB contamination was expected to be the discharge of spring water from Neal's Landfill to Conard's Branch, partly treated and partly bypassing the water treatment system during storm events (Section 1.1). However, post-removal confirmation sampling was not performed, so the possibility of residual contamination in the banks or floodplain cannot be ruled out. From Conard's Branch, the discharge then flows into Richland Creek. The FERA focuses on risks to mammalian and avian piscivores (mammals and birds feeding on fish) because these receptors are more highly exposed to PCBs as compared to aquatic receptors (fish, aquatic insects, or aquatic plants) or herbivores (animals feeding on aquatic vegetation). Actions based on risk to piscivores are expected to be protective for fish, in addition to the animals feeding on fish, because the PCB levels in fish that may harm piscivores are usually lower than the levels potentially causing harm to the fish themselves.

2.1 PCB Characteristics

2.1.1 Structure and Names

The term "polychlorinated biphenyl" (PCB) refers to a class of chemicals comprised of two six-carbon rings (phenyls) attached side-by-side by a single carbon-carbon bond with various numbers of chlorine (Cl) atoms attached to the outside of the rings. There are 209 types of PCB chemicals differentiated by the number of Cl atoms and their positions on the rings. The different types are referred to as "congeners". The congeners have been numbered for convenience by the International Union of Pure and Applied Chemistry (IUPAC). The structures and numbers of the PCB congeners are presented in Eisler and Belisle (1996) and ATSDR (2000). Another term used in the literature is "homolog", which refers to the congeners with the same number of Cl atoms that differ only in the positions of the Cl atoms on the phenyl rings (e.g., all 46 five-chlorine congeners are homologs).

Commercial PCBs mixtures were marketed under several names, Aroclor is best-known in the U.S.⁸. Aroclors are congener mixtures designated by four numbers—the first two are usually “12” to indicate biphenyls, and the second two give the overall percentage by weight of Cl atoms in the mixture⁹, for example, Aroclor 1248 has 48 % Cl. Aroclor batches with the same number may differ in the specific congener composition so long as the overall Cl percentage remains the same, which means that different batches of the same Aroclor may have different toxicological properties. Aroclors often contain variable proportions of other contaminants in addition to PCB congeners, for example, chlorinated dibenzofurans.

A small subset of PCB congeners cause dioxin-like toxic effects because the geometry of these congeners is similar enough to that of dioxin so they behave similarly once absorbed within the cells of exposed animals. An important characteristic for dioxin-like behavior is that the two phenyl rings orient in the same plane, referred to as planar or coplanar congeners. The PCB coplanar congeners that best mimic dioxin behavior have no Cl atoms attached to the closest positions on the phenyl rings to the bond holding the two rings together. This is called the *ortho* position, and congeners with no *ortho* Cl are called non-*ortho* (coplanar congeners 77, 81, 126, and 169). Another class of coplanar PCBs have one Cl in the *ortho* position, and are called mono-*ortho* (coplanar congeners 105, 114, 118, 123, 156, 157, 167, and 189) (Van den Berg, et al. 1998). Each of the twelve non- and mono-*ortho* congeners listed here also possess the remaining characteristics required for dioxin-like activity—two Cl in the *para* positions (attached to the phenyl rings directly opposite from the point of attachment of the two rings) and two or more Cl in the *meta* positions (located between the *ortho* and *para* positions). The *ortho* and mono-*ortho* coplanar congeners listed above are referred to as “dioxin-like PCB congeners”.

2.1.2 PCB Fate and Transport

The fate and transport discussion is based on ATSDR (2000), which should be consulted for a more thorough treatment. The following discussion focuses on the fate of PCBs released to surface water.

PCBs consist of mixtures of various congeners that have significantly different chemical properties. For example, the lower chlorinated congeners (ones with few Cl atoms) are more volatile and degrade more readily than higher chlorinated congeners (ones with many Cl atoms). Also the positions of the Cl atoms on the phenyl rings can affect the partitioning behavior of the congeners between water and lipids or other organic matter, and susceptibility to degradation.

Overall, PCBs are persistent in water. Lower chlorinated congeners have higher volatilization rates than the higher chlorinated congeners, and are subject to photolysis (light-catalyzed breakdown) and aerobic biodegradation (by bacteria that require oxygen). However, the congeners with 4 or more Cl are much less affected by these processes, which means that the most toxic PCB congeners do not readily degrade and slowly volatilize from surface waters. Some of the higher chlorinated congeners may be biodegraded by anaerobic (without oxygen) bacteria in subsurface sediments, but the rates become very slow at PCB concentrations still much higher than considered environmentally protective (anaerobic degradation may also be inhibited at very high PCB concentrations). Although sediments act

⁸ Some other names for commercial PCB mixtures include Clophen, Phenoclor, Pyralene, Kanechlor, and Fenclor.

⁹ With the exception of Aroclor 1016, a PCB distillation product, for which “16” does not indicate Cl percentage.

as a sink for PCBs, since PCBs degrade slowly, sediments also act as a continuously available reserve source of PCBs to the water column (PCB desorption from contaminated sediments increases whenever water column PCB concentrations drop).

The recalcitrant (persistent) congeners have low water solubilities, which means that they tend to partition from water to organic matter in sediments or in suspension in water, and to lipids (fat) of aquatic organisms. Despite the pronounced partitioning behavior, the proportion of dissolved PCBs ranges from 1/3 to 3/4 of the total PCBs in the water column (the remainder are bound to suspended organic matter). The dissolved phase PCBs are readily absorbed directly from the water column by aquatic organisms (bioconcentration). The concentrations of Aroclors in freshwater fish are as much as several hundred thousand times greater than the concentration in water, and the concentrations of individual congeners may be as much as a million times greater than the water concentrations. Plankton also acts as suspended organic matter and accumulates high levels of PCBs from surface water.

PCB concentrations tend to increase (biomagnify) through trophic transfer, that is, animals tend to have higher PCB concentrations compared to their prey. Again there is a difference in the behavior of lower and higher chlorinated congeners—the lower chlorinated congeners are more readily metabolized and eliminated, but the higher chlorinated congeners are poorly metabolized and slowly eliminated. Dioxin-like congeners tend to be poorly eliminated and therefore bioaccumulate more than non-coplanar congeners that do not exhibit dioxin-like toxicity. This often results in an increase in dioxin-like toxicity as PCBs are transferred from prey to predators.

2.1.3 PCB Ecotoxicity

Recent reviews of the ecotoxicity¹⁰ of PCBs include Bosveld and Van den Berg (1994), Barron, et al. (1995), Leonards, et al. (1995), Eisler and Belisle (1996), Hoffman, et al. (1996), Giesy and Kannan (1998), Henshel (1998), Kannan, et al. (2000), and ATSDR (2000). See Section 4.2.2 for discussion of selected toxicological studies.

PCBs have been associated with a range of adverse effects in wildlife including growth, neurobehavioral, hormonal, reproductive, embryotoxic, immunotoxic, and lethal effects. Many, but not all, adverse effects appear to be mediated through the same mode of action as dioxins, and are therefore attributed to the dioxin-like coplanar congeners. However, non-dioxin-like congeners also may be responsible for toxic effects through different modes of action (Fisher, et al. 1998).

One of the most sensitive adverse effects related to PCB exposure is impaired reproduction. Reduced reproductive success results from delayed estrus, delayed hatching, increased mortality (either before or after birth or hatch), increased deformities, and reduced growth (slow growth decreases the long-term survivability of offspring). The parental generation may show no overt signs of toxicity while exposed to PCBs at the doses that impair reproduction. In addition to the direct toxicity of PCBs to embryos and chicks, sublethal neurobehavioral effects in parents (inattentiveness) is a contributing factor in the reduced reproductive success of exposed birds. PCBs have also been associated with impaired immune functions, endocrine (hormonal) disruptions, altered vitamin A regulation, brain asymmetries, and other behavioral effects. There are significant differences in PCB

¹⁰ Ecotoxicity refers to the toxicity of chemicals to ecological receptors (wildlife and plants).

sensitivities between species. Of the relatively few avian species tested, chickens are the most sensitive, followed by pheasants/turkey, ducks, and gulls, in descending order. Of mammals, mink, seals, and primates are particularly sensitive.

2.2 Conceptual Site Model

The conceptual model for PCB exposure to ecological receptors downstream of Neal's Landfill is shown in Figure 1. The PCB exposure routes include direct absorption from water by aquatic organisms, food chain exposures through consumption of PCB-contaminated prey (the "Aquatic prey" category includes anything fish feed on), and sediment ingestion. Fish are shown as receiving an integrated exposure through all exposure routes. Direct absorption of a chemical from water is called "bioconcentration", exposure through food is known as "trophic transfer" or "biomagnification", and the integrated exposure through all routes is referred to as "bioaccumulation" (in this example, the combined effects of bioconcentration, trophic transfer, and sediment ingestion).

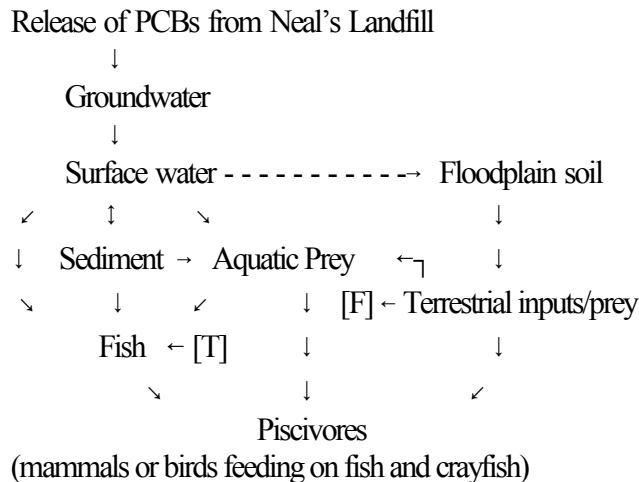


Figure 1. Conceptual Site Model, Neal's Landfill, Bloomington, IN

The symbols [T] and [F] represent terrestrial inputs from the riparian corridor that contribute to the diets of fish. Examples include plant litter (leaves, seeds, flowers, bark, stems, or branches) (Wallace, et al. 1997), and land-based insects or other invertebrates that find themselves, intentionally or otherwise, in the water, or captured from near the water's surface by aquatic animals (Kawaguchi, et al. 2003). The importance of terrestrial inputs is greatest in first order streams, defined as the smallest headwater streams (such as Conard's Branch), and decreases downstream as streams get larger (Lotrich 1973). Terrestrial inputs are assumed to be a minor source of PCB exposure at the site, but data are not available for quantifying the terrestrial contribution to PCB burdens in fish or in piscivores.

The details of the PCB accumulation in the various prey the fish feed on are not shown (various combinations of bioconcentration, sediment exposure, and trophic transfer for each prey species) since, as described below, bioaccumulation in fish is directly measured (not modeled). Although sediment interactions are shown in the conceptual model (exchange of PCBs between surface water and sediment, and sediment ingestion by aquatic organisms), a significant reservoir of sediment-bound PCBs is thought to be unlikely at this site because the main

contaminated sediment deposits were excavated from Conard's Branch and a portion of Richland Creek, Richland Creek is predominantly hard-bottomed (does not have extensive sediment depositional areas), and sediment sampling indicates relatively low PCB levels (Section 1.1). However, confirmation sampling was not performed following the 1987 removal of sediments and banks. Subsequent sampling shows decreasing PCB levels in sediments (Section 1.1), but there is little information concerning bank or floodplain residuals.

Some piscivores may also have terrestrial-based exposures, for example, mink will opportunistically hunt on land as well as in water. The dotted line from Surface water to Floodplain soil in the conceptual site model indicates possible deposition of PCBs in the floodplain during flood events. Terrestrial animals may be exposed to floodplain soil PCBs through direct exposure (soil ingestion and/or dermal absorption), by feeding on soil organisms (for example, earthworms, insect larvae, and other soil invertebrates), and by preying on other animals exposed to soil PCBs (for example, preying on garter snakes that accumulated PCBs by feeding on earthworms). Significant exposure through plant uptake is unlikely because terrestrial plants poorly absorb PCBs from soil (Puri, et al. 1997). Data on PCB levels in terrestrial prey were not collected at the site, and are assigned a value of 0 because of this data gap. Since the primary source of PCBs is through groundwater discharge to surface waters, PCB concentrations are expected to be less in terrestrial animals than fish. This uncertainty does not affect the kingfisher risk estimate because kingfishers feed exclusively on aquatic prey.

The sole aquatic-based PCB exposure route¹¹ considered for piscivorous (fish-eating) mammals and birds is trophic transfer, that is, the exposure from feeding on contaminated fish and crayfish. Additional aquatic-based exposure routes to piscivores include ingestion of water or sediments, and inhalation of PCBs that have volatilized from the water surface. These routes are excluded because their potential contributions are minor compared to the PCB exposure through fish consumption.

Partitioning of PCBs between water and the fat of an aquatic organism is about one to a million, that is, the PCB concentration in fat is expected to be approximately one million times greater than the concentration in water (the comparison is with the "dissolved", "free", or "available" fraction of PCB in water). The fish in Conard's Branch have approximately 5 % lipid content (average of 2001 and 2003 means, Section 3.1). Fish with 5 % lipid content are expected to have about 50,000 times greater PCB concentration compared to the concentration in the water from which they are caught¹². In order to get equivalent exposures, for every pound of fish consumed, an animal would have to drink 6,000 to 8,600 gallons of the water the fish inhabited¹³. This is implausible, for example, mink ingest

¹¹ An exposure route is the specific "gateway" through which a receptor absorbs a contaminant—for animals, either through ingestion, inhalation, dermal absorption (skin or eyes), or injection. The exposure route is the final step of a complete exposure pathway.

¹² $PCB_{\text{fish lipid}} = PCB_{\text{water}} * 1,000,000$, and $PCB_{\text{whole fish}} = PCB_{\text{fish lipid}} * 0.05_{\text{lipid content}}$, so $PCB_{\text{whole fish}} = 1,000,000 * 0.05 * PCB_{\text{water}}$

¹³ One L water weighs 1 kg, 1 lb = 0.4536 kg, and the PCB concentration in fish is about 50,000 times greater than the concentration in water, so $50,000 \text{ L per kg fish} * 0.4536 \text{ kg/lb} = 22,680 \text{ L}$ for the same amount of PCBs in 1 lb of fish. There are 0.264 gal/L, so $22,680 \text{ L} = 5988 \text{ gal}$. This calculation assumes that all the PCBs in surface water are freely dissolved. For the Great Lakes, the dissolved PCB fraction is about 0.7 of the total PCBs in water (USEPA 1997b), which adjusts the calculation to 8,554 gal water for each lb of fish.

only about 0.5 gal water per lb food ¹⁴, which is less than 1 ten-thousandth of the amount required to get the same dose of PCBs from water as from fish consumption.

Similarly, although PCBs will volatilize from contaminated surface water to the atmosphere, the concentrations in air are much less than the concentrations in water—about 0.008 by volume (less than 1 hundredth) according to the results of a global survey (Iwata, et al. 1993 cited in Duursma and Carroll 1996). This means the volatilized air concentration over the creek is likely to be about 2 ten-millionth the concentration in fish with 5 % fat content ¹⁵. Volatilization is a key process influencing the global distribution of PCBs, but is not a significant contributor to animals that feed on contaminated fish because PCBs are so highly magnified in fish tissue.

Incidental sediment ingestion is excluded because of the prior sediment excavation. Even if considered, since sediment ingestion by animals feeding on fish is unlikely to exceed a few percent of their dry-weight diet, the direct contribution of sediments is unlikely to significantly affect the estimated PCB exposure to piscivores based on their consumption of fish ¹⁶. This does not mean that contaminated sediments present no risk to piscivorous wildlife, but rather that the risks associated with contaminated sediments are much greater through a fish consumption pathway as compared to a direct sediment ingestion pathway for chemicals that strongly biomagnify (such as PCBs).

2.3 Assessment Endpoints

The assessment endpoints of the FERA are the reproductive conditions of piscivorous (fish-eating) mammals and birds that inhabit or potentially inhabit Conard's Branch and Richland Creek. Protection of piscivorous wildlife from PCB-related risks is expected to be protective of aquatic organisms (fish, invertebrates, plants), because animals that feed on fish (piscivores) commonly receive higher exposures to PCBs compared to animals that feed on aquatic insects (insectivores) or plants (herbivores), and some piscivores have been shown to be highly sensitive to the effects of PCBs (e.g., mink, seals). For these reasons, federal ambient water quality criteria for protection of wildlife from PCBs are based on effects in piscivores, not on direct effects in aquatic organisms (USEPA 1980, 1995, 1997b, 1999). The same rationale is followed in this FERA—protection of piscivorous wildlife from PCB-related risks is expected to be protective of aquatic organisms as well. Therefore, the health and reproductive condition of fish or aquatic invertebrates (important food source for fish) are not included as assessment endpoints in the FERA. Note that this simplifying assumption is only valid in the river segments in which risk to piscivores is assessed for full utilization of the reach (Area Use Factor of 100 %). An important caveat associated with this approach is that the potential risks of PCB releases to fish and other aquatic organisms are unknown in reaches in

¹⁴ Male mink estimated food ingestion rate of 0.22 kg/kg_{BW}-d, and water ingestion rate of 0.1 kg/kg_{BW}-d (USEPA 1993). Assuming 1 kg mink bodyweight, the food ingestion rate equals 0.485 lb/d, and the water ingestion rate 0.026 gal/d (see the preceding footnote for the units conversion factors). 0.026 gal/d ÷ 0.485 lb/d = 0.54 gal water / lb food.

¹⁵ $PCB_{water} = PCB_{whole\ fish} / (1,000,000 * 0.05)$, and $PCB_{air} = PCB_{water} * 0.008$, so $PCB_{air} = PCB_{whole\ fish} * 0.00000016$ (assuming the ratio of PCB concentrations in water and air by volume are proportional to the ratio of the concentrations by mass.)

¹⁶ This probably would not be true for other receptor groups, for example, direct sediment ingestion could be a significant exposure pathway to an animal feeding on the tubers or rhizomes of aquatic plants (muskrat, geese, swans, and some ducks), partly because of potentially large sediment ingestion, but also because aquatic macrophytes (plants other than algae or fungi) usually do not accumulate high levels of PCBs (therefore not overwhelming the contribution from sediment ingestion).

which only partial utilization by piscivores is assessed (Section 2.6). To address this uncertainty, direct risk to fish is evaluated as additional risk information (Section 5.2.9.2).

Impaired reproduction has obvious implications for the long-term viability of exposed populations, from complete reproductive suppression in extreme cases to reduced reproductive capacity in intermediate cases. Depending on species and habitat, partial reduction of reproductive capacity may suffice in normal (good) years, but may be insufficient for carrying a population through adverse years. Reduced reproductive capacity could also result in a population “sink”—an area that supports adults but not a locally self-sustaining population because of an imbalance between reproduction and mortality. The persistence of a species in a population sink depends on continued immigration of new members from outside “source” areas favorable for population growth. If the habitat in a population sink appears favorable to the receptor, animals may be attracted to the sink despite the negative effects on population, in other words, the population sink becomes a “trap” (Pulliam 1996). For these reasons, any significant reduction in reproductive endpoints is considered to be an ecologically significant adverse effect.

2.4 Risk Question

The risk question for the Neal’s Landfill FERA is whether exposure to PCBs by piscivores feeding on Conard’s Branch/Richland Creek fish and crayfish equals or exceeds the levels reported to cause reproductive impairment in published ecotoxicological studies performed with the same or surrogate species.

Ideally, the risk questions should be stated as testable hypotheses, that is, as refutable statements. Testable hypotheses are often stated in the form of null hypotheses, which describe the situation when no effects occur. However, the null hypothesis approach is not always most appropriate or even feasible depending on the particular research question (Ford 2000). For comparison of measured contaminant levels (or exposures) to regulatory standards or toxicological benchmark values, the recommended procedure is to state the testable hypothesis as the effect of concern (e.g., contaminant concentration $\geq$ benchmark value), and no effects as the alternate hypothesis (e.g., concentration $<$ benchmark value) (USEPA 2000a, b).

The alternate hypothesis describes the situation when the testable hypothesis is contradicted (refuted, rejected) by the results of the investigation. In this case, the alternate hypothesis is that exposures to PCBs by piscivores feeding on Conard’s Branch/Richland Creek fish and crayfish are less than the levels reported to cause reproductive impairment.

2.5 Measurement Endpoints

Two piscivorous (fish-eating) receptors are assessed: mink (*Mustela vison*) to represent mammalian piscivores, and belted kingfisher (*Ceryle alcyon*) to represent avian piscivores.

The Richland Creek/Conard’s Branch habitat is suitable for mink. “Mink inhabit wetland areas of all kinds, including banks of rivers, streams, ... ditches, and other waterways” (Chapman and Feldhamer 1982). The potential presence of mink at the site is demonstrated by a road-killed mink collected from the Highway 48 bridge over Richland Creek on 7/31/04 (between Reaches 2 and 3, see Section 3.2) (Sparks 2005), and a visual sighting of mink near the intersection of Oard Road and Vernal Pike in April 2002 reported by a U.S. Fish and Wildlife Service biologist (King 2005) (near Reach 0). Mink also serve as a surrogate for river otter (*Lutra canadensis*)

which is successfully being reintroduced throughout Indiana. Otter are reported to be “relatively abundant in major nonpolluted river systems and in the lakes and tributaries that feed them” (Chapman and Feldhammer 1982). Assessment of risk to mink is protective for other mammals that include fish, crayfish, or turtles in their diets (usually in lower proportions of the total diet compared to mink), such as raccoon (*Procyon lotor*), striped skunk (*Mephitis mephitis*), or opossum (*Didelphis virginiana*). As discussed in Section 2.3, assessment of mink is expected to be protective for aquatic receptors (fish and invertebrates) in reaches in which full utilization of the reach by mink is assumed.

Kingfisher inhabit wooded waterways with clear and relatively shallow water (USEPA 1993), and have been observed in Richland Creek including the upper portion near Conard’s Branch by USEPA and USFWS biologists. Kingfisher also serve as a surrogate for other piscivorous birds such as herons or egrets. Great blue heron (*Ardea herodias*) was reported in Richland Creek near the confluence with Conard’s Branch (Viacom 2002). Green heron ¹⁷ (*Butorides striatus*) is another potential piscivorous receptor at the site because it feeds in creeks and streams (Kaufman 1996; Robinson 1996).

The measures of exposure are based on site-specific measurements of PCB concentrations in Conard’s Branch and Richland Creek fish (2001, 2002, 2003), and modeled (2001, 2002) or measured (2003) PCB concentrations in crayfish. The fish and crayfish concentrations are used directly to evaluate dietary exposure to mink, and are the basis for modeling dose to kingfisher. The two approaches are necessary because PCB toxicological studies have been performed with mink, allowing direct comparison of dietary concentrations in the field with those in controlled feeding studies, but PCB studies have not been performed with kingfisher, therefore requiring extrapolation of toxic effects between species. Interspecific extrapolation of toxicological data is best performed by converting the dietary concentrations to dose (PCBs ingested per receptor bodyweight per day). In addition to dose to adult birds, kingfisher exposure is also assessed by modeling accumulation of PCBs in kingfisher eggs.

The measures of effects for mink are studies of the reproductive effects of feeding PCB-contaminated fish to mink, and, for kingfisher, laboratory studies with chicken, pheasant, doves, and cormorants, and a field study of bald eagles (described in Section 4.2.2).

2.6 Exposure Assumptions

The following assumptions are made for assessing exposure and risk:

Dietary Composition: Mink—66 % fish and 13 % crayfish by weight based on gut content data for 31 mink collected from Michigan streams (Alexander 1977). Fish (61 %) and amphibian (5 %) categories are combined into the overall fish percentage, and crustacean (11 %) and insect (2 %) categories are combined into the crayfish percentage. The remaining 21 % of the mink diet included birds and mammals (17 %) and unidentified (4 %) ¹⁸. The latter portion of the diet is not modeled in the FERA because site-specific PCB data for terrestrial mammals

¹⁷ Also known as green-backed heron.

¹⁸ There are errors in the data summaries in USEPA (1993) for the Alexander (1977) study. The values reported above are taken directly from Alexander (1977).

and birds are not available. Mink exposure to PCBs is therefore underestimated because of the unknown concentrations in bird/mammal prey, but probably by less than 20 % because terrestrial animals are expected to have less exposure to PCBs, and therefore accumulate less PCBs compared to aquatic animals at this site. The differences in exposure and accumulation between terrestrial and aquatic animals relate to the pathway for release of PCBs from Neal's Landfill—discharge of contaminated groundwater to Conard's Branch and then Richland Creek. An alternative dietary estimate of 88 % fish and amphibians, 4 % crayfish, and 8 % birds, mammals, vegetation and unidentified for mink in riverine habitats in Michigan (Alexander 1977), is not selected because Conard's Branch and the upper portion of Richland Creek are not rivers. The stream data are more applicable to Conard's Branch and Richland Creek.

Dietary Composition: Kingfisher—80 % fish and 20 % crayfish based on a variety of studies reported in USEPA (1993). Alexander (1977) reported 5 % crustacea and insects on a wet weight basis for 17 kingfisher collected from Michigan streams, with the remainder various species of fish (86 %) and amphibians (9 %). Other kingfisher dietary estimates for Midwestern states include 13 % crayfish of the total number of prey items in Ohio creeks (Davis 1982 cited in USEPA 1993), and 41 % crayfish and insects by volume in Michigan streams (Sayler and Langer 1946 cited in USEPA 1993). In both studies, the remainder of the diet consisted of various fish. Assuming roughly equivalent sizes and specific gravities¹⁹ for fish and crayfish prey of kingfisher, the mean value of the various estimates for creeks and streams is 20 % crayfish. Although not directly applicable to Conard's Branch or Richland Creek, Alexander (1977) reported 20 % crayfish by weight in kingfisher gut contents collected from Michigan rivers.

Opportunistic fish consumption: The receptors are assumed to have no dietary preference among the different species of fish, that is, they will feed equally on the fish species collected in a sampling location (stream reach).

Aquatic-based diet: 100 % of the kingfisher diet and 79 % of the mink diet consist of aquatic prey, based on the studies used for dietary composition.

Home range²⁰: Mink—1 stream mile. The home range of radiotracked adult female mink along Swedish streams ranged between 0.6 to 1.7 stream miles, with a mean value of 1.1 miles (Gerell 1970)²¹. The mean home range for adult male mink was 40 % longer, but since the risk estimates for mink are based on reproductive effects, the female home range is used to evaluate site utilization. Home range estimates in stream miles for female mink along Midwestern streams were not located.

Home range: Kingfisher—1 stream mile. Kingfisher breeding territory lengths of 0.64 and 1.36 mile were reported for Ohio and Pennsylvania streams, respectively (Brooks and Davis 1987 cited by USEPA 1993), for a mean value of 1 stream mile. The foraging length is much smaller after the breeding season, for example, a mean value of 0.24

¹⁹ Specific gravity is a measure of the weight per volume of a substance, expressed as the ratio to that of water.

²⁰ "Home range is the area over which an animal normally travels in pursuit of its routine activities" (Jewell 1966 as cited by Gerell 1970). In the FERA, this is a surrogate for the area over which an animal feeds (foraging area).

²¹ The citation for Gerell (1970) in USEPA (1993) is incorrect (conflated with an earlier Gerell publication).

stream miles was reported for Ohio streams in late summer and fall (Davis 1980 cited by USEPA 1993). The breeding season value is used because the risk estimates for kingfisher are based on reproductive effects.

Site utilization: Richland Creek—The fish sample locations in Richland Creek are all greater than 1 mile apart. Since this exceeds the home range of both kingfisher and female mink, the area use factor (AUF) for Richland Creek reaches is 1 (100 % foraging within a reach).

Site utilization: Conard's Branch—The overall length of Conard's Branch is 0.84 miles (4420 ft) from Neal's Landfill to the confluence with Richland Creek. Although this approaches the size of the mink and kingfisher home range, not all of Conard's Branch is likely to support adequate fish populations due to shallowness in the upper portions. While frogs may be present anywhere along Conard's Branch, fish are present in appreciable numbers in the lower (and deeper) portion. For example, Viacom, Inc., reported that significant numbers of fish were first seen about 0.4 mile from the origin of Conard's Branch in August, 2002, although some fish were observed upstream of this location (Viacom 2002). Since 2002 was a dry year, this indicates that at a minimum, 0.44 stream mile of Conard's Branch is capable of supporting appreciable numbers of fish. The proportion supporting significant fish numbers may be greater in wetter years. In 2003, a beaver started to dam Conard's Branch, which would have significantly increased the area supporting fish (the dam was dismantled). Assuming 0.5 mile is capable of supporting significant fish numbers, the AUF would be 0.5 (50 % foraging in Conard's Branch).

Viacom, Inc., initially proposed modeling kingfisher foraging 50 % in Conard's Branch and 50 % in Richland Creek, and mink 25 % in Conard's Branch and 75 % in Richland Creek (Viacom 2002). Viacom, Inc., later withdrew the foraging proposals²², but they are reasonable starting points under the assumption that kingfisher may be more likely to feed on the small fish in Conard's Branch compared to mink. Therefore, the 50-50 and 25-75 allocations between Conard's Branch and Richland Creek foraging by kingfisher and mink, respectively, are used to assess feeding in Conard's Branch and the first downstream reach of Richland Creek as a single exposure unit (referred to as "Reaches 1 & 2", see Section 3.2 for reach information). The AUF for the combined Conard's Branch and first Richland Creek reach is 1 (100 % foraging within the combined exposure unit).

²² In comments on a previous draft of the FERA, Viacom, Inc. stated that the original proposals for allocating utilization between Conard's Branch and Richland Creek were "based solely on the length of Conard's Branch that contained fish relative to published home ranges as a reasonable starting point" (Viacom 2004). According to Viacom (2004), the proposals were withdrawn for two reasons: 1) that Viacom considers the water depth in Conard's Branch to be inappropriate for use by kingfisher, and 2) that USEPA did not concur with another Viacom proposal to average together Richland Creek fish PCB data collected from both above and below the confluence with Conard's Branch for modeling exposure to piscivores. USEPA concurs with the former assertion (1) for the upper portion of Conard's Branch, but not the lower portion in which deep pools are present. The latter proposal (2) was not accepted by USEPA because it meant that risk to mink and kingfisher would be assessed by averaging together unexposed fish (upstream of the source of PCBs) with exposed fish (Conard's Branch and Richland Creek downstream of the confluence). This would be equivalent to averaging together air monitoring data both upwind and downwind of an air pollution source before assessing the potential risks of the air emissions. The appropriate method for investigating the potential risks associated with on-going releases of a pollutant from a stationary source is to assess the risks in the exposed area (in this case, downstream of the source), not arbitrarily diluting the exposure with data from unexposed areas. It should be noted that Viacom (2004) separately argued that kingfisher are unlikely to utilize the "very upper reach of Richland Creek", which directly contradicts Viacom's earlier proposal to include unexposed fish upstream of the Richland Creek-Conard's Branch confluence in the modeled kingfisher diet. Kingfisher have been observed in Richland Creek upstream of Reach 2 (between the Vernal Pike bridge and the confluence with Conard's Branch) by USEPA (James Chapman) and USFWS (Daniel Sparks) biologists.

3 Methods

3.1 Fish Collection and Analysis

3.1.1 2001 Samples

Through a collaborative effort of the Indiana Department of Environmental Management (IDEM), U.S. Fish and Wildlife Service (USFWS), and U.S. Environmental Protection Agency (USEPA), whole fish were collected August 7-9, 2001, from Conard's Branch and Richland Creek, by electro-shocking. The electro-shocker was either towed behind the fish collectors (lower three locations) or carried in a backpack unit by one of the collectors (upper three locations) depending on the depth of the reach. Both whole fish and fish fillet samples were collected, but only the whole-fish data are used in the FERA because piscivorous wildlife consume whole fish. The fillet samples were collected for human health purposes and are not discussed in this report.

Samples were analyzed by Paradigm Analytical Laboratories for total Aroclor using 4-peak quantitation²³ (Aroclor 1248 was the only Aroclor detected) (USEPA method 8082), dioxin-like PCB congeners (IUPAC #77, 81, 105, 114, 118, 123, 126, 156/157, 167, 169, 170, 180/193, and 189)²⁴ (USEPA method 1668 - Revision A), and lipid content (percent fat). The data for congeners 170 and 180/193 are not used in the FERA because they are no longer considered to have significant dioxin-like toxicity (Van den Berg, et al. 1998). The species analyzed on a whole-fish basis include black redhorse (*Moxostoma duquesnei*), common carp (*Cyprinus carpio*), creek chub (*Semotilus atromaculatus*), green sunfish (*Lepomis cyanellus*), longear sunfish (*Lepomis megalotis*), and white sucker (*Catostomus commersoni*). The majority of the whole-fish samples were of individual fish, but some samples were composited when single fish did not provide sufficient mass for laboratory analysis (a composite sample consists of several individuals of a single species collected from a particular reach). The sample identification number of composite samples is underlined in Appendix A1.

At each sample location, an attempt was made to collect 8 omnivorous fish and 5 forage fish for whole-fish analysis. The omnivorous species are green and longear sunfish, and the forage species carp, redhorse, sucker and creek chub. The particular species and numbers collected at each location are shown in Appendix A1. If feasible, a single omnivore and a single forage fish species were collected within a given reach. Two species in the same category were collected within a single reach if there were insufficient individuals of a single species to achieve the target numbers (e.g., Reach 4) (see Section 3.2 for locations), or if a particular species of interest was caught in low numbers but sampled along with a more numerous species of secondary interest (carp versus black redhorse in Reaches 5 and 6). Reach 3S was intended for human health assessment only, but whole fish were collected there as well in case insufficient fish were caught in the upstream locations (1 and 2). Since the sample number from Reach 1 was low, two whole fish samples were analyzed from Reach 3.

²³ Since Aroclors consist of mixtures of large numbers of PCB congeners (more than 100 in environmental samples), the concentration of an Aroclor in a sample is determined by assessing several key peaks in a chromatograph. The chromatograph peaks represent individual or clustered groups of PCB congeners. In this case, 4 key chromatograph peaks are used to determine the Aroclor concentration of the sample.

²⁴ Two congener numbers joined by a slash means that the combined concentration of those two congeners was measured and reported as a single concentration.. This happens when the analytical procedure cannot separate overlapping signals of two congeners.

Upper trophic level fish species were also collected, but were analyzed as fillets for human health risk, and therefore are not discussed in this FERA.

See Sprenger, et al. (2002) for more detailed discussion of field collection and processing methods and analytical results.

3.1.2 2002 Samples

Fish were collected November, 2002, by Normandeau Associates for Viacom, Inc., from Conard's Branch, and Richland Creek both upstream and downstream of the confluence with Conard's Branch. Creek chubs, white suckers, and longear sunfish were collected. The fish were analyzed by Paradigm Analytical Laboratories for total Aroclor (EPA method 8082) by two approaches: 4-peak quantitation and 5-peak quantitation. Both Aroclor results are reported and used in the FERA. A subset of samples from each sample location were also analyzed for dioxin-like PCB congeners (EPA method 1668A). A very small subset was analyzed for total PCBs as the sum of all detected PCB congeners (out of 209 possible congeners), but these data are not used in the FERA because they are too sparse for risk assessment purposes. Lipid contents are low compared to other sampling dates: the mean values for Conard's Branch fish are 0.7 % in 2002, 4.4 % in 2001, and 6.7 % in 2003 (calculated from Appendices B3, A3, and C3). The 2002 lipid data were thought to be anomalously low at first, but samples collected by USFWS in November 2003 also show low lipid content (mean of 1 % in Conard's Branch). Low November lipid content therefore appears to be a seasonal phenomenon, and the lipid data are used as reported. The whole fish data are reported in Appendix B1.

3.1.3 2003 Samples

Fish were collected May 27 and 28, 2003, by Normandeau Associates for Viacom, Inc., from Conard's Branch, and Richland Creek downstream of the confluence with Conard's Branch. Creek chub, white sucker, longear sunfish, shorthead redhorse (*Moxostoma macrolepidotum*), and crayfish (species not reported) were collected. The fish and crayfish were analyzed by Paradigm Analytical Laboratories for dioxin-like congeners (EPA method 1668A), total PCBs as the sum of all the detected PCB congeners, and lipid content. Note that the basis for total PCBs is different in 2003 (sum of detected congeners) from 2001 and 2002 (Aroclor analysis). Additional fish samples were taken by Tetra Tech, contractors providing oversight of the collection effort for USEPA, which were analyzed for dioxin-like congeners by EPA method 1668A by Axys Analytical Services. The Tetra Tech samples are not true "split samples" because they consist of whole fish (a split sample would be homogenized in the field and then divided into two portions for separate analysis), although they were collected at the same location and time as the Normandeau samples. For this reason, the Tetra Tech samples are treated as additional samples in the FERA. The whole fish data are reported in Appendix C1.

3.2 Sample Locations

The sample locations (reaches) are described in Table 1 and shown in Figures 2 and 3.

Table 1. Fish Sample Locations in Richland Creek (RC) and Conard's Branch (CB), Neal's Landfill, Bloomington, IN			
Location Number	Stream miles from RC headwater	Description	Years Sampled for FERA
0	< 0.5	RC upstream of the confluence with CB	2002
1	0.5 (at the confluence)	CB upstream of the confluence with RC	2001, 2002, 2003
2	2.0	RC south of West Vernal Pike bridge	2001, 2002, 2003
3S	3.0	State (IDEM) sample location, RC south of the intersection of Routes 43 and 48	2001
3V	5.5	Viacom sample location, RC downstream of future fire station/community center	2002
4	12.7	RC, Route 43 Bridge south of Hendricksville	2001
5	21.8	RC, northwest of Solsberry near 490N and 800	2001
6	34.5	RC, Route 54 bridge east of Bloomfield	2001



Figure 2. Sampling Locations 0 - 3, Conard's Branch and Richland Creek, Bloomington, IN.



Figure 3. Sampling Locations 4 - 6, Richland Creek, Bloomington, IN

3.3 Data Transformations and Modeling

3.3.1 Non-detection Values

Non-detection values are set equal to 0.1 method quantitation limit (MQL) if any sample within the same reach (sample location) showed a positive detection for that analyte (either an Aroclor or a PCB congener). The rationale is that the laboratory would report an estimated value as low as 0.2 MQL, one-half of which is 0.1 MQL. If an analyte was not detected in any sample within a given reach, it is assigned a zero value. The sole exception is total Aroclor in reach 6 (2001), which was not detected in any sample, but is assigned 0.1 MQL for comparative purposes. The non-detection values are shown in italics in Appendices A, B, and C (0.1 MQL is given).

3.3.2 Dioxin Toxic Equivalent Concentration (TEC) or Quantity (TEQ)

The toxic equivalent concentration approach is based on a shared mechanism of action. Both dioxin and the dioxin-like PCB congeners initiate toxic effects by binding with a particular receptor in cells (aryl hydrocarbon receptor or AhR) which sets off a chain of events that affects the DNA of the cell, which in turn results in hormonal and other changes associated with a variety of adverse effects (Denison and Heath-Pagliuso 1998).

To assess risk to mink, the congener data for each fish sample are converted to World Health Organization (dioxin) toxic equivalent concentrations (WHO-TEC) for mammals according to Van den Berg, et al. (1998). The procedure is to multiply the concentration of each PCB dioxin-like congener by its toxic equivalency factor (TEF), which is the relative potency of that congener in causing toxic responses compared to the most toxic of the dioxin congeners (2,3,7,8-tetrachlorinated dibenzo-*p*-dioxin or TCDD) (Table 2). After each dioxin-like congener concentration in a sample has been multiplied by its respective TEF, the products are summed to give the toxic equivalent concentration (TEC) of the sample (Appendices A1, B1, C1), that is, the concentration of TCDD that would be expected to have the same overall toxicity as the mixture of dioxin-like PCB congeners in the sample.

A similar process is followed to convert modeled PCB dioxin-like congener concentrations in kingfisher eggs to WHO-TECs for birds (Appendices A5, B5, C5), except that the TEFs are based on relative toxicity in bird eggs (Table 2). Since the WHO avian TEFs are based on egg studies, they are not directly applicable to bird diets, but require modeling of congener bioaccumulation into bird tissue (or direct analysis of tissue congener concentrations) before applying the avian TEFs. For the FERA, kingfisher egg TECs are modeled by use of diet-to-egg biomagnification factors (Section 3.3.6).

The WHO mammalian TEFs are used for calculating TEQ ingestion dose to kingfisher because avian dietary ingestion-based TEFs are unavailable, and because, historically, mammalian TEFs have been used in studies of birds with good success.

“From our own research experience, the TEF concept has been applied successfully in a series of experiments to determine the potential risk of increased levels of TCDD and TCDD-like compounds to bird populations, namely, great blue heron (Bellward *et al.*, 1990; Hart *et al.*, 1991; Sanderson *et al.*, 1994a), and double-crested cormorants (Sanderson *et al.*, 1994b) in Canada, and great cormorants (Van den Berg *et al.*, 1994) in The Netherlands. These studies demonstrated that statistically highly significant correlations were observed between TEQ levels, using mammalian TEF values (Safe, 1990), and several

(potentially) Ah receptor-mediated endpoints in several widely different populations of fish-eating birds. ... Recalculation of TEQs using TEFs based on EROD induction in primary chicken hepatocytes (Kennedy *et al.*, 1996) did not improve the statistical correlations significantly ...” [scientific names deleted] (Sanderson and Van den Berg 1999).

For additional risk information, TEFs for fish are used to assess the risk of dioxin-like PCB congeners to the fish themselves (Section 5.2.9.2). Lipid-normalized congener data for whole fish samples (Section 3.3.5) are multiplied by the WHO-TEFs for fish to calculate lipid-normalized TEC in fish (Appendices A4, B4, C4). In this case, only four key congeners (77, 81, 126, and 169) are used to calculate TEC. The remaining dioxin-like congeners are assigned very small TEFs (less than 5 millionth) (Van den Berg, *et al.* 1998), because they have not been associated with adverse effects in fish at the highest concentrations tested.

Table 2. Toxic Equivalency Factors (TEF) for Calculating World Health Organization Toxic Equivalent Concentrations (TEC) (Van den Berg, <i>et al.</i> 1998)			
TEF	IUPAC PCB Congener Numbers		
	Mammals ^a	Birds ^b	Fish ^c
0.1	126	81, 126	
0.05		77	
0.01	169		
0.005			126
0.001		169	
0.0005	114, 156, 157		81
0.0001	77, 81, 105, 118, 123, 189	105, 114, 156, 157	77
0.00005			169
0.00001	167	118, 123, 167, 189	
< 0.000005			105, 114, 118, 123, 156, 157, 167, 189
0	170, 180		

a) Used for dietary TEC exposure to mink and for kingfisher TEQ ingestion dose.

b) Used for kingfisher egg TEC.

c) Used for fish TEC to assess risks to fish.

3.3.3 Species and Dietary UCL and Mean Values

Average values are calculated as the “95 percent upper confidence limit of the mean” (UCL) with the statistical program SYSTAT[®] 8.0 (1998) SPSS Inc., Chicago (see Section 1.2 for an explanation of UCL and the rationale for its use in risk assessments). Two UCL values are calculated—species UCL and dietary UCL (Appendices A2, A4, B2, B4, C2, C4).

Species UCL is the upper estimate of the mean concentration (PCB or TEC) in a given species of fish within a single reach. Species UCLs for crayfish are either extrapolated from fish UCLs in the same reach (2001 and 2002) (Section 3.3.4), or calculated directly from crayfish data collected from the site (2003).

Dietary UCL represents the overall PCB concentration or TEC in the mink or kingfisher diet, which is the aggregate of the concentrations in the different species of fish collected from a reach and the modeled or measured concentration in crayfish. The concentration in the fish component is calculated as the arithmetic average of the species UCLs within a single reach (PCB or TEC), that is, each species of fish analyzed in a particular reach contributes equally to the fish component of the diet regardless of their relative abundances. The overall dietary UCL for a given reach is calculated by weighting the fish and crayfish components by the dietary composition of the receptor (Section 2.6):

$$\text{Mink dietary UCL} = (0.66 * \text{mean fish species UCLs}) + (0.13 * \text{crayfish UCL}) \quad [1]$$

$$\text{Kingfisher dietary UCL} = (0.8 * \text{mean fish species UCLs}) + (0.2 * \text{crayfish UCL}) \quad [2]$$

Similarly, species UCLs are calculated for each of the dioxin-like PCB congeners (Appendices A4, B4, C4) on a lipid-normalized basis (Section 3.3.5) as a step in modeling uptake in kingfisher eggs (Section 3.3.6) and assessing direct risk to fish (Section 5.2.9.2).

Note that the UCL calculation itself is only performed for one step (calculation of species UCL) and is not reapplied in subsequent operations such as calculating dietary UCL (which would result in compounded conservative calculations). Only the fish contaminant data (PCBs, TEC, and PCB congener concentrations) are assessed as UCLs, the other exposure data are not modified (ingestion rate or biomagnification factors).

For comparative purposes, the analyses are repeated with mean values instead of UCL values (Appendices A7, A8, B7, B8, C7, C8). Species mean is the arithmetic average concentration in a given species of fish within a single reach. Dietary mean is calculated the same as dietary UCL, except that it is based on species mean data instead of species UCLs.

3.3.4 Modeled PCB Uptake by Crayfish

Site-specific data on PCB levels in crayfish were not collected in 2001 or 2002. Crayfish Aroclor concentrations are modeled in 2001 and 2002 by dividing measured fish concentrations by species-specific fish-crayfish PCB concentration ratios based on crayfish and fish data collected in Conard's Branch and Richland Creek in August 1982 and May 1998 (Appendix D). Fish-crayfish PCB concentration ratios are calculated by date and location, for example, mean PCB concentration in creek chubs collected 8/82 at stream mile 0.4 are divided by the crayfish PCB concentration collected on the same date and location. The mean fish-crayfish ratios are calculated by taking the average of all of the available location- and date-specific ratios for the species under consideration, for example, the mean creek chub/crayfish ratio of 2.65 is the arithmetic average of 4 creek chub/crayfish ratios—2.06 at mile 0.4 (8/82), 3.95 at mile 0.5 (5/98), 2.60 at mile 1 (8/82), and 2.00 at mile 1 (5/98) (Appendix D). The fish-crayfish PCB ratios for longear and green sunfish are averaged together to increase the sample size for the mean sunfish-crayfish PCB ratio.

The preferred ratios for modeling Aroclor concentrations in crayfish are based on the largest number of comparisons (creek chub with 4 comparisons, and combined longear and green sunfish with 4 comparisons). When both creek chub and sunfish data are available at a 2001 or 2002 sample location, the mean of the concentrations in creek chubs and sunfish is divided by the mean PCB ratio for creek chub-longear sunfish-green sunfish/crayfish in 8/82 and 5/98 (Appendix D).

In one case, Aroclor concentration in crayfish is modeled from white sucker data (location 0, 2002), because Aroclor was not detected in creek chub at that location (Richland Creek upstream of the confluence with Conard's Branch). Extrapolation of Aroclor concentration in crayfish from non-detect values in creek chubs would not be informative. Aroclor was detected in white suckers at that location. Two white sucker/crayfish PCB ratios are available in the historic data base, but they differ by an order of magnitude—0.39 at 0.5 stream miles (5/98) and 3.51 at 1 stream mile (5/98) (Appendix D). The ratio at stream mile 0.5 is based on a single white sucker, and at stream mile 1 on 3 white suckers. Data based on a single fish sample is highly uncertain, so, instead of using the mean ratio of the two comparisons, the 3.51 ratio at stream mile 1 is used to model crayfish Aroclor concentration at location 0.

The same PCB ratios used to model Aroclor concentration in crayfish also are used to model TEC in crayfish from fish TEC in the same reaches (Appendices A2, A7, B2, B7), under the assumption that relative differences in TEC between species is proportional to the relative differences in total PCBs.

Lipid-normalized concentrations of the dioxin-like PCB congeners are necessary for modeling uptake in kingfisher eggs (Sections 3.3.5 and 3.3.6). UCL lipid-normalized congener concentrations in 2001 and 2002 crayfish are modeled by dividing fish species UCL lipid-normalized congener concentrations by the respective ratios of the mean lipid-normalized congener concentrations in fish and crayfish in 2003 (for either creek chub or longear sunfish) (Appendices A4 and B4, based on Appendix C8). Mean lipid-normalized congener concentrations in 2001 crayfish are modeled in the same manner from fish species mean lipid-normalized congener concentrations (Appendices A8 and B8).

3.3.5 Lipid Normalization

Lipid normalization is performed by dividing the concentration of PCBs or PCB congeners in a fish or crayfish sample by the lipid (fat) content of that sample. The result is the concentration of PCBs or individual congeners in the fat tissues of the animal that was sampled. The reason for this data transformation is that PCBs are highly lipophilic (fat-loving), which means that most of the PCBs absorbed by an animal reside in its fat tissue. Lipid normalization is necessary for the FERA because uptake of PCB congeners in kingfisher eggs is modeled on a lipid-normalized basis (from lipid-normalized dietary concentration to lipid-normalized egg concentration) (Section 3.3.6). Also, the toxicity reference value (TRV) for evaluating direct risk to fish is reported as a lipid-normalized value (Section 5.2.9.2).

Lipid content was not reported for some of the 2001 fish samples. In order to perform lipid normalization for each of the samples, the missing lipid values are set equal to the mean lipid content of other samples of the same species collected from within the same reach in 2001. For example, 3 green sunfish samples were collected from Location 1, but lipid content was reported for 2 samples (2.93 and 3.80 %). The lipid content of the third sample is set equal to the average of the reported lipid contents for green sunfish at location 1 (3.36 %) (Appendix A3). The calculated lipid values are shown in bold in Appendix A3.

3.3.6 Modeled PCB Uptake in Kingfisher Eggs

Uptake of dioxin-like PCB congeners in kingfisher eggs is modeled by multiplying the congener-specific lipid-normalized dietary concentrations (Appendices A4, A8, B4, B8, C4, C8) by the respective diet-to-egg biomagnification factors (BMF), which gives the lipid-normalized concentration in eggs for each congener (Appendices A5, A9, B5, B9, C5, C9). The BMFs are empirical, calculated for each dioxin-like congener as the geometric mean of the lipid-normalized BMFs from several field studies of PCB congener concentrations in eggs of piscivorous birds (eagle, guillemot, gulls) and their prey (salmon, herring, carp, shad, silver bass, smallmouth bass) by Blankenship and Giesy (2002), with missing congener BMFs supplemented by diet-to-egg BMFs from a field study of great cormorant by Murata, et al. (2003) (Appendix A5).

The lipid-normalized avian TEC of kingfisher eggs is calculated by multiplying the modeled dioxin-like congener concentrations in the eggs by the respective avian TEFs (Table 2) and summing the results (Appendices A5, A9, B5, B9, C5, C9). The avian TEFs are based on the relative potencies of PCB congeners and TCDD injected in bird eggs (Van den Berg, et al. 1998), and are therefore the appropriate TEFs for calculating the dioxin-like potency of PCB congeners in bird eggs.

Lipid-normalized diet-to-egg BMFs are available for most of the dioxin-like PCB congeners with the exception of 123, 167, and 189 (Appendix A5). The omitted congeners have a relatively small effect on the overall TEC in the eggs because their TEFs are very small (0.00001). In one example, 98 to 99 % of the total TEC attributable to PCBs in tern eggs was contributed by congeners 77, 105, 118, 126, and 169, and only 1 to 2 % of the total TEC was contributed by the remaining dioxin-like PCB congeners detected in the eggs (calculated from Smith, et al. 1990) (the eggs were collected from tern colonies at Green Bay, WI, with elevated PCB exposures related to paper company operations along the Fox River).

3.3.7 Outlier Data

Data are used as reported with a single exception. A “background” location, Richland Creek upstream of the confluence with Conard’s Branch (Reach 0), was sampled in 2002. One of the white suckers collected in Reach 0 appears to have recently moved upstream from Reach 2 (downstream of Conard’s Branch). The TEC for sample ID 15 (9.4 pg/g) is an order of magnitude (10X) larger than the mean TEC for the remaining Reach 0 white sucker samples (0.5 pg/g), a difference equivalent to 80 standard deviations of the remaining Reach 0 white sucker samples (Appendix B7). The sample ID 15 white sucker therefore appears to have recently moved into Reach 0, supported by the similarity between the sample ID 15 contaminant concentrations with the lower range for 2002 Reach 2 white sucker (12 - 14 pg/g TEC, sample IDs 23 and 24) (Appendix B1). For this reason, sample ID 15 is excluded from all Reach 0 calculations.

4 Site Investigation and Data Analysis

Exposure and effects are characterized both for total PCBs and TEC for both mink and kingfisher.

4.1 Characterization of Exposure

4.1.1 Exposure Methods

The exposures for both receptors (mink and kingfisher) are based on the measured PCB concentrations in whole-body fish collected from the site, and modeled or measured PCB concentrations in crayfish. The exposure data are handled differently for the two receptors. PCB toxicological studies have been performed with mink, so the risk may be directly estimated by comparing the dietary concentrations in the field with the dietary concentrations used in controlled feeding studies. Since PCB toxicological studies have not been performed with kingfishers, it is necessary to model total PCB and dioxin toxic equivalent (TEQ)²⁵ doses to kingfishers so that the estimated doses in the field can be compared to the doses given to the surrogate species that have been tested. The PCB or TEC concentrations in the kingfisher diet are converted to kingfisher doses by multiplying the reach dietary concentration by a kingfisher food ingestion rate of 0.5 kilogram food per kilogram bodyweight per day (kg food/kg_{BW}-d) (Alexander 1977) (Appendices A2, A7, B2, B7, C2, C7).

The method for modeling uptake of dioxin-like PCB congeners in kingfisher eggs is described in Section 3.3.6.

4.1.2 Exposure Characterization

4.1.2.1. Fish and Crayfish Data

The measured Aroclor or total PCB, measured dioxin-like PCB congener concentrations, and calculated TEC in individual fish samples are shown in Appendices A1, B1, and C1. The UCL and mean concentrations by species are summarized in Tables 3 (Aroclor and total PCB) and 4 (TEC) for Conard's Branch and the first two downstream reaches of Richland Creek. See Appendices A2 and A7 for the UCL and mean concentrations in fish and crayfish in Reaches 4 - 6 in 2001 (these reaches were not sampled in 2002 or 2003). Note that the UCL and mean concentrations in crayfish are modeled in 2001 and 2002, but measured in 2003 (Section 3.3.4).

The fish data show a very pronounced spatial gradient consistent with a PCB source from Neal's Landfill. The concentrations of both total PCBs and TEQ are highest in the fish in Conard's Branch (Reach 1), and exponentially decline to the farthest downstream locations (Reaches 5 and 6). Spatial trends in mean concentrations of PCBs and TEC in sunfish in 2001 are illustrated in Figures 4 and 5 (see Table 1 for the sample locations corresponding to the distances in the figures). Sunfish data are used to illustrate spatial trends because sunfish were collected from all but one of the 6 sample locations in 2001. The mean PCB concentration in sunfish in Conard's Branch (7.6 mg/kg)²⁶ is 2 orders of magnitude (100 times) higher than in sunfish in Reach 5 (0.07 mg/kg), the farthest downstream location in which Aroclor was above the detection limit for fish samples. Similarly, the mean TEC is 500 times higher in Conard's Branch sunfish (196 pg/g) compared to Reach 6 sunfish (0.4 pg/g)²⁷.

²⁵ TEC is used for dioxin toxic equivalent concentrations (Section 3.3.2). TEQ is used for the total quantity (not concentration) of dioxin toxic equivalents. In the FERA, TEC is used for concentrations in fish, crayfish, diet, and kingfisher eggs, but TEQ is used for kingfisher dose because a dose is the amount of a toxicant ingested (or otherwise absorbed) by an animal per kilogram bodyweight per day.

²⁶ 1 mg/kg = 1000 µg/kg

²⁷ The detection limits for dioxin-like PCB congeners are lower than for Aroclor, so dioxin-like congeners were detected in Reach 6 fish even though Aroclor was not.

The recent fish data do not exhibit a consistent temporal trend²⁸. The following comments are overall impressions of the data, and are not based on formal trend analyses. The November 2002 samples have the lowest PCB and TEC concentrations of the years sampled for the FERA, but the May 2003 samples have the highest PCB concentrations (Tables 3 and 4). Part of the difference may be due to the change in analytical procedure (Aroclor analysis in 2001 and 2002, and sum of total congeners in 2003), as well as differences in fish lipid content (low in November), seasonal effects (PCB solubility is higher in warmer water), and changes in rainfall patterns between sample events (affecting release of PCBs from Neal's Landfill). On a TEC-basis, the August 2001 samples mostly have the highest concentrations of the years sampled for the FERA, but the reduction in TEC observed in November 2002 was reversed in May 2003, which mostly showed TEC values intermediate between those of 2001 and 2002.

4.1.2.2. Mink and Kingfisher Exposures

UCL PCB and TEC exposure estimates (mink dietary concentration, kingfisher dose, and kingfisher egg concentration) are summarized in Tables 5 - 9. See Appendices A7, A9, B7, B9, C7, and C9 for mean exposure estimates.

Mink dietary concentrations, kingfisher doses, and kingfisher egg concentrations reflect similar trends as the fish data because they are modeled from Conard's Branch and Richland Creek fish (and 2003 crayfish) PCB data. The modeled exposures are lowest in November 2002, and generally highest in May 2003 on a PCB-basis and August 2001 on a TEC-basis.

The exposure estimates are affected not only by annual and seasonal differences in PCB concentrations in fish (Section 4.1.2.1), the change in analytical technique for determining total PCBs (Section 3.1.3), and the shift from modeled uptake of PCBs by crayfish (Section 3.3.4) to measured PCB concentration in crayfish (Section 3.1.3), but also by differences in the species composition collected at each sample location (Table 5). Sunfish and creek chub were collected from Conard's Branch (Reach 1) in 2001, but only creek chub was collected in 2002 and 2003. Conversely, creek chub, sunfish, and white sucker were collected from Richland Creek (Reach 2) in 2002 and 2003, but only sunfish and white sucker in 2001. Creek chub was collected from Reach 3S in 2001, but shorthead redhorse was collected from Reach 3V in 2003. The change in species composition affects the exposure estimate because it is assumed that mink and kingfisher do not discriminate among the fish species collected from a sample location, that is, the exposure modeling is based on equal contribution of each of the fish species analyzed from a particular reach in a given year (Section 2.6).

²⁸ A formal trend analysis is outside of the scope of the FERA, but is being looked at in a separate effort.

Table 3. PCB Concentrations in Fish and Crayfish in Reaches 1 - 3 (µg/kg ww), Neal's Landfill Site, Bloomington, IN^a				
Species	Basis	August 2001 ^b	November 2002 ^c	May 2003 ^d
Conard's Branch–Reach 1				
CRC	UCL	7668	6381 - 7689	12,015
	mean	6233	4050 - 4893	10,360
GS	UCL	11,114	NA	NA
	mean	7633	NA	NA
CF	UCL	3141 ^e	2408 - 2902 ^e	8509
	mean	2319 ^e	1528 - 1847 ^e	3833
Richland Creek–Reach 2				
CRC	UCL	NA	471 - 587	1572
	mean	NA	269 - 330	1107
LS	UCL	804	915 - 1143	1895
	mean	599	476 - 590	1101
WS	UCL	3390	1913 - 2348	2712
	mean	2100	1186 - 1473	2127
CF	UCL	242 ^e	232 - 289 ^e	401
	mean	180 ^e	102 - 154 ^e	343
Richland Creek–Reaches 3S (2001) and 3V (2003)				
CRC	UCL	347	NA	NA
	mean	220	NA	NA
SRH	UCL	NA	NA	915
	mean	NA	NA	448
CF	UCL	131 ^e	NA	NA
	mean	83 ^e	NA	NA

a) Appendices A2, A7, B2, B7, C2, C7

b) Aroclor based on 4-peak analysis

c) Aroclor based on 4-peak and 5-peak analyses

d) Total PCBs based on the sum of all detected PCB congeners

e) Modeled from fish data

Table 4. Dioxin Toxic Equivalent Concentration (TEC) in Fish and Crayfish (pg/g, ww) in Reaches 1 - 3, Neal's Landfill, Bloomington, IN ^a				
Species	Basis	August 2001	November 2002	May 2003
Conard's Branch–Reach 1				
CRC	UCL	278	109	150
	mean	203	71	127
GS	UCL	414	NA	NA
	mean	196	NA	NA
CF	UCL	116 ^b	41 ^b	67
	mean	67 ^b	27 ^b	48
Richland Creek–Reach 2				
CRC	UCL	NA	10	28
	mean	NA	7	20
LS	UCL	59	25	30
	mean	33	14	19
WS	UCL	59	40	40
	mean	26	25	31
CF	UCL	18 ^b	6 ^b	8
	mean	10 ^b	4 ^b	7
Richland Creek–Reaches 3S (2001) and 3V (2003)				
CRC	UCL	10	NA	NA
	mean	5	NA	NA
SRH	UCL	NA	NA	16
	mean	NA	NA	7
CF	UCL	4 ^b	NA	NA
	mean	2 ^b	NA	NA

a) Appendices A2, A7, B2, B4, C2, C7

b) Modeled from fish data

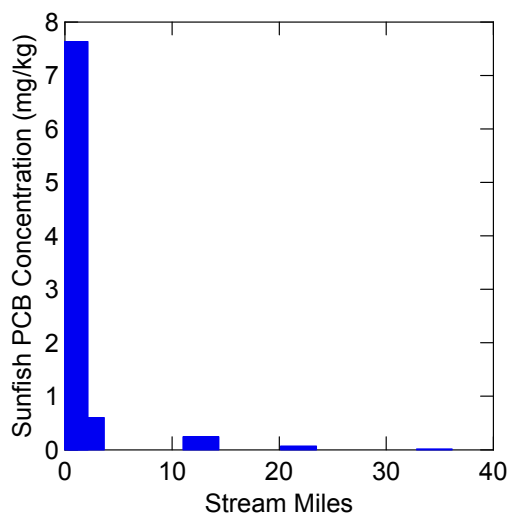


Figure 4. Mean Aroclor Concentration in Green and Longear Sunfish in Conard's Branch and Richland Creek, 8/01, Neal's Landfill Site, Bloomington, IN (Appendix A7).

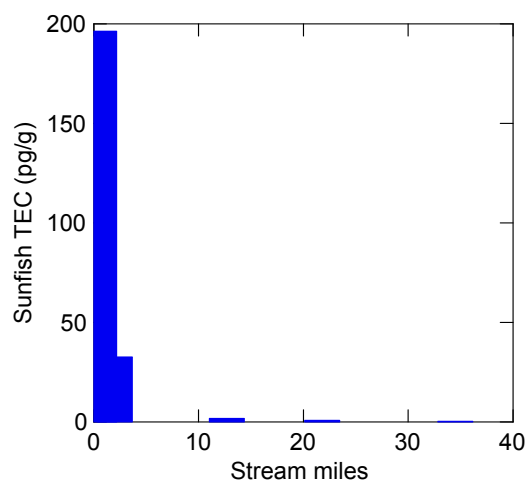


Figure 5. Mean Dioxin Toxic Equivalent Concentration (TEC) in Green and Longear Sunfish in Conard's Branch and Richland Creek, 8/01, Neal's Landfill Site, Bloomington, IN (Appendix A7).

Table 5. Mink Dietary UCL PCB ($\mu\text{g/kg}$, ww), and Species Sampled						
Reach	August 2001		November 2002		May 2003	
	Aroclor ^a	Species ^b	Aroclor ^a	Species ^b	Congener Sum ^a	Species ^b
0	NA		192 - 216	CRC, WS	NA	
1	6606	CRC, GS	4524 - 5452	CRC	9036	CF, CRC
2	1416	LS, WS	756 - 935	CRC, LS, WS	1411	CF, CRC, LS, WS
1 & 2	2713		1698 - 2064		3318	
3S	246	CRC	NA		NA	
3V	NA		NA		723	SRH
4	324	BRH, LS, WS	NA		NA	
5	111	BRH, CP, LS	NA		NA	
6	11	BRH, CP, LS	NA		NA	

a) Appendices A2, B2, C2

b) The species acronyms are defined in the footnotes to Appendices A1, B1, C1

Table 6. Mink Dietary UCL TEC (pg/g, ww) ^a			
Reach	August 2001	November 2002	May 2003
0	NA	3	NA
1	243	77	108
2	41	17	23
1 & 2	92	32	44
3S	7	NA	NA
3V	NA	NA	13
4	4	NA	NA
5	0.6	NA	NA
6	0.4	NA	NA

a) Appendices A2, B2, C2

Table 7. Kingfisher UCL PCB Dose ($\mu\text{g}/\text{kg}_{\text{BW}}\text{-d}$)^a			
Reach	August 2001 (Aroclor)	November 2002 (Aroclor)	May 2003 (sum of congeners)
0	NA	119 - 134	NA
1	4070	2793 - 3366	5657
2	863	463 - 573	864
1 & 2	2467	1628 - 1969	3260
3S	152	NA	NA
3V	NA	NA	458
4	198	NA	NA
5	68	NA	NA
6	7	NA	NA

a) Appendices A2, B2, C2

Table 8. Kingfisher UCL TEQ Dose ($\text{ng}/\text{kg}_{\text{BW}}\text{-d}$)^a			
Reach	August 2001	November 2002	May 2003
0	NA	2	NA
1	150	48	67
2	25	11	32
1 & 2	88	29	40
3S	4	NA	NA
3V	NA	NA	8
4	2	NA	NA
5	0.4	NA	NA
6	0.3	NA	NA

a) Appendices A2, B2, C2

Table 9. Kingfisher UCL Egg TEC ($\mu\text{g/kg}$ lipid) ^a			
Reach	August 2001	November 2002	May 2003
0	NA	6	NA
1	670	1200	226
2	226	254	73
1 & 2	448	728	149
3S	53	NA	NA
3V	NA	NA	24
4	27	NA	NA
5	3	NA	NA
6	0.8	NA	NA

a) Appendices A5, B5, C5

4.2 Characterization of Effects

4.2.1 Effects Methods

Characterization of PCB effects is based on laboratory and field toxicological studies. In addition to describing potential effects, the studies are used to derive toxicity reference values (TRVs). TRVs are the threshold levels of a chemical (either concentration or dose) that bound the occurrence of adverse effects in animals or plants. For dietary concentrations, the TRVs are the no and lowest observed adverse effect concentrations (NOAEC and LOAEC, respectively) in food. The NOAEC is the highest dietary concentration of a toxic substance in a study that did not result in adverse effects, and the LOAEC is the lowest dietary concentration that resulted in adverse effects. The same terms are used for the TRVs for egg concentration. For doses, the TRVs are the no and lowest observed adverse effect levels (NOAEL and LOAEL, respectively). The NOAEL is the highest dose in a study that did not result in adverse effects, and the LOAEL is the lowest dose that resulted in adverse effects.

In the FERA, the terms “NOAEC”, “NOAEL”, “LOAEC” and “LOAEL” are used when the TRVs are based on statistical comparisons among treatments. Statistical comparisons address whether or not the incidence (frequency) or severity of an adverse effect in an exposed (treatment) group is statistically greater than the effect in a less exposed (alternate treatment) or unexposed (control) group (a control group should be maintained the same as exposed groups except for the difference in exposure). The terms “no effect” and “low effect” are used when the TRVs are interpolated from dose-response or exposure-response curves based on aggregated data from multiple studies (meta-analysis). The interpolation approach is based on the shape of the dose-response or exposure-response

relationship, so it is no longer restricted to the observed effects at the specific concentrations tested, but can address the responses at intermediate concentrations between those tested ²⁹.

At least four approaches may be followed when the results of multiple toxicological studies are available for a particular chemical and receptor: 1) select the single study that has the highest scientific quality or applicability to the site, 2) average the TRVs from the studies that are judged to be scientifically valid, 3) combine all the valid study results into a single exposure-response or dose-response plot which is then used to interpolate the threshold concentrations or doses corresponding to selected effect levels (meta-analysis approach), or 4) compile all the TRVs in ascending order for comparison with exposure at the site.

All of these approaches are used in the FERA for one or more TRVs, depending on the available data. For approach 2, the TRVs from multiple studies of equivalent scientific quality are averaged together by taking the geometric mean ³⁰. For approach 3, the no effect level is defined as a 10 % decrease in a reproductive or growth endpoint compared to control values, and the low effect level as a 25 % decrease. Effect size is the amount of decrease in response of animals exposed to a chemical, as compared to unexposed controls, that is selected as the level of concern for assessing risk. A pragmatic consideration is to avoid choosing an effect size for which interpolation may be strongly model dependent. In an examination of aquatic toxicity data sets, Moore and Caux (1997) concluded that interpolation becomes strongly model-dependent for less than 10 % decreases in response compared to that of controls (see also Scholze, et al. 2001). The various regression models gave reasonably consistent results for response differences of at least 10 % compared to controls. A related consideration is the effect size commonly associated with statistically-determined LOAECs in well-designed toxicity studies. The LOAECs of the toxicity studies for the ambient water quality criteria (AWQC) and pesticide programs generally correspond to 20 to 25 % effect sizes (Suter, et al. 2000), and interpolation of the 25 % effect size is recommended for effluent toxicity testing (e.g., Klemm, et al. 1994). Another pragmatic consideration is consistency with the basis for regulatory decision-making in other programs that utilize toxicity testing results. A *de minimis* effect size of 20 % was identified in one such review (summarized in Suter, et al. 2000) ³¹. This indicates that a reasonably detectable effect size consistent with Agency practices in other programs would fall between 20 and 25 %. The higher of these values is chosen for the FERA to ensure that the low effect size represents a non-trivial departure from the control response. Similar to the rationale for the choice of low effect size, 10 % is chosen for no effect size because it is unlikely to be identified as a LOAEL in a reasonably well-designed toxicity study, is lower than the *de minimis* effect-level identified in a review of regulatory decision-making, but is at the minimum size so that the interpolated TRV is not strongly model-dependent (various regression techniques will likely give similar values).

An issue regarding consistency with TRVs used in other ERAs was raised in meetings with Viacom, Inc.. To address this issue, a detailed review of PCB and TEC TRVs and their derivations in recent ERAs was performed

²⁹ The interpolation approach is referred to as ED_x (ED - effective dose, x - the selected effect level of concern) or EC_x (effective concentration).

³⁰ One way to calculate a geometric mean is to multiply each of the values together, then take the root set equal to the reciprocal of the number of values being averaged. For example, to take the geometric mean of 3, 5, and 10, first multiply them together (3 x 5 x 10 = 150), then take the cubic root (because 3 values are being averaged) ($150^{1/3} = 5.3$). The arithmetic average for this example is 6.

³¹ 20 % is not a standard written in the regulations, but is the minimum effect size associated with regulatory actions in practice.

(Appendix E). The review focused on ERAs in USEPA Region 5 (Great Lakes Initiative water quality criteria for protection of wildlife; Fox River, WI; Kalamazoo River, MI; Sheboygan River and Harbor, WI; and Upper Green Bay, WI), but also included the Hudson River, NY, because it is a high profile PCB Superfund site, and the results of a recent mink feeding study performed for the Housatonic River, MA. Of these, the following ERAs were externally peer-reviewed: Great Lakes Initiative, Fox River, Housatonic River, and Hudson River. The meta-analysis of mink and avian PCB TRVs from aggregated studies performed with Aroclors (approach 3 above) was internally peer-reviewed by USEPA (Appendix D of the ERA for the Kalamazoo River, and the FERA Addendum). One non-governmental ERA is included (Blankenship and Giesy 2002) because the methodology and TRVs are applied in the FERA (Appendix E6). In addition to the review of TRVs from previous ERAs, two new TRV derivations are included in Appendix E: mink TEC TRV (Appendix E2), and revised avian TEC TRV (Appendix E4).

In the cases in which large divergences in TRVs occur with no clear basis for selecting between the values, both sets of TRVs are selected for the FERA (kingfisher PCB dose TRVs and kingfisher egg TEC TRVs). Single values are selected for the remaining TRVs (mink dietary PCB and TEC TRVs, and kingfisher TEQ dose TRVs) (Table 10).

4.2.2 Toxicity Reference Values

The toxicity reference values used in the FERA are summarized in Table 10, and discussed in Sections 4.2.21 through 4.2.2.5.

4.2.2.1 Mink Dietary PCB TRVs

Mink PCB TRVs and their derivations are shown in Appendix E1, both on a dose and dietary basis. The dietary TRVs selected for the FERA are 500 µg/kg no effect and 600 µg/kg low effect (Kalamazoo River ERA). The details of the TRV derivation are summarized in the FERA Addendum. The TRVs are interpolated (Section 4.2.1 approach 3) from an exposure-response plot based on the combined results of three mink feeding studies performed with Aroclor 1254 added to the mink food (Aulerich and Ringer 1977; Kihiström, et al. 1992; Wren, et al. 1987a,b). The toxicological endpoints are live kit production per mated female (which integrates the effects on whelping rate³², litter size, and the proportion of live kits at birth), and kit bodyweight. The exposure durations of the Aroclor feeding studies did not exceed a single breeding season, however, mink studies with Clophen A50 (European PCB product) and field-contaminated prey have shown increased adverse effects when exposures continue over two breeding seasons or generations compared to the effects of exposure over one breeding season (Section 4.2.2.2). Since no mink Aroclor feeding studies were located that continued exposure beyond a single breeding season, the interpolated single-breeding season exposure TRVs were adjusted for continuous exposure over multiple breeding seasons or generations by multiplying by the mean ratio of the interpolated 2-season or 2-generation exposure TRVs for the Clophen A50 (Brunström, et al. 2001)³³ and field-contaminated prey studies

³² Proportion of mated females giving birth.

³³ The Brunström, et al. (2001) single-breeding season exposure effects data are supplemented with the results of a Clophen A50 treatment by Kihiström, et al. (1992).

(Restum, et al. 1998) divided by the respective single-breeding season exposure TRVs (mean ratio of 0.52 for live kit production, kit bodyweight, and kit survival endpoints). The details of the exposure duration adjustment are presented in the FERA Addendum.

Table 10. Toxicity Reference Values (TRVs), Neal's Landfill, Bloomington, IN						
TRV	PCBs			Dioxin Toxic Equivalents		
	Mink Dietary TRVs					
Type	no effect	low effect	Units	NOAEC	LOAEC	Units
Value	500	600	µg PCB / kg diet	4.6	18	pg TEC / g diet
	Kingfisher Ingestion Dose TRVs					
Type	no effect	low effect	Units	NOAEL	LOAEL	Units
Value	400	500	µg PCB / kg _{BW} -d	1.4	14	ng TEQ / kg _{BW} -d
Type	NOAEL	LOAEL	Units			
Value	110	1120	µg PCB / kg _{BW} -d			
	Kingfisher Egg TEC TRVs					
Type				NOAEC	LOAEC	Units
Value				1.8	5.3	µg TEC / kg lipid
Value				17	68	µg TEC / kg lipid

Of the other ERAs reviewed in Appendix E1, the LOAECs for the Great Lakes Initiative (GLI) and the Housatonic River are markedly higher, the LOAEC for the Hudson River is markedly lower, and the LOAEC for the Fox River is consistent with the low effect TRV in the FERA. The LOAEC for the GLI (2000 µg/kg) resulted in complete reproductive suppression, so is inadequately protective for regulatory purposes (the GLI water quality criteria are based solely on no effect levels, and the LOAEC served only as a starting point for estimating a NOAEC). The Hudson River LOAEC of 250 µg/kg is based on the Restum, et al. (1998) 2-breeding season and 2-generation exposure study, but interpretation of the results on an Aroclor basis is complicated by possible additive or multiplicative effects of co-contaminants other than PCBs (the study was performed with field-contaminated carp collected from Saginaw Bay, MI). The same consideration applies to the Heaton, et al. (1995) study, also performed with Saginaw Bay carp, but over a single-breeding season exposure duration.

A site-specific mink feeding study was performed for the Housatonic River Superfund site (Bursian, et al. 2006a). The dietary concentration of the treatment resulting in decreased kit survival (3700 µg PCB/kg diet) is higher than the LOAEC TRVs used at other Superfund sites, but resulted in high kit mortality (54 %). The investigators

performed probit regression analysis³⁴ to calculate the dietary concentrations lethal to 20 % and 10 % of kits (LC₂₀ and LC₁₀, respectively) and the associated 95 % confidence intervals (CI). The LC₂₀ is 984 µg PCB/kg diet (CI: 472 – 1940 µg/kg), and the LC₁₀ is 231 µg PCB/kg diet (CI: 26 – 483 µg/kg) (Bursian, et al. 2006a). The Bursian, et al. (2006a) LC₂₀ differs from the FERA low effect TRV by less than a factor of 2, reasonably consistent with the observed difference in toxicity between PCB exposure over 1 breeding season versus exposure over 2 breeding seasons. In contrast, the Bursian, et al. (2006a) LC₁₀ is lower (more conservative) than the FERA no effect TRV. However, the rounded 95 % confidence intervals for the Bursian, et al. (2006a) LC₂₀ and LC₁₀ include the values of the low effect (600 µg PCB/kg diet) and no effect (500 µg/kg) TRVs, respectively, used in the FERA.

The mink feeding study performed for the Housatonic River Superfund site is a high quality single-breeding season exposure study, but the applicability to the Neal's Landfill site is uncertain because of the differences in the major Aroclors released at each site. The predominant Aroclor released to the Housatonic River was Aroclor 1260 (USACE/USEPA 2004), reflected in both Housatonic River aquatic biota (Yanik, et al. 2003) and tree swallows (Custer and Read 2006).

Aroclor 1260 differs in composition and toxicity from Aroclor 1242, the predominant Aroclor disposed in Neal's Landfill. The dioxin-like toxicity of Aroclor 1260 is less than one-half of that of Aroclor 1242, as shown by bioassays that provide an integrated measure of total dioxin-like activity (Tillitt, et al. 1992). Aroclor 1260 differs in having a larger polychlorinated dibenzofuran (PCDF) co-contaminant component compared to other Aroclors – 12 times as much as Aroclor 1242 (Wakimoto, et al. 1988). Most of the TEQ of Aroclor 1260 is due to PCDFs (70 %), not PCB coplanar congeners (29 %), in contrast to Aroclors 1248 and 1254 in which most of the TEQs are due to PCB coplanar congeners (at least 90 %) with only small PCDF contributions (less than 10 %) (Yamashita, et al. 2000). PCDFs also bioaccumulate less overall compared to PCB coplanar congeners. The reported biota-sediment accumulations factors (BSAFs) in field studies are lower for PCDFs than for PCBs (Niimi 1996; van der Oost, et al. 1996; Marvin, et al. 2002; Naito, et al. 2003; Burkhard, et al. 2004).

Overall, the FERA mink PCB low effect TRV is a median value among the LOAECs based on single studies at other sites. The estimated risk to mink would increase more than 2-fold if the FERA low effect TRV were replaced with the LOAEC TRV from the Hudson River site, the risk estimate would not change if the Fox River LOAEC TRV were used, and about 40 % less risk would be estimated based on the LC₂₀ of the Housatonic River study with no adjustment for exposure duration.

Most of the NOAECs in the other ERAs reviewed in Appendix E1 are lower than the value selected for the FERA. This is because the FERA value is derived from an exposure-response curve, which exhibits the very steep slope between no effects and severe effects characteristic of dioxin-like toxicity. The low NOAEC values are the result of wide dose spacing in individual experiments that missed the actual dose at which adverse effects begin to be observed. The LC₁₀ of the Housatonic mink feeding study is also lower than the FERA no effect TRV, but the FERA value is consistent with the 95 % confidence limit of the Housatonic mink LC₁₀.

³⁴ Probit is a method for transforming (converting) the results of toxicological tests so that the dose-response relationship is a straight line (Rand, et al. 1995). The transformation facilitates interpolation of exposure concentrations or doses that result in specific outcomes, such as the dietary concentration lethal to 20 % of exposed kits (LC₂₀).

4.2.2.2 Mink Dietary TEC TRVs

Mink TEC TRVs and their derivations are shown in Appendix E2, both on a dose and dietary basis. The dietary TRVs selected for the FERA are 4.6 pg TEC/g diet NOAEC and 18 pg/g LOAEC (multiple-season or generation exposure TRVs). The TRVs are calculated as the geometric mean of the TRVs in Brunström, et al. (2001) and Restum, et al. (1998) mink feeding studies (Section 4.2.1 approach 2).

Brunström, et al. (2001) reported TEQ consumption per mink per day, which is converted to dietary TEC by dividing by the reported mink food consumption rate of 130 g/mink/d. Restum, et al. (1998) did not report TEC, however, the contaminated feed came from the same collection of Saginaw Bay carp used by Heaton, et al. (1995), which was homogenized and frozen for use in multiple studies. Tillitt, et al. (1996) reported the dioxin, chlorinated dibenzofuran, and dioxin-like PCB congener composition of the diets in the Heaton, et al. (1995) study (contaminated carp were mixed in varying proportions with uncontaminated feed for the different exposure treatments). For the FERA, the TECs of the Heaton, et al. (1995) treatments are recalculated with the current mammalian WHO-TEFs (Table 2), and a regression is calculated between TEC and PCB concentrations (footnote m in Appendix E2). The regression is then applied to the reported PCB concentrations for the Restum, et al. (1998) treatments to calculate the treatment dietary TECs.

The rationale for deriving mink TRVs based on 2-breeding season or generation exposure durations is as follows. Brunström, et al. (2001) showed a dramatic decrease in mink whelping frequency from 90 % of mated females for the first breeding season during PCB exposure to 39 % for the second season in their Clophen “A50 high” treatment (Clophen is a commercial PCB product). The control whelping frequency was 93 % in both years. Live litter size per whelping female decreased nearly by half between the two exposure periods for the same treatment from 3.8 live kits/whelped female the first year to 2.0 the second year (control values 4.0 and 4.4, respectively). Mean kit bodyweight also decreased for this treatment from 7.9 g in the first year to 6.7 g in the second (control values 9.6 and 8.9, respectively). Only the “A50 high” kit bodyweight was statistically discernible from the control in the first breeding season, but, in addition to kit bodyweight, both whelping frequency and live litter size per mated female were also statistically discernible from control values in the second breeding season. For the “A50 low” treatment, there were no significant differences with controls the first year for any outcome, but, in the second year of exposure, kit bodyweight and kit survival at 2 weeks were statistically less than control values: second-year A50 low 2-week kit bodyweight of 48 g (70 g control), and 2-week kit survival of 36 % (73 % control). Brunström, et al. (2001) wrote:

“In the second season, the effects on reproduction were more pronounced and clearly dose dependent... In our study, the concentration in the feed was the same during the two reproduction seasons, resulting in a reduced frequency of whelping females in the second season only. This finding suggests that the PCB concentration in the animals increased from the first to the second reproduction season, showing the relevance of long-term exposure for estimation of a LOAEL.”

Similarly, in the Restum, et al. (1998) study, whelping frequency, litter size, and kit survival were lower after exposure over 2 breeding seasons than for a single breeding season, and lower for the second generation of female mink exposed (combined natal and post-natal exposure) than for the initial female generation exposed only as adults.

Similar increases in PCB toxicity have been reported in multi-generational studies with other mammals.

McCoy, et al. (1995) reported the PCB body burden in oldfield mice (*Peromyscus polionotus*) approximately doubled between generations at a constant exposure concentration, and was associated with increasingly adverse effects.

"These observations indicate that, even at this low level, chronic exposure to PCBs has pronounced reproductive effects on mammals and that these effects are amplified through multigeneration exposure. ... It is apparent that continued exposure at a low level results in amplified body burdens over three generations. For wild populations that remain in the same area for many generations, cumulative effects may have serious consequences. ... In studies of wild populations, it is evident that the roles of maternal exposure and increasing body burdens must be considered in assessing the long term effects of PCB exposure."

Linzey (1988) reported for white-footed mice (*Peromyscus leucopus*) that

"Reproductive success of second generation PCBs-treated white-footed mice was reduced in comparison with performance of the parental generation reported by Linzey (1987). ... These results confirm the expectation that effects of chronic exposure to PCBs are cumulative through generations, probably due to length of exposure as well as to exposure during critical periods of growth and development."

These findings are consistent with an increase in the reproductive toxicity of dioxin (TCDD) associated with exposure to multiple generations of rats compared to exposure to a single generation (f_0 is the initial generation tested, f_1 is the offspring from f_0 , and f_2 is the offspring from f_1) (Murray, et al. 1979).

"At the intermediate dose of 0.01 μg TCDD/kg/day, many adverse effects seen in the f_1 and f_2 adults and their litters were not evident in the f_0 adults or their offspring. The most obvious difference is that the f_0 rats were given TCDD beginning at about 7 weeks of age whereas subsequent generations were exposed to TCDD, at least theoretically, from the time of conception. This suggests that some of the effects observed were initiated, perhaps, during the neonatal period."

In addition to guiding selection of mink TEC TRVs, these considerations also support the rationale for adjusting the mink single-breeding season exposure Aroclor TRVs (Section 4.2.2.1). Multi-breeding season exposure or multi-generation mink studies have not been performed with commercial Aroclors, but since increased toxicity in mink has been reported for exposure over multiple breeding seasons or generations in both controlled (Clophen) and field-contaminated feeding studies, a similar exposure duration effect is expected to occur with Aroclors.

Most of the TRVs of the other ERAs reviewed in Appendix E2 are based on single-breeding season exposures, out-dated TEFs, or were not performed with mink. The TEC NOAEC and LOAEC of the Housatonic River mink feeding study (single breeding season exposure) are higher than reported in other mink feeding studies (16.1 and 68.5 pg/g, respectively), but resulted in relatively high kit mortality (54 %). The investigators performed probit regression analysis to calculate the dietary concentrations lethal to 20 % and 10 % of kits (LC_{20} and LC_{10} , respectively) and the associated 95 % confidence intervals (CI). The LC_{20} is 16.2 pg/g (CI: 9.4 – 33.5 pg/g), and the

LC₁₀ is 4.2 pg/g diet (CI: 1.4 – 7.5 pg/g) (Bursian, et al. 2006a). The Housatonic River TEC LC₂₀ and LC₁₀ for exposure over a single breeding season are remarkably consistent with the FERA LOAEC and NOAEC TRVs (18 and 4.6 pg/g, respectively) calculated as the geometric means of two studies performed with exposures over two breeding seasons or generations.

4.2.2.3 Kingfisher Dose PCB TRVs

Avian PCB dose TRVs and their derivations are shown in Appendix E3. Two sets of TRVs are selected for the FERA because they cluster around two different values, excluding an inappropriate TRV derivation. The kingfisher dietary TRVs selected for the FERA are 400 µg/kg_{BW}-d no effect and 500 µg/kg_{BW}-d low effect (Kalamazoo River ERA), and 110 µg/kg_{BW}-d NOAEL and 1120 µg/kg_{BW}-d LOAEL (Fox River and Upper Green Bay ERAs).

The details of the 400 - 500 µg/kg_{BW}-d TRV derivation are presented in Appendix D of the Kalamazoo ERA, and summarized in the FERA Addendum. The TRVs are interpolated (Section 4.2.1 approach 3) from a dose-response plot based on the combined results of three chicken feeding studies performed with Aroclor 1248 added to the feed (Cecil, et al. 1974; Lillie, et al. 1974 and 1975; Scott 1977). The chicken dietary PCB concentrations are converted to bodyweight-normalized doses by multiplying by the food ingestion rate reported in each study, or by a default leghorn hen food ingestion rate of 0.067 kg feed/kg_{BW}-d (Medway and Kare 1959 as cited in USEPA 1995) if no food ingestion rate is given. The toxicological endpoint is hatchability.

The 500 µg/kg_{BW}-d TRV is intermediate between the avian PCB LOAEL used in the Great Lakes Initiative (600 µg/kg_{BW}-d), based on pheasants exposed to Aroclor 1254 (Dahlgren, et al. 1972) with an uncertainty factor of 0.33 for extrapolating between species, and that of the Sheboygan River and Harbor ERA (400 µg/kg_{BW}-d), based on chicken exposed to field-contaminated feed (Summer, et al. 1996) (Appendix E3). The NOAELs of these two ERAs are significantly lower than 400 µg/kg_{BW}-d no effect TRV selected for the FERA. This is because the FERA value is derived from a dose-response curve, which has the characteristically very steep slope between no effects and severe effects observed for dioxin-like toxicity. The low NOAELs values used at other sites are the result of wide dose spacing in the original studies that missed the actual dose at which adverse effects begin to be observed.

The 110 - 1120 µg/kg_{BW}-d TRV derivation is based on impairment of courtship and nesting behaviors in selected studies of doves (Peakall and Peakall 1973; Tori and Peterle 1983) (Section 4.2.1 approach 1). Parental inattentiveness was shown to contribute to the decreased survival of the offspring of PCB-exposed doves. Birds with impaired courtship behavior are less likely to successfully mate. Even though the LOAEL is double the alternate value selected for the FERA, the NOAEL is much lower than the interpolated value.

The remaining TRVs reviewed in Appendix E3 are the exceptionally high values used in the Hudson River ERA, which are an order of magnitude (10X) larger than the TRVs used by the GLI based on the same study (Appendix E3). The discrepancy is mainly due to use of different toxicological endpoints—egg production (Hudson) versus hatchability (GLI), and, to a lesser degree, to a difference in interspecific extrapolation—no adjustment (Hudson) versus division of the initial TRV by a factor of 3 (GLI). The Hudson River ERA avian PCB dose TRVs are not selected for use in the FERA because egg production is an insensitive response of birds to PCB exposure. For example, chicken, the bird species so far shown to be the most sensitive to PCBs, do not exhibit a coherent dose-response relation between PCBs and egg production (FERA Addendum Figure A2).

4.2.2.4 Kingfisher Dose TEQ TRVs

Avian dose TEQ TRVs are shown in Appendix E4. The TRVs selected for the FERA are 1.4 ng/kg_{BW}-d NOAEL and 14 ng/kg_{BW}-d LOAEL. The TRVs are based on a study of TCDD (dioxin) in pheasant (Nosek, et al. 1992a) (Section 4.2.1 approach 1). The toxicological endpoints are fertility and embryo mortality. The initial TRVs from Nosek, et al. (1992a) are adjusted by dividing by a factor of 10 to account for the short duration of the experiment (subchronic-to-chronic extrapolation) for use by both the GLI and Hudson River ERAs. The subchronic-to-chronic adjustment is based on a study of TCDD kinetics in pheasants showing a half-life for whole-body elimination from non-laying pheasant of nearly one year (Nosek, et al. 1992b). The 10-week exposure period of the toxicity study (Nosek, et al. 1992a), therefore would have only resulted in about one-tenth of the steady-state TCDD accumulation of long-term exposure (USEPA 1995).

The Nosek, et al. (1992a) TEQ TRVs without the subchronic-to-chronic exposure adjustment were presented in the Sheboygan River and Harbor terrestrial ERA for comparative purposes, and were not used for decision-making purposes. TEQ TRVs based on the Summer, et al. (1996) study with field-contaminated prey are presented on three different bases: HII4E bioassay TEQ (performed with a rat cell line), recalculated TEQ using Kennedy, et al. (1996) TEFs, and recalculated TEQ using the current WHO-TEFs for mammals (Appendix E4). The HII4E bioassay gives the lowest TRV values, and the current WHO-TEF-based TEQ the highest. The oral TEQ dose to chicken is not calculated with avian WHO-TEFs because they are based on relative effects of dioxin-like congeners accumulated in eggs, not the relative effects as fed to the adult birds. Also note that the original TEQs presented by Summer, et al. (1996), based on the HII4E bioassay, are for mammalian toxicity. With the current mammalian WHO-TEFs, the TRVs for the Summer, et al. (1996) study are remarkably similar to the TRVs selected for the GLI and Hudson River ERAs.

4.2.2.5 Kingfisher Egg TEC TRVs

Avian egg TEC TRVs are shown in Appendix E6 for both whole-egg concentration and lipid-normalized concentration. Two sets of TRVs are selected for the FERA because they cluster around two different values. The kingfisher egg TRVs selected for the FERA are 1.8 µg/kg lipid NOAEC and 5.3 µg/kg lipid LOAEC (Blankenship and Giesy 2002), and 17 µg/kg lipid NOAEC and 68 µg/kg lipid LOAEC (Hudson River ERA).

The whole-egg concentration TRVs are converted to lipid-normalized TRVs by dividing by the lipid contents reported in the study, or, if lipid content was not reported, by the lipid content reported by functional category by Carey, et al. (1980) (see footnotes to Appendix E6). Carey, et al. (1980) summarize egg lipid contents, based on their analyses combined with the results of an extensive literature review, for different functional categories of birds related to whether the chicks can immediately leave the nest (precocial–10.3 % lipid), are initially helpless (altricial–5.9 % lipid), or fall into intermediate categories (semi-precocial–9.5 % lipid, semi-altricial–6.3 % lipid). The species in the toxicological studies are assigned by genus to the functional categories in Carey, et al. (1980). A chicken egg lipid content of 11.2 % is based on Pennington and Church (1985).

The Blankenship and Giesy (2002) TRVs are based on a field study of bald eagle eggs (Elliot, et al. 1996) (Section 4.2.1 approach 1). The TEC is recalculated from the original study with the current WHO-TEFs. The toxicological endpoint is hepatic cytochrome P4501A induction (enzymes produced in response to exposure dioxin-like chemicals). The 5 µg/kg lipid LOAEC is intermediate between the two LOAECs of the Fox River

ERA, 3 - 4 µg/kg lipid based on field studies of egg lethality in wood duck, double-crested cormorant, and caspian tern (Giesy, et al. 1994a, 1995) (Section 4.2.1 approach 3), and 6 µg/kg lipid based on deformities in double-crested cormorant (Ludwig, et al. 1996) (Section 4.2.1 approach 1). Both of the Fox River TRVs are H4IIE-bioassay TECs. Also, the Hudson River LOAEC for great blue heron eggs (8 µg/kg lipid), based on chick bodyweight (Sanderson, et al. 1994a) is higher, and the Sheboygan River and Harbor LOAEC for robin eggs (1.4 µg/kg lipid), based on embryo mortality (Powell, et al. 1996) is lower, than the Blankenship and Giesy (2002) LOAEC. The Sanderson, et al. (1994a) TRV is based on an older TEF scheme, and the Powell, et al. (1996) TRV on TCDD injected in chicken eggs. Although the endpoint of the Blankenship and Giesy (2002) TRVs (enzyme induction) is not, of itself, necessarily harmful, the TRVs are selected for the FERA because they represent middle values among the cluster of single-digit lipid-normalized egg TRVs that are based on adverse effects in birds (mortality, deformities, reduced growth).

Two additional avian egg TEC TRVs in the Hudson River ERA are an order of magnitude (10X) greater than the rest of the TRVs reviewed in Appendix E6: belted kingfish TRVs of 17 µg/kg lipid NOAEC and 68 µg/kg lipid LOAEC, based on embryo mortality in double-crested cormorant eggs injected with TCDD (Powell, et al. 1997) (Section 4.2.1 approach 1), and bald eagle TRVs of 13 µg/kg lipid NOAEC, based on a field study of egg hatchability (Elliot, et al. 1996), and 79 µg/kg lipid LOAEC based on embryo mortality of kestrel eggs injected with PCB congener 77 (one of the dioxin-like congeners) (Hoffman, et al. 1998). The Hudson River kingfisher TRVs are selected for the FERA to represent the less conservative avian egg TEC TRVs.

5 Risk Characterization

5.1 Risk Characterization Methods

5.1.1 Hazard Quotient (HQ)

The initial step in characterizing risk is to calculate the hazard quotients (HQ) in each reach:

$$\text{HQ} = \text{exposure estimate} / \text{toxicity reference value (TRV)} \quad [3]$$

where the exposure estimate is dietary PCB concentration or TEC, PCB or TEQ dose, or egg TEC. Mink HQs are based on UCL or mean PCB or TEC dietary concentrations divided by mink dietary TRVs (Appendices A2, A7, B2, B7, C2, C7). Kingfisher HQs are based on modeled UCL or mean PCB or TEQ doses divided by avian dose TRVs (Appendices A2, A7, B2, B7, C2, C7). Kingfisher egg HQs are based on modeled UCL or mean TEC in eggs divided by egg TRVs (Appendices A5, A9, B5, B9, C5, C9).

The HQs for the Richland Creek reaches (2 through 6) directly characterize risk to mink or kingfisher because the sample locations are greater than 1 stream mile apart. Since the home range of female mink and kingfisher is 1 stream mile (Section 2.6), they are likely to forage completely within a single Richland Creek reach. In contrast, female mink and kingfisher are unlikely to forage exclusively within Conard's Branch (Reach 1), so the risks associated with Reach 1 cannot be directly characterized by the Reach 1 HQs. Instead, risks to mink or kingfisher feeding in part on Conard's Branch prey are evaluated through combined reach foraging models: 25 % Conard's Branch (Reach 1) prey and 75 % Richland Creek (Reach 2) prey for mink; and 50 % Conard's Branch prey and 50 % Richland Creek prey for kingfisher (Section 2.6). The combined reach models are designated as "Reaches 1

& 2". The combined Reaches 1 & 2 exceeds the home range for female mink and kingfisher, so the Reaches 1 & 2 HQs directly characterize risk to these receptors.

Interpretation of HQs depends on the TRV used in the calculation. For NOAEC, NOAEL, or no-effect TRVs, a HQ of 1 or less indicates that adverse effects are not expected (the exposure is as low or lower than levels that have not been associated with adverse effects). For LOAEC, LOAEL, or low-effect TRVs, a HQ of 1 or more indicates that adverse effects are expected (the exposure is as high or higher than levels that have been shown to cause adverse effects).

There is a gray area for the exposures that fall between NOAEC and LOAEC TRVs, that is, when the NOAEC HQ is greater than 1 but the LOAEC HQ is less than 1. In this case, the exposure is higher than levels not associated with problems, but lower than the levels known to cause problems. The gray area bounds the exposure threshold associated with the onset of adverse effects, in other words, the threshold for adverse effects is somewhere between the NOAEC and LOAEC TRVs, but the location of the threshold within that range is unknown. Therefore, risk is uncertain when the exposure falls between the NOAEC and LOAEC TRVs, other than to state that the exposures are lower than levels shown to cause adverse effects but higher than levels shown not to cause problems.

5.1.2 Percent Allowable Consumption (PAC)

To address uncertainty over the exposure assumptions for mink and kingfisher, in particular, the allocation of foraging between Conard's Branch and Richland Creek in the combined Reach 1 & 2 assessment, an additional approach is used for risk characterization. This approach calculates the percent allowable consumption (PAC) for a reach, which is the percent diet an animal can take from a stream reach without exceeding TRVs. The technique is modified from the percent allowable daily intake (PADI) approach by Giesy, et al. (1994b). Giesy, et al. (1994b) gave the following equation for PADI:

$$\text{PADI} = ((\text{NOAED} / \text{Conc.}_{\text{fish}}) / \text{Daily food consumption}) * 100 \quad [4]$$

where NOAED is the no observed adverse effect dose ($\mu\text{g}/\text{day}$), $\text{Conc.}_{\text{fish}}$ is the contaminant concentration in fish ($\mu\text{g}/\text{kg}$), and Daily food consumption is expressed as $\text{kg food}/\text{day}$. Since $\text{NOAED} (\mu\text{g}/\text{d}) = \text{dietary NOAEC} (\mu\text{g}/\text{kg}) * \text{Daily food consumption} (\text{kg}/\text{d})$, equation 4 simplifies to:

$$\text{PAC} = (\text{NOAEC} / \text{Conc.}_{\text{diet}}) * 100 \quad [5]$$

Where $\text{Conc.}_{\text{diet}}$ is the dietary concentration to allow for consumption of prey other than fish. The name is changed to percent allowable consumption (PAC) because the daily food consumption term no longer appears in the equation. PAC may be calculated directly from the hazard quotient (HQ) because $\text{HQ} = \text{Contaminant conc.} / \text{TRV}$, or, in this case, $\text{HQ} = \text{Conc.}_{\text{diet}} / \text{NOAEC}$. This means that equation 5 may be further simplified to:

$$\text{PAC} = (1 / \text{HQ}) * 100 \quad [6]$$

PADI or PAC give the percent diet an animal can consume from a contaminated source without exceeding toxic levels assuming that the remainder of the diet has zero contamination. This assumption is unrealistic for PCBs

because they are present even in apparently uncontaminated areas due to global circulation driven by volatilization from PCB sources to the atmosphere, transportation through the atmosphere, and deposition over widespread areas through precipitation (wet deposition), settling on surfaces (dry deposition), and gaseous absorption (Duursma and Carroll 1996; Lohmann, et al. 2001; Meijer, et al. 2003).

Equation 6 is modified to account for the “background” exposure to PCBs by subtracting the HQ for foraging in areas with no local source of PCBs ($HQ_{\text{off-site}}$) before dividing by the HQ for the area under investigation (HQ_{site}). PAC is then calculated as:

$$PAC = ((1 - HQ_{\text{off-site}}) / HQ_{\text{site}}) * 100 \quad [7]$$

For the FERA, the 2002 Reach 0 data collected from Richland Creek upstream of the confluence with Conard’s Branch is used to represent “background” PCB exposure (HQ_{upstream}):

$$PAC = ((1 - HQ_{\text{upstream}}) / HQ_{\text{reach}}) * 100 \quad [8]$$

where HQ_{reach} is the HQ in the stream reach for which the PAC is being calculated. Reach 0 UCL HQs are shown in Appendices B2 and B5 and mean HQs in Appendices B7 and B9. PAC calculations are made only with TEC (mink diet and kingfisher egg) or TEQ (kingfisher dose) data because they are fully comparable across years. In contrast, the analytical methods for PCBs changed each of the 3 years samples were collected for the FERA.

Since the 2002 fish data for Reach 0 is used in the FERA to represent the contaminant loads in fish not directly exposed to PCBs discharged from Neal’s Landfill, the individual sample data for this reach are examined for evidence of movement of fish from reaches directly receiving Neal’s Landfill outflow (Appendix B1). The creek chub data are highly consistent—TEC ranging from 0.30 to 0.39 pg/g, and no positive detections for Aroclors. The white sucker data are mostly consistent, with one conspicuous outlier—sample number 15. For this reason, sample ID 15 is excluded from all Reach 0 calculations (Section 3.3.7).

Equation 8 produces negative values if the upstream HQ is greater than 1. This occurs twice in Reach 0 for UCL kingfisher egg HQs for 1.79 µg/kg lipid NOAEC and 5.33 µg/kg lipid LOAEC TRVs (Appendix B5), and once for mean kingfisher egg HQ for 1.79 µg/kg lipid NOAEC (Appendix B9). For these three TRVs, PAC is calculated by Equation 6, that is, percent allowable consumption is calculated without taking account of off-site exposure. This means that the calculated PACs for UCL kingfisher egg for the 1.79 µg/kg lipid NOAEC and 5.33 µg/kg lipid LOAEC TRVs, and for mean kingfisher egg for the 1.79 µg/kg lipid NOAEC TRV, are potentially overestimated because off-site exposure is assumed to be zero.

5.1.3 Additional Risk Information

Two additional lines of evidence regarding risk are considered as supporting evidence. One is evaluation of the PCB concentration and TEC in the liver of a road-killed mink collected from the Highway 48 bridge over Richland Creek (Sparks 2005). The lipid-normalized liver concentrations are compared to published mink and otter liver values associated with adverse effects.

The other additional line of evidence is assessment of potential risks of PCB exposures to fish based on comparison of fish body burdens with fish tissue-based toxicity reference values (TRVs). Fish were not included as one of the original assessment endpoints at this site under the assumption that evaluation of risk to piscivores (specifically, mink) would be protective for fish and other aquatic organisms as well (Section 2.3). This assumption is valid only in the river segments in which the mink area utilization factor (AUF) is 100 % (forage entirely within a reach), but does not necessarily hold for segments in which mink or kingfisher forage only part of the time, such as Conard's Branch (Section 2.6). Also, Viacom, Inc., has argued that findings of risk to piscivores in the upper Richland Creek reach closest to Neal's Landfill are not meaningful for decision-making purposes because the upper reach is too small to support a population of mink or kingfisher. This line of argument focuses solely on the specific characteristics of the assessment endpoints, and disregards the initial assumption that selection of a particular assessment endpoint will ensure protectiveness for other species in the same ecosystem.

5.2 Risk Characterization Results

The range of risk estimates for the first 3 reaches downstream of Neal's landfill are summarized in Tables 11 and 12 for mink and kingfisher UCL and mean exposures to total PCBs (Aroclor or sum of congeners), dioxin toxic equivalent concentration (TEC), and dioxin toxic equivalents (TEQ). The HQs in Tables 11 and 12 are rounded to a single significant digit³⁵. The individual UCL HQs are reported in Appendices A2, A5, B2, B5, C2, and C5, and the mean HQs in Appendices A7, A9, B7, B9, C7 and C9.

The HQ ranges shown in Tables 11 and 12 have different sources. For mink, the range is between HQs calculated for PCB and TEC (1 set of TRVs each). For kingfisher dose, the range is between TEQ HQ (1 set of TRVs) and 2 estimates of PCB HQs (2 sets of TRVs). For kingfisher eggs, the range is between 2 estimates of TEC HQs (2 sets of TRVs). An additional source for the range in PCB HQs is that the Aroclor analysis in 2002 was performed twice (4-peak and 5-peak), with both sets of results carried through the risk calculations.

5.2.1. Risk Characterization for PCBs versus TEC or TEQ

There is no consistent relationship between the HQs based on total PCBs (dietary concentration or dose) and the HQs based on TEC (dietary concentration) or TEQ (dose), that is, neither TEC/TEQ or PCB results in consistently more or less conservative risk estimates. For mink, the NOAEC HQs are larger for TEC than for PCB in all years, but, conversely, the LOAEC HQs are larger for PCB than for TEC in 2002 and 2003, and have equivalent values in 2001. For kingfisher dose, the NOAEL HQs are much larger for TEQ than for PCB in 2001, somewhat larger for TEQ than for PCB in 2002, and only slightly larger to no difference for TEQ and PCB in 2003. Kingfisher TEQ dose LOAEL HQs are greater than or equivalent to PCB dose LOAEL HQs in 2001, intermediate within the PCB HQ range in 2002, and less than or equivalent to PCB HQs in 2003. The altered basis for analyzing total PCBs in different years (4-peak Aroclor in 2001, 4- and 5-peak Aroclor in 2002, and sum of congeners in 2003) is largely responsible for the changing relationship between TEC/TEQ and PCB risk estimates between years.

³⁵ A single significant digit means that there is only one non-zero digit in a number. For example, in rounding to one significant digit, 0.042 becomes 0.04, 0.873 becomes 0.9, 6.81 becomes 7, 32.77 becomes 30, and 127.64 becomes 100.

5.2.2. Risk Characterization for 4-peak and 5-peak Aroclor Analysis

The two types of Aroclor analysis in 2002 produced similar risk estimates, even though the 5-peak analysis resulted in 17 to 19 % lower mean Aroclor concentrations in Reach 1 and 2 fish compared to 4-peak analysis (Appendix B2). In some cases, the nearly 20 % difference in exposure concentration had no effect on the rounded HQs, for example, mink low effect HQs for combined Reaches 1 & 2 (Appendices B2 and B7), or kingfisher mean low effect or LOAEL HQs for Reach 1 (Appendices B7). In most cases, the exposure difference resulted in small HQ differences, for example, Reach 1 mink UCL low effect HQs of 9 (4-peak analysis) and 8 (5-peak analysis), or kingfisher LOAEL HQs of 3 (4-peak analysis) and 2 (5-peak analysis) (Appendix B2). There are no cases for which a choice of 4- versus 5-peak analytical results leads to opposite risk conclusions, that is, the same set of reaches are at risk regardless of the Aroclor analytical method. The few cases in which the 4-peak HQ is 1 and the 5-peak HQ is 0.9 are not exceptions because either result indicates that the exposures are close to the levels associated with adverse effects—kingfisher UCL low effect HQ in Reach 2 (Appendix B2) and mean LOAEL HQ in combined Reaches 1 & 2 (Appendix B7).

5.2.3 Risk Characterization for Mean and Upper 95th Percent Confidence Limit of the Mean (UCL) Exposure Estimates

The difference between HQs based on UCL exposure data versus those based on mean exposure data is relatively small, generally less than 2-fold (Tables 11 and 12). The Reach 3 locations are exceptions with up to 3-fold differences between UCL and mean exposure HQs. The numbers of fish sample are low for both Reach 3 locations—2 creek chub in 3S (2001) and 3 shorthead redhorse in 3V (2003) (Appendices A1 and C1).

5.2.4. Risk Characterization for Kingfisher Dose versus Egg, and Kingfisher Consensus HQ Range

Risks are estimated for kingfisher through two methods: dose modeling to adult birds (TEQ and PCB), and egg accumulation modeling (TEC only). In addition, two sets of TRVs are used to estimate the risks of PCB dose to adult birds, two sets of TRVs for assessing TEC in eggs, and a single set of TRVs for kingfisher TEQ dose. In 2003 and most of 2001, the range of dose HQs overlap with the corresponding egg HQs (Tables 11 and 12). The HQs of the two approaches do not overlap in 2002 (neither do low effect UCL HQs in Reach 2 in 2001, but they barely miss overlapping, unlike the larger gap between 2002 HQs) (Tables 11 and 12). The difference is due to seasonal fluctuations in fish lipid content, which is very low in November (2002). Very low lipid level has opposite effects on the two modeling approaches – decreasing risk on a whole-diet basis as in the dose approach³⁶, but increasing risk on a lipid-normalized basis as in the egg approach³⁷. Kingfisher do not lay eggs in November, so the egg-based risk estimates are theoretical and comparative, and do not indicate any expectation that kingfisher

³⁶ The same amount of fish consumed per day with low lipid content will present less risk because of the decrease in dietary PCB levels compared to eating fatter fish. This holds only if the total amount of fish eaten does not change with lipid content.

³⁷ The November fish lipid contents are so low that the lipid-normalized PCB concentrations are higher than in fish in other months, and therefore the modeled PCB accumulation in eggs is highest in November. An assumption for lipid-normalized diet-to-egg BMFs is that the lipid content of the diet remain relatively constant. This would imply higher food consumption rates of low-fat fish compared to fatty fish, probably a more likely scenario than constant food ingestion rate regardless of fat content.

would produce eggs in November. The late-season kingfisher dose estimates are of interest because the kingfisher foraging area (home range) appears to decrease significantly (smaller area) in the non-breeding season (Section 2.6).

The kingfisher egg modeling approach tends to show higher estimates of risk compared to ingestion dose modeling to adult kingfishers. In most cases, the kingfisher dose and egg HQ ranges overlap, that is, the more conservative of the kingfisher dose HQs overlap with the less conservative of the egg HQs. Unlike the situation with mink, the effects of PCBs have not been tested in kingfisher, and the kingfisher TRVs are based on studies of other bird species with uncertain applicability to kingfisher. Therefore, there is no *apriori* reason for considering one of the kingfisher assessment approaches to be more reliable for estimating risk than the other. The overlap between the dose and egg HQs defines the kingfisher HQ range with the lowest uncertainty because there is agreement between two separate exposure modeling approaches evaluated with independent sets of TRVs. For the FERA, the HQs in the overlap between kingfisher dose and egg HQs are referred to as “consensus” HQs. In the few cases in which the dose and egg HQs do not overlap, the highest HQ of the lower of the two HQ ranges, and the lowest HQ of the higher HQ range are shown. The gap between approaches is mostly a factor of 3 or less (Tables 13 and 14), similar to the range of overlap in 2001 and 2003.

The kingfisher dose and egg HQ ranges in Tables 11 and 12 are modified to show the range of consensus HQs (the overlap or gap between the kingfisher dose and egg HQ ranges) in Tables 13 and 14. The kingfisher consensus HQ range and the mink HQ range are remarkably similar, particularly for LOAEC and LOAEL HQs, although the kingfisher range tends to be somewhat wider than the mink range.

5.2.5 Risk Characterization of Richland Creek Reaches 4 through 6

The HQs for Reaches 4 through 6 are not included in Tables 11 and 12 because they were only sampled once (2001) for the FERA, and do not show excess risk for mink or, by weight-of-evidence, for kingfisher. All but one of the mink and kingfisher HQs are less than 1 in Reaches 5 and 6, the sole exception is one of the kingfisher egg NOAEC HQs which equals 1 (Appendices A2, A5, A7, A9). This indicates that mink and kingfisher are not at risk when feeding in Reaches 5 and 6 (22 miles or more downstream from Neal’s Landfill)³⁸. Dioxin-like PCB congeners were detected at the farthest downstream reach sampled. The UCL TEC/TEQ-based HQs in Reach 6 are 0.09 NOAEC and 0.02 LOAEC for mink, and from 0.01 NOAEC to 0.1 LOAEC for kingfisher (Appendices A2, A5). Aroclors were detected as far as Reach 5. The UCL Aroclor-based HQs in Reach 5 are 0.2 for mink, and no greater than 0.6 NOAEL and 0.1 LOAEL for kingfisher.

All of the mink HQs are also less than 1 in Reach 4 (Appendices A2, A7), which means that mink feeding in Reach 4 or further downstream, with a dietary composition of 66 % fish, 13 % crayfish, and 21 % non-aquatic prey, would not be exposed to PCBs at levels that could potentially result in reproductive impairment. Of the 5 kingfisher UCL LOAEL-, low effect-, or LOAEC-based HQs in Reach 4 (1 for TEQ dose, 2 for Aroclor dose, and 2 for egg TEC), only 1 indicates potential risk (HQ of 5 for the more conservative egg LOAEC), the other 4 HQs are less than 1 (Appendices A2, A5). Conversely, all but 1 of the 5 kingfisher UCL NOAEL-, no effect-, or NOAEC-based HQs are greater than 1 in Reach 4. This means that kingfisher exposures to PCBs along Reach 4

³⁸ A NOAEC-based HQ of 1 indicates no risk because it means that exposure is equal to the level that did not result in adverse effects compared to unexposed controls.

fall in the grey area of our knowledge of toxicity—mostly less than exposures associated with adverse reproductive effects, but mostly greater than exposures shown to have no statistical effects on reproduction. Potential risk to kingfishers in Reach 4 cannot be ruled out, but the likelihood of adverse effects appears to be low (13 miles or more downstream from Neal's Landfill).

5.2.6 Risk Characterization of Richland Creek Reach 3

The risk characterizations for the two Reach 3 locations should be interpreted cautiously because they are based on small sample sizes—2 creek chub in 3S (2001) and 3 shorthead redhorse in 3V (2003). Excess risk to mink or kingfisher cannot be ruled out in Reach 3 because the upper range of UCL no effect HQs exceeds 1 for both receptors (Table 11), and the upper range of the UCL low effect HQs meets or exceeds 1 for mink (2003), and kingfisher dose (2003)³⁹ and eggs (both years) (Table 12). Conclusions are similar for the kingfisher consensus HQs, except that the upper range of the UCL low effect HQs approaches (2001) or just meets (2003) a value of 1 (Tables 13 and 14).

Mink and kingfisher exposures to PCBs along Reach 3 fall in the gray area of our knowledge of toxicity—mostly exceeding no effect levels, but less than or just equal to low effect levels (mink HQ range, and kingfisher consensus HQ range). The limited fish sampling data for the Reach 3 locations indicates there are possible risks to mink or kingfisher (3 to 6 miles downstream of Neal's Landfill), with considerable uncertainty in either direction (risk may be greater or less than estimated in the FERA).

The 2001 TEC/TEQ-based risk estimates for Reach 3 might be underestimated because PCB congener 126 was not detected in Reach 3 creek chubs (Appendix A1), but was detected in 2 of the 3 shorthead redhorse collected in 2003 (Appendix C1). In 2003, congener 126 accounted for 43 % of the total mammalian TEC in fish (calculated from Appendix C1), and 20 % of the avian TEC in eggs (calculated from Appendix C9). If congener 126 was present in Reach 3 fish in 2001 and made the same proportional contribution to fish or egg TEC as in 2003, the 2001 fish TEC would be 1.8 times greater than reported, the estimated dietary TEC would be approximately 1.4 times greater for mink, and 1.8 times greater for kingfisher, and the estimated egg TEC would be 1.3 times greater. As it turns out, this would not appreciably change the risk conclusions for Reach 3 in 2001—the low effect HQs remain less than 1 for mink, and the upper range of the kingfisher consensus low effect HQs just equals 1 in 2001.

The 2003 data, which have no detection issues, indicate that exposures to mink or kingfisher in Reach 3 are mostly greater than no effect levels, and range from less than to equal to levels associated with adverse reproductive effects.

³⁹ HQ of 0.9 is equivalent to 1, that is, the dose is expected to result in adverse reproductive effects. Stated another way, a 10 % reduction in a dose shown to cause adverse effects is not a sufficiently smaller exposure to expect to avoid the adverse effects.

Year	Exposure Basis	Conard's Branch Reach 1			Richland Creek Reach 2			Conard's and Richland Combined Reaches 1 & 2			Richland Creek Reach 3S (2001) or 3V (2003)		
		Mink	Kingfisher		Mink	Kingfisher		Mink	Kingfisher		Mink	Kingfisher	
			Dose	Egg		Dose	Egg		Dose	Egg		Dose	Egg
2001	UCL	10 - 50	10 - 100	40 - 400	3 - 9	2 - 20	10 - 100	5 - 20	6 - 60	30 - 300	0.5 - 2	0.4 - 3	3 - 30
	Mean	10 - 30	8 - 60	20 - 200	2 - 4	1 - 9	7 - 70	4 - 10	4 - 40	10 - 100	0.3 - 0.7	0.2 - 1	0.9 - 8
2002	UCL	9 - 20	7 - 30	70 - 700	2 - 4	1 - 8	20 - 100	3 - 7	4 - 20	40 - 400	NA	NA	NA
	Mean	6 - 10	4 - 20	40 - 400	0.9 - 2	0.7 - 5	8 - 80	2 - 5	3 - 10	30 - 200	NA	NA	NA
2003	UCL	20	10 - 50	10 - 100	3 - 5	2 - 10	4 - 40	7 - 10	8 - 30	9 - 80	1 - 3	1 - 6	1 - 10
	Mean	20	10 - 40	10 - 100	2 - 4	2 - 7	3 - 30	5 - 8	6 - 20	7 - 60	0.7 - 1	0.6 - 3	0.6 - 6

Year	Exposure Basis	Conard's Branch Reach 1			Richland Creek Reach 2			Conard's and Richland Combined Reaches 1 & 2			Richland Creek Reach 3S (2001) or 3V (2003)		
		Mink	Kingfisher		Mink	Kingfisher		Mink	Kingfisher		Mink	Kingfisher	
			Dose	Egg		Dose	Egg		Dose	Egg		Dose	Egg
2001	UCL	10	4 - 10	10 - 100	2	0.8 - 2	3 - 40	5	2 - 6	7 - 90	0.4	0.1 - 0.3	0.8 - 10
	Mean	8	3 - 6	5 - 60	1 - 2	0.5 - 1	2 - 20	3	2 - 4	3 - 40	0.2 - 0.3	0.1 - 0.2	0.2 - 3
2002	UCL	4 - 9	2 - 7	20 - 200	1 - 2	0.4 - 1	4 - 50	2 - 3	1 - 4	10 - 100	NA	NA	NA
	Mean	3 - 6	2 - 4	10 - 100	0.7 - 0.9	0.2 - 0.7	2 - 30	1 - 2	0.9 - 2	7 - 80	NA	NA	NA
2003	UCL	6 - 20	5 - 10	3 - 40	1 - 2	0.8 - 2	1 - 10	2 - 6	3 - 7	2 - 30	0.7 - 1	0.4 - 0.9	0.4 - 4
	Mean	5 - 10	4 - 9	3 - 30	0.9 - 2	0.6 - 1	0.9 - 9	2 - 4	1 - 5	2 - 20	0.3 - 0.6	0.2 - 0.3	0.2 - 2

Appendices A2, A5, A7, A9, B2, B5, B7, B9, C2, C5, C7, C9 (all HQs are rounded to one significant digit)

Table 13. No Effect-based Hazard Quotient Range with Consensus HQs for Kingfisher Dose and Egg Modeling Approaches, Neal's Landfill, Bloomington, IN									
Year	Exposure Basis	Conard's Branch Reach 1		Richland Creek Reach 2		Conard's and Richland Combined Reaches 1 & 2		Richland Creek Reach 3S (2001) or 3V (2003)	
		Mink	Kingfisher	Mink	Kingfisher	Mink	Kingfisher	Mink	Kingfisher
2001	UCL	10 - 50	40 - 100	3 - 9	10 - 20	5 - 20	30 - 60	0.5 - 2	3
	Mean	10 - 30	20 - 60	2 - 4	7 - 9	4 - 10	10 - 40	0.3 - 0.7	0.9 - 1
2002	UCL	9 - 20	30 - 70 ^a	2 - 4	8 - 20 ^a	3 - 7	20 - 40 ^a	NA	NA
	Mean	6 - 10	20 - 40 ^a	0.9 - 2	5 - 8 ^a	2 - 5	10 - 30 ^a	NA	NA
2003	UCL	20	10 - 50	3 - 5	4 - 10	7 - 10	9 - 30	1 - 3	1 - 6
	Mean	20	10 - 40	2 - 4	3 - 7	5 - 8	7 - 20	0.7 - 1	0.6 - 3

Table 14. Low Effect-based Hazard Quotient Range with Consensus HQs for Kingfisher Dose and Egg Modeling Approaches, Neal's Landfill, Bloomington, IN									
Year	Exposure Basis	Conard's Branch Reach 1		Richland Creek Reach 2		Conard's and Richland Combined Reaches 1 & 2		Richland Creek Reach 3S (2001) or 3V (2003)	
		Mink	Kingfisher	Mink	Kingfisher	Mink	Kingfisher	Mink	Kingfisher
2001	UCL	10	10	2	2 - 3 ^a	5	6 - 7 ^a	0.4	0.3 - 0.8 ^a
	Mean	8	5 - 6	1 - 2	1 - 2 ^a	3	3 - 4	0.2 - 0.3	0.2
2002	UCL	4 - 9	7 - 20 ^a	1 - 2	1 - 4 ^a	2 - 3	4 - 10 ^a	NA	NA
	Mean	3 - 6	4 - 10 ^a	0.7 - 0.9	0.7 - 2 ^a	1 - 2	2 - 7 ^a	NA	NA
2003	UCL	6 - 20	5 - 10	1 - 2	1 - 2	2 - 6	3 - 7	0.7 - 1	0.4 - 0.9
	Mean	5 - 10	4 - 9	0.9 - 2	0.9 - 1	2 - 4	2 - 5	0.3 - 0.6	0.2 - 0.3

Tables 11 and 12—all HQs are rounded to one significant digit. Range of overlap between kingfisher HQs based on ingestion dose and egg accumulation approaches. a—no overlap between dose and egg HQ ranges. The highest HQ of the lower range and the lowest HQ of the higher range are shown..

A road-killed mink was collected near Reach 3, and the liver analyzed for total PCBs and dioxin-like PCB congeners for calculating liver TEC (discussed in Section 5.2.9.1). The measured PCB concentration in the liver is consistent with modeled PCB uptake by mink foraging along Reaches 3 or 4. The liver TEC of the road-killed mink exceeds thresholds for reproductive impairment (litter size, kit survival, kit growth) defined as the geometric mean of the NOAEC and LOAEC liver TRVs (Appendix G). This means that the liver TEC of the road-killed mink is intermediate between the liver TECs of mink not showing adverse effects and those of mink with adverse effects. The road-killed mink supports the same conclusions reached in the FERA based on modeled dietary exposure to Reach 3 fish, crayfish, and terrestrial prey—that exposures mostly fall in the gray area between levels not causing adverse effects and levels associated with impaired reproduction.

5.2.7 Risk Characterization of Richland Creek Reach 2

In all three sample years, the mink UCL no effect HQs exceed 1 in Reach 2 (2 to 9), and the mean no effect HQs mostly exceed 1 (0.9 to 4) (Table 13). The mink UCL low effect HQs equal or exceed the levels associated with reproductive impairment in all three sample years (1 to 2), as do all but one of the mean low effect HQs (1 to 2 in 2001, 0.7 to 0.9 in 2002, and 0.9 to 2 in 2003)⁴⁰ (Table 14). This indicates that mink feeding in Reach 2 are potentially at risk of reproductive impairment (approximately 2 miles downstream of Neal's Landfill).

The kingfisher consensus no effect HQs show similar results as for mink, but with a wider range—UCL HQs of 4 to 20 and mean HQs of 3 to 9 (Table 13). The kingfisher consensus UCL low effect HQs all equal to exceed 1 (1 to 4) as do all but one mean low effect HQ (0.7 to 2) (Table 14). This indicates that kingfisher feeding in Reach 2 are potentially at risk of reproductive impairment (approximately 2 miles downstream of Neal's Landfill).

Piscivores feeding in Richland Creek Reach 2 may be expected to include as part of their prey fish and crayfish from Conard's Branch, which changes the risk estimates since Conard's Branch prey have much higher PCB concentrations (Figures 4 and 5). The combined exposure scenario is assessed in Section 5.2.8.

5.2.8 Risk Characterization of Conard's Branch (Reach 1) and Combined Reaches 1 & 2

5.2.8.1 Conard's Branch Hazard Quotient

The fish and crayfish in Conard's Branch have very high concentrations of PCBs (Tables 3 and 4), therefore the hypothetical risks to piscivores feeding exclusively on Conard's Branch prey are also high—mink UCL no effect HQs of 9 to 50, mean no effect HQs of 6 to 30, UCL low effect HQs of 4 to 20, and mean low effect HQs of 3 to 10; and kingfisher consensus UCL no effect HQs of 10 to 100, mean no effect HQs of 10 to 60, UCL low effect HQs of 5 to 10, and mean low effect HQs of 4 to 9 (Tables 13 and 14). These risk estimates are considered hypothetical because Conard's Branch is too small to solely support mink or kingfisher (Section 2.6).

Since neither mink or kingfisher are expected to forage exclusively in Conard's Branch, the potential risks of including Conard's Branch prey as part of the diets are assessed in two ways—HQs for the combined Reaches 1 & 2 scenario with fixed proportions of prey from each reach (Section 2.6), and the percent allowable consumption

⁴⁰ See footnote 39.

(PAC) approach that calculates the percent of the diet that can be taken from Conard's Branch (or the combined Reaches 1 & 2) without exceeding TRVs (Section 5.1.2).

5.2.8.2 Combined Reaches 1 & 2 Hazard Quotient

The foraging allocations in the combined Reaches 1 & 2 scenario are 25 % prey from Conard's Branch and 75 % from Richland Creek Reach 2 for mink, and 50 % from Conard's Branch and 50 % from Richland Creek Reach 2 for kingfisher (Section 2.6). The dietary compositions do not change for the two receptors—66 % fish, 13 % crayfish, and 21 % non-aquatic prey for mink; and 80 % fish and 20 % crayfish for kingfisher (Section 2.6).

In the combined Reaches 1 & 2 scenario, all of the mink and consensus kingfisher HQs indicate that exposures meet or exceed the levels associated with adverse effects (Tables 13 and 14). The mink UCL no effect HQs range from 3 to 20, and the mean no effect HQs from 2 to 10. The mink UCL low effect HQs range from 2 to 6, and the mean low effect HQs from 1 to 4. Similarly, the kingfisher consensus HQs range from 9 to 60 for UCL no effect, 7 to 40 for mean no effect, 3 to 7 for UCL low effect, and 2 to 7 for mean low effect. This indicates that mink or kingfisher feeding on a mixed diet of prey from Conard's Branch and the first Richland Creek reach downstream of the confluence with Conard's Branch are potentially at risk of reproductive impairment (0.5 to 2 miles downstream).

5.2.8.3 Conard's Branch Percent Allowable Consumption

The percent allowable consumption (PAC) results are summarized in Tables 15 and 16 for Conard's Branch (Reach 1), Richland Creek Reaches 2 through 4, and the combined Reaches 1 & 2. The PACs for Reaches 5 and 6 are presented in Appendices A6 and A10.

The Conard's Branch PACs assume that the only exposure mink or kingfisher have to PCBs released from Neal's Landfill is through consumption of Conard's Branch fish and crayfish, with the remainder of the aquatic prey in the diets taken from streams that do not directly receive Neal's Landfill discharge, as represented by the data for Richland Creek upstream of the confluence with Conard's Branch. The PAC values therefore represent the percentage of the mink or kingfisher diet taken from Conard's Branch that would result in a NOAEC- or LOAEC-based HQ of 1. Note that a NOAEC HQ of 1 is not associated with adverse effects, but a LOAEC HQ of 1 means that the exposures are the lowest likely to cause potentially adverse effects.

The Conard's Branch (Reach 1) NOAEC-based PACs for mink range from 2 to 5 % for UCL exposures between 2001 and 2003 (Table 15), and from 3 to 8 % for mean exposures (Table 16). The mink LOAEC-based PACs range from 7 to 23 % for UCL exposures (Table 15), and from 13 to 35 % for mean exposures (Table 16). The results indicate that, to stay within no effect dietary concentrations, mink should forage along Conard's Branch for no more than a few percent of the total diet, and potentially adverse effects may be expected if approximately 10 to 20 % of the total diet comes from Conard's Branch (based on the UCL exposures, which account for the variability of the sample data used in determining the mean TEC in Conard's Branch fish and crayfish in each of the sample years). Viewed from another perspective, the UCL PACs indicate that mink may be at risk from foraging along Conard's Branch even if they obtain as much as 80 to 90 % of the total diet from areas not receiving direct PCB loading from Neal's Landfill.

Table 15. Percent Allowable Consumption (PAC) Based on UCL TEC or TEQ Exposure					
Reach	Year	NOAEC-NOAEL		LOAEC-LOAEL	
		Mink	Kingfisher	Mink	Kingfisher
1	2001	2	0.3 - 2	7	0.8 - 9
	2002	5	0.1 - 2	23	0.4 - 29
	2003	4	0.8 - 5	16	2 - 27
2	2001	10	0.8 - 5	43	2 - 54
	2002	25	0.7 - 11	100	2 - 100
	2003	20	2 - 15	78	7 - 99
1 & 2	2001	5	0.4 - 2	19	1 - 16
	2002	13	0.2 - 4	55	0.7 - 47
	2003	10	1 - 7	40	4 - 91
3	2001	60	3 - 27	100	10 - 100
	2003	33	7 - 45	100	22 - 100
4	2001	100	7 - 50	100	20 - 100

Appendices A6, B6, C6

Table 16. Percent Allowable Consumption (PAC) Based on Mean TEC or TEQ Exposure					
Reach	Year	NOAEC/NOAEL		LOAEC/LOAEL	
		Mink	Kingfisher	Mink	Kingfisher
1	2001	3	0.5 - 4	13	0.5 - 19
	2002	8	0.2 - 4	35	0.2 - 44
	2003	5	1 - 8	20	0.9 - 37
2	2001	21	2 - 11	86	1 - 100
	2002	40	1 - 19	100	1 - 100
	2003	26	4 - 27	100	3 - 100
1 & 2	2001	8	0.8 - 6	35	0.7 - 28
	2002	21	0.4 - 6	86	0.4 - 73
	2003	12	2 - 12	51	1 - 81
3	2001	100	12 - 89	100	11 - 100
	2003	76	16 - 100	100	15 - 100
4	2001	100	26 - 100	100	24 - 100

Appendices A10, B10, C10

The mink PACs do not represent the allowable percentage of Conard's Branch fish and crayfish in a mink diet because another assumption is that aquatic prey from all sources represent 79 % of the mink diet (66 % fish and 13 % crayfish), with 21 % of the diet with no PCBs (bird and small mammal prey) (Section 2.6). This means that one-fifth of the diet is assumed to have no PCB contamination regardless of where the mink forage. The allowable percent consumption of fish and crayfish can be calculated by multiplying the mink PACs by 0.79. The percentage of Conard's Branch fish and crayfish that would result in a mink LOAEC-based HQ of 1 is 6 to 18 % for UCL exposure, and 10 to 28 % for mean exposure. Therefore, mink may be at risk even if as much as approximately 80 to 95 % of the total diet consists of prey not directly exposed to the release of PCBs from Neal's Landfill (based on UCL exposure).

Another way to consider this is to calculate the number of fish required to reach the LOAEC. For a mink food ingestion rate of 160 g/mink/d (Bleavins and Aulerich 1981 as cited in USEPA 1993, assuming 1 kg bodyweight), 6 to 18 % of the diet is 10 to 29 g aquatic prey per day from Conard's Branch. The fish component of the allowable Conard's Branch aquatic prey is 8 to 24 g/d⁴¹. The mean weight of the individual creek chubs and green sunfish collected from Conard's Branch in 2001 is 11.4 g (calculated from Sprenger, et al. 2002), which means that less than 1 to 3 small fish per day on average from Conard's Branch, along with just 2 to 5 g crayfish, could result in potential reproductive impairment in mink—hypothetically assuming that the remainder of the diet is somehow entirely free of PCBs from Neal's Landfill.

The kingfisher diet is 100 % aquatic prey, so the kingfisher PACs directly represent the percent allowable consumption of fish and crayfish from the reach under consideration. In contrast to the kingfisher consensus HQs, the kingfisher PACs are based on the full range of results for TEQ dose and egg TEC. The kingfisher PACs range over about 1 order of magnitude (10-fold) at any particular sample location and date due to the wide range in egg TEC TRVs (Section 4.2.2.5). The kingfisher LOAEC-based PAC range includes the corresponding mink PAC for each individual reach (Tables 15 and 16).

For Conard's Branch, the kingfisher NOAEC-NOAEL-based PACs range from 0.1 to 5 % for UCL exposures, and 0.2 to 8 % for mean exposures. The LOAEC-LOAEL-based PACs range from 0.4 to 29 % for UCL exposures, and 0.2 to 44 % for mean exposures, which bracket the corresponding mink PAC ranges for Conard's Branch. Another way to consider this is to calculate the number of fish required to reach the LOAEC. For a kingfisher food ingestion rate of 0.5 g/g_{BW}-d (Appendix A2) and a kingfisher bodyweight of 147 g (mean adult values cited in USEPA 1993), 0.4 to 29 % of the diet corresponds to 0.3 to 21 g aquatic prey per day from Conard's Branch⁴². The fish component of the allowable Conard's Branch aquatic prey is 0.2 to 17 g/d⁴³. The mean weight of the individual creek chubs and green sunfish collected from Conard's Branch in 2001 is 11.4 g (calculated from

⁴¹ $(66\% \text{ fish} / (66\% \text{ fish} + 13\% \text{ crayfish})) * 100 = 83.5\% \text{ fish of the total aquatic prey}$. Therefore, 10 g of aquatic prey * 0.835 = 8.4 g fish, and 29 g * 0.835 = 24.2 g fish.

⁴² $0.5 \text{ g food/g}_{\text{BW}}\text{-d} * 147 \text{ g}_{\text{BW}} * 0.004 \text{ allowable aquatic prey fraction of diet} = 0.29 \text{ g allowable aquatic prey per day}$, and $0.5 \text{ g food/g}_{\text{BW}}\text{-d} * 147 \text{ g}_{\text{BW}} * 0.29 \text{ allowable aquatic prey fraction of diet} = 21.3 \text{ g allowable aquatic prey per day}$

⁴³ $0.3 \text{ g aquatic prey} * 0.8 \text{ fraction of fish} = 0.24 \text{ g fish}$, and $21 \text{ g aquatic prey} * 0.8 \text{ fraction of fish} = 16.8 \text{ g fish}$.

Sprenger, et al. 2002), which means that as little as 1 small fish every 7 weeks from Conard's Branch ⁴⁴ along with 0.1 g crayfish over the same time period ⁴⁵, to 10 fish per week ⁴⁶ with 5 g crayfish ⁴⁷, could result in potential reproductive impairment in kingfisher--hypothetically assuming that the remainder of the diet is somehow free of Neal's Landfill PCBs.

Of course, piscivores foraging in the vicinity of Conard's Branch would necessarily forage along Richland Creek as well. This means that much of the remaining diet taken outside of Conard's Branch will include prey that have accumulated PCBs from Neal's Landfill releases. The combined exposures are assessed in Section 5.2.8.4.

5.2.8.4 Reaches 1 & 2 Percent Allowable Consumption

Most of the mink and kingfisher LOAEC-based PACs do not overlap in combined reach 1 & 2 because of differences in the apportionment of foraging between Conard's Branch and Richland Creek (Section 2.6). The combined Reaches 1 & 2 NOAEC-based PACs for mink range from 5 to 13 % for UCL exposures (Table 15), and from 8 to 21 % for mean exposures (Table 16). The mink LOAEC-based PACs range from 19 to 55 % for UCL exposures (Table 15), and from 35 to 86 % for mean exposures (Table 16). The results indicate that to stay within no effect dietary concentrations, mink should forage along the combined Conard's Branch and Richland Creek reach for no more than approximately 5 to 10 % of the total diet, and that potentially adverse effects may be expected if mink obtain more than approximately 20 to 60 % of the total diet from the combined reach (based on the UCL exposures, which account for the variability of the sample data used in determining the mean TEC in Conard's Branch and Richland Creek fish and crayfish in each of the sample years). Since the combined reach length is approximately double the home range for female mink (Section 2.6), these results indicate that mink are likely to be potentially at risk of reproductive impairment even with much less intensive use of the combined Reaches 1 & 2 than assumed for risk characterization in the FERA. Viewed from another perspective, the UCL PACs indicate that mink may be at risk from foraging in the combined Reaches 1 & 2 even if they forage for as much as 40 to 80 % of the total diet from areas not receiving direct PCB loading from Neal's Landfill.

The mink PACs do not represent the allowable percentage of combined Reaches 1 & 2 fish and crayfish in a mink diet because of the assumption that aquatic prey from all sources represents 79 % of the mink diet (Section 2.6). This means that one-fifth of the diet is assumed to have no PCB contamination regardless of where mink forage. The percentage of fish and crayfish from the combined Reaches 1 & 2 that would result in a mink LOAEC-based HQ of 1 is 15 to 43 % for UCL exposure, and 28 to 68 % for mean exposure (the calculation is described in Section 5.2.8.3). Potentially adverse effects may be expected if mink obtain more than approximately 15 to 40 % of the total diet as fish and crayfish from the combined reach (based on UCL PACs), which also means that mink may

⁴⁴ 11.4 g per fish / 0.24 g fish allowable consumption per day = 47.5 days allowable consumption per fish, and 47.5 days per fish / 7 days/week = 6.8 weeks per fish

⁴⁵ 0.3 g allowable aquatic prey per day - 0.24 g allowable fish per day = 0.06 g allowable crayfish per day

⁴⁶ 16.8 g fish allowable consumption per day / 11.4 g per fish = 1.47 fish allowable consumption per day, and 1.47 fish per day * 7 days/week = 10.3 fish per week.

⁴⁷ 21.3 g allowable aquatic prey per day - 16.8 g allowable fish per day = 4.5 g allowable crayfish per day

be at risk even if as much as 60 to 85 % of the total diet consists of prey not directly exposed to the release of PCBs from Neal's Landfill.

For kingfisher in the combined Reaches 1 & 2, the NOAEC-NOAEL-based PACs range from 0.2 to 7 % for UCL exposures, and 0.4 to 12 % for mean exposures. The LOAEC-LOAEL-based PACs range from 0.7 to 90 % for UCL exposures, and 0.4 to 80 % for mean exposures. This indicates that kingfisher are likely to exceed no effect levels with only a small portion (less than 12 %) of the diet obtained by foraging along both Richland Creek Reach 2 and Conard's Branch. The full range of kingfisher risk estimates for foraging over both Richland Creek and Conard's Branch result in PACs ranging from about 1 % to 90 %, indicating high to moderate risk of exceeding low effect levels.

5.2.9 Additional Risk Information

5.2.9.1 Road-killed Mink

A road-killed mink was collected 7/31/04 from the Highway 48 bridge over Richland Creek (near the upstream end of Reach 3, see Section 3.2). The mink's liver was analyzed by USFWS for total PCBs, dioxin-like PCB congeners, and lipid content (Sparks 2005). The liver PCB concentration and TEC are compared to tissue-based toxicity reference values (TRVs) from studies of mink and otter. Since only a single sample is available, the liver TEC and PCB concentrations are compared to an array of mink and otter liver TRVs (Section 4.2.1 approach 4) (Appendix G). The road-killed mink has a lower liver lipid content (2.6 %) than a default value of 5 % representative of healthy mink populations (Kannan, et al. 2000)⁴⁸. Therefore, to minimize confounding by differences in liver lipid content, comparisons with TRVs are made on a lipid-normalized basis.

The road-killed mink liver lipid-normalized TEC of 3010 pg/g lipid exceeds NOAECs for hepatic (liver) vitamin A, kit survival, and kit growth; exceeds LOAECs for enzyme induction and hepatic retinoids (vitamin A and precursors), is within the range associated with jaw lesions (mandibular and maxillary squamous cell proliferation); and exceeds threshold concentrations for adverse effects on litter size, kit survival, and kit growth (the threshold is defined as the geometric mean of NOAEC and LOAEC TRVs) (Appendix G).

The road-killed mink liver lipid-normalized PCB concentration of 34 µg/g lipid exceeds NOAECs for hepatic vitamin A, litter size, kit survival, and kit growth; exceeds the maximum PCB concentrations associated with reduced baculum (penis bone) length; exceeds the critical level (EC₉₀) for vitamin A deficiency, and exceeds LOAECs for adverse effects on kit production, kit survival, and kit growth (Appendix G).

Based on these comparisons, the road-killed mink was at increased risk of vitamin A deficiency (possible effects include impaired disease resistance, reproduction, development, and vision), jaw lesions (which can result in loss of teeth), and reproductive impairment. The TEC-based results for reproductive endpoints are similar to the risk

⁴⁸ Kannan, et al. (2000) cite Poole, et al. (1995) for the value of 5 % lipid content in livers of healthy mink populations, however, the mink mean liver lipid content in Poole, et al. (1995) is 6 % (calculated from their Table 1). The 5 % value is used in this memo for consistency with Kannan, et al (2000).

estimates for Reach 3 mink based on fish data—exposures to Reach 3 mink fall in the gray area between no effect levels and levels associated with adverse reproductive effects (Section 5.2.6).

The history of the road-killed mink is unknown, specifically, where it foraged prior to its death. The expected mink liver PCB concentration and TEC are modeled from the mink dietary PCB and PCB congener concentrations in the FERA, using the mean diet-to-liver biomagnification factors (BMFs) reported for 3 exposure treatments in a mink feeding study (Tillitt, et al. 1996). The Tillitt, et al. (1996) BMFs are not lipid-normalized, so mink dietary exposures are calculated on a whole-tissue wet-weight basis for modeling uptake in mink livers. First, UCL lipid-normalized mink dietary PCB and PCB congeners are calculated for the mink dietary composition of 66 % fish and 13 % crayfish from the fish and crayfish data in Appendices A4 and C4 ⁴⁹ (Appendix H2). Next, the lipid-normalized dietary concentrations are converted to whole-tissue wet-weight dietary concentrations (Appendix H2) by multiplying by the fraction of lipid in the modeled mink diet (Appendix H1). Then the wet-weight dietary concentrations are multiplied by the respective mean diet-to-liver BMFs of 2.57 (total PCB), 0.13 (77), 1.17 (81), 5.4 (105), 4.03 (114), 4.6 (118), 0.57 (123), 11.77 (126), 11.25 (156/157), 6.1 (167), 13.6 (169), and 10 (189) (congener numbers in parentheses) (BMFs calculated from Table 5 in Tillitt, et al. 1996). The modeled liver congener concentrations are multiplied again by the respective mammalian TEFs (Section 3.3.2), and summed to calculate the modeled mink liver TEC. Modeling is based on data for August 2001 and May 2003 because these sample dates are more representative of conditions during the season in which the mink was killed (July) than the data collected in November 2002.

The liver data for the road-killed mink (near Reach 3) are consistent with the modeled accumulation of PCBs in mink feeding predominantly along Reaches 3 or 4 of Richland Creek, and is inconsistent with modeled uptake for reaches further upstream or downstream (Figures 6 and 7). The road-killed mink liver total PCB (0.9 µg/g) is intermediate between the modeled range of 0.8 µg/g (August 2001) and 1.5 µg/g (May 2003) for Reach 3, and the liver TEC (77 pg/g) is intermediate between the modeled range of 27 pg/g (2001) and 87 pg/g (2003). The 2001 Reach 4 modeled mink liver concentrations are similar to the Reach 3 values for both total PCB (1.1 µg/g) and TEC (21 pg/g). Modeled mink liver concentrations are much lower further downstream (0.2 to 0.3 µg/g PCB and 4 to 5 pg/g TEC in Reaches 5 and 6 in 2001), and much higher further upstream (3 to 4 µg/g PCB and 280 to 489 pg/g TEC in Reach 2 in 2001 and 2003) than the levels in the road-killed mink. The liver uptake modeling is performed with the same mink exposure assumptions used in the FERA, specifically, a dietary composition of 66 % fish, 13 % crayfish, and 21 % terrestrial prey (Section 2.6), with missing crayfish data modeled from other crayfish data from the site. Terrestrial prey are assumed to have 0 PCBs or TEC.

⁴⁹ The lipid-normalized dietary concentrations differ from those reported in Appendices A4 and C4 because A4 and C4 report values for kingfisher based on a different dietary composition (80 % fish and 20 % crayfish) from that of mink.

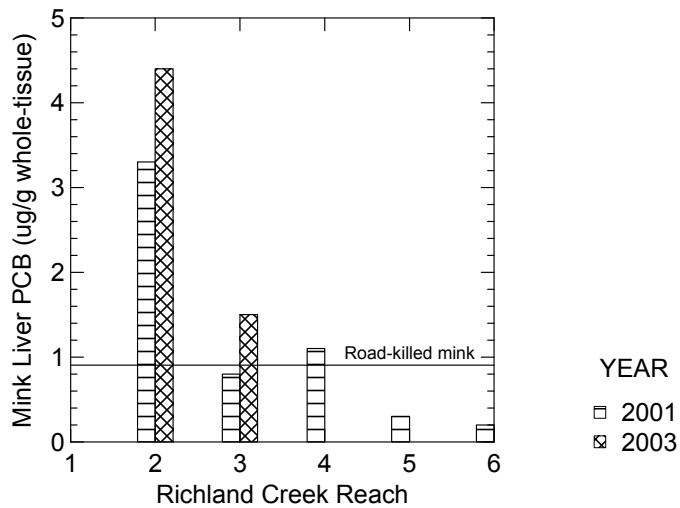


Figure 6. Modeled Accumulation of Total PCBs in Mink Livers in Richland Creek Reaches (August 2001 and May 2003) Compared to Liver PCBs Measured in a Road-killed Mink Collected July 2004 from the Highway 48 Bridge Over Richland Creek.

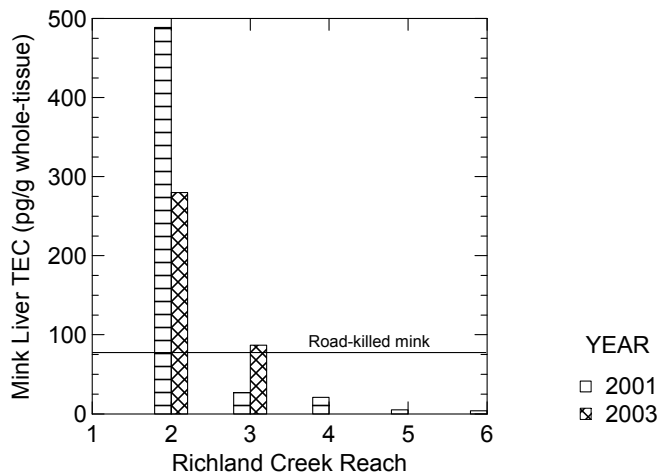


Figure 7. Modeled Accumulation of Dioxin Toxic Equivalent Concentration (TEC) in Mink Livers in Richland Creek Reaches (August 2001 and May 2003) Compared to Liver TEC Based on PCB Dioxin-like Congeners Measured in a Road-killed Mink Collected July 2004 from the Highway 48 Bridge Over Richland Creek.

The liver uptake modeling also indicates that mink could potentially receive lethal or near lethal exposure to PCBs by foraging in Reach 2. The modeled liver accumulation of PCBs from Reach 2 (3 to 4 µg/g whole tissue for 2001 and 2003, respectively) is close or equal to the mean PCB concentrations in the livers of mink that died during PCB feeding experiments (4 to 5 µg/g whole tissue) (Aulerich, et al. 1973). Exposure is much greater with a small portion (25 % of diet) of Conard's Branch foraging included with Richland Creek Reach 2 (75 % of diet), resulting in modeled liver uptake of 8 to 11 µg/g whole tissue, which greatly exceeds the liver concentrations associated with mortality. The modeling results for the upper portion of Richland Creek provide a possible explanation why mink signs have not been observed along Richland Creek Reach 2.

Uncertainties are discussed in Section 6.5.1.

5.2.9.2 Risk to Fish

As discussed in Section 2.3, fish were not selected as one of the original assessment endpoints under the expectation that fish would be adequately protected by assessing risk to piscivores. This expectation should hold when piscivores are assessed for full utilization of a reach (area use factor = 100 %), but the protectiveness to fish is unknown in reaches in which risk to piscivores is assessed for partial utilization (area use factor < 100 %), or if findings of piscivore risks are dismissed because the areal extent of the reaches with elevated risk is claimed to be insufficiently large to result in population-level impacts. In these situations, the potential risks to fish, or other components of the aquatic ecosystem, are highly uncertain. The purpose of the additional risk information in this section is to reduce the uncertainty by bounding the potential risks of PCB exposures to fish in the reaches closest to the source of PCB releases.

The TRV for assessing risk to fish, 14.1 ng TCDD/kg lipid (fillet), is the LOAEC for reduced survival and adverse (listless and unresponsive) behavioral effects in adult female rainbow trout (*Oncorhynchus mykiss*) following 200 days dietary exposure to TCDD, as reported by Giesy, et al. (2002) and Jones, et al. (2001). A tissue-based NOAEC was not reported. Exposure of Conard's Branch and Richland Creek fish to dioxin-like activity is characterized as lipid-normalized TEC based on whole-fish congener data and WHO fish TEFs (Section 3.3.2). Potential risk is characterized as the hazard quotient of tissue TEC divided by tissue TCDD LOAEC, on a lipid-normalized basis (Appendices A4, A8, B4, B8, C4, C8).

Fish TECs are 20 to 100 times greater than the fish tissue LOAEC for growth and behavioral impairment in Conard's Branch, 6 to 40 times greater in Richland Creek Reach 2, 2 to 4 times greater in Reach 3, and 1 to 4 times greater in Reach 4, based on UCL TEC in whole fish (Table 17). Risk estimates are one-third to one-half lower based on mean TEC in whole fish, but still indicate elevated potential risk in Conard's Branch (HQs of 10 to 60) and Richland Creek Reaches 2 (HQs of 3 to 20) and 3 (HQ of 1)⁵⁰ (Table 18).

The main uncertainty associated with this approach is the relative susceptibility to dioxin-like effects of the species sampled in Conard's Branch and Richland Creek as compared to rainbow trout. Salmonids (including trout) are known to be highly sensitive to dioxin-like effects. The Jones, et al. (2001) / Giesy, et al. (2002) study is the first to

⁵⁰ The fish TRV is a LOAEC, that is, the tissue concentration associated with adverse effects, which means that adverse effects may occur at a HQ of 1.

report survival and behavioral effects in adult fish resulting from long-term chronic exposure to dioxin, so there is no presently available database for directly comparing species relative susceptibilities of chronically-exposed adults. Comparisons of species sensitivities to dioxin-like effects are available for fish eggs and fish juveniles.

Elonen, et al. (1998), in a comparison of the egg sensitivities of ten species of freshwater fish to dioxin, reported up to 2 orders-of-magnitude (180-fold) range between the most sensitive (lake trout eggs) and least sensitive (zebrafish eggs) to acute effects (LC_{50}) on a lipid-normalized basis, and a 38-fold range on a whole-egg basis⁵¹. Rainbow trout eggs are approximately 6-fold less sensitive than lake trout eggs to dioxin on a whole-egg or lipid-normalized basis⁵², for an overall range in fish egg sensitivities relative to rainbow trout eggs of 30-fold on a lipid-normalized basis and 6-fold on a whole-egg basis. White sucker eggs are 15-fold less sensitive than rainbow trout eggs on a lipid-normalized basis, and 4-fold less sensitive on a whole-egg basis (calculated from Elonen, et al. 1998). No other species collected from Conard's Branch or Richland Creek are included in the Elonen, et al. (1998) paper.

The range of sensitivities to dioxin-like effects appears to be much smaller for juvenile fish than for eggs (Monosson 1999). Kleeman, et al. (1988) determined acute LC_{50} values for six species of freshwater fish (intraperitoneal injection of TCDD at 4 doses, with mortality, growth, and lesions assessed for 80 days post-injection). An overall 5-fold range of sensitivities was reported between species (on a whole-tissue basis). An important difference with egg toxicity studies is that salmonids are not the most sensitive juvenile fish to dioxins – common carp, yellow perch (*Perca flavescens*), and black bullhead (*Ictalurus melus*) are more sensitive than rainbow trout. Bluegill sunfish (*Lepomis macrochirus*), a related species to green and longear sunfish collected from Conard's Branch and Richland Creek, are only 1.6 times less sensitive to the acute effects of dioxin compared to rainbow trout, and common carp are 3 times *more* sensitive than rainbow trout (calculated from Kleeman, et al. 1988).

If the relative sensitivities of juvenile or young adult fish to acute dioxin-like effects are reliable indicators of the relative sensitivities to chronic effects, sunfish would still be at risk of growth and behavioral impairment in Conard's Branch and Richland Creek Reach 2, but not Reach 4 (sunfish data are not available for Reach 3). The adjusted HQs would be 20 to 50 in Conard's Branch, 3 to 20 in Reach 2, and 0.4 to 0.6 in Reach 4 (range of UCL and mean risk estimates for all available sample dates). If the relative sensitivities of fish eggs to acute dioxin-like effects are reliable indicators of the relative sensitivities of adult fish to chronic effects, white sucker would not be at risk based on 2001 or 2003 data (adjusted HQs < 1), but would still be at risk in Reach 2 based on 2002 data (adjusted HQ of 3). Adult creek chub would have to be more than an order-of-magnitude less sensitive than adult rainbow trout to not be at risk in Reach 2, and as much as 2 orders-of-magnitude less sensitive to not be at risk in Conard's Branch.

The risk-to-fish assessment indicates that fish are at risk of direct adverse effects of PCB exposure in Conard's Branch and the upper reaches of Richland Creek. The distance downstream to which fish remain at risk depends on the relative sensitivities of the fish species at the site compared to rainbow trout, but, even after adjusting for reported differences in sensitivities among fish, the downstream extent of potential risk to fish appears to be similar

⁵¹ The difference in the magnitude of the ranges is due to species differences in egg lipid content.

⁵² Rainbow trout egg lipid content of 8.7 % (Niimi 1983).

as for mink or kingfisher, that is, elevated risk in Conard's Branch and Richland Creek Reach 2, possible, but more uncertain risk in Reach 3 due to data limitations, and low or no risk further downstream.

Uncertainties are discussed further in Section 6.5.2.

Table 17. Risk to Fish—Hazard Quotients (HQ) Based on 95 % Upper Confidence Limit (UCL) Whole-Fish Lipid-normalized TEC and Lowest Observed Adverse Effect Concentration (LOAEC)								
Species	Year	Conard's Branch	Richland Creek Reach					
			0	2	3	4	5	6
Creek Chub	2001	50			4			
	2002	100	0.8	10				
	2003	20		6				
Longear or Green Sunfish	2001	70		30		1	0.4	0.1
	2002			10				
	2003			7				
White Sucker	2001			7		4		
	2002		0.4	40				
	2003			6				
Black or Shorthead Redhorse	2001					1	0.3	0.1
	2003				2			
Common Carp	2001						0.1	0.2

TRV - 14.1 ng TCDD/kg lipid (Giesy, et al. 2002; Jones, et al. 2001)

Hazard quotients are rounded to one significant digit.

Table 18. Risk to Fish–Hazard Quotients (HQ) Based on Mean Whole-Fish Lipid-normalized TEC and Lowest Observed Adverse Effect Concentration (LOAEC)

Species	Year	Conard's Branch	Richland Creek Reach					
			0	2	3	4	5	6
Creek Chub	2001	20			1			
	2002	60	0.4	9				
	2003	10		4				
Longear or Green Sunfish	2001	40		20		0.6	0.2	0.04
	2002			8				
	2003			5				
White Sucker	2001			3		0.6		
	2002		0.3	20				
	2003			4				
Black or Shorthead Redhorse	2001					0.6	0.2	0.03
	2003				0.9			
Common Carp	2001						0.1	0.04

See notes to Table 17.

5.3 Whole-fish PCB and TEC Preliminary Goals

5.3.1 Richland Creek Reaches

Protective whole-fish concentrations of 0.7 to 0.9 mg/kg PCBs or 7 to 26 pg/g TEC, NOAEC to LOAEC, respectively, are calculated from the mink dietary TRVs. These values account for the modeled mink dietary composition of 66 % fish, 13 % crayfish, and 21 % without PCB contamination, incorporate the site-specific ratio of PCB concentrations in fish and crayfish, and apply to any of the Richland Creek reaches.

Protective whole-fish concentrations of 0.3 to 0.9 mg/kg PCBs (NOAEL) to 1 to 3 mg/kg PCBs (LOAEL), or 3 to 32 pg/g TEC (NOAEL to LOAEL) are calculated from the range of kingfisher ingestion dose TRVs. These values account for the modeled kingfisher dietary composition of 80 % fish and 20 % crayfish, incorporate the site-specific ratio of PCB concentrations in fish and crayfish, and apply to any of the Richland Creek reaches.

5.3.1.1 Mink

Protective whole-fish concentrations for mink are calculated as follows:

$$\text{TRV}_{\text{diet}} = 0.66 C_f + 0.13 C_c + 0.21 C_t \quad [9]$$

where TRV_{diet} is the dietary toxicity reference value for mink, C_f is the protective concentration in whole fish, C_c is the protective concentration in crayfish, and C_t is the concentration in terrestrial prey (assumed to be 0). Since the mean fish (creek chub, green and longear sunfish) PCB conc. / mean crayfish PCB conc. = 2.99 (Appendix D), the protective concentrations in fish and crayfish are expected to have the same ratio.

$$C_c = C_f / 2.99 \quad [10]$$

Combining Equations 9 and 10, entering 0 for C_t , and simplifying:

$$C_f = \text{TRV}_{\text{diet}} / 0.7 \quad [11]$$

Equation 11 is used to calculate the protective fish concentrations corresponding to mink dietary TRVs. The fish goals represent the average concentration in the mix of available species over a reach.

Table 19. Whole-body Fish Goals Protective for Mink				
Basis	PCB NOAEC	PCB LOAEC	TEC ^a NOAEC	TEC ^a LOAEC
Units	mg/kg		pg/g	
TRV_{diet}	0.5	0.6	4.6	18
Fish goal	0.7	0.9	7	26

a) TEC based on WHO mammalian TEFs (Van den Berg, et al. 1998).

5.3.1.2 Kingfisher

The first step for calculating protective whole-fish goals for kingfisher is to convert the kingfisher ingestion dose TRVs (TRV_{dose}) to dietary TRVs (TRV_{diet}):

$$\text{TRV}_{\text{diet}} = \text{TRV}_{\text{dose}} / \text{FD}_{\text{ingest}} \quad [12]$$

where $\text{FD}_{\text{ingest}}$ is the kingfisher food ingestion rate (0.5 kg food/kg_{BW}-d).

$$\text{TRV}_{\text{diet}} = 2 \text{TRV}_{\text{dose}} \quad [13]$$

For a kingfisher dietary composition of 80 % fish and 20 % crayfish, the dietary exposure is

$$\text{TRV}_{\text{diet}} = 0.8 C_f + 0.2 C_c \quad [14]$$

Combining Equations 10 and 14, simplifying, and rearranging:

$$C_f = \text{TRV}_{\text{diet}} / 0.87 \quad [15]$$

Combining Equations 13 and 15, and simplifying:

$$C_f = 2.3 \text{TRV}_{\text{dose}} \quad [16]$$

Equation 16 is used to calculate the protective fish concentrations corresponding to kingfisher ingestion dose TRVs. The fish goals represent the average concentration in available species over a reach.

Table 20. Whole-body Fish Goals Protective for Kingfisher Based on Ingestion Dose						
Basis	PCB NOAEL		PCB LOAEL		TEC ^a NOAEL	TEC ^a LOAEL
TRV units	mg/kg _{BW} -d				ng/kg _{BW} -d	
TRV _{dose}	0.11	0.4	0.5	1.12	1.4	14
Fish goal	0.25	0.9	1.2	2.6	3	32
Fish units	mg/kg				pg/g	

a) TEC based on WHO mammalian TEFs (Van den Berg, et al. 1998).

A different procedure is necessary to calculate protective fish goals from the kingfisher egg TRVs because the egg TRVs are lipid-normalized, and because egg TEC is calculated with avian TEFs, in contrast to the mammalian TEFs used to calculate mink diet and kingfisher ingestion dose. To derive protective whole-body fish TEC (mammalian) from lipid-normalized egg TEC (avian), the empirical ratio between fish TEC (mammalian) and egg TEC (avian) is needed, and the lipid content of the kingfisher diet. The calculations are performed with the 2003 data because crayfish were sampled at that time (not modeled as in 2001 and 2002).

First, the mean lipid-normalized dietary TEC (mammalian) is calculated from the data in Appendix C8, using WHO mammalian TEFs, and the 80 % fish and 20 % crayfish dietary composition for kingfisher (the outlier CF 15 is excluded from all the calculations). The calculated lipid-normalized TEC (mammalian) of the 2003 kingfisher diet in Reach 2 is 0.78 µg/kg lipid. The 2003 Reach 2 egg TEC (avian) is 49.34 µg/kg lipid (Appendix C9), and the ratio of egg TEC (avian) divided by fish TEC (mammalian) is 62.92. This ratio can be used to calculate the dietary TECs (mammalian) that correspond to the egg TEC (avian) TRVs. The two egg NOAEC TRVs of 1.79 and 17 µg/kg lipid correspond to dietary TEC (mammalian) of 0.03 and 0.27 µg/kg lipid, and the two egg LOAEC TRVs of 5.33 and 68 µg/kg lipid correspond to dietary TEC (mammalian) of 0.08 and 1.08 µg/kg lipid. The next step is to convert the lipid-normalized TEC to whole-diet TEC. The lipid content of the 2003 Reach 2 kingfisher diet is 3.16 % (calculated from Appendix C3 excluding the CF 15 outlier). The whole-diet TEC is then calculated by multiplying the lipid-normalized dietary TEC by the dietary lipid content (as a fraction), and multiplying again by 1000 to convert µg/kg to pg/g. Although these calculations include crayfish and fish together, the crayfish contribution to PCB or TEC in the diet is small (less than 10 % of the total), and therefore the protective dietary TEC values may be treated as protective fish goals without the additional complication of disaggregating the crayfish portion.

The two egg NOAEC TRVs of 1.79 and 17 µg/kg lipid correspond to whole-diet TEC (mammalian) of 0.9 and 8.5 pg/g, and the two egg LOAEC TRVs of 5.33 and 68 µg/kg lipid correspond to whole-diet TEC (mammalian) of 2.7 and 34.2 pg/g. In both cases, the egg-based protective fish goals bracket the ingestion dose-based fish goals of 3 pg/g (NOAEL) and 32 pg/g (LOAEL) (Table 20), and bracket the diet-based mink fish goals of 7 pg/g (NOAEC) and 26 pg/g (LOAEC) (Table 19).

5.3.2 Conard's Branch

Additional calculations are performed for fish goals in Conard's Branch since it is too small to fully support mink or kingfisher. Whole-fish concentrations of 1.8 to 2.3 mg/kg PCBs or 17 to 68 pg/g TEC (NOAEC to LOAEC) are protective for mink foraging 25 % in Conard's Branch, and 75 % in Richland Creek Reach 2. The corresponding values for kingfisher foraging 50 % in Conard's Branch are 0.5 to 2 mg/kg PCBs (NOAEC), 2.4 to 5 mg/kg PCBs (LOAEC), and 6 to 63 pg/g TEC (NOAEC to LOAEC).

5.3.2.1 Mink

An additional calculation is performed for fish goals in Conard's Branch since it is too small to fully support a mink. For goal-setting purposes, the mink are assumed to forage 25 % in Conard's Branch and 75 % in Richland Creek.

$$C_{f1\&2} = 0.25 C_{CB} + 0.75 C_{RC} \quad [17]$$

where $C_{f1\&2}$ is the mean fish concentration in Reaches 1 and 2, C_{CB} is the fish concentration in Conard's Branch, and C_{RC} is the fish concentration in Richland Creek (the reach immediately downstream of the confluence with Conard's Branch).

To solve for C_{CB} , the ratio of mean concentrations in fish in Conard's Branch and Richland Creek is needed. This was calculated from the mean values reported in Tables 3 and 4. First, the mean concentrations of the fish species collected within a reach were averaged (reach mean), and then the Conard's Branch reach mean was divided by the Richland Creek (Reach 2) reach mean separately for 2001, 2002, and 2003. For PCBs, the ratios for the respective years are 3.7, 6.1, and 4.9, for an overall ratio of 4.9; and for TEC, the ratios are 6.8, 4.6, and 5.5, for an overall ratio of 5.6.

$$PCB C_{f1\&2} = 0.25 C_{CB} + 0.75 (C_{CB} / 4.9) \quad [18]$$

$$C_{CB} = PCB C_{f1\&2} / 0.40 \quad [19]$$

$$TEC C_{f1\&2} = 0.25 C_{CB} + 0.75 (C_{CB} / 5.6) \quad [20]$$

$$C_{CB} = TEC C_{f1\&2} / 0.38 \quad [21]$$

Equations 19 and 21 are used to calculate the protective fish concentrations in Conard's Branch corresponding to mink dietary TRVs for mink foraging over both Conard's Branch and Richland Creek.

Table 21. Conard's Branch (CB) Whole-body Fish Goals Protective for Mink Primarily Foraging in Richland Creek (RC)				
Basis	PCB NOAEC	PCB LOAEC	TEC ^a NOAEC	TEC ^a LOAEC
Units	mg/kg		pg/g	
Fish goal	0.7	0.9	6.6	25.7
CB fish goal	1.8	2.3	17	68

a) TEC based on WHO mammalian TEFs (Van den Berg, et al. 1998).

5.3.2.2 Kingfisher

An additional calculation is performed for fish goals in Conard's Branch since it is too small to fully support a kingfisher. For goal-setting purposes, the kingfisher are assumed to forage 50 % in Conard's Branch and 50 % in Richland Creek.

$$C_{f1\&2} = 0.5 C_{CB} + 0.5 C_{RC} \quad [22]$$

Using the mean ratio of PCB concentrations or TECs in Conard's Branch and Richland Creek fish presented above:

$$PCB C_{f1\&2} = 0.5 C_{CB} + 0.5 (C_{CB} / 4.9) \quad [23]$$

$$C_{CB} = PCB C_{f1\&2} / 0.51 \quad [24]$$

$$TEC C_{f1\&2} = 0.5 C_{CB} + 0.5 (C_{CB} / 5.6) \quad [25]$$

$$C_{CB} = TEC C_{f1\&2} / 0.51 \quad [26]$$

Equations 24 and 26 are used to calculate the protective fish concentrations in Conard's Branch corresponding to ingestion dose TRVs for kingfisher foraging over both Conard's Branch and Richland Creek.

Table 22. Conard's Branch (CB) Whole-body Fish Goals Protective for Kingfisher Also Foraging in Richland Creek						
Basis	PCB NOAEL		PCB LOAEL		TEC ^a NOAEL	TEC ^a LOAEL
Fish goal	0.25	0.9	1.2	2.6	3	32
CB fish goal	0.5	2	2.4	5	6	63
Fish units	mg/kg				pg/g	

a) TEC based on WHO mammalian TEFs (Van den Berg, et al. 1998).

6 Uncertainty Analysis

Uncertainties are discussed under several categories that could influence the outcome of the FERA.

6.1 Fish and Crayfish Contaminant Data

The measured total PCB and PCB congener concentrations in fish and the associated dioxin toxic equivalent concentrations (TEC) are the site-specific data that drive the FERA. Several sources of uncertainty include the adequacy of the sampling effort for determining species concentrations within reaches (inadequate sample designs may result in excessively variable data), the fidelity of fish within a given reach (movement of fish between reaches could obscure spatial patterns in contaminant accumulation), species differences in accumulation (different combinations of sampled species will change the dietary concentrations and dose calculations that are the basis of the exposure characterizations), seasonal differences in PCB accumulation and/or fish lipid content (each of the sample events occurred in different months), annual changes in PCB loading from Neal's Landfill, and changes in the analytical procedures for determining total PCB concentrations (2 Aroclor methods and the sum of congeners method). Each concern is discussed below.

The moderate difference between UCL and mean exposure estimates (Section 5.2.3) indicates that sampling variability is not likely to have a major influence on risk characterization. In other words, most of the fish contaminant data are reasonably consistent within reaches and adequately characterized by the sampling effort. This can be seen by calculating the coefficient of variation (CV), which is the standard deviation (SD) divided by the mean. CVs are calculated for 55 mean PCB concentrations (including 4-peak and 5-peak Aroclor, and sum of congeners) and TEC values between 2001 and 2003 (Appendix F). Two CVs are shown for white sucker in Reach 0 in 2002. The first value is for the reduced data set excluding sample ID 15 (Section 3.3.7), the second value (in parentheses) is for the entire white sucker data set. The Reach 0 white sucker CVs for the reduced data set are included in the discussion below.

The CV is less than 0.5 for more than two-thirds (69 %) of the calculated means, which shows that sample variability is low for most of the analyses. The CV is as large or larger than the mean (defined here as rounded CV ≥ 0.9) for only 5 % of the analyses, which includes TEC in longear sunfish (0.97) and white sucker (1.02) in Reach 2, and Aroclor in longear sunfish (1.38) in Reach 5 in 2001 (CVs in parentheses). This indicates that a relatively small proportion of the data exhibits very high variability.

When the data are considered by sampling year, 2002 shows the greatest proportion of means with higher variability (defined here as CV > 0.5): 42 % in 2002 (5 of 12 means considering 4- and 5-peak Aroclor analyses together) compared to 22 % in 2001 (5 of 23 means) and 14 % in 2003 (2 of 14 means). This shows that overall the 2002 data are more variable compared to 2001 or 2003 data.

Although variability is not considered uncertainty (Section 1.2), the relatively low variability of the most of the contaminant data indicates that the uncertainty over the species mean concentrations within a reach at a particular time is generally low. This indicates that additional studies to reduce the uncertainty over the range of variability of fish contaminant levels at any one time is unlikely to significantly change the outcome of the FERA.

The low variability of mean PCB and TEC concentrations also indicates that the fish or crayfish sampled for analysis mostly stayed within a given reach prior to capture. Possible exceptions, based on high CV, include longear sunfish in Reaches 2 and 5, and white sucker in Reaches 0 and 2. Movement of fish in or out of Reach 5 is not a concern because exposures are well below risk levels. Movement of fish into Reach 2 from Conard's Branch may explain the higher variability, or it may reflect a water PCB concentration gradient along the length of the reach (higher concentrations near the confluence with Conard's Branch). This does not affect the findings of the FERA because piscivores foraging along Reach 2 will be exposed to the mix of fish contaminant concentrations regardless of its origin, and the risks of foraging in both reaches is assessed in the combined Reaches 1 & 2 scenario. The exceptionally high variability for white sucker in Reach 0 (CV of 1.7 for the complete data set) indicates that fish may have moved into Reach 0 from a reach downstream of the confluence with Conard's Branch, and inspection of the individual sample data indicates that one of the sampled white fish likely moved upstream from a more contaminated reach (Sections 3.3.7 and 5.1.2). Conversely, the data also show that about 90 % of the Reach 0 fish samples stayed upstream of the Conard's Branch confluence. Overall, possible movement of fish between sample locations is not a significant source of uncertainty in the FERA.

The fish contribution to mean dietary PCB exposure is calculated as the unweighted average of the species mean concentrations within a reach. One of the assumptions of the FERA is that piscivores show no preference for any particular species of fish (Section 2.6). Since contaminant levels may vary between species, differences in the particular combination of species sampled in a reach may introduce uncertainty in the estimated exposure to animals feeding on fish. The main differences in species composition between sample years are that green sunfish were sampled in Conard's Branch in 2001, but not thereafter; and creek chub were sampled in Reach 2 in 2002 and 2003, but not 2001 (Tables 3 and 4). Green sunfish had somewhat higher mean PCB concentration compared to creek chub in 2001 (Table 3), which indicates that the 2002 and 2003 exposure estimates for Conard's Branch might be low because they do not include sunfish data, but other considerations indicate that sunfish are unlikely to appreciably change exposure estimates. Mean TECs are similar in green sunfish and creek chubs in Conard's Branch in 2001 (Table 4). Although the Reach 2 mean TEC and Aroclor concentrations in longear sunfish are greater than in creek chub in 2002, the means are nearly identical in 2003 (Tables 3 and 4). The historic data tell a similar story, longear sunfish and creek chub had nearly identical mean Aroclor concentrations at the 1-stream mile sample location in 1982, but mean concentrations in sunfish were somewhat (40 - 60 %) higher than in creek chub in 1998 at the same location (Appendix D). The relationship between PCB uptake in sunfish and creek chub is inconsistent, so firm conclusions cannot be made regarding the effect of changes in the sampled species composition in Conard's Branch or Reach 2 between sampling years. The weight of evidence indicates that the effect is more likely to be small than large.

There are insufficient site-specific data to draw general conclusions regarding potential seasonal effects on PCB accumulation in fish. For the FERA, samples have been taken once each in three different months—May, August, and November. Of the three, the November data show the lowest PCB or TEC concentrations. This is mainly due to the very low lipid content of the November samples (fish with low lipid content have less storage capacity for lipophilic chemicals such as PCBs), and perhaps secondarily to temperature-dependent influences on PCB uptake. The 2002 lipid data were initially rejected as anomalously low, but no errors were found in the review of the laboratory calculations, and subsequent sampling by USFWS showed similarly low lipid contents in November (Section 3.1.2).

There is no obvious evidence of consistent temporal trends in fish PCB concentrations or TECs (based on visual inspection of data, and not on formal trend analysis) (Section 4.1.2.1). Both were lowest in November 2002, TECs were generally highest in August 2001, but PCB concentrations were generally highest in May 2003. May 2003 TECs were mostly intermediate between those in August 2001 and November 2002. The comparisons are confounded by season, fish lipid content, the analytical procedure for total PCB (4- and 5-peak Aroclor, and sum of congeners), and differences in annual rainfall patterns that affect the discharge of PCBs from Neal's Landfill. The data show fluctuations in PCB uptake by fish, but do not demonstrate a consistent declining trend as would be expected if PCB discharges from Neal's Landfill were diminishing through natural processes⁵³.

6.2 PCB versus TEC-TEQ

Potential uncertainties are associated with risk characterization based either on total PCBs (measured by Aroclors or sum of congeners) or TEC. Two issues involving Aroclors are that 1) the mix of congeners retained in animal tissue often differs from the commercial mixture characteristic of a particular Aroclor, and 2) the toxicity reference values (TRVs) may be based on different Aroclors from the ones released to the environment. The issues for PCBs are addressed by basing the PCB TRVs for mink dietary concentration and kingfisher dose on the more toxic Aroclors to account for the frequently reported observation that transfer of PCBs through foodchains increases its dioxin-like toxic potency (Jones, et al. 1993; Koslowski, et al. 1994; Ludwig, et al. 1996; Guruge and Tanabe 1997; Leonards, et al. 1997; Giesy and Kannan 1998; Trowbridge and Swackhamer 2002). An advantage of Aroclor toxicity studies is that all of the potential toxic effects of PCBs contribute to the observed effects, both ones acting through dioxin-like toxic mechanism and those acting through non-dioxin-like mechanisms.

TECs have different concerns: 1) since the procedure is based on dioxin-like effects, it may not account for toxic effects caused by a different process, and 2) there is uncertainty over the toxic equivalent factors (TEFs) used to convert the congener data to TECs, and the assumption of additivity in calculating TEC. An important advantage of the TEC approach is that it avoids the uncertainty over the specific congener composition associated with Aroclor analyses of fish tissue. Since TEC is measured in fish (actually the dioxin-like PCB congeners are measured, from which TEC is calculated), there is no uncertainty over how bioaccumulation may have affected dioxin-like toxicity. The influences of bioaccumulation and environmental processes are directly revealed by the levels of dioxin-like congeners in biota samples.

Instead of considering each issue separately, the aggregate influence of the various uncertainties is assessed by comparing the risk estimates based on TEC and PCB (Aroclor or sum of congeners). There is no consistent relationship between the HQs based on PCBs versus those based on TEC or TEQ (Section 5.2.1). Since there is no systematic bias associated with either approach (either may be more or less conservative than the other in any particular sampling event or endpoint), the range of risk estimates from each approach are evaluated in the FERA as supporting lines of evidence.

⁵³ Natural processes are often misleadingly referred to as "natural attenuation", but attenuation is but one possible outcome of natural processes. Other outcomes include no effects over reasonable time frames ("natural inertia"); or increased release, bioavailability, or toxicity over time ("natural potentiation").

The absence of large systematic differences between the TEC- and PCB-based risk estimates increases confidence in the overall results of the FERA.

An additional issue is that chemicals besides PCBs may also contribute to the TEC at a site. Technical PCB mixtures contain byproducts, such as polychlorinated dibenzofurans (PCDF) and polychlorinated naphthalenes (PCN), that contribute to the total TEC. This means that commercial Aroclors may have had greater TEC than expected from the dioxin-like PCB congener composition alone. PCDF accounts for more than twice as much of the total TEC as the dioxin-like PCB congeners do in Aroclor 1260, but PCDF and PCN contribute less than 10 % of the TEC of Aroclors with lower overall chlorine content (1248 or 1254) (Yamashita, et al. 2000). Other chemicals, such as dioxin, capable of affecting TEC may be present in the environment from sources other than PCB mixtures. Therefore, TEC may be underestimated if the contributions of dioxin and polychlorinated dibenzofuran congeners are unknown. This uncertainty partly holds for Aroclor-based risk assessments. Feeding studies performed with Aroclors include all of the byproducts found in the PCB mixture. The second uncertainty, potential contribution of non-PCB related chemicals to TEC, remains for Aroclor-based assessments.

Dioxins or polychlorinated dibenzofurans have not been analyzed in Conard's Branch or Richland Creek fish, which means that the TEC of the samples may be underestimated. Based on experience at other PCB sites, dioxin-like PCB congeners are expected to be the predominant contributors to the total dioxin-like toxicity, but, without site-specific data, the potential contribution of dioxins or polychlorinated dibenzofurans to the TEC is uncertain.

Viacom, Inc., questioned whether the calculated TECs are over-estimated by summing the contributions of all PCB dioxin-like congeners when non-additive interactions might occur between PCB congeners, specifically, antagonism by di-*ortho*-substituted PCB congeners⁵⁴ (Viacom 2004). An antagonistic interaction is when the integrated effect of two chemicals dosed simultaneously is less than the sum of the separate effects of each chemical dosed individually. Antagonism by di-*ortho*-substituted PCB congeners is species-specific, for example, in bioassays, mouse cell line is sensitive, guinea pig and human cell lines insensitive, and rat cell line intermediate in sensitivity to antagonism by di-*ortho*-substituted PCB congeners (Aarts, et al. 1995). The relevance of purported antagonism by di-*ortho*-substituted PCB congeners to mink or kingfisher is unknown. In addition to species differences, the effect also depends on the particular endpoint being assessed, for example, in bioassays, luciferase induction is a less sensitive endpoint than EROD induction to the antagonistic effect of di-*ortho*-substituted PCB congeners, even though luciferase is a more sensitive endpoint to dioxin exposure compared to EROD induction (Sanderson, et al. 1996).

Although antagonism is a potential uncertainty in calculating TEC or TEQ, this usually is not a significant complication in practice.

“... the value of the TEF approach for risk management is dependent on minimal non-additive interactions among the PCB, PCDD, and PCDF congeners. However, antagonism has been demonstrated only when relatively high doses of TCDD are employed... Also, it is important to note that synergism has also been observed for certain endpoints... Clearly, more research is needed on this question,

⁵⁴ Di-*ortho*-substituted means that the PCB congeners have Cl attached to two ortho positions in addition to whatever Cl is attached to meta or para positions (see Section 2.1.1).

but at present large deviations from additivity at environmentally relevant exposure levels have not been demonstrated.” (Compton and Sigal 1999).

“The studies of chemical pairs or complex mixtures of organochlorine chemicals found in the environment have largely shown additive interactions for embryo lethality. Some non-additive responses have been observed when endpoints other than mortality have been measured. However, ecological risk assessments to protect the integrity of fish and wildlife populations are most often conducted using organismal level responses... Clearly, additional studies are needed to more precisely understand the limitations of application of the TEF/TEQ approach. Yet, current documentation in fish and wildlife species indicates that the uncertainties associated with not using such an approach are greater than the uncertainties of using it.” (Tillitt 1999).

Leonards, et al. (1995) found a closer relationship between body burden and reproductive toxicity in a meta-analysis of mink PCB toxicity tests when the body burden was expressed as TEC as compared to total PCB. The body burden was calculated by modeling mink uptake of dioxin-like PCB congeners and calculating TEC with the usual assumption of additivity amongst congeners. The finding of an improved relationship between TEC body burden and toxicity compared to PCB body burden would be unlikely if the TEFs and the additivity assumption used to calculate the TEC body burdens of the various studies were introducing significant errors.

6.3 Toxicity Reference Values (TRVs)

Some of the sources of uncertainty in TRVs include: 1) extrapolation of toxicity between species, 2) extrapolation of the results of laboratory studies to field situations, 3) differences between pure or commercial chemical products used in testing and the chemical mixtures in the environment, and, conversely, 4) the potential for additional toxic effects caused by contaminants other than the chemical under investigation in studies using field-contaminated prey.

6.3.1 Mink TRVs

There is no issue over interspecific extrapolation for mink since the toxicological studies were performed with mink, however, the mink TRVs are based on captive mink feeding studies. There are some indications that the effects reported in laboratory studies are similar to those in the field. For example, changes in otter populations in Sweden between the 1960s and 1990s have been correlated with PCB levels in otter muscle samples, but not with other contaminants. The empirical threshold for improved otter population status is reported to be about 10 ppm PCBs in muscle tissue on a lipid-normalized basis (northern Sweden), with levels around 30 ppm associated with poorer recovery (southern Sweden—the proportion of juveniles is much lower compared to the northern otters) (Roos, et al. 2001). This compares reasonably well with the estimated lipid-normalized PCB concentration of 40-60 ppm in mink for causing a 50 % reduction in litter size, based on an extensive review of mink feeding studies (Leonards, et al. 1995), which indicates that TRVs based on the reproductive effects of PCBs in captive mink are not over-protective for wild populations of a related piscivorous mustelid⁵⁵.

⁵⁵ Mink and otter are in the weasel family (Mustelidae).

None of the studies selected for the mink PCB TRVs used field-contaminated prey for their exposed treatment groups (Section 4.2.2.1), so the observed toxicities were not confounded with the effects of other contaminants.

Adjustment of the Aroclor-based TRVs for an expected increase in toxic effects with continuous exposure through 2 breeding seasons or generations is a potential source of uncertainty. Two mink feeding studies show such an effect, although performed with Clophen (European PCB product) and field-contaminated prey instead of an Aroclor product. Two studies performed with two different species of mice show the same effects, as does a multi-generation rat study performed with dioxin (Section 4.2.2.2). In addition to empirical support, there is theoretical support that the toxic effects of chemical exposures vary both with exposure concentration and exposure duration. One expression of the relationship is known as Haber's Law or Rule, and has been recommended for routine analysis of toxicity data (Rozman and Doull 2001). Other researchers caution that Haber's Rule, or other equations relating dose and time to toxic effects, cannot be routinely used because the form of the interaction varies in different applications, but agree that "dose, response intensity, and time are all important and interrelated parameters that have to be considered in the design of toxicological experiments" (Bunce and Remillard 2003). In other words, Bunce and Remillard consider the interaction of dose and time to be important, but caution that the specific relationship for one class of chemicals may not apply to a different class of chemicals. This caution also applies to different receptors, that is, species-specific processes may change the form of the dose-time interaction. In the absence of a universal equation that can be applied with confidence to the mink PCB toxicity data, the empirical approach in the FERA is to use the mean difference in toxicity observed in the two long-term mink studies performed with PCB-contaminated food to adjust the results of the Aroclor studies performed over only a single breeding season.

The alternative is to disregard the empirical evidence for increased PCB toxicity in mink with exposure duration, which then entails the uncertainty that the TRVs are not adequately protective for sustainable use of the site by mink. This is contrary to the requirement in the NCP to select remedies that "maintain protection over time" (Section 1.2). The selected mink TRVs are intermediate between the TRVs chosen in recent ERAs (Section 4.2.2.1), and so are neither markedly more or less conservative compared to previous choices. The Housatonic River mink feeding study resulted in a higher LOAEC compared to the FERA low effect TRV, but the FERA value is within 2-fold of the LC_{20} for the Housatonic feeding study, consistent with the difference in toxicity between PCB exposure over one breeding season versus over two breeding seasons or generations (Section 4.2.2.1).

The mink TEC TRVs are derived from the two long-term mink feeding studies (Section 4.2.2.2), and therefore there is no uncertainty regarding extrapolation of single breeding season results to longer exposure durations. One uncertainty associated with TEC TRVs, differences in TEF values put forward at different times or by different research groups, is minimized in the FERA by recalculating the published TECs or TEQs with a common set of TEFs, the WHO-TEFs developed through a consensus process (Van den Berg, et al. 1998).

6.3.2 Kingfisher TRVs

No PCB studies with kingfisher were located, so the kingfisher TRVs are based on studies of other bird species. The uncertainty of extrapolating toxicity data between species is addressed by selecting two sets of TRVs for kingfisher PCB dose—one set based on chicken studies, the most sensitive of the few bird species tested so far with PCBs, and a second set based on behavioral effects in doves, which are less sensitive to PCBs (Section 4.2.2.3). Both sets of dose TRVs are based on controlled feeding studies performed with Aroclor product, so a

confounding effect by other chemicals is not a concern. The set of dose TRVs derived from chicken studies are based on the most toxic Aroclor to chicken (Aroclor 1248), to account for the frequently reported increase in the dioxin-like toxicity of PCBs with biomagnification (Section 6.2). The dose TRVs derived from dove studies are based on Aroclor 1254, which is one of the more toxic Aroclors. The two sets of TRVs are representative of the range of TRVs used in recent ERAs (Appendix E3), with the exception of the exceptionally high values in the Hudson River ERA, which are based on an inappropriate endpoint (Section 4.2.2.3).

The kingfisher TEQ dose TRVs are based on a study of pheasant injected with dioxin (TCDD), with the dose adjusted to account for the kinetics of dioxin accumulation in pheasant (Section 4.2.2.4). The TRVs might be insufficiently protective for kingfisher because “pheasant ... is one of the more tolerant species” to dioxin-like effects (Giesy, et al. 1995)⁵⁶. However, the other study considered for TRVs, chicken fed field-contaminated prey (Summer, et al. 1996a,b), results in nearly identical TRVs when the TEQs are recalculated with the current WHO-TEFs (Appendix E4). Confounding effects by other contaminants is not an issue in the pheasant study selected for the TRV.

Similarly, the uncertainties associated with the kingfisher egg TEC TRVs are addressed by selecting 2 sets of TRVs to represent higher and lower sensitivity to dioxin-like effects (4.2.2.5). The lower (more sensitive) set of TRVs represents the results of field studies of bald eagle, double-crested cormorant, and caspian tern; and a chicken egg injection study with dioxin. The higher (less sensitive) set is based on a cormorant egg injection study with dioxin, and is also representative for the results of kestrels and bald eagle studies. The range of HQs from the two sets of TRVs represents the range of uncertainty in estimating kingfisher risk to TEC.

The protectiveness of the selected range of egg TRVs can be assessed through the results of an independent review of egg toxicity data. USEPA (2003) derived egg TEC TRVs through a different procedure—species sensitivity distribution. This procedure utilizes the data from all tested species, and provides estimates of the proportion of species at risk for particular TRVs. Based on laboratory studies, the hazard concentration for 5 % of species (HC_5)⁵⁷ is 0.1 to 0.2 $\mu\text{g TEQ/kg}$ whole egg for embryo mortality (NOAEC to LOAEC, respectively), and 0.06 to 0.15 $\mu\text{g TEQ/kg}$ whole egg for developmental effects. The HC_5 values are somewhat lower (more conservative) than the lowest set of egg TRVs in the FERA—1.8 to 5.3 $\mu\text{g TEQ/kg lipid}$, which convert to 0.1 to 0.4 $\mu\text{g TEQ/kg}$ whole egg (NOAEC to LOAEC, respectively) (Appendix E6). The higher set of egg TRVs in the FERA—17 to 68 $\mu\text{g TEQ/kg lipid}$, which convert to 1 to 4 $\mu\text{g TEQ/kg}$ whole egg (NOAEC to LOAEC, respectively) (Appendix E6)—are an order of magnitude higher (less conservative) than the HC_5 .

Viewed another way, the two kingfisher egg LOAECs in the FERA (0.4 and 4 $\mu\text{g TEQ/kg}$ whole egg) correspond to the HC_{10} and HC_{50} for embryo mortality, respectively, which means the lower value is protective for 90 % of tested bird species, and the higher value for 50 % of species (USEPA 2003 Tables 3-4 and 3-5). This analysis supports the choice of egg TRVs in the FERA, and demonstrates that the range is not overly

⁵⁶ A similar statement is made in Bowerman, et al. (1995).

⁵⁷ The HC_5 is protective for 95 % of tested species, but may result in adverse effects in 5 % of tested species, in other words, it is protective for all but the most sensitive species.

conservative. As intended, the range of egg TRVs represents a range of species susceptibility to PCBs from fairly high sensitivity (but not extreme sensitivity) (HC_{10}) to middling sensitivity (HC_{50}).

Multiple HQs are calculated for kingfisher at each stream reach in any particular year: 5 estimates each for NOAEC-NOAEL or LOAEC-LOAEL (1 TEQ dose, 2 PCB dose, and 2 egg TEC). In most cases, the range of dose HQs overlaps with the range of egg HQs at a particular location and time, when the ranges do not overlap, the gap between the ranges is relatively small. The region of overlap between the dose and egg HQs, or the gap between non-overlapping HQs, are selected as the “consensus HQs” (Section 5.2.4). The uncertainty is less for the consensus HQs than for the entire range of kingfisher HQs because the consensus HQs represent the range in which the results of all approaches converge.

Viacom, Inc. recommended assessing kingfisher risk on the basis of a field study of kingfisher along the Housatonic River, another PCB site (Viacom 2004). This recommendation was not implemented because the Housatonic field study is limited by several shortcomings in design, including an insufficiently broad exposure gradient, lack of a control or reference population, and a method of evaluation that is subject to confounding because the results of the field study are compared to that of a single study from the literature for a different location. Sanderson and Van den Berg (1999) discuss the first two issues:

“... in order to be able to study potential dose-dependent, contaminant-related adverse effects in a wild population, which is subject to a large degree of biological variability, it is essential to choose a wide range of levels of contaminant exposure. If this is not done, the problem arises that, in effect, one is looking for potential adverse effects within what is virtually a single exposure group, in which the variation in contamination is smaller than the variation in all other influences combined. A variation of this problem is the common lack of a true control group in environmental monitoring studies.”

According to the Housatonic River ERA (USACE/USEPA 2004 §H.4.5.2):

“The belted kingfisher field study methods generally followed accepted protocols. There were, however, several shortcomings that resulted in uncertainties regarding the conclusions of the study. No information was provided regarding nest search intensity, important endpoints like clutch size and hatching success were not measured, and there were few visits to the nests during the reproductive cycle. These shortcomings limit the ability to draw rigorous conclusions from the field study results.

The lack of reference area and small sample size (i.e., $n=6$) with the statistics used is considered a major source of uncertainty in the kingfisher field study. The statistics used, including assumptions and limitations, were not clearly described in the report.

The approach used to estimate dose in the belted kingfisher field study had a number of shortcomings. ... The lack of reference area concentrations further compounds uncertainties in the dose gradient. Thus, there are major uncertainties in the dose gradient achieved by this approach, as it is likely too narrow to detect a significant dose-response relationship and the dose associated with a given nest is unknown.

The belted kingfisher field study results do not definitively support the conclusions of low risk because the data collected in the study 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 available in the literature (Brooks and Davis 1987) and no site-specific reference area. It introduces significant uncertainties 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 tPCBs, it fails to acknowledge the limitations associated with the use of only one metric (fledging success) to evaluate the assessment endpoint, survival, growth, and reproduction. The potential magnitude and direction of the uncertainty associated with lack of additional metrics is unknown, but considered to be of moderate importance.”

For these reasons, the Housatonic kingfisher field study is not considered an adequate study for reducing uncertainty over the relative sensitivity of kingfisher to PCBs. Another complication is Aroclor 1260 is a significant portion of the source PCBs at the Housatonic site. Aroclor 1260 differs from less chlorinated Aroclors in that most of the TEQ is due to polychlorinated dibenzofuran (PCDF) byproduct, not PCB congeners (Yamashita, et al. 2000). Also, diet-to-egg biomagnification factors (BMFs) for birds are lower overall for PCDFs compared to PCB coplanar congeners (Hoffman, et al. 1996; Henny, et al. 2003; Murata, et al. 2003). The predominant role of PCDFs in causing toxicity at the Housatonic River site is supported by a field study of tree swallows:

“The hypothesis that the dioxin and furan congeners are contributing to reduced hatching success *more so* than the PCB congeners is further supported by the two-variable model results. In these models, TEQs associated with PCBs become nonsignificant in both analyses, while the WHO TEQs for dioxin/furans remained as a significant variable” (Custer, et al. 2003) [emphasis added].

The approach used in the FERA of evaluating TRV ranges corresponding to ranges of sensitivities in birds is retained because it captures the uncertainty regarding kingfisher sensitivity and explicitly carries it through the risk assessment process.

In a related comment, Viacom, Inc., questioned whether the toxicity of PCBs is exaggerated in laboratory studies compared to actual effects in the field, which would mean that laboratory-based TRVs may be overly conservative (Viacom 2004). Contrary to this assertion, USEPA (2003), in a comparison of avian species sensitivity distributions for reproductive effects in birds exposed to dioxin-like compounds, found that field studies result in a *lower* HC₅ (egg TEC protective for 95 % of species) compared to the HC₅ based on laboratory studies. This was true even when laboratory studies with chicken (the most sensitive bird species tested to date) were included in the comparison. The comparison by USEPA (2003) indicates that use of laboratory-based PCB TRVs may be under-protective for effects in the field. This uncertainty (possible under-protectiveness) may apply to the kingfisher dose TRVs and the higher set of egg TRVs, which are based on laboratory studies, but not to the lower set of kingfisher egg TRVs, which are based primarily on field studies.

6.4 Exposure Assumptions

6.4.1. Mink Dietary Composition and Foraging Assumptions

The dietary composition for mink is uncertain because mink are opportunistic predators and will shift their diets in response to the availability of prey. Mink “feed on any animal material that they can find and kill”, and the “responsiveness ... to available prey results in a diet that is extremely variable by season and location” (Chapman and Feldhammer 1982). This means that mink foraging along Richland Creek may consume higher or lower percentages of fish and crayfish in their diets than assumed for the FERA. The proportion of aquatic prey is of particular importance in the FERA because PCB exposure is only modeled through consumption of fish (also representing amphibians) and crayfish (also representing other aquatic invertebrates), and all other prey (small mammals and birds) are assumed to have no PCB contamination. The mink dietary composition of 66 % fish, 13 % crayfish, and 21 % birds and mammals chosen for the FERA has a robust foundation—gut content analysis of 31 mink taken from Michigan streams (Section 2.6). The proportion of aquatic prey was significantly higher (92 %) in mink taken from Michigan rivers for the same study, but the stream mink data were selected for the FERA because they are based on habitat similar to that of Richland Creek.

Although mink may consume less aquatic prey than modeled in the FERA, selection of a very low dietary proportion of aquatic prey would be inappropriate for modeling PCB exposure to mink because there is no guarantee that mink will feed in this manner in Richland Creek. There would be low confidence in a finding of no risk to mink based on a risk assessment that assumed a low proportion of aquatic prey in the mink diet because the no risk conclusion would only hold if mink did not deviate from the assumed predominantly terrestrial diet. If the assumption were incorrect, the risk conclusions would be meaningless. The mink dietary proportions chosen for the FERA are middle values, not the highest in the literature (which were measured for mink in habitats much different from Richland Creek), or so low that a no risk conclusion would only hold true so long as mink strictly complied with the assumed dietary selection.

The 13 % contribution of crayfish to the mink diet also affects the risk findings because crayfish accumulate less PCBs compared to fish. Again, the value chosen for the FERA is not the most conservative value available for studies of mink in the Midwest. Crayfish contributed only 4 % of the diets of mink in Michigan rivers (Section 2.6), which is less than one-third of the value chosen for the FERA, but the higher value for mink in Michigan streams is selected because it is based on habitat similar to that of Richland Creek.

The remaining 21 % of the mink diet for which PCB uptake is not modeled (mainly bird and mammal prey with a small unidentified component) (Section 2.6), is more problematic because it is non-conservative, that is, PCB exposure to mink is underestimated because PCB uptake is not modeled for one-fifth of the mink diet. Presumably terrestrial prey at this site would have less PCB exposure than aquatic prey because aquatic animals are more continuously exposed to PCBs discharge from Neal’s Landfill, while the primary exposure to terrestrial animals would occur through PCB deposition in the floodplain during flooding and the subsequent accumulation through terrestrial food chains. If this assumption is valid, then the underestimation of mink exposure to PCBs would be less than 20 % of the modeled uptake. However, in the absence of site-specific terrestrial data, an uncertainty remains over the extent to which PCB exposure to mink is underestimated.

Limited field evidence in support of the mink exposure estimates is that the measured liver PCB concentration and TEC in a road-killed mink are consistent with modeled uptake of total PCBs and dioxin-like congeners from the nearest Richland Creek reach calculated with the mink dietary composition used in the FERA (Section 5.2.9.1).

A potential source of uncertainty is the selection of a 1-mile home range for female mink along streams (Section 2.6). Since the original study reported a range from 0.6 to 1.7 stream miles (Gerell 1970), in theory the risk estimates could vary with different home range assumptions. In practice, the home range selection has little effect on the outcome because the Richland Creek sampling locations are further apart than the longest reported female mink stream home range. This means that the AUF for mink in each of the Richland Creek reaches remains the same (100 %) for the entire range of reported female mink home ranges in streams.

Conard's Branch is assumed to be incapable of solely supporting either mink or kingfisher, so the allocation of mink foraging between Conard's Branch (25 %) and Richland Creek (75 %) in the Reaches 1 & 2 mixed foraging model is another source of uncertainty. PAC analysis is performed to address this uncertainty (Sections 5.1.2). If mink otherwise forage in areas that receive no direct discharge of PCBs from Neal's Landfill, their dietary exposures would meet the no observed adverse effect concentrations by foraging along Conard's Branch only 2 to 5 % of the time, and would equal the lowest observed adverse effect concentrations by foraging only 7 to 23 % of the time along Conard's Branch (assuming 21 % of the diet has zero PCBs in either location) (Section 5.2.8.3). In terms of fish and crayfish, the overall mink dietary exposure would equal the LOAEC if Conard's Branch fish and crayfish contribute only 6 to 18 % of the total diet, when the remaining 82 to 94 % of the diet consists of prey not directly exposed to PCBs released from Neal's Landfill. This means that mink may be at risk of adverse reproductive effects by foraging in Conard's Branch *less* than assumed in the Reaches 1 & 2 scenario.

The PAC analysis indicates that uncertainty over the proportion of prey mink may take from Conard's Branch does not significantly affect the conclusions of the FERA. Conard's Branch fish are so highly contaminated that only a small contribution to an otherwise uncontaminated diet will raise the total dietary concentration to levels of concern. Of course, the more realistic model is mink foraging in Richland Creek (Reach 2) with some contribution from Conard's Branch. The conclusions of the PAC analysis for Conard's Branch hypothetically assuming the remainder of the prey are unexposed apply to the more realistic model as well, that is, uncertainty over the likely proportions of Conard's Branch prey and Richland Creek prey does not affect whether or not mink are at risk of reproductive impairment.

6.4.2 Kingfisher Dietary Composition and Foraging Assumptions

Although belted kingfisher "occasionally takes prey away from water" (Kaufman 1996), the terrestrial component of the diet is so small that it can be neglected with no uncertainty. Only 3 of 7 studies of kingfisher diets summarized in USEPA (1993) reported non-aquatic prey, and the proportion in the diet is uniformly low—only 1 to 2 % of the diet. This means that there is no significant un-modeled component of the kingfisher diet lacking PCB data.

The main uncertainty regarding the kingfisher dietary composition is the relative proportion of fish and crayfish. The kingfisher dietary composition used in the FERA, 80 % fish (including amphibians) and 20 % crayfish (including other aquatic invertebrates), is based on the mean values from 3 studies of kingfisher in the midwest (Section 2.6). Two of the studies reported lower crayfish-invertebrate contributions, 5 % by weight for Michigan streams (Alexander 1977) and 13 % by number of prey for Ohio creeks (Davis 1982), and one reported a higher contribution, 41 % by volume in Michigan streams (Sayler and Langler 1946). The different bases for reporting dietary composition introduces uncertainty in calculating a mean value from the 3 studies. Since kingfisher dose is

calculated from PCB concentration or TEC per unit weight of prey, only the weight-based dietary composition is directly applicable to the exposure modeling in the FERA. The prey number-based dietary composition is treated as a weight-based composition by assuming that the fish and crayfish prey are of approximately equal sizes, and the volumetric-based dietary composition by assuming that the specific gravities are approximately equal. Both assumptions introduce uncertainty. The 41 % crayfish and insect volumetric value reported by Sayler and Langer (1946) appears to be an outlier. No other study of kingfisher dietary composition summarized in USEPA (1993) reports values as high, and many report no insect or crayfish prey (see volume II). This indicates that kingfisher may be more likely to consume less than 20 % crayfish of the total diet than to consume significantly more than 20 %, which in turn indicates that kingfisher risk is more likely to be somewhat underestimated in the FERA due to uncertainty over the dietary composition, and less likely to be overestimated due to this uncertainty.

A potential source of uncertainty is the selection of a 1-mile home range for kingfisher along streams (Section 2.6). Since the original study reported a range from 0.6 to 1.4 stream miles (Brooks and Davis 1987), in theory the risk estimates could vary with different home range assumptions. In practice, the home range selection has little effect on the outcome because the Richland Creek sampling locations are further apart than the longest reported kingfisher stream home range. This means that the AUF for kingfisher in each of the Richland Creek reaches remains the same (100 %) for the entire range of reported kingfisher home ranges in streams.

The very short kingfisher home range of 0.24 stream miles reported for OH streams in late summer and fall (Davis 1980), raises an uncertainty that kingfisher may forage over an area extending only one-fourth the length assumed in the FERA. If a seasonally shortened home range were located near the confluence of Conard's Branch and Richland Creek, the kingfisher PCB exposure and risk would probably exceed the estimates made in the FERA for Reach 2. However, this would not affect kingfisher risk in the further downstream reaches of Richland Creek.

Another uncertainty concerns the allocation of kingfisher foraging between Conard's Branch (50 %) and Richland Creek (50 %) in the Reaches 1 & 2 mixed foraging model. PAC analysis is performed to address this uncertainty (Section 5.1.2). If kingfisher otherwise forage in areas that receive no direct discharge of PCBs from Neal's Landfill, their doses would meet the no observed adverse effect levels by foraging along Conard's Branch only 0.5 to 15 % of the time, and would equal the lowest observed adverse effect levels by foraging 2 to 60 % of the time along Conard's Branch (Section 5.2.8.3). This means that kingfisher may be at risk of adverse reproductive effects by foraging in Conard's Branch as little as four-hundredth of the time assumed for the Reaches 1 & 2 scenario (for the most conservative TRV) to as much as 1.2 times more than assumed (for the least conservative TRV), when the remaining 40 to 98 % of the diet consists of prey not directly exposed to PCBs released from Neal's Landfill.

The PAC analysis indicates that uncertainty over the proportion of prey kingfisher may take from Conard's Branch may affect the risk conclusions based on the least conservative of the kingfisher TRVs, but is unlikely to significantly affect conclusions based the remaining (more conservative) kingfisher TRVs. In the FERA, risk to kingfisher is assessed by the consensus HQs, which discards the highest and lowest risk estimates and retains only the range of HQs in common between the kingfisher dose and egg modeling approaches (Section 5.2.4). The lowest risk estimates are the ones generated with the least conservative TRVs. Since these are dropped from the consensus HQs, the uncertainty over the proportion of time kingfisher forage in Conard's Branch is unlikely to significantly affect the conclusions of the FERA.

6.5 Additional Risk Information

6.5.1 Road-killed Mink

The main uncertainties associated with the road-killed mink are that the history of the mink is unknown, specifically where it foraged prior to its death, a range of mink/otter liver TRVs are available from published studies, and the road-killed mink is a single sample.

Uncertainty over foraging history is partly addressed by the agreement between the road-killed mink liver contents and modeled uptake in mink foraging over Richland Creek Reach 3 with the exposure assumptions used in the FERA (Section 5.2.9.1). This does not prove that the road-killed mink fed along the Richland Creek near where it was killed, but it shows that this possibility cannot be excluded. Starting with the assumption that the road-killed mink was locally exposed, unless data show otherwise, is commonsense, but potential movement from other foraging areas also cannot be excluded. However, if the road-killed mink was a recent immigrant from another area, it had to have been exposed to PCBs to a similar level as expected for a mink foraging in Richland Creek Reaches 3 or 4, which would indicate that mink might be receiving integrated exposures from multiple stream systems with elevated PCBs. If this is a common occurrence in the Bloomington area, the mink risk estimates in the FERA are likely to be underestimated.

Another uncertainty is that a range of mink liver TRVs are reported in the literature (Appendix G). Much of the range occurs because different endpoints are being considered. Part of the range is due to a limitation of controlled studies—effects can only be reported at the particular exposure levels tested. Researchers testing different exposure treatments will necessarily report different effect levels. Part of the difference between studies is also due to differences in PCB toxicity related to differences in source PCBs, byproducts, or co-contaminants, differences in sensitivity of different strains of mink, or differences in experimental or field conditions. Selective use of particular studies can be used to either support no effect findings or support adverse effect findings. Overall, the road-killed mink liver TEC falls within the gray area of liver TRVs between no effects and the onset of adverse effects.

An important limitation of the road-killed mink data is there is only a single sample. This is analogous to a medical case study in which a single patient is discussed, which can be useful in providing insights, but, by itself, is not conclusive. Accordingly, the road-killed mink findings are not considered definitive proof that mink foraging along Richland Creek are at risk of adverse effects from PCBs, rather they indicate that mink could be receiving exposures at or close to adverse levels by foraging along Richland Creek near Highway 48. Besides this observation, the road-killed mink is used to evaluate the approaches in the FERA for assessing risk to mink in two ways: 1) as a field check of the exposure modeling assumptions for mink, and 2) as a field check for the risk estimates for mink. The road-killed mink liver data are consistent with the exposure models and risk estimates used in the FERA. This supports the approaches used in the FERA for assessing risk to mink (or, at the least, does not contradict the FERA approaches), and contributes to the weight-of-evidence that PCB exposures in the upper reaches of Richland Creek may result in excess risk to mink. Put another way, the road-killed mink does not, of itself, prove that mink may be harmed by PCB releases from Neal's Landfill, but it raises confidence that the procedures for assessing exposure and risk in the FERA are reasonable.

6.5.2 Risk to Fish

The main uncertainty associated with assessing risk to fish based on the effects of PCBs on rainbow trout is the relative susceptibility to dioxin-like effects of the species sampled in Conard's Branch and Richland Creek as

compared to rainbow trout. Salmonids are known to be highly sensitive to dioxin-like effects, and therefore risk estimates to non-salmonid species might be overestimated when based on toxicity data for a salmonid species, such as rainbow trout, the source of the toxicity reference value (TRV) used in the FERA. Data are not available for comparing species sensitivities to long-term chronic exposures to adult fish resulting in sub-lethal effects (growth or behavior), but comparisons are available for certain species based on acute (short-term) exposures to eggs or to juvenile/young adult fish resulting in lethal effects (LC_{50}).

Adult white sucker could be about 15-fold less sensitive to PCB dioxin-like effects as adult rainbow trout if the relative sensitivities of their eggs are valid predictors of the relative sensitivities of the adults of the two species, and if relative rankings for acute lethal effects are valid predictors of the relative rankings for chronic growth and behavioral effects. Relative egg sensitivity could predict relative adult sensitivity if the differences between species are due to differences in intracellular processes that are conserved through growth and development from eggs to adults, for example, species differences in AhR affinity/responsiveness to dioxin-like molecules. The relative species rankings based on susceptibility of eggs will not hold for adults if adult susceptibilities are significantly affected by species differences in pharmacokinetics not apparent in eggs; for example, species differences in the tissue/organ distribution, metabolism, sequestration, or excretion of dioxin-like molecules; or if developmental changes affect susceptibility to dioxin-like effects. In these cases, the relative susceptibilities of adult white sucker and rainbow trout could not be directly predicted from the relative egg susceptibilities. Of the relatively few comparisons of species sensitivities to dioxin, the decrease in the relative sensitivity of salmonids from the most sensitive species based on effects in eggs (Elonen, et al. 1998) to intermediate sensitivity based on effects in juvenile/young adult stages (Kleeman, et al. 1988), indicates that species relative sensitivities of juvenile/young adult fish may be poorly predicted by the relative sensitivities of eggs. Accordingly, the prediction that adult white sucker would be about 15-fold less sensitive than adult rainbow trout based on the relative sensitivities of the eggs, is highly uncertain.

Adult sunfish could be about 1.6-fold less sensitive to PCB dioxin-like effects as adult rainbow trout if the relative sensitivities of their juvenile/young adult stages are valid predictors of the relative sensitivities of the adults of the two species, and if relative rankings for acute lethal effects are valid predictors of the relative rankings for chronic growth and behavioral effects. The applicability of relative sensitivities to acute lethal effects for projecting relative sensitivities to chronic sub-lethal effects remains uncertain, but there is more confidence in projecting adult relative sensitivities from studies of juveniles or young adults than there is from studies of eggs because juveniles and adults share the same body plan and major organs. This means there is greater confidence in the conclusion that sunfish are still at risk of adverse effects in the upper reaches of Richland Creek even after accounting for differences in sensitivity compared to rainbow trout, than in the egg-based comparison for white sucker.

Another potential uncertainty is that chemicals in addition to administered TCDD may have contributed to the total TEC in the diet of the rainbow trout study used for the fish TRV, which would mean the reported TRV is underestimated. Giesy, et al. (2002) calculated a total TEC in the control diet of 2.3 ng/kg ww, and, since the lowest dietary treatment resulting in reduced adult survival was 1.8 ng added TCDD/kg diet, estimated that the “actual, absolute value is at least 2.3-fold greater than the concentration of TCDD in the diet”. However, the control TEC is based on non-detection of TCDD (reported as < 0.2 ng/kg) or PCB (reported as < 0.2 mg/kg) in the control diet, and calculated by assuming 0.1 ng/kg TCDD and 0.1 mg/kg Aroclor 1254 at 10.83 ng TEC/mg Aroclor 1254 product (Giesy, et al. 2002). This is correctly noted as “an upper estimate of the concentration of TEQ in the control diet” (Giesy, et al. 2002), which means that the 2.3-fold value is a maximum, not minimum,

estimate of the possible contribution of the base feed to the TEC. The actual dietary TEC is therefore *less* than 2-fold greater than the TCDD concentration reported in Giesy, et al. (2002). The uncertainty related to undetected co-contaminants in the feeding study used for the TRV appears to be less important than the uncertainty related to differences in species sensitivities.

6.6 Whole-fish PCB and TEC Goals

The preliminary fish goals have the same uncertainties as the FERA, discussed in the previous sections. The range of preliminary goals is wider for kingfisher than for mink reflecting the greater uncertainty over kingfisher sensitivity to PCBs. PCB toxicity is better understood in mink because mink have been used in PCB toxicity studies. The preliminary fish goals for mink are consistent with (bracketed by) the goals for kingfisher.

7 Conclusions

All of the receptors evaluated in the risk assessment (mink, kingfisher, and fish) show a similar pattern of risk—elevated risk to at least 2 miles downstream from Neal’s Landfill, low risk beyond 13 miles downstream, and possible, but sparsely documented, risk 3 to 6 miles downstream.

PCB releases from Neal’s Landfill have been reduced by the interim and remedial actions taken to date at the site, but, between 2001 and 2003, fish in Conard’s Branch and the upper portion of Richland Creek accumulated sufficient PCBs to raise dietary exposures to levels as high or greater than those shown to cause reproductive impairment in mink, or projected to cause reproductive impairment in kingfisher. Risk estimates are generally similar for total PCBs as for dioxin toxic equivalent concentration (TEC) or dose (TEQ).

Although piscivores (fish-eating animals) are not expected to feed exclusively in Conard’s Branch (Reach 1), PCB burdens are so elevated in Conard’s Branch fish that relatively small proportions in an otherwise uncontaminated diet will raise exposures to levels of concern. Consumption of fish and crayfish from Conard’s Branch could result in reproductive impairment in mink even if they contribute as little as 10 to 20 % of the total diet, under the unrealistic assumption that the remaining 80 to 90 % of the prey have no direct exposure to PCBs released from Neal’s Landfill. Consumption of fish and crayfish from Conard’s Branch could result in reproductive impairment in kingfisher if they contribute as little as 2 to as much as 60 % of the total diet, under the unrealistic assumption that the remaining 40 to 98 % of the prey have no direct exposure to PCBs released from Neal’s Landfill.

The hazard quotients (HQ) for mink in Richland Creek Reach 2, the first downstream reach from the confluence with Conard’s Branch, range from 2 to 9 for no observed adverse effect concentration (NOAEC) and from 1 to 2 for lowest observed adverse effect concentration (LOAEC) for 95-percent upper confidence limit of the mean (UCL) dietary exposures between 2001 and 2003. Piscivores feeding in Richland Creek Reach 2 are expected to partially feed in the lower portion of Conard’s Branch as well. The mink UCL HQs for combined foraging in Conard’s Branch and Richland Creek (Reaches 1 & 2) range from 3 to 20 (NOAEC) and from 2 to 6 (LOAEC).

Risk to kingfisher is modeled with two approaches: total PCB or TEQ dose to adult birds, and TEC in eggs, both of which are evaluated with two sets of toxicity reference values (TRVs) to account for uncertainty over the sensitivity of kingfisher to PCBs. The overlap or gap between the HQ range for dose modeling and the HQ range for egg modeling are referred to as the kingfisher “consensus HQs”. Uncertainty is minimized for the consensus

HQs because they represent agreement in risk calculations through separate modeling approaches with independent sets of TRVs.

The UCL consensus HQs for kingfisher in Richland Creek Reach 2, the first reach downstream from the confluence with Conard's Branch, range from 4 to 20 for no observed adverse effect level (dose NOAEL) or concentration (egg NOAEC), and from 1 to 3 for lowest observed adverse effect level (dose LOAEL) or concentration (egg LOAEC) between 2001 and 2003. The kingfisher UCL HQs for combined feeding in Conard's Branch and Richland Creek (Reaches 1 & 2) range from 9 to 60 (NOAEL-NOAEC) and from 3 to 10 (LOAEL-LOAEC).

The mink and kingfisher results are supported by estimates of direct risk to fish. Adjusted for differences in PCB sensitivity between sunfish and rainbow trout (source of the fish tissue TRV), the UCL LOAEC-HQs for sunfish are 20 to 50 in Conard's Branch, 3 to 20 in Richland Creek Reach 2, and 0.4 to 0.6 in Reach 4. Creek chub also appear to be at risk with unadjusted UCL LOAEC-HQs of 20 to 100 in Conard's Branch, 6 to 10 in Reach 2, and 4 in Reach 3 (the sensitivity of creek chubs to PCBs relative to rainbow trout is not known, but would have to be more than an order-of-magnitude less sensitive to not be at risk in Reach 2, and as much as 2 orders-of-magnitude less sensitive not to be at risk in Conard's Branch).

The results of the focused ecological risk assessment (FERA) show elevated risk to mammals or birds feeding on fish and crayfish through at least 2 miles downstream of Neal's Landfill, and indicate low or no risk beyond 13 miles downstream. The justification for further remedial action might be questioned since the PCBs discharged from Neal's Landfill are progressively diluted as they move downstream in Richland Creek. The problem with this reasoning is that PCBs are persistent (degrade slowly under special conditions) and highly bioaccumulative so they do not simply disappear from the environment. For example, Giesy, et al. (1994a) wrote:

“...because the rate of loss of these compounds through burial and degradation is slow and environmental sorting mechanisms tend to keep the more toxic Ah-active p-PCH [dioxin-like polychlorinated hydrocarbons] in circulation, these toxicants will be active in the ecosystem for many years. For this reason, and because, once released, they cannot be effectively removed from the environment, every effort must be made to minimize any further releases of these compounds. This means immediate cleanup of ... landfills and spill areas which continue to be a source of these chemicals and adoption of sufficiently protective WQC [water quality criteria] to minimize any further releases. Only in this way will the exposure to wildlife and humans be reduced effectively. Simply because the manufacture of PCBs has been stopped does not mean that nothing more can be done to protect wildlife from the effects of p-PCH.”

The same point was more emphatically made by Ludwig, et al. (1993):

“Because they [dioxin-like PCB congeners] cannot be removed effectively from the Great Lakes once released, extraordinary efforts should be made to minimize *any* further point source releases of these compounds and any other PCHs with similar modes of action to the Great Lakes.” [emphasis in the original].

The risks to mink may be underestimated because PCB accumulation is not modeled for one-fifth of the diet due to data gaps (site-specific data were not collected for terrestrial prey). Risk to mink or kingfisher also may be underestimated because dioxin and chlorinated dibenzofurans, which contribute to TEC or TEQ, have not been analyzed in Conard's Branch or Richland Creek fish or crayfish.

Other sources of uncertainty do not appear to significantly affect the outcome of the risk assessment.

For mink in Richland Creek, preliminary fish protective goals are 0.7 to 0.9 mg/kg PCBs and 7 to 26 pg/g TEC (mammalian) for no effect to low effect. The goals pertain to the total fish component of the diet, therefore, represent the mean concentrations in the available fish in a reach. Assuming partial use of Conard's Branch and primary use of Richland Creek by mink, preliminary fish protective goals in Conard's Branch fish are 2 mg/kg PCBs (no effect or low effect) and 17 to 68 pg/g TEC (mammalian) for no effect to low effect.

The protective fish goals for kingfisher are similar to those for mink, but with a wider range due to uncertainty over the relative sensitivity of kingfisher to PCBs. For Richland Creek, preliminary fish protective goals are 0.3 to 0.9 mg/kg PCBs (NOAEL), 1 to 3 mg/kg PCBs (LOAEL), and 3 to 32 pg/g TEC (mammalian) for no effect to low effect. Assuming equal use of Conard's Branch and Richland Creek by kingfisher, preliminary fish protective goals in Conard's Branch fish are 0.5 to 2 mg/kg PCBs (NOAEL), 2 to 5 mg/kg PCBs (LOAEL) and 6 to 63 pg/g TEC (mammalian) (NOAEL to LOAEL).

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Appendix A

**Aroclor, PCB Congener, and TEC Concentrations in Fish
Collected from Conard's Branch and Richland Creek, August 2001,
and Risk Estimates for Mink and Kingfisher**

Appendix A1. PCB Congener, Aroclor, and TEC Concentrations in Whole Fish Samples, 8/01, Neal's Landfill Site, Bloomington, IN.																		
Loc.	Spp.	ID	PCB Congeners										Mammalian WHO-TEC		Aroclor			
			77	81	105	114	118	123	126	156/157	167	169	189	pg/g				
			_____ pg/g _____											pg/g				
1	CRC	A06782	60900	3630	176000	15800	294000	10400	1250	7980	2390	72	171	192.14	5900			
1	CRC	A06783	98100	5080	266000	18100	442000	11700	1390	13100	3950	3.9	263	236.99	6900			
1	CRC	A06784	80500	4990	207000	17100	340000	14300	1020	8480	2840	4.3	196	179.56	5900			
1	GS	A06785	46500	2510	121000	10500	187000	7700	617	4980	1300	4.1	83.4	105.97	9000			
1	GS	A06786	90000	5560	228000	21700	342000	15300	1190	9050	2290	3.7	142	202.54	7700			
1	GS	A06787	128000	6910	302000	27500	466000	19900	1680	12800	3470	6.8	203	280.55	6200			
2	LS	A06709	41700	2820	127000	10200	223000	8340	598	5730	2070	U	164	108.09	360			
2	LS	A06710	10700	468	48000	2760	79100	2570	158	2340	793	U	63.6	32.45	380			
2	LS	A06711	9120	433	36700	2290	57600	1850	3	1620	552	U	44.2	12.84	560			
2	LS	A06712	20000	996	73300	4830	118000	3890	2.9	3520	1180	U	92.8	26.10	570			
2	LS	A06713	10400	449	32900	2140	54500	1940	2.6	1570	517	U	44.5	12.14	320			
2	LS	A06714	13400	645	60300	4980	97900	4070	4.3	3230	1170	U	86.1	22.19	780			
2	LS	A06715	5390	298	20300	1680	35000	1290	72.4	1090	399	U	36	14.86	840			
2	LS	A06716	10000	512	45300	3670	67500	3010	161	2280	689	U	53.2	31.72	980			
2	WS	A06717	233	4.1	390	4.1	675	4.1	4.1	4.1	4.1	U	4.1	0.55	1200			
2	WS	A06718	19500	1160	58100	4090	95800	3380	243	2390	801	U	63.5	45.35	3600			
2	WS	A06719	8710	491	27500	1980	48100	1350	116	1360	504	U	51.1	21.90	1800			
2	WS	A06720	15500	1380	71700	5240	145000	4210	319	3320	1360	U	106	59.98	2700			
2	WS	A06721	771	4.7	2710	207	4540	186	4.7	185	81.2	U	4.7	1.49	1200			
3S	CRC	A06729	2590	133	12000	888	20200	613	U	498	215	U	25.5	4.25	210			
3S	CRC	A06730	5035	268	13500	948	23000	910	U	623	272	U	25.5	5.06	230			
4	BRH	A06746	1590	97.9	6450	330	11400	372	U	330	129	U	4	2.32	320			
4	BRH	A06747	2050	129	7620	531	14000	458	U	384	162	U	2.4	2.89	390			

Appendix A1. PCB Congener, Aroclor, and TEC Concentrations in Whole Fish Samples, 8/01, Neal's Landfill Site, Bloomington, IN.																	
Loc.	Spp.	ID	PCB Congeners										Mammalian WHO-TEC		Aroclor		
			77	81	105	114	118	123	126	156/157	167	169	189	pg/g			
			_____ pg/g _____														
	4BRH	A06748	1650	98.5	6210	452	11300	396		U	330	139	U	2.5	2.36	310	
	4LS	A06749	758	37.3	5380	351	10700	382		U	373	186	U	2.4	2.09	200	
	4LS	A06750	325	2.4	3450	246	7380	269		U	403	201	U	28	1.47	250	
	4LS	A06776	871	48.9	5300	341	10700	370		U	420	213	U	26.7	2.11	220	
	4LS	A06777	965	2.4	7140	471	15700	488		U	587	305	U	35.8	2.97	350	
	4LS	A06778	1100	56.9	4710	285	9420	318		U	317	162	U	2.5	1.86	350	
	4LS	A06779	482	24.6	3920	277	8600	276		U	321	167	U	2.4	1.63	200	
	4LS	A06780	787	37.6	2550	158	5170	187		U	166	85.3	U	2.5	1.04	300	
	4LS	A06781	96.6	2.4	976	73.5	2240	76.8		U	183	91.3	U	2.4	0.47	260	
	4WS	A06744	1910	96.9	5700	378	10200	381		U	278	134	U	4.5	2.16	290	
	4WS	A06745	823	3.8	1510	108	2710	102		U	105	51.8	U	3.8	0.62	340	
	5BRH	A06697	437	31.3	2080	156	3620	151		U	155	68.7	U	2.4	0.79	230	
	5BRH	A06698	563	31	2570	198	4440	183		U	179	75.8	U	2.4	0.97	160	
	5BRH	A06699	794	46.9	3710	269	6310	237		U	240	102	U	2.4	1.37	210	
	5BRH	A06700	480	39.6	2330	170	4270	157		U	163	75.2	U	2.4	0.90	210	
	5CP	A06696	217	2.4	713	50.9	1230	48.4		U	54	24.4	U	2.4	0.27	170	
	5LS	A06701	281	2.6	1830	141	3560	134		U	216	104	U	2.6	0.76	200	
	5LS	A06702	390	2.6	1790	135	3320	122		U	178	87.7	U	2.6	0.72	240	
	5LS	A06703	98.8	2.5	843	68.6	1730	68.6		U	107	50.9	U	2.5	0.36	160	
	5LS	A06704	111	2.5	1130	90.5	2180	77.6		U	127	58	U	2.5	0.46	200	
	5LS	A06705	261	2.7	1200	88.7	2310	86		U	124	62.3	U	2.7	0.49	190	
	5LS	A06706	243	2.4	1460	115	3100	115		U	297	157	U	28.2	0.70	220	
	5LS	A06707	309	2.7	2160	167	4600	158		U	413	212	U	33.7	1.02	160	
	5LS	A06708	312	2.4	2710	147	5380	185		U	340	172	U	30.3	1.11	180	

Appendix A2. UCL Aroclor and TEC Concentrations in Whole Fish and Reach-specific Risk to Mink and Kingfisher, 801, Neal's Landfill Site, Bloomington, IN.																				
Loc.	Spp.	N	Mink						Kingfisher											
			Species UCL			Dietary UCL			PCB HQ			Dose			TEQ HQ			PCB HQ		
			TEC	Aroclor	µg/kg	TEC	Aroclor	µg/kg	TEC	Aroclor	µg/kg	TEQ	Aroclor	µg/kg-d	NOAEL	TEQ HQ	NOAEL	no effect	NOAEL	low effect
			pg/g			pg/g			pg/g			ng/kg			ratio	ratio	ratio	ratio	ratio	ratio
1	CRC	3	277.89	7667.55	243.22	6606.24	53	14	11	299.72	8140.80	149.86	4070.40	107	11	10	37	8	4	4
1	GS	3	413.60	11114.08																
1	CF		115.63	3140.74																
2	LS	8	58.94	803.85	41.12	1415.57	9	2	3	50.59	1726.11	25.30	863.06	18	2	2	8	2	0.8	
2	WS	5	58.67	3390.38																
2	CF		17.75	242.12																
1&2	Receptor																			
1&2	Fish		130.54	3920.54	91.64	2713.24	20	5	5	175.16	4933.46	87.58	2466.73	63	6	6	22	5	2	
1&2	CF		42.22	966.78																
1&2	Receptor																			
1&2	Fish		202.27	5743.96																
1&2	CF		66.69	1691.43																
3S	CRC	2	9.80	347.06	6.95	246.09	2	0.4	0.5	0.4	8.58	303.84	4.29	151.92	3	0.3	0.4	1	0.3	0.1
3S	CF		3.70	130.97																
4	BRH	3	3.31	448.28	3.79	323.93	0.8	0.2	0.6	0.5	4.63	396.89	2.31	198.45	2	0.2	0.5	2	0.4	0.2
4	LS	8	2.33	332.33																
4	WS	2	11.17	632.66																
4	CF		0.70	100.10																
5	BRH	4	1.41	305.17	0.61	110.59	0.1	0.03	0.2	0.2	0.75	136.01	0.38	68.00	0.3	0.0	0.2	0.6	0.1	0.1
5	CP	1	0.27	17.00																
5	LS	8	0.92	153.24																
5	CF		0.28	46.16																
6	BRH	3	0.75	17.54	0.43	11.02	0.09	0.02	0.02	0.02	0.53	13.56	0.26	6.78	0.2	0.02	0.02	0.06	0.01	0.01

Appendix A3. Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish Samples, 8/01, Neal's Landfill Site, Bloomington, IN.																	
Loc.	Spp.	ID	Lipid Conc.	PCB Congeners												Aroclor mg/kg lipid	
				77	81	105	114	118	123	126	156/157	167	169	189			
				————— µg/kg lipid —————													
1	CRC	A06782	4.87	1250.51	74.54	3613.96	324.44	6036.96	213.55	25.67	163.86	49.08	1.48	3.51	121.15		
1	CRC	A06783	3.89	2521.85	130.59	6838.05	465.30	11362.47	300.77	35.73	336.76	101.54	0.10	6.76	177.38		
1	CRC	A06784	6.28	1281.85	79.46	3296.18	272.29	5414.01	227.71	16.24	135.03	45.22	0.07	3.12	93.95		
1	GS	A06785	2.93	1587.03	85.67	4129.69	358.36	6382.25	262.80	21.06	169.97	44.37	0.14	2.85	307.17		
1	GS	A06786	3.8	2368.42	146.32	6000.00	571.05	9000.00	402.63	31.32	238.16	60.26	0.10	3.74	202.63		
1	GS	A06787	3.36	3803.86	205.35	8974.74	817.24	13848.44	591.38	49.93	380.39	103.12	0.20	6.03	184.25		
2	LS	A06709	1.08	3843.32	259.91	11705.07	940.09	20553.00	768.66	55.12	528.11	190.78	U	15.12	33.18		
2	LS	A06710	1.08	986.18	43.13	4423.96	254.38	7290.32	236.87	14.56	215.67	73.09	U	5.86	35.02		
2	LS	A06711	1.08	840.55	39.91	3382.49	211.06	5308.76	170.51	0.28	149.31	50.88	U	4.07	51.61		
2	LS	A06712	1.08	1843.32	91.80	6755.76	445.16	10875.58	358.53	0.27	324.42	108.76	U	8.55	52.53		
2	LS	A06713	1.08	958.53	41.38	3032.26	197.24	5023.04	178.80	0.24	144.70	47.65	U	4.10	29.49		
2	LS	A06714	1.08	1235.02	59.45	5557.60	458.99	9023.04	375.12	0.40	297.70	107.83	U	7.94	71.89		
2	LS	A06715	1.14	472.81	26.14	1780.70	147.37	3070.18	113.16	6.35	95.61	35.00	U	3.16	73.68		
2	LS	A06716	1.03	970.87	49.71	4398.06	356.31	6553.40	292.23	15.63	221.36	66.89	U	5.17	95.15		
2	WS	A06717	4.74	4.92	0.09	8.23	0.09	14.24	0.09	0.09	0.09	0.09	U	0.09	25.32		
2	WS	A06718	4.28	455.25	27.08	1356.42	95.49	2236.58	78.91	5.67	55.80	18.70	U	1.48	84.05		
2	WS	A06719	3.22	270.50	15.25	854.04	61.49	1493.79	41.93	3.60	42.24	15.65	U	1.59	55.90		
2	WS	A06720	4.28	361.87	32.22	1673.93	122.33	3385.21	98.29	7.45	77.51	31.75	U	2.47	63.04		
2	WS	A06721	4.89	15.77	0.10	55.42	4.23	92.84	3.80	0.10	3.78	1.66	U	0.10	24.54		
3S	CRC	A06729	2.58	100.39	5.16	465.12	34.42	782.95	23.76	U	19.30	8.33	U	0.99	8.14		
3S	CRC	A06730	3.38	148.96	7.93	399.41	28.05	680.47	26.92	U	18.43	8.05	U	0.75	6.80		

Appendix A3. Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish Samples, 8/01, Neal's Landfill Site, Bloomington, IN.																			
Loc.	Spp.	ID	Lipid Conc.	PCB Congeners												Aroclor			
				77	81	105	114	118	123	126	156/157	167	169	189					
				_____ µg/kg lipid _____															
			%																
	4 BRH	A06746	3.00	53.00	3.26	215.00	11.00	380.00	12.40	U	11.00	4.30	U	0.13	10.67				
	4 BRH	A06747	2.35	87.23	5.49	324.26	22.60	595.74	19.49	U	16.34	6.89	U	0.10	16.60				
	4 BRH	A06748	3.65	45.21	2.70	170.14	12.38	309.59	10.85	U	9.04	3.81	U	0.07	8.49				
	4 LS	A06749	1.18	64.24	3.16	455.93	29.75	906.78	32.37	U	31.61	15.76	U	0.20	16.95				
	4 LS	A06750	0.38	85.53	0.63	907.89	64.74	1942.11	70.79	U	106.05	52.89	U	7.37	65.79				
	4 LS	A06776	0.8	108.88	6.11	662.50	42.62	1337.50	46.25	U	52.50	26.62	U	3.34	27.50				
	4 LS	A06777	0.72	134.03	0.33	991.67	65.42	2180.56	67.78	U	81.53	42.36	U	4.97	48.61				
	4 LS	A06778	0.94	117.02	6.05	501.06	30.32	1002.13	33.83	U	33.72	17.23	U	0.27	37.23				
	4 LS	A06779	1.05	45.90	2.34	373.33	26.38	819.05	26.29	U	30.57	15.90	U	0.23	1.90				
	4 LS	A06780	1.8	43.72	2.09	141.67	8.78	287.22	10.39	U	9.22	4.74	U	0.14	16.67				
	4 LS	A06781	0.7	13.80	0.34	139.43	10.50	320.00	10.97	U	26.14	13.04	U	0.34	37.14				
	4 WS	A06744	1.99	95.98	4.87	286.43	18.99	512.56	19.15	U	13.97	6.73	U	0.23	14.57				
	4 WS	A06745	1.99	41.36	0.19	75.88	5.43	136.18	5.13	U	5.28	2.60	U	0.19	17.09				
	5 BRH	A06697	2.24	19.51	1.40	92.86	6.96	161.61	6.74	U	6.92	3.07	U	0.11	10.27				
	5 BRH	A06698	1.74	32.36	1.78	147.70	11.38	255.17	10.52	U	10.29	4.36	U	0.14	9.20				
	5 BRH	A06699	4.25	18.68	1.10	87.29	6.33	148.47	5.58	U	5.65	2.40	U	0.06	4.94				
	5 BRH	A06700	2.63	18.25	1.51	88.59	6.46	162.36	5.97	U	6.20	2.86	U	0.09	0.80				
	5 CP	A06696	2.53	8.58	0.09	28.18	2.01	48.62	1.91	U	2.13	0.96	U	0.09	0.67				
	5 LS	A06701	0.77	36.49	0.34	237.66	18.31	462.34	17.40	U	28.05	13.51	U	0.34	2.60				
	5 LS	A06702	1.41	27.66	0.18	126.95	9.57	235.46	8.65	U	12.62	6.22	U	0.18	17.02				
	5 LS	A06703	1.04	9.50	0.24	81.06	6.60	166.35	6.60	U	10.29	4.89	U	0.24	1.54				
	5 LS	A06704	1.03	10.78	0.24	109.71	8.79	211.65	7.53	U	12.33	5.63	U	0.24	1.94				
	5 LS	A06705	0.39	66.92	0.69	307.69	22.74	592.31	22.05	U	31.79	15.97	U	0.69	4.87				
	5 LS	A06706	1.54	15.78	0.16	94.81	7.47	201.30	7.47	U	19.29	10.19	U	1.83	14.29				

Appendix A4. UCL Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish, and TEC and Risk in Fish, 801, Neal's Landfill Site, Bloomington, IN.																
Loc.	Spp.	N	PCB Congeners											Aroclor mg/kg lipid	Fish TEC ng/kg lipid	Fish HQ ratio
			77	81	105	114	118	123	126	156/157	167	169	189			
			µg/kg lipid													
1 CRC	3	3	3486.06	171.97	9450.69	602.04	15726.08	363.62	50.09	482.91	143.43	2.55	9.43	236.52	685.18	49
1 GS	3	3	5379.55	294.44	12437.92	1152.68	19154.04	828.56	70.46	529.53	144.74	0.27	8.28	396.05	1037.48	74
1 CF			2485.45	129.50	5852.57	347.18	11819.66	230.47	30.47	269.96	94.25	na	5.13	149.54		
1 Dietary			4043.34	212.46	9925.96	771.33	16315.98	522.97	54.31	458.97	134.12	1.41	8.11	282.94		
2 LS	8	8	2282.88	140.49	7691.97	589.82	13021.45	484.24	27.27	361.98	127.27	U	9.99	74.77	434.89	31
2 WS	5	5	474.59	33.44	1722.31	124.14	3229.77	99.13	7.47	77.45	29.82	U	2.43	82.35	101.55	7
2 CF			665.85	50.09	1363.74	109.24	2751.21	97.09	9.50	60.15	25.90	na	1.47	19.32		
2 Dietary			1236.16	79.59	4038.46	307.43	7050.73	252.76	15.80	187.80	68.02	U	5.26	66.71		
1&2 Mean			2639.75	146.03	6982.21	539.38	11683.36	387.86	35.06	323.39	101.07	0.71	6.68	174.82		
3S CRC	2	2	433.25	24.14	849.73	71.70	1382.78	45.42	U	24.39	9.97	U	2.40	15.98	55.40	4
3S CF			308.69	18.18	526.21	41.35	1039.29	28.79	na	13.64	6.55	na	1.30	10.11		
3S Dietary			408.37	22.95	785.02	65.63	1314.08	42.09	U	22.24	9.29	U	2.18	14.81		
4 BRH	3	3	117.34	7.48	433.39	31.07	798.83	25.69	U	21.51	9.11	U	0.18	22.35	15.48	1
4 LS	8	8	111.50	4.60	787.41	52.91	1675.87	56.57	U	73.33	37.15	U	4.45	48.36	13.45	1
4 WS	2	2	415.68	32.26	1518.80	98.36	2715.55	101.21	U	64.83	30.90	U	0.46	31.84	57.70	4
4 CF			32.52	1.64	135.60	9.80	354.08	11.34	na	12.19	7.56	na	0.65	12.50		
4 Dietary			178.38	12.16	758.48	50.58	1454.88	51.19	U	45.02	22.09	U	1.49	29.85		
5 BRH	4	4	33.01	1.90	150.51	11.62	260.29	10.80	U	10.58	4.51	U	0.15	13.19	4.25	0.3
5 CP	1	1	8.58	0.09	28.18	2.01	48.62	1.91	U	2.13	0.96	U	0.09	0.67	0.90	0.06

Appendix A5. Modeled UCL Congener and TEC Concentrations in Kingfisher Eggs, and Estimated Risk, 8/01, Neal's Landfill Site, IN.

Reach	PCB Congeners										Avian		NOAEC	
	77	81	105	114	118	123	126	156/157	167	169	189	WHO-TEC µg/kg lipid	HQ	LOAEC HQ — ratio —
1	3598.57	3144.42	78911.36	11107.09	426662.94	1615.29	9730.16	44.11				670.19	374	39 126 10
2	1100.18	1177.93	32105.78	4427.01	184376.58	469.81	3981.38	U				225.68	126	13 42 3
1&2	2349.37	2161.18	55508.57	7767.05	305519.76	1042.55	6855.77	22.05				447.93	250	26 84 7
3S	363.45	339.67	6240.95	945.12	34363.15	U	471.51	U				53.25	30	3 10 0.8
4	158.76	179.89	6029.90	728.39	38045.15	U	954.35	U				27.08	15	2 5 0.4
5	24.86	10.23	1236.25	149.26	7842.17	U	352.08	U				2.52	1	0.2 0.5 0.04
6	11.66	U	703.29	79.34	4575.38	U	432.72	U				0.75	0.4	0.04 0.1 0.01
Diet-Egg BMF	0.89	14.8	7.95	14.4	26.15	NA	29.74	21.2	NA	31.25	NA	TRV:	1.79	17 5.33 68
BMF Source	BG	M	BG	M	BG	BG	BG	M		BG				— µg/kg lipid —
Bird Egg TEFs	0.05	0.1	0.0001	0.0001	—0.00001—	0.1	0.0001	0.00001	0.00001	0.001	0.00001			

Egg concentration = reach mean lipid-normalized fish concentration * Diet-egg BMF

Diet-Egg BMF- geometric mean lipid-normalized diet-to-egg biomagnification factors based on various studies (Blankenship and Giesy 2002).

BMF Source: BG – geometric mean of various studies (Blankenship and Giesy 2002); M – great cormorant (Murata, et al. 2003)

NA - not available

Avian TEC = $[0.1 * (\#81 + 126)] + [0.05 * \#77] + [0.001 * \#169] + [0.0001 * (\#105 + 114 + 156/157)] + [0.00001 * (\#118 + 123 + 167 + 189)]$ (Van den Berg, et al. 1998)

HQ - hazard quotient = egg concentration / egg toxicity reference value (TRV)

TEC TRVs - 1.79 ug/kg lipid NOAEC, 5.33 ug/kg lipid LOAEC (Blankenship and Giesy 2002 based on Elliott, et al. 1996); and 17 ug/kg lipid NOAEC, 68 ug/kg lipid LOAEC (Powell, et al. 1997) for a cormorant egg lipid content of 5.9 % (altricial species) (Carey, et al. 1980)

1&2 - weighted UCL fish congener concentration for kingfisher foraging 50 % each in Reaches 1 and 2, and the corresponding HQs.

Remaining symbols and definitions as in Appendices A1 and A2. All concentrations are lipid-normalized (lw).

Appendix A6. Percent Allowable Consumption (UCL TEC Basis), 8/01, Neal's Landfill Site, IN.

Reach	Mink Diet		Kingfisher Dose		Kingfisher Egg		Kingfisher Range			
							NOAEL-NOAEC		LOAEL-LOAEC	
	NOAEC	LOAEC	NOAEL	LOAEL	NOAEC	LOAEC	lowest	highest	lowest	highest
----- % -----										
1	2	7	0.8	9	0.3	2	0.8	0.3	2	0.8
2	10	43	5	54	0.8	5	2	0.8	5	2
1&2	5	19	1	16	0.4	2	1	0.4	2	1
3S	60	100	27	100	3	20	10	3	27	10
4	100	100	50	100	7	40	20	7	50	20
5	100	100	100	100	71	100	100	71	100	100
6	100	100	100	100	100	100	100	100	100	100
Egg TRVs					1.79	17	5.33	68		
TRV units					----- ug/kg lipid -----					

Percent Allowable Consumption (PAC) = ((1 - HQ upstream in 2002) / HQ reach) * 100

HQ upstream (based on 2002 UCL TEC in location 0): mink dietary NOAEC HQ - 0.09, LOAEC HQ - 0.02; kingfisher dose NOAEL HQ - 0.17, LOAEL HQ - 0.02 (Appendix B2); and kingfisher egg NOAEC HQs - 3.5 (1.79 TRV) and 0.37 (17 TRV), LOAEC HQs - 1.2 (5.33 TRV) and 0.09 (68 TRV) (Appendix B5)

PACs for kingfisher egg NOAEC (1.79 TRV) and LOAEC (5.33 TRV) are not adjusted for upstream HQ because the 2002 upstream HQs are greater than 1. The equation becomes: PAC = (1 / HQ reach) * 100

1&2 - PAC for fixed reach foraging models (Appendix A2).

Appendix A8. Mean Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish, and TEC and Risk in Fish, 8/01, Neal's Landfill Site, Bloomington, IN.																	
Loc.	Sp.	N	PCB Congeners										Aroclor		Fish TEC	Fish HQ	ratio
			77	81	105	114	118	123	126	156/157	167	169	189	1248			
			µg/kg lipid										mg/kg lipid	ng/kg lipid			
1 CRC	3		1684.74	94.86	4582.73	354.01	7604.48	247.34	25.88	211.88	65.28	0.55	4.46	130.83	345.34	24	
1 GS	3		2586.44	145.78	6368.14	582.22	9743.56	418.94	34.10	262.84	69.25	0.15	4.21	231.35	502.04	36	
1 CF			1201.16	71.44	2837.97	204.15	5715.50	156.77	15.74	118.45	42.90	na	2.43	82.71			
1 Dietary			1948.70	110.54	4947.94	415.32	8082.32	297.87	27.14	213.58	62.39	0.35	3.95	161.41			
2 LS	8		1393.82	76.43	5129.49	376.32	8462.16	311.73	11.60	247.11	85.11	U	6.75	55.32	235.62	17	
2 WS	5		221.66	14.95	789.61	56.73	1444.53	44.60	3.38	35.88	13.57	U	1.15	50.57	46.54	3	
2 CF			406.54	27.25	909.43	66.70	1787.91	62.50	4.04	41.06	17.32	na	0.99	14.30			
2 Dietary			727.50	42.00	2549.52	187.16	4320.26	155.04	6.80	121.41	42.94	U	3.35	45.21			
1&2 Mean			1338.10	76.27	3748.73	301.24	6201.29	226.45	16.97	167.49	52.66	0.17	3.65	103.31			
3S CRC	2		124.68	6.54	432.26	31.23	731.71	25.34	U	18.87	8.19	U	0.87	7.47	15.74	1	
3S CF			88.89	4.93	267.69	18.01	549.95	16.06	na	10.55	5.38	na	0.47	4.72			
3S Dietary			117.52	6.22	399.35	28.59	695.36	23.49	U	17.20	7.63	U	0.79	6.92			
4 BRH	3		61.81	3.82	236.46	15.33	428.44	14.25	U	12.13	5.00	U	0.10	11.92	8.09	0.6	
4 LS	8		76.64	2.63	521.69	34.81	1099.42	37.33	U	46.42	23.57	U	2.11	31.47	8.98	0.6	
4 WS	2		68.67	2.53	181.16	12.21	324.37	12.14	U	9.62	4.67	U	0.21	15.83	8.13	0.6	
4 CF			22.35	0.94	92.49	6.45	232.29	7.49	na	7.71	4.80	na	0.31	8.13			
4 Dietary			59.70	2.58	268.98	17.92	540.39	18.49	U	19.72	9.82	U	0.71	17.42			
5 BRH	4		22.20	1.45	104.11	7.78	181.90	7.20	U	7.26	3.17	U	0.10	6.30	2.94	0.2	
5 CP	1		8.58	0.09	28.18	2.01	48.62	1.91	U	2.13	0.96	U	0.09	0.67	0.91	0.1	
5 LS	8		31.42	0.31	208.06	14.28	410.90	14.81	U	26.24	13.11	U	1.49	5.88	3.30	0.2	

Appendix A10. Percent Allowable Consumption (Mean TEC Basis), 801, Neal's Landfill Site, Bloomington, IN.

Reach	Mink Diet		Kingfisher Dose				Kingfisher Egg				Kingfisher Range			
			NOAEL		LOAEL		NOAEC		LOAEC		NOAEL-NOAEC		LOAEL-LOAEC	
	NOAEC	LOAEC	NOAEL	LOAEL	NOAEC	NOAEC	LOAEC	LOAEC	lowest	highest	lowest	highest		
	_____ % _____						_____ % _____							
1	3	13	1	16	0.5	4	0.5	19	0.5	4	0.5	19		
2	21	86	10	100	2	11	1	54	2	11	1	100		
1&2	8	35	2	28	0.8	6	0.7	28	0.8	6	0.7	28		
3S	100	100	59	100	12	89	11	100	12	89	11	100		
4	100	100	100	100	26	100	24	100	26	100	24	100		
5	100	100	100	100	100	100	95	100	100	100	95	100		
6	100	100	100	100	100	100	100	100	100	100	100	100		
Egg TRVs													68	
TRV units													_____ ug/kg lipid _____	
					1.79	17	5.33							

See footnotes to Appendix A6.

HQ upstream (based on 2002 mean TEC): mink dietary NOAEC HQ - 0.07, LOAEC HQ - 0.02; kingfisher dose NOAEL HQ - 0.14, LOAEL HQ - 0.01 (Appendix B7); and kingfisher egg NOAEC HQs - 2.0 (1.79 TRV) and 0.21 (17 TRV), LOAEC HQs - 0.69 (5.33 TRV) and 0.054 (68 TRV) (Appendix B9)

PAC for kingfisher egg NOAEC (1.79 TRV) is not adjusted for upstream HQ because the 2002 upstream HQ is greater than 1.

The equation becomes: $PAC = (1 / HQ \text{ reach}) * 100$

Appendix B

**Aroclor, PCB Congener, and TEC Concentrations in Fish
Collected from Conard's Branch and Richland Creek, November 2002,
and Risk Estimates for Mink and Kingfisher**

Appendix B1. PCB Congener, Aroclor, and TEC Concentrations in Whole Fish Samples, 11/02, Neal's Landfill Site, Bloomington, IN.																		
Loc.	Spp.	ID	PCB Congeners												Mammalian WHO-TEC	4-peak Aroclor	5-peak Aroclor	
			77	81	105	114	118	123	126	156/157	167	169	189					
			pg/g															
			17	na	na	na	na	na	na	na	na	na	na	na	na	pg/kg		
0	CRC	17	na	na	na	na	na	na	na	na	na	na	na	na	na	83	83	
0	CRC	18	na	na	na	na	na	na	na	na	na	na	na	na	na	87	87	
0	CRC	19	45.7	2.1	453	34	818	38	2.1	58.4	40.1	U	2.1	0.39	78	78	78	
0	CRC	20	2.3	2.3	183	2.3	317	2.3	2.3	40.8	25	U	2.3	0.30	88	88	88	
0	CRC	21	2.4	2.4	260	2.4	480	25.5	2.4	55.8	35.9	U	2.4	0.35	98	98	98	
0	CRC	22	2.2	2.2	184	2.2	353	2.2	2.2	39.7	26.1	U	2.2	0.30	88	88	88	
0	WS	10	na	na	na	na	na	na	na	na	na	na	na	na	200	190	190	
0	WS	11	na	na	na	na	na	na	na	na	na	na	na	na	500	440	440	
0	WS	12	214	1.9	1180	80	2110	77.2	1.9	190	112	U	23.8	0.69	150	190	190	
0	WS	13	126	2.4	666	43.7	1160	53.5	2.4	102	67	U	2.4	0.51	120	160	160	
0	WS	14	81.9	2.3	442	27.9	766	38.8	2.3	81.1	50.4	U	2.3	0.42	110	150	150	
0	WS	15	2560	219	12500	1080	21900	607	47.1	656	231	U	26.2	9.36	740	610	610	
0	WS	16	98.6	2.5	606	40.7	1070	48.2	2.5	129	70.8	U	2.5	0.52	93	150	150	
1	CRC	1	na	na	na	na	na	na	na	na	na	na	na	na	na	6700	5500	5500
1	CRC	2	na	na	na	na	na	na	na	na	na	na	na	na	na	10000	8200	8200
1	CRC	3	na	na	na	na	na	na	na	na	na	na	na	na	na	2200	1800	1800
1	CRC	4	na	na	na	na	na	na	na	na	na	na	na	na	na	640	550	550
1	CRC	5	45800	2580	134000	9960	234000	7790	565	7410	2380	U	164	107.64	5200	4200	4200	
1	CRC	6	22400	1360	84200	6090	150000	5250	293	4240	1600	U	124	60.81	2700	2200	2200	
1	CRC	7	42300	2340	110000	7850	192000	6650	472	5450	2110	U	153	89.22	11000	9300	9300	
1	CRC	8	29300	1760	85200	6750	152000	5490	384	4810	1620	U	123	71.58	3100	2600	2600	
1	CRC	9	9940	557	38500	2980	69900	2590	128	2190	889	U	69.8	27.55	2500	2100	2100	
2	CRC	35	na	na	na	na	na	na	na	na	na	na	na	na	na	750	600	600

Appendix B3. Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish Samples, 11/02, Neal's Landfill Site, Bloomington, IN.																				
Loc.	Spp.	Sample ID	Lipid Conc.	PCB Congeners										Aroclor						
				%	77	81	105	114	118	123	126	156/157	167	169	189	4-peak	5-peak	mg/kg lipid		
0	CRC	17	0.21	na	na	na	na	na	na	na	na	na	na	na	na	na	39.52	39.52	39.52	39.52
0	CRC	18	0.26	na	na	na	na	na	na	na	na	na	na	na	na	na	33.46	33.46	33.46	33.46
0	CRC	19	0.35	13.06	0.60	129.43	9.71	233.71	10.86	0.60	16.69	11.46	U	0.60	0.60	0.60	22.29	22.29	22.29	22.29
0	CRC	20	0.28	0.82	0.82	65.36	0.82	113.21	0.82	0.82	14.57	8.93	U	0.82	0.82	0.82	31.43	31.43	31.43	31.43
0	CRC	21	0.15	1.60	1.60	173.33	1.60	320.00	17.00	1.60	37.20	23.93	U	1.60	1.60	1.60	65.33	65.33	65.33	65.33
0	CRC	22	0.19	1.16	1.16	96.84	1.16	185.79	1.16	1.16	20.89	13.74	U	1.16	1.16	1.16	46.32	46.32	46.32	46.32
0	WS	10	1.28	na	na	na	na	na	na	na	na	na	na	na	na	na	15.63	15.63	15.63	14.84
0	WS	11	0.5	na	na	na	na	na	na	na	na	na	na	na	na	na	100.00	100.00	100.00	88.00
0	WS	12	0.7	30.57	0.27	168.57	11.43	301.43	11.03	0.27	27.14	16.00	U	3.40	3.40	3.40	21.43	21.43	21.43	27.14
0	WS	13	1.4	9.00	0.17	47.57	3.12	82.86	3.82	0.17	7.29	4.79	U	0.17	0.17	0.17	8.57	8.57	8.57	11.43
0	WS	14	0.5	16.38	0.46	88.40	5.58	153.20	7.76	0.46	16.22	10.08	U	0.46	0.46	0.46	22.00	22.00	22.00	30.00
0	WS	15	0.6	426.67	36.50	2083.33	180.00	3650.00	101.17	7.85	109.33	38.50	U	4.37	4.37	4.37	123.33	123.33	123.33	101.67
0	WS	16	0.61	16.16	0.41	99.34	6.67	175.41	7.90	0.41	21.15	11.61	U	0.41	0.41	0.41	15.25	15.25	15.25	24.59
1	CRC	1	0.89	na	na	na	na	na	na	na	na	na	na	na	na	na	752.81	752.81	752.81	617.98
1	CRC	2	0.73	na	na	na	na	na	na	na	na	na	na	na	na	na	1369.86	1369.86	1369.86	1123.29
1	CRC	3	0.59	na	na	na	na	na	na	na	na	na	na	na	na	na	372.88	372.88	372.88	305.08
1	CRC	4	0.45	na	na	na	na	na	na	na	na	na	na	na	na	na	142.22	142.22	142.22	122.22
1	CRC	5	0.64	7156.25	403.13	20937.50	1556.25	36562.50	1217.19	88.28	1157.81	371.88	U	25.63	25.63	25.63	812.50	812.50	812.50	656.25
1	CRC	6	0.36	6222.22	377.78	23388.89	1691.67	41666.67	1458.33	81.39	1177.78	444.44	U	34.44	34.44	34.44	750.00	750.00	750.00	611.11
1	CRC	7	0.97	4360.82	241.24	11340.21	809.28	19793.81	685.57	48.66	561.86	217.53	U	15.77	15.77	15.77	1134.02	1134.02	1134.02	958.76
1	CRC	8	0.75	3906.67	234.67	11360.00	900.00	20266.67	732.00	51.20	641.33	216.00	U	16.40	16.40	16.40	413.33	413.33	413.33	346.67
1	CRC	9	0.73	1361.64	76.30	5273.97	408.22	9575.34	354.79	17.53	300.00	121.78	U	9.56	9.56	9.56	342.47	342.47	342.47	287.67
2	CRC	35	0.28	na	na	na	na	na	na	na	na	na	na	na	na	na	267.86	267.86	267.86	214.29
2	CRC	36	0.28	na	na	na	na	na	na	na	na	na	na	na	na	na	164.29	164.29	164.29	132.14

Appendix B3. Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish Samples, 11/02, Neal's Landfill Site, Bloomington, IN.																			
Loc.	Spp.	Sample ID	Lipid Conc. %	PCB Congeners										Aroclor					
				77	81	105	114	118	123	126	156/157	167	169	189	4-peak	5-peak	mg/kg lipid		
2 CRC		37	0.25	776.00	50.80	4560.00	404.00	7760.00	272.00	18.16	284.00	106.00	U	10.00		80.00	64.00		
2 CRC		38	0.34	335.29	17.00	2279.41	193.24	3882.35	135.29	10.09	162.65	58.53	U	0.68		52.94	47.06		
2 CRC		39	0.31	364.52	18.90	2467.74	207.74	4290.32	158.71	8.77	170.00	67.74	U	0.77		22.90	20.97		
2 CRC		40	0.46	450.00	24.57	2391.30	202.39	4065.22	131.52	9.57	147.83	55.65	U	5.28		69.57	56.52		
2 LS		41	1.16	na	na	na	na	na	na	na	na	na	na	na		74.14	59.48		
2 LS		42	0.47	na	na	na	na	na	na	na	na	na	na	na		21.28	19.79		
2 LS		43	1.08	579.63	29.07	2083.33	164.81	3712.96	138.89	8.10	120.37	45.28	U	4.13		138.89	111.11		
2 LS		44	0.8	905.00	45.88	3475.00	275.00	6150.00	237.50	13.13	200.00	67.75	U	6.46		45.00	36.25		
2 LS		45	0.81	382.72	18.15	1802.47	145.68	3296.30	117.41	6.16	110.49	43.21	U	4.84		70.37	56.79		
2 LS		46	0.82	273.17	13.05	914.63	73.41	1585.37	59.63	3.99	54.15	21.83	U	0.27		18.29	14.63		
2 WS		23	0.57	617.54	51.40	2491.23	201.75	4000.00	151.40	12.81	119.82	40.35	U	3.63		245.61	192.98		
2 WS		24	0.5	274.00	54.40	4180.00	384.00	7920.00	248.00	12.42	256.00	91.60	U	8.42		108.00	86.00		
2 WS		25	0.44	na	na	na	na	na	na	na	na	na	na	na		184.09	147.73		
2 WS		26	0.4	na	na	na	na	na	na	na	na	na	na	na		165.00	130.00		
2 WS		27	1	1070.00	89.40	4580.00	351.00	7490.00	269.00	22.40	210.00	69.20	U	5.96		250.00	200.00		
2 WS		28	0.44	3159.09	217.05	9954.55	765.91	16522.73	572.73	44.77	456.82	157.50	U	13.64		681.82	568.18		
2 WS		29	0.64	946.88	92.81	4640.63	362.50	8046.88	278.13	22.81	240.63	82.50	U	7.23		218.75	171.88		

See notes to Appendices A3 and B1. All concentrations are lipid-normalized.

Appendix B4. UCL Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish and Crayfish, and TEC and Risk in Fish, 11/02, Neal's Landfill Site, Bloomington, IN.

Loc.	Spp.	N	PCB Congeners										Aroclor				Fish HQ ratio			
			77	81	105	114	118	123	126	156/157	167	169	189	4-peak	5-peak	N		Fish TEC		
			µg/kg lipid										mg/kg lipid		ng/kg lipid					
0	CRC	4	13.61	1.74	189.73	10.12	351.21	20.00	1.74	38.65	24.98	U	1.74	55.37	55.37	6	10.92	0.8		
0	WS	4	32.41	0.54	180.97	12.25	323.22	12.33	0.54	31.31	17.98	U	3.55	26.82	36.35	6	6.20	0.4		
0	CF		9.71	1.31	117.49	5.83	263.97	12.68	1.06	21.61	16.41	U	0.95	35.01	35.01					
0	Dietary		20.35	1.17	171.77	10.11	322.57	15.47	1.12	32.30	20.47	U	2.30	39.88	43.69					
1	CRC	5	7391.18	429.56	23781.06	1741.08	41966.45	1439.02	92.72	1247.62	436.64	U	32.45	983.94	811.95	9	1417.47	101		
1	CF		5269.68	323.49	14727.01	1004.05	31541.82	912.09	56.39	697.45	286.92	U	17.66	622.09	513.35					
1	Dietary		6966.68	408.35	21970.25	1593.67	39881.52	1333.64	85.45	1137.59	406.70	U	29.49	911.57	752.23					
2	CRC	4	803.37	52.73	4663.82	413.53	7939.89	279.69	18.61	290.73	108.99	U	11.24	204.91	164.29	6	199.75	14		
2	LS	6	976.33	49.65	3758.02	297.42	6682.36	256.25	14.06	216.82	74.38	U	8.10	108.22	86.70	6	192.73	14		
2	WS	5	2617.57	185.00	8663.42	673.79	14544.51	500.70	39.35	410.27	141.99	U	12.40	440.91	362.80	7	551.01	39		
2	CF		572.78	39.71	2888.18	238.48	5967.59	177.28	11.32	162.53	71.62	U	6.11	129.55	103.87					
2	Dietary		1287.16	84.58	5133.70	416.96	8971.32	311.89	21.47	277.26	101.09	U	9.69	226.99	184.45					
Fish TEFs			0.0001	0.0005	-----<0.000005-----										0.0005	<0.000005	0.00005	<0.000005	Fish TRV:	14.1
																			ng TEC/kg lipid	
CRC/CF																				
2003			1.40	1.33	1.61	1.73	1.33	1.58	1.64	1.79	1.52	na	1.84	1.58	1.58					
Location 0 WS UCL calculated excluding the sample ID 15 outlier																				
N - sample size: left side - congener, right side - Aroclor																				
See notes to Appendix A4. All concentrations are lipid-normalized.																				

See footnotes to Appendices B1 and B2. All concentrations are fresh weight (fw).

Appendix B8. Mean Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish and Crayfish, and TEC and Risk in Fish, 11/02, Neal's Landfill Site, Bloomington, IN.

Loc	Spp.	N	PCB Congener										Aroclor				Fish TEC ng/kg lipid	Fish HQ ratio
			77	81	105	114	118	123	126	156/157	167	169	189	4-peak mg/kg lipid	5-peak mg/kg lipid	N		
0	CRC	4	4.16	1.04	116.24	3.32	213.18	7.46	1.04	22.34	14.51	U	1.04	39.72	39.72	6	6.16	0.4
0	WS	4	18.03	0.33	100.97	6.70	178.22	7.63	0.33	17.95	10.62	U	1.11	30.48	32.67	6	3.61	0.3
0	CF		2.97	0.79	71.98	1.92	160.22	4.73	0.64	12.49	9.54	U	0.57	25.12	25.12			
0	Dietary		9.47	0.71	101.26	4.39	186.61	6.96	0.68	18.61	11.96	U	0.98	33.10	33.96			
1	CRC	5	4601.52	266.62	14460.11	1073.08	25573.00	889.58	57.41	767.76	274.33	U	20.36	676.68	558.78	9	880.53	62
1	CF		3280.74	200.79	8954.78	618.83	19220.57	563.84	34.92	429.20	180.26	U	11.06	427.83	353.29			
1	Dietary		4337.37	253.45	13359.05	982.23	24302.51	824.43	52.91	700.04	255.51	U	18.50	626.91	517.68			
2	CRC	4	481.45	27.82	2924.61	251.84	4999.47	174.38	11.65	191.12	71.98	U	4.18	109.59	89.16	6	120.29	9
2	LS	6	535.13	26.54	2068.86	164.73	3686.16	138.36	7.84	121.25	44.52	U	3.93	61.33	49.68	6	106.00	8
2	WS	5	1213.50	101.01	5169.28	413.03	8795.92	303.85	23.04	256.65	88.23	U	7.78	264.75	213.82	7	287.07	20
2	CF		343.26	20.95	1811.14	145.23	3757.59	110.53	7.06	106.84	47.30	U	2.28	69.29	56.37			
2	Dietary		663.34	45.62	3072.30	250.27	5413.26	186.53	12.76	173.11	64.05	U	4.69	130.04	105.32			
Fish	TEFs		0.0001	0.0005	-----	<0.000005	-----	0.005	<0.000005	0.00005	<0.000005						Fish TRV: ng TEC/kg lipid	14.1
CRC/CF																		
2003			1.40	1.33	1.61	1.73	1.33	1.58	1.64	1.79	1.52	na	1.84	1.58	1.58			

Location 0 WS mean calculated excluding the sample ID 15 outlier
See notes to Appendix A4. All concentrations are lipid-normalized.

Appendix B9. Modeled Mean Congener and TEC Concentrations in Kingfisher Eggs, and Estimated Risk, 11/02, Neal's Landfill Site, IN.																			
Reach	77	81	105	114	PCB Congeners					126	156/157	167	169	189	Avian		LOAEC HQ ratio		
					118	123	126	135	141						WHO-TEC	NOAEC HQ			
	0	8.43	10.46	806.19	63.26	4932.06				20.11	394.58		U		3.65	2	0.2	0.7	0.05
	1	3860.25	3751.13	106204.42	14144.14	635510.69				1573.67	14840.93		U		745.37	416	44	140	11
	2	590.37	675.18	24424.75	3603.94	141556.85				379.45	3669.89		U		139.57	78	8	26	2
	1&2	2225.31	2213.16	65314.59	8874.04	388533.77				976.56	9251.41		U		442.47	247	26	83	7
Diet-Egg																			
BMF	0.89	14.8	7.95	14.4	26.15	NA			29.74	21.2	NA	31.25	NA	TRV:	1.79	17	533	68	
Bird Egg																			
TEFs	0.05	0.1	0.0001	0.0001	0.00001	0.00001	0.1	0.0001	0.00001	0.0001	0.00001	0.001	0.00001	— µg/kg lipid	—	—	—	—	—

See notes to Appendix A9. All concentrations are lipid-normalized.

See notes to Appendix A9. All concentrations are lipid-normalized.

Appendix B10. Percent Allowable Consumption (Mean TEC Basis), 11/02, Neal's Landfill Site, IN.												
Reach	Mink		Kingfisher Dose		Kingfisher Egg		Kingfisher Range		Kingfisher Range		Kingfisher Range	
	NOAEC	LOAEC	NOAEL	LOAEL	NOAEC	LOAEC	NOAEL-NOAEC	LOAEL-LOAEC	lowest	highest	lowest	highest
1	8	35	4	44	0.2	0.2	9	0.2	4	0.2	44	44
2	40	100	19	100	1	1	46	1	19	1	100	100
1&2	21	86	6	73	0.4	0.4	15	0.4	6	0.4	73	73
Egg TRVs: 1.79 17 5.33 68												
TRV units ——— µg/kg lipid ———												

See footnotes to Appendices A10 and B6

Appendix C

**PCB and TEC Concentrations in Fish and Crayfish
Collected from Conard's Branch and Richland Creek, May 2003,
and Risk Estimates for Mink and Kingfisher**

Appendix C1. PCB Congener, Total PCB, and TEC Concentrations in Whole Fish Samples, 5/03, Neal's Landfill Site, Bloomington, IN.																
Loc.	Spp.	ID	PCB Congeners										Mammalian WHO-TEC	Total PCB		
			77	81	105	114	118	123	126	156/157	167	169			189	
			pg/g													
1	CRC	4	46200	2460	87200	9750	108000	8190	540	5350	2170	U	175	86.79	6700	
1	CRC	5	61700	3470	95000	12700	119000	12100	897	8290	3590	U	285	129.39	10800	
1	CRC	6	52000	3840	82000	14700	105000	14400	983	9020	3850	U	334	135.96	11000	
1	CRC	7	53400	4760	85200	19600	107000	19500	1340	12900	5540	U	461	177.34	12700	
1	CRC	8	53500	3280	81000	14900	101000	12700	928	9570	4140	U	343	130.26	9700	
1	CRC	9	47900	2470	75400	10300	95600	10500	685	6530	2780	U	236	100.15	8500	
1	CRC	10	45000	2130	74500	9380	95900	8730	536	6070	2470	U	212	84.00	7800	
1	CRC	11	58800	2990	91300	11800	108000	10900	830	7520	3430	U	285	119.92	10500	
1	CRC	12	39800	4060	64100	18600	87700	22200	969	8710	3740	U	360	132.41	11400	
1	CRC	TT1	88600	4610	181000	14600	328000	15300	1020	9880	3720	U	306	176.06	14500	
1	CF	1	21700	1120	55800	5170	93600	4370	309	2940	1230	U	116	52.64	2900	
1	CF	2	18300	1070	34500	4970	44400	4880	250	2410	978	U	88.6	39.02	2600	
1	CF	3	21400	1120	57000	5200	89300	4280	309	2880	1200	U	111	52.27	6000	
2	CRC	17	10800	547	32100	3360	40900	2870	165	1610	527	U	47	27.72	1700	
2	CRC	18	9390	426	25800	2240	35200	1940	126	1270	444	U	51.9	21.64	1400	
2	CRC	19	3560	187	9180	743	16700	747	42	430	187	U	12	7.83	470	
2	CRC	20	7580	376	27800	2300	41200	1930	132	1310	449	U	42.1	22.90	940	
2	CRC	21	8210	376	29200	2600	41500	2270	139	1520	564	U	57.8	24.13	1300	
2	CRC	22	5750	272	22200	1810	38200	1650	83.7	1070	414	U	43.8	16.63	830	
2	LS	42	3050	138	17200	1440	30600	1350	30	1010	362	U	34.4	9.47	460	
2	LS	43	7160	317	31400	2810	44300	2590	92.1	1590	623	U	62	20.00	1100	
2	LS	44	2860	135	23400	1800	39400	1900	38.6	1210	436	U	43.5	12.14	400	
2	LS	45	1290	54.5	5160	408	8390	376	12.2	212	70.2	U	12.2	3.06	150	
2	LS	46	9270	400	36700	3340	47300	2870	108	2070	766	U	77.7	23.17	1200	
2	LS	47	15200	685	38500	3540	49800	3680	180	1900	730	U	73.8	31.52	1900	

Appendix C2. UCL PCB and TEC Concentrations in Whole Fish and Reach-specific Risk to Mink and Kingfisher, 5/03, Neal's Landfill Site, Bloomington, IN.																							
Loc.	Spp.	N	Mink						Kingfisher														
			Species UCL			Dietary UCL			TEC HQ			PCB HQ			Dose			TEQ HQ			PCB HQ		
			TEC	PCB	pg/kg	TEC	PCB	pg/kg	NOAEC	LOAEC	no effect	low effect	no effect	low effect	TEQ	PCB	ng/kg-d	NOAEL	LOAEL	no effect	low effect		
1	CRC	10	150.38	12014.87	108.01	9036.03	23	6	18	15	133.78	11313.77	66.89	5656.89	48	5	14	51	11	5			
1	CF	3	67.38	8509.41																			
2	CRC	6	27.64	1572.49	22.57	1411.46	5	1	3	2	27.72	1727.86	31.86	863.93	10	1	2	8	2	0.8			
2	LS	7	29.62	1894.51																			
2	WS	7	40.38	2711.94																			
2	CF	3	8.40	400.70																			
1&2	Receptor																						
1&2	Fish		62.00	4548.45	43.93	3317.60	10	2	7	6	80.75	6520.82	40.37	3260.41	29	3	8	30	7	3			
1&2	CF		23.14	2427.88																			
1&2	Receptor																						
1&2			91.46	7037.25																			
1&2			37.89	4455.06																			
3V	SRH	3	15.91	915.34	12.57	723.11	3	0.7	1	1	15.91	915.34	7.95	457.67	6	0.6	1	4	0.9	0.4			

Appendix C3. Lipid-normalized PCB Congener and Total PCB Concentrations in Whole Fish Samples 5/03, Neal's Landfill Site, Bloomington, IN.																			
Loc.	Spp.	ID	Lipid Conc.	PCB Congeners												Total PCBs			
				77	81	105	114	118	123	126	156/157	167	169	189					
				_____ µg/kg lipid _____															
			%																
1	CRC	4	9.8	470.28	25.04	887.63	99.25	1099.35	83.37	5.50	54.46	22.09	U	1.78	68.20	mg/kg lipid			
1	CRC	5	6.8	904.44	50.87	1392.57	186.16	1744.38	177.37	13.15	121.52	52.62	U	4.18	158.31				
1	CRC	6	6.2	838.71	61.94	1322.58	237.10	1693.55	232.26	15.85	145.48	62.10	U	5.39	177.42				
1	CRC	7	5.7	937.07	83.53	1495.10	343.94	1877.65	342.19	23.51	226.37	97.22	U	8.09	222.88				
1	CRC	8	5.1	1043.41	63.97	1579.74	290.59	1969.80	247.69	18.10	186.64	80.74	U	6.69	189.18				
1	CRC	9	8.1	593.49	30.60	934.22	127.62	1184.50	130.10	8.49	80.91	34.44	U	2.92	105.32				
1	CRC	10	6.4	703.12	33.28	1164.06	146.56	1498.44	136.41	8.38	94.84	38.59	U	3.31	121.88				
1	CRC	11	6.4	918.75	46.72	1426.56	184.38	1687.50	170.31	12.97	117.50	53.59	U	4.45	164.06				
1	CRC	12	3.8	1047.37	106.84	1686.84	489.47	2307.89	584.21	25.50	229.21	98.42	U	9.47	300.00				
1	CRC	TT1	8.4	1054.76	54.88	2154.76	173.81	3904.76	182.14	12.14	117.62	44.29	U	3.64	172.62				
1	CF	1	1.7	1288.75	66.52	3313.92	307.04	5558.84	259.53	18.35	174.60	73.05	U	6.89	172.23				
1	CF	2	1.6	1147.13	67.07	2162.63	311.54	2783.21	305.90	15.67	151.07	61.31	U	5.55	162.98				
1	CF	3	1.5	1444.40	75.59	3847.23	350.98	6027.32	288.88	20.86	194.39	80.99	U	7.49	404.97				
2	CRC	17	4.1	261.39	13.24	776.91	81.32	989.89	69.46	3.99	38.97	12.75	U	1.14	41.14				
2	CRC	18	4.4	214.54	9.73	589.47	51.18	804.24	44.32	2.88	29.02	10.14	U	1.19	31.99				
2	CRC	19	3.5	100.31	5.27	258.66	20.93	470.54	21.05	1.18	12.12	5.27	U	0.34	13.24				
2	CRC	20	1.4	535.06	26.54	1962.35	162.35	2908.24	136.24	9.32	92.47	31.69	U	2.97	66.35				
2	CRC	21	3.5	237.61	10.88	845.10	75.25	1201.08	65.70	4.02	43.99	16.32	U	1.67	37.62				
2	CRC	22	2.8	203.04	9.60	783.91	63.91	1348.89	58.26	2.96	37.78	14.62	U	1.55	29.31				
2	LS	42	1.0	315.01	14.25	1776.44	148.73	3160.41	139.43	3.10	104.31	37.39	U	3.55	47.51				
2	LS	43	1.7	430.76	19.07	1889.08	169.05	2665.17	155.82	5.54	95.66	37.48	U	3.73	66.18				
2	LS	44	1.0	293.40	13.85	2400.52	184.66	4041.90	194.91	3.96	124.13	44.73	U	4.46	41.03				
2	LS	45	0.5	247.18	10.44	988.71	78.18	1607.61	72.05	2.34	40.62	13.45	U	2.34	28.74				
2	LS	46	1.2	750.64	32.39	2971.78	270.46	3830.11	232.40	8.75	167.62	62.03	U	6.29	97.17				
2	LS	47	4.4	346.63	15.62	877.98	80.73	1135.68	83.92	4.10	43.33	16.65	U	1.68	43.33				

Appendix C4. UCL Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish and Crayfish, and TEC and Risk in Fish, 503, Neal's Landfill Site, Bloomington, IN.

Loc.	Spp.	N	PCB Congener													Total PCBs mg/kg lipid	Fish TEC ng/kg lipid	Fish HQ ratio
			77	81	105	114	118	123	126	156/157	167	169	189					
1 CRC	10	995.25	73.73	1669.40	312.42	2461.92	331.78	19.01	179.95	77.13	U	6.73	213.93	231.46	16			
1 CF	3	1662.79	82.36	5246.73	383.24	9145.64	343.04	24.74	227.23	96.38	U	9.11	587.35					
1 Diet		1128.76	75.46	2384.86	326.59	3798.66	334.03	20.16	189.40	80.98	U	7.20	288.61					
2 CRC	6	412.15	20.24	1474.51	125.67	2181.03	106.47	6.97	70.70	24.56	U	2.39	54.95	86.20	6			
2 LS	7	572.79	26.30	2457.72	211.35	3690.66	194.66	6.59	134.02	49.35	U	4.93	78.99	103.36	7			
2 WS	7	355.78	27.18	1427.61	136.55	2108.89	117.53	6.54	70.91	25.13	U	2.31	69.79	81.88	6			
2 CF	3	295.05	16.54	777.98	67.92	1411.40	70.77	3.99	37.62	16.57	U	1.24	36.06					
2 Diet		416.54	22.97	1584.88	139.87	2410.43	125.80	6.16	81.03	29.72	U	2.81	61.54					
1&2 Mean		772.65	49.21	1984.87	233.23	3104.55	229.91	13.16	135.22	55.35	U	5.01	175.08					
3V SRH	3	143.56	7.78	464.02	35.64	982.33	36.34	1.77	22.60	10.70	U	1.19	20.04	27.12	2			
Fish TEFs		0.0001	0.0005	—————<0.000005				0.005	<0.000005	0.000005	0.00005	<0.000005	Fish TRV:	14.1	ng TEC/kg lipid			

CF lipid-normalized concentrations are calculated from site-specific measurements, not estimated.
Symbols and definitions as in Appendix C1. All concentrations are lipid-normalized (lw).

CF lipid-normalized concentrations are calculated from site-specific measurements, not estimated.

Symbols and definitions as in Appendix C1. All concentrations are lipid-normalized (lw).

Appendix C5. Modeled UCL Congener and TEC Concentrations in Kingfisher Eggs, and Estimated Risk, 503, Neal's Landfill Site, IN.

Reach		PCB Congener										Avian		Hazard Quotient			
		77	81	105	114	118	123	126	156/157	167	169	189	WHO-TEC µg/kg lipid	NOAEC	LOAEC	ratio	—
	1	1004.59	1116.74	18959.68	4702.85	99334.96		599.54	4015.35		U		225.62	126	13	42	3
	2	370.72	339.89	12599.83	2014.15	63032.83		183.14	1717.77		U		73.10	41	4	14	1
	1&2	687.66	728.31	15779.75	3358.50	81183.90		391.34	2866.56		U		149.36	83	9	28	2
	3V	127.77	115.21	3688.92	513.27	25687.91		52.79	479.14		U		23.91	13	1	4	0.4
Diet-Egg BMF		0.89	14.8	7.95	14.4	26.15	NA	29.74	21.2	NA	31.25	NA	TRV: 1.79	17	5.33	68	
Bird Egg TEFs		0.05	0.1	0.0001	0.0001	0.00001		0.1	0.0001	0.00001	0.001	0.00001	— µg/kg lipid	—	—	—	—

Symbols and definitions as in Appendices A5 and C1. All concentrations are lipid-normalized (lw).

Appendix C6. Percent Allowable Consumption (UCL TEC Basis), 503, Neal's Landfill Site, IN.

Reach	Mink	Kingfisher				Egg				Kingfisher Range			
		NOAEC	LOAEC	NOAEL	LOAEL	NOAEC	NOAEL	LOAEC	LOAEL	NOAEL-NOAEC	LOAEL-LOAEC	lowest	highest
	1	4	16	2	21	0.8	5	2	27	0.8	5	2	27
	2	20	78	8	99	2	15	7	85	2	15	7	99
	1&2	10	40	3	34	1	7	4	41	1	7	4	41
	3V	33	100	15	100	7	45	22	100	7	45	22	100
Egg TRVs													
TRV units													
1.79													
5.33													
68													
— µg/kg lipid													

See footnotes to Appendix A6

Appendix C7. Mean PCB and TEC Concentrations in Whole Fish and Reach-specific Risk to Mink and Kingfisher, 503, Neal's Landfill Site, Bloomington, IN.																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																								
Loc.	Spp.	N	Species TEC						Species PCB				Mink						Kingfisher																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																					
			Mean		SD		pg/g		Mean		SD		µg/kg		TEC		PCB		TEC HQ		PCB HQ		Mean Dose		TEQ HQ		PCB HQ																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																													
			Mean	SD	pg/g	Mean	SD	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC	PCB	µg/kg	TEC	PCB	pg/g	TEC

Appendix C8. Mean Lipid-normalized PCB Congener and Aroclor Concentrations in Whole Fish and Crayfish, 503, Neal's Landfill Site, Bloomington, IN.																
Loc.	Spp.	N	77	81	PCB Congener								Total PCBs	Fish TEC	Fish HQ	
					105	114	118	123	126	156/157	167	169				189
_____µg/kg lipid_____																
1 CRC	10	851.14	55.77	1404.41	227.89	1896.78	228.60	14.36	137.46	58.41	U	4.99	167.98	184.79	13	
1 CF	3	1293.43	69.73	3107.93	323.19	4789.79	284.77	18.29	173.35	71.78	U	6.65	246.73			
1 Diet		939.60	58.56	1745.11	246.95	2475.38	239.84	15.15	144.64	61.09	U	5.32	183.73			
1 CRC/CF		0.66	0.80	0.45	0.71	0.40	0.80	0.78	0.79	0.81	na	0.75	0.68			
2 CRC	6	258.66	12.54	869.40	75.83	1287.15	65.84	4.06	42.39	15.13	U	1.48	36.61	52.43	4	
2 LS	7	413.02	18.96	1765.38	148.18	2689.66	139.57	4.66	91.60	33.34	U	3.43	57.07	74.06	5	
2 WS	7	255.19	17.31	916.63	84.18	1364.36	71.40	4.02	42.67	15.29	U	1.41	46.85	54.26	4	
2 CF	3	120.47	6.76	312.99	27.44	568.28	27.98	1.62	15.22	6.79	U	0.50	14.75			
2 Diet		291.38	15.86	1135.40	98.43	1735.26	89.99	3.79	56.75	20.81	U	2.04	44.52			
2 CRC/CF		2.15	1.86	2.78	2.76	2.26	2.35	2.50	2.78	2.23	na	2.92	2.48			
2 LS/CF		3.43	2.80	5.64	5.40	4.73	4.99	2.87	6.02	4.91	na	6.80	3.87			
182 Mean		615.49	37.21	1440.26	172.69	2105.32	164.91	9.47	100.69	40.95	U	3.68	114.13			
3V SRH	3	67.34	3.62	219.36	16.38	452.48	17.09	0.73	10.33	4.90	U	0.55	10.07	12.22	0.9	
Fish TEFs		0.0001	0.0005	_____	<0.000005	_____	0.005	<0.000005	0.00005	<0.000005	<0.000005			Fish TRV:	14.1	
Mean CRC/CF		1.40	1.33	1.61	1.73	1.33	1.58	1.64	1.79	1.52	na	1.84	1.58	ng TEC/kg lipid		
Symbols and definitions as in Appendix C4. All concentrations are lipid-normalized (lw).																

Symbols and definitions as in Appendix C4. All concentrations are lipid-normalized (lw).

Appendix C9. Modeled Mean Congener and TEC Concentrations in Kingfisher Eggs, and Estimated Risk, 5/03, Neal's Landfill Site, IN.

Reach	77	81	105	114	PCB Congener					167	169	189	Avian WHO-TEC µg/kg lipid	NOAEC HQ	LOAEC HQ
					118	123	126	156/157	µg/kg lipid						
1	836.24	866.67	13873.63	3556.06	64731.29	450.43	3066.27			U			176.22	98	33
2	259.33	234.70	9026.46	1417.41	45377.04	112.81	1203.18			U			49.34	28	9
1&2	547.79	550.68	11450.04	2486.74	55054.17	281.62	2134.72			U			112.78	63	21
3V	59.94	53.62	1743.92	235.87	11832.45	21.84	219.00			U			10.88	6	2
Diet-Egg															
BMF	0.89	14.8	7.95	14.4	26.15	NA	29.74	21.2	NA	31.25	NA	TRV:		1.79	5.33
Bird Egg															
TEFs	0.05	0.1	—0.0001	—	0.00001	0.1	0.0001	0.00001	0.0001	0.0001	0.00001			—	µg/kg lipid

Symbols and definitions as in Appendices A9 and C1. All concentrations are lipid-normalized (lw).

Appendix C10. Percent Allowable Consumption (Mean TEC Basis), 5/03, Neal's Landfill Site, IN.

Reach	Mink		Kingfisher Dose		Kingfisher Egg		Kingfisher Range	
	NOAEC	LOAEC	NOAEL	LOAEL	NOAEC	LOAEC	NOAEL-NOAEC lowest	LOAEL-LOAEC highest
1	5	20	2	25	1	8	1	0.9
2	26	100	12	100	4	27	4	3
1&2	12	51	7	81	2	12	2	1
3V	76	100	34	100	16	100	16	15
Egg TRVs: 1.79 17 5.33 68								
TRV units — µg/kg lipid —								

See footnotes to Appendix A10

Appendix D

Comparison of Historic Data on PCB Concentrations in Fish and Crayfish in Conard's Branch and Richland Creek

Appendix D1 Fish-crayfish PCB Concentration Ratios, Neal's Landfill, Bloomington, IN							
Date	Species	Location	PCB Conc	Species Mean PCB Conc.	Fish-Crayfish PCB Conc. ratio by Location and Date	Mean Fish-Crayfish PCB ratio	
		stream miles	ppm, ww	ppm	ratio	ratio	number of comparisons
8/82	crayfish	0.4	56	52.5		creek chub / crayfish	
8/82	crayfish	0.4	49			2.65	4
8/82	creek chub	0.4	125	108	2.06	longear and green sunfish / crayfish	
8/82	creek chub	0.4	91			3.32	4
8/82	darter	0.4	279	279	5.31	stoneroller / crayfish	
5/98	crayfish	0.5	2.4	3.57		5.36	2
5/98	crayfish	0.5	3.6			white sucker / crayfish	
5/98	crayfish	0.5	4.7			1.95	2
5/98	longear sunfish	0.5	16	16	4.49	rock bass / crayfish	
5/98	white sucker	0.5	1.4	1.4	0.39	4.11	2
5/98	creek chub	0.5	20	14.09	3.95	Mean creek chub, green sunfish, and longear sunfish / crayfish ratios	
5/98	creek chub	0.5	1.7				
5/98	creek chub	0.5	25			2.99	3
5/98	creek chub	0.5	11				
5/98	creek chub	0.5	12				
5/98	creek chub	0.5	20				
5/98	creek chub	0.5	8.9				
5/98	stoneroller	0.5	22	17	4.77		
5/98	stoneroller	0.5	13				
5/98	stoneroller	0.5	16				
8/82	crayfish	1	2.56	2.39			
8/82	crayfish	1	2.21				
8/82	creek chub	1	4.35	6.21	2.60		
8/82	creek chub	1	8.06				
8/82	longear sunfish	1	3.46	6.28	2.63		
8/82	longear sunfish	1	9.09				
8/82	rock bass	1	5.49	12.05	5.05		
8/82	rock bass	1	18.6				
5/98	crayfish	1	0.17	0.2			
5/98	crayfish	1	0.2				

Appendix D1 Fish-crayfish PCB Concentration Ratios, Neal's Landfill, Bloomington, IN							
Date	Species	Location	PCB Conc	Species Mean PCB Conc.	Fish-Crayfish PCB Conc. ratio by Location and Date	Mean Fish-Crayfish PCB ratio	
		stream miles	ppm, ww	ppm	ratio	ratio	number of comparisons
5/98	crayfish	1	0.18	0.41	2.00		
5/98	crayfish	1	0.26				
5/98	creek chub	1	0.32				
5/98	creek chub	1	0.43				
5/98	creek chub	1	0.29				
5/98	creek chub	1	0.5				
5/98	creek chub	1	0.49				
5/98	stoneroller	1	0.91	1.21	5.95		
5/98	stoneroller	1	1.5				
5/98	green sunfish	1	0.58	0.58	2.86		
5/98	longear sunfish	1	0.98	0.67	3.28		
5/98	longear sunfish	1	0.35				
5/98	white sucker	1	0.96	0.71	3.51		
5/98	white sucker	1	0.58				
5/98	white sucker	1	0.59				
5/98	rock bass	1	1.4	0.64	3.17		
5/98	rock bass	1	0.5				
5/98	rock bass	1	0.39				
5/98	rock bass	1	0.3				
5/98	rock bass	1	0.41				
5/98	rock bass	1	0.85				

Appendix E

Comparison of PCB Toxicity Reference Values in Recent Ecological Risk Assessments

Appendix E1. Mink PCB Toxicity Reference Values (TRV) in Recent Ecological Risk Assessments										
Site or Application	NOAEL--NOAEC ^a		LOAEL--LOAEC ^b			Endpoint	UF ^d	Contam Source ^e	Exposure Duration	Reference
	Dose	Diet ^c	Dose	Diet ^c	number of breeding seasons or generations					
	mg/kg _{BW} -d	µg/kg	mg/kg _{BW} -d	µg/kg						
Fox River, WI	0.05	250	0.1		500 - 700	kit survival	none	field	1 (NOAEL); 2 (LOAEL)	Heaton et al 1995; Restum et al 1998
Great Lakes Initiative (GLI) Water Quality Criteria	0.03	200	0.3 ^f		2000 ^f	whelping rate; kit production and BW ^g	0.1 (LOAEL-to-NOAEL)	product (A1254)	1	Aulerich and Ringer 1977
Housatonic River, MA	0.17	1600	0.41		3700	kit survival	none	field	1	Bursian et al 2006a
(Confidence interval)	LD ₁₀ 0.02 ^{hi} (0.003-0.05)	LC ₁₀ 230 ^j (30-480)	LD ₂₀ 0.11 ^{ki} (0.05-0.21)		LC ₂₀ 980 ^l (470-1940)					
	0.004	25	0.04		250	kit BW	none	field	2	Restum et al 1998
Kalamazoo River, WI	ED ₁₀ 0.08 ^{mn}	EC ₁₀ 500 ^o	ED ₂₅ 0.1 ^{pn}		EC ₂₅ 600 ^q	live kit production and BW	0.52 (single-to-multiple season or generation exposure) ^r	product (A1254)	1	Aulerich and Ringer 1977; Kihström et al 1992; Wren, et al 1987
Sheboygan River and Harbor, WI, aquatic ERA	0.004	15	0.146		720	gestation duration; kit BW and survival	none	field	1	Heaton et al 1995
Upper Green Bay, WI	0.004	15	0.134		720	kit survival	none	field	1	Heaton et al 1995

a) NOAEL – no observed adverse effect level (dose); NOAEC – no observed adverse effect concentration (diet).

b) LOAEL – lowest observed adverse effect level (dose); LOAEC – lowest observed adverse effect concentration (diet).

c) Dietary concentration on a fresh-weight (fw) basis.

d) Uncertainty factor expressed as a multiplier.

e) Contaminant source of the study used for TRVs: product – Aroclor added to feed; field – field-contaminated prey.

f) The GLI calculated water quality criteria solely on a NOAEL basis, so did not evaluate the appropriateness of using the LOAEL by itself for decision making. In this case, the LOAEL served as a starting point for calculating the NOAEL.

g) BW – bodyweight

- h) LD₁₀ - lethal dose to 10 % of kits (0 to 6 weeks) (Bursian, et al. 2006a) and the 95% confidence interval (in parentheses).
- i) Mean normalized food ingestion rate of 0.107 kg food/kg_{BW}-d (Bursian, et al. 2006a).
- j) LC₁₀ - lethal concentration to 10 % of kits (0 to 6 weeks) and the 95% confidence interval (in parentheses), probit analysis by Bursian, et al. (2006a).
- k) LD₂₀ - lethal dose to 20 % of kits (0 to 6 weeks) (Bursian, et al. 2006) and the 95% confidence interval (in parentheses).
- l) LC₂₀ - lethal concentration to 20 % of kits (0 to 6 weeks) and the 95% confidence interval (in parentheses), probit analysis by Bursian, et al. (2006a).
- m) ED₁₀ - 10 % effective dose, the dose associated with a 10 % decrease in live kit production and kit bodyweight.
- n) Farm-raised female mink food ingestion rate of 0.16 kg/kg_{BW}-d (Bleavins and Aulerich 1981 as cited in USEPA 1993).
- o) EC₁₀ - 10 % effective concentration, the dietary concentration associated with a 10 % decrease in live kit production and kit bodyweight based on combined exposure-response data, adjusted for continuous exposure over multiple breeding seasons or generations (FERA Addendum; Appendix D of the Kalamazoo ERA).
- p) ED₂₅ - 25 % effective dose, the dose associated with a 25 % decrease in live kit production and kit bodyweight.
- q) EC₂₅ - 25 % effective concentration, the dietary concentration associated with a 25 % decrease in live kit production and kit bodyweight based on combined exposure-response data, adjusted for continuous exposure over multiple breeding seasons or generations (FERA Addendum; Appendix D of the Kalamazoo ERA).
- r) Based on the mean difference in interpolated EC₂₅ for continuous exposures over 1 versus 2 breeding seasons and 1 versus 2 generations of females in mink feeding studies with Clophen A50 (Brunström, et al. 2001; Kihström, et al. 1992) and field-contaminated prey (Restum, et al. 1998).

Appendix E2. Mink Dioxin Toxic Equivalent Concentration (TEC) Toxicity Reference Values (TRVs) in Recent Ecological Risk Assessments											
Site or Application	NOAEL/NOAEC		LOAEL/LOAEC		Endpoint	Test Species	UF	Contam Source	TEF/TEC ^a	Exposure Duration	Reference
	Dose	Diet	Dose	Diet						no. breeding seasons or generations	
	µg/kg _{BW} -d	pg/g ww	µg/kg _{BW} -d	pg/g ww							
Single-season exposure TRV	0.001 ¹	7.0 ^m	0.004 ¹	28 ⁿ	kit BW, survival, and abnormality	mink	none	field (Heaton, Restum, Bursian); Clophen A50 (Brunström)	Van den Berg et al 1998 (Brunström, Bursian); recalculated with TEFs from Van den Berg et al 1998 (Heaton, Restum)	1	Heaton et al 1995; Restum et al 1996; Brunström et al 2001; Bursian, et al. 2006a
Multiple-season or generation exposure TRV	0.0008 ¹	4.6 ^o	0.003 ¹	18 ^p	whelping rate, live kits/mated female, kit BW and survival	mink	none	field (Restum); Clophen A50 (Brunström)	Berg et al 1998 (Heaton, Restum)	2	Restum et al 1996; Brunström et al 2001

a) TEF – dioxin toxic equivalency factor; TEC – dioxin toxic equivalent concentration .

b) Mink food ingestion: $0.22 \text{ kg/kg}_{\text{BW}}\text{-d} = (0.159 + 0.0177 \text{ kg/d}) / 0.8 \text{ kg}_{\text{BW}}$ (USEPA 1995)

c) The GLI calculated water quality criteria solely on a NOAEL basis, so did not evaluate the appropriateness of using the LOAEL by itself for decision making.

d) Interspecific extrapolation of toxicity data between species.

e) TCDD – 2,3,7,8-tetrachlorinated dibenzo-*p*-dioxin.

f) LD₁₀ - lethal dose to 10 % of kits (0 to 6 weeks) (Bursian, et al. 2006a) and the 95% confidence interval (in parentheses).

g) Mean normalized food ingestion rate of 0.107 kg food/kg_{BW}-d (Bursian, et al. 2006a).

h) LC₁₀ - lethal concentration to 10 % of kits (0 to 6 weeks) and the 95% confidence interval (in parentheses), probit analysis by Bursian, et al. (2006a).

i) LD₂₀ - lethal dose to 20 % of kits 6 weeks after whelping (Bursian, et al. 2006) and the 95% confidence interval (in parentheses).

j) LC₂₀ - lethal concentration to 20 % of kits 6 weeks after whelping and the 95% confidence interval (in parentheses), probit analysis by Bursian, et al. (2006a).

k) The recalculated TEC is identical to the value obtained with the mammalian WHO-TEFs (Van den Berg, et al. 1998).

l) Farm-raised female mink food ingestion rate of 0.16 kg/kg_{BW}-d (Bleavins and Aulerich 1981 as cited in USEPA 1993)

m) Geometric mean NOAEC–1 breeding season or generation exposure: 3.2 pg/g (Brunström, et al. 2001), 0.89 pg/g (Heaton, et al. 1995; Restum, et al. 1998), and 16.1 pg/g (Bursian, et al. 2006a). Brunström, et al. (2001) dietary concentration calculated by dividing the reported TEC/mink/d by the reported daily feed consumption of 130 g/d. Restum, et al. (1998) TEC is calculated from the following regression of total PCB (mg/kg) and TEC (pg/g) (data from Tillitt, et al. 1996 recalculated with mammalian WHO-TEFs): $\text{TEC} = (32.594 * \text{PCB}) - 1.577$ $r^2 = 0.99$, $p = 0.005$, for a PCB range 0.015–2.56 mg/kg. The Restum, et al. (1998) and Heaton, et al. (1995) studies were performed with the same collection of field-

contaminated prey (homogenized and frozen in large batches for multiple feeding studies), so the PCB-TEC regression for the feed used by Heaton, et al. (1995), as reported by Tillitt, et al. (1996), applies to the Restum, et al. (1998) treatments as well.

n) Geometric mean LOAEC-1 breeding season or generation exposure: 64.6 pg/g (Brunström, et al. 2001), 22 pg/g (Heaton, et al. 1995), 6.6 pg/g (Restum, et al. 1998), and 68.5 pg/g (Bursian, et al. 2006a).

o) Geometric mean NOAEC-2 breeding seasons or generations exposure: 3.2 pg/g (Brunström, et al. 2001) and 6.6 pg/g (Restum, et al. 1998).

p) Geometric mean LOAEC-2 breeding seasons or generations exposure: 22.3 pg/g (Brunström, et al. 2001) and 14.7 pg/g (Restum, et al. 1998).

Other definitions as in Appendix E1.

Appendix E3. Avian PCB Dose Toxicity Reference Values (TRVs) ($\mu\text{g}/\text{kg}_{\text{BW}}\text{-d}$) in Recent Ecological Risk Assessments								
Site or Application - Receptor	NOAEL	LOAEL	Endpoint	Contaminant Source	Test Species	UF	Reference	
Great Lakes Initiative (GLI) Water Quality Criteria - belted kingfisher, bald eagle, herring gull	200	600 ^a	hatchability	product (A1254)	pheasant	0.33 (inter-specific); 0.33 (LOAEL-to-NOAEL)	Dahlgren et al 1972	
Fox River, WI - piscivorous and carnivorous birds	110	1120	courtship and nesting behaviors	product (A1254)	ring dove; mourning dove	0.1 (LOAEL-to-NOAEL)	Peakall and Peakall 1973; Tori and Peterle 1983	
Hudson River, NY - belted kingfisher, great blue heron, bald eagle	1800	7100	egg production	product (A1254)	pheasant	none	Dahlgren et al 1972	
Kalamazoo River, MI	ED ₁₀ 400 ^b	ED ₂₅ 500 ^c	hatchability	product (A1248)	chicken	none	Lillie et al 1974, 1975; Cecil et al 1974; Scott 1977	
Sheboygan River and Harbor, WI, aquatic ERA - great blue heron	46	400	hatchability; deformity	field	chicken	none	Summer et al 1996	
Sheboygan River and Harbor, WI, terrestrial ERA - robin	42	360	hatchability; deformity	field	chicken	none	Summer et al 1996	
Upper Green Bay, WI - caspian tern, double-crested cormorant	110	1120	courtship and nesting behaviors	product (A1254)	ring dove; mourning dove	0.1 (LOAEL-to-NOAEL)	Peakall and Peakall 1973; Tori and Peterle 1983	

a) The GLI calculated water quality criteria solely on a NOAEL basis, so did not evaluate the appropriateness of using the LOAEL by itself for decision making. In this case, the LOAEL served as a starting point for calculating the NOAEL.

b) ED₁₀ - 10 % effective dose (the dose associated with a 10 % decrease in hatchability based on combined dose-response data) (FERA Addendum; Appendix D of the Kalamazoo ERA).

c) ED₂₅ - 25 % effective dose (the dose associated with a 25 % decrease in hatchability based on combined dose-response data) (FERA Addendum; Appendix D of the Kalamazoo ERA).

Other definitions as in Appendix E1

Appendix E4. Avian Dioxin Toxic Equivalent Concentration (TEC) Dose Toxicity Reference Values (TRVs) (ng/kg _{BW} -d) in Recent Ecological Risk Assessments									
Site or Application - Receptor	NOAEL	LOAEL	Endpoint	Contam Source	Test Species	UF	TEF/TEC	Reference	
GLI Water Quality Criteria - belted kingfisher, bald eagle, herring gull	1.4	14	fertility; embryo mortality	TCDD	pheasant	0.1 (subchronic-to-chronic)	1 (by definition)	Nosek et al 1992a	
Hudson River, NY - belted kingfisher, great blue heron, bald eagle	1.4	14	fertility; embryo mortality	TCDD	pheasant	0.1 (subchronic-to-chronic)	1 (by definition)	Nosek et al 1992a	
Sheboygan River and Harbor, WI, aquatic ERA - great blue heron	2.9	28	hatchability; deformity	field	chicken	none	recalculated with TEFs from Kennedy et al 1996	Summer et al 1996	
Sheboygan River and Harbor, WI, terrestrial ERA - robin	1.44	323	hatchability; deformity	field	chicken	none	HII4E bioassay ^a	Summer et al 1996	
Revised Summer, et al. (1996) TRVs	14	140	hen and embryo mortality	TCDD	pheasant	none	1 (by definition)	Nosek et al 1992a	
	1.4	12	hatchability; deformity	field	chicken	none	recalculated with mammalian TEFs from Van den Berg et al. 1998 ^b	Summer et al 1996	

a) TEC as measured by the H4IIE rat hepatoma cell line bioassay (Tillitt, et al. 1996).

b) TEC is calculated from the following regression of total PCB (mg/kg) and TEC (pg/g) (data from Tillitt, et al. 1996 recalculated with mammalian WHO-TEFs): $TEC = (32.594 * PCB) - 1.577$ $r^2 = 0.99$, $p = 0.005$, for a PCB range 0.015–2.56 mg/kg. The Heaton, et al. (1995) and Summer, et al. (1996) studies were performed with the same collection of field-contaminated prey (homogenized and frozen in large batches for multiple feeding studies), so the PCB-TEC regression for the feed used by Heaton, et al. (1995), as reported by Tillitt, et al. (1996), applies to the Summer, et al. (1996) treatments as well. The avian TEFs reported by Van den Berg, et al. (1998) are not used for recalculating the Summer, et al. (1996) TECs because the avian TEFs are based on egg studies, which means that congeners should be modeled or measured in eggs to apply the avian TEFs. Summer, et al. (1996) did not report egg TECs, only the dietary TECs as determined by the HII4E bioassay performed with a rat hepatoma cell line. Since Summer, et al. (1996) originally reported the dietary TECs on a mammalian basis, and because ingestion-based avian TEFs are unavailable, the dietary TEC is recalculated with the updated mammalian TEFs. Dietary TEC is converted to dose by converting to $\mu\text{g/kg}$ and multiplying by the mean daily food consumption over the exposure period (weeks 3–10): 0.0548 kg/kg-d (high-dose treatment) and 0.0553 kg/kg-

d (low-dose treatment). An additional uncertainty for the high-dose treatment is that the dietary PCB concentration (6.6 mg/kg) is greater than the highest PCB concentration (2.56 mg/kg) reported by Tillitt, et al. (1996) in the data used to develop the PCB-TEC regression.

Other definitions as in Appendices E1 and E2

Appendix E5. Avian PCB Egg Toxicity Reference Values (TRVs) in Recent Ecological Risk Assessments										
Site or Application - Receptor	NOAEC		LOAEC		Endpoint	Contam Source	Test Species	UF	Reference	
	mg/kg	mg/kg lipid	mg/kg	mg/kg lipid						
Fox River, WI - all birds	4.7	49 ^a	7.6	80 ^a	hatchability	field	common tern	none	Hoffman et al 1993	
Fox River, WI - all birds	0.8	14 ^b	8	136 ^b	deformity	field	double-crested cormorant	10 (NOAEC-to-LOAEC)	Ludwig et al 1996	
Hudson River, NY - belted kingfisher	4.7	49 ^a	7.6	80 ^a	hatchability	field	common tern	none	Hoffman et al 1993	
Hudson River, NY - great blue heron	2	32 ^c	7.6	80 ^a	hatchability	field	GBH (NOAEC); common tern (LOAEC)	none	Halbrook et al 1999 (NOAEC); Hoffman et al 1993 (LOAEC)	
Hudson River, NY - bald eagle	5.5	73 ^d	8.7	116 ^d	nest success	field	bald eagle	none	Wiemeyer et al 1993	
Kalamazoo River, MI - general	1	8.9 ^e	1.5	13.4 ^e	hatchability	product (A1242)	chicken	none	Britton and Huston 1973	
Kalamazoo River, MI - great blue heron	5.8 ^f	61 ^a	20.6 ^g	217 ^a	hatchability, population size or reproductive success	field	Forster's tern	none	Barron et al 1995	
Kalamazoo River, MI - bald eagle	1.5	20 ^d	7.7 ^h	103 ^d	hatchability, nesting success, population size or reproductive success	field	bald eagle	0.2 (LOAEC-to-NOAEC) ⁱ	Wiemeyer et al 1984; various secondary sources ^h	

Appendix E5. Avian PCB Egg Toxicity Reference Values (TRVs) in Recent Ecological Risk Assessments									
Site or Application - Receptor	NOAEC		LOAEC		Endpoint	Contam Source	Test Species	UF	Reference
	mg/kg	mg/kg lipid	mg/kg	mg/kg lipid					
Kalamazoo River, MI - robin	2.8 ^j	25 ^e	6.2 ^k	55 ^e	hatchability, egg production, fertility, deformity	product (A1242, A1248, A1254) (Barron); field (Summer)	chicken	none	Barron et al 1995; Summer et al 1996
Sheboygan River and Harbor, WI, terrestrial ERA - robin	5	45 ^e	24	214 ^e	hatchability; deformity	field	chicken	none	Summer et al 1996
Upper Green Bay, WI - caspian tern, double-crested cormorant	4.7	49 ^a	7.6	80 ^a	hatchability; deformity	field	common tern	none	Hoffman et al 1993
USEPA Region 5 proposed	0.7	6 ^e	1.3	12 ^e	hatchability	product (A1248)	chicken	none	Lillie et al 1974; Cecil et al 1974; Scott 1977

a) Tern egg lipid content of 9.5 % (semi-precocial) (Carey, et al. 1980)

b) Cormorant egg lipid content of 5.9 % (altricial) (Carey, et al. 1980)

c) Heron egg lipid content of 6.3 % (semi-altricial) (Carey, et al. 1980)

d) Bald eagle egg lipid content of 7.5 % (Blankenship and Giesy 2002)

e) Chicken egg fat content of 11.2 % (5.6 g fat in 50 g egg - Pennington and Church 1985)

f) Arithmetic mean NOAEC: 4.5 mg/kg (Kubiak, et al. 1989 as cited in Barron, et al. 1995) and 7.0 mg/kg (Bosveld and Van den Berg, in press as cited in Barron, et al. 1995). Note: the article as published by Bosveld and Van den Berg (1994) show an egg PCB NOAEC of 2.3 mg/kg for Forster's tern hatching success based on King, et al. (1991).

g) Arithmetic mean LOAEC: 22.2 mg/kg (Kubiak, et al. 1989 as cited in Barron, et al. 1995) and 19 mg/kg (Bosveld and Van den Berg, in press as cited in Barron, et al. 1995). Note: the published article by Bosveld and Van den Berg (1994) show an egg PCB LOAEC of 19.2 mg/kg for Forster's tern hatching success based on Kubiak, et al. (1989).

h) Arithmetic mean LOAEC: 4.0 mg/kg (Wiemeyer, et al. 1984 as derived by Ludwig, et al. 1993 as cited by Barron, et al. 1995), 4.5 mg/kg (40 % decrement in productivity, Wiemeyer, et al. 1984) 13 mg/kg (unsuccessful nests, Wiemeyer, et al. 1984), 13 mg/kg (reproductive success, Wiemeyer, et al. 1984 as cited by Bosveld and Van den Berg 1994). Note: all of the values are estimates of the LOAEC of the same study.

i) Table 4-9 of the Kalamazoo ERA mistakenly states that the NOAEC is "est. from mean LOAEC/10", but the actual divisor was 5.

j) Arithmetic mean NOAEC: 0.36 mg/kg (Scott 1977 as cited by Barron, et al. 1995), 0.95 mg/kg (Britton and Huston 1973 as cited by Barron, et al. 1995), <5 (entered as 5) mg/kg (Platanow and Reinhart 1973 as cited by Barron, et al. 1995), and 5 mg/kg (Summer, et al. 1996).

- k) Arithmetic mean LOAEC: 1.5 mg/kg (Britton and Huston 1973 as cited by Barron, et al. 1995) [note: Table 4-9 of the Kalamazoo ERA mistakenly cites this as 'Britton 1973"', 2.5 mg/kg (Scott 1977 as cited by Barron, et al. 1995), 3 mg/kg (RCB/Hagler, Bailly, Inc 1994), 4 mg/kg (Tumasonis, et al. 1973 as cited by Barron, et al. 1995), 4.8 mg/kg (RCB/Hagler, Bailly, Inc 1994), 5 mg/kg (Platanow and Reinhart 1973 as cited by Barron, et al. 1995), and 24 mg/kg (Summer, et al. 1996).

Appendix E6. Avian Dioxin Toxic Equivalent Concentration (TEC) Egg Toxicity Reference Values (TRVs) in Recent Ecological Risk Assessments										
Site or Application - Receptor	NOAEC		LOAEC		Endpoint	Contam Source	Test Species	UF	TEFs	Reference
	µg/kg	µg/kg lipid	µg/kg	µg/kg lipid						
Blankenship & Giesy 2002 - bald eagle	0.134	1.79 ^a	0.4	5.33 ^a	P4501A induction	field	bald eagle	none	Recalculated with TEFs from Van den Berg et al 1998	Elliott et al. 1996
Fox River, WI - all birds	0.007	0.04 ^b	0.19 - 0.31 ^c	2.5 - 4.1 ^d	egg lethality	field	wood duck (NOAEC), double-crested comorant, caspian tern (LOAEC) ^e	unknown (treatment-to-LC ₅₀) ^f , 0.1 (LC ₅₀ -to-NOAEC); none ^g (LOAEC)	USEPA 1989 (NOAEC); H4IIE bioassay (LOAEC) ^h	Giesy et al 1995 (NOAEC), Giesy et al 1994a (LOAEC)
Fox River, WI - all birds	0.038	0.6 ⁱ	0.38	6.4 ⁱ	deformity	field	double-crested comorant	10 (NOAEC-to-LOAEC)	H4IIE bioassay	Ludwig et al 1996
Hudson River, NY - belted kingfisher	1	17 ⁱ	4	68 ⁱ	embryo mortality	TCDD injection	double-crested comorant	none	1 (by definition)	Powell et al 1997
Hudson River, NY - great blue heron	0.3	4.8 ^j	0.5	7.9 ^j	chick BW	field	great blue heron	none	Safe et al 1990	Sanderson et al 1994a
Hudson River, NY - bald eagle	0.214 ^k	12.8 ^l	5	79 ^m	hatch rate (NOAEC); embryo mortality (LOAEC)	field (NOAEC); PCB 77 injection (LOAEC)	bald eagle (NOAEC); kestrel (LOAEC)	none	Ahlborg et al 1994 (NOAEC); Van den Berg et al 1998 (LOAEC)	Elliott et al 1996 (NOAEC); Hoffman et al 1998 (LOAEC)
Sheboygan River and Harbor, WI, terrestrial ERA - robin	0.08	0.7 ⁿ	0.16	1.4 ⁿ	embryo mortality	TCDD injection	chicken	none	1 (by definition)	Powell et al 1996

a) Bald eagle egg lipid content of 7.5 % (Blankenship and Giesy 2002)

- b) Wood duck egg lipid content of 18 % (White and Seginak 1994)
- c) Lethal concentration to 20 % (LC_{20}) and 30 % (LC_{30}) of eggs based on a linear regression of data on field-exposed double-crested cormorant and caspian tern colonies (Table 9 in Giesy, et al. 1994a) and the NOAEC from Giesy, et al. (1995). Note—the data are incorrectly attributed in the Fox River ERA Figure 6-4 (Giesy and Tillitt are transposed), Table 6-7 (should read “Giesy, et al. 1994b”), and Table 6-5 (should read “derived from Giesy, et al. 1994b and 1995”; the regression based on Tillitt, et al. 1992 data was not used in the derivation).
- d) Based on the following regression: egg $LC_{50} = 0.006$ lipid-normalized TEC (pg/g) + 5.282, $r^2 = 0.99$, $p < 0.01$, where LC_{50} is the lethal concentration in eggs associated with mortality in n % of eggs. Each egg TEC datum in Table 6-7 of the Fox River ERA was lipid-normalized by species: wood duck (see b), cormorant (see g), and caspian tern (semi-precocial-egg lipid content of 9.5 %, Carey, et al. 1980).
- e) The regression includes the wood duck NOAEC value in addition to the cormorant and tern data.
- f) Giesy, et al. (1995) wrote: “The LC_{50} for wood ducks (*Aix sponsa*) has been reported to be approximately 70 ng TCDD-EQ/kg in the eggs of wood ducks (White and Setinak [sic] 1994). If this value is divided by an application factor of 10, the estimated NOAEC for eggs is estimated to be approximately 7 ng TCDD-EQ/kg.” However, LC_{50} values are not presented in White and Seginak (1994) or the companion paper by White and Hoffman (1995). The nearest datum to a LC_{50} is 55 % eggs hatched at >50 ppt TEC (Table 4 in White and Seginak 1994). Giesy, et al. (1995) do not discuss the procedure for deriving a LC_{50} of 70 ppt from White and Seginak (1994).
- g) The cormorant and tern data were used without UFs (6 data points). One data point in the regression was derived with UFs (the wood duck NOAEC).
- h) Six of the 7 TEC data points used in the regression are based on the HII4E bioassay (Giesy, et al. 1994a), but one datum (the Giesy, et al. 1995 NOAEC derived from White and Seginak 1994) is based on USEPA 1989 TEFs.
- i) Cormorant egg lipid content of 5.9 % (altricial) (Carey, et al. 1980)
- j) Heron egg lipid content of 6.3 % (semi-altricial) (Carey, et al. 1980)
- k) Average of whole egg TECs at Powell River (210 ng/kg) and East Vancouver (217 ng/kg – calculated from 13,000 ng TEC/kg lipid ÷ 60 [yolk lipid concentration-to-whole egg concentration reported by Elliott, et al. 1996]).
- l) Average of lipid-normalized egg yolk TECs at Powell River (12,600 ng/kg lipid) and East Vancouver (13,000 ng/kg lipid – estimated from Figure 4 of Elliott, et al. 1996).
- m) Falcon egg lipid content of 6.3 % (semi-altricial) (Carey, et al. 1980)
- n) Chicken egg fat content of 11.2 % based on 5.6 g fat in a 50-g egg (Pennington and Church 1985)

Appendix E7. Ecological Risk Assessment Sources

- Fox - Final Baseline Human Health and Ecological Risk Assessment, Lower Fox River and Green Bay, Wisconsin, Remedial Investigation and Feasibility Study, 12/2002, prepared by The RETEC Group for Wisconsin Dept. of Natural Resources. <http://dnr.wi.gov/org/water/wm/foxriver/reportsanddocs.html> choose "All Risk Assessment Reports", then "Section 6".
- Great Lakes Initiative - USEPA. 1995. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, PCBs. Office of Water. EPA-820-B-95-008. <http://www.epa.gov/gliclearinghouse/> choose "GLI Documents", then "Technical Support Documents".
- Hudson - Hudson River PCB Reassessment, Phase 2 Report, Further Characterization and Analysis, 11/2000, prepared by TAMS Consultants and Menzie-Cura Assoc. for USEPA Region 2 and USACE, Kansas City District. <http://www.epa.gov/udson/reports.htm>
- Housatonic - Bursian, S., C. Sharma, R. Aulerich, B. Yamini, R. Mitchell, C. Orazio, D. Moore, S. Svirsky, and D. Tillitt. 2006a. Dietary Exposure of Mink (*Mustela vison*) to fish from the Housatonic River, Berkshire County, Massachusetts, USA: effects on reproduction, kit growth, and survival. Environ Toxicol Chem 25: 1533-1540; and Bursian, S., R. Aulerich, B. Yamini, and D. Tillitt. 2003. Dietary Exposure of Mink to Fish from the Housatonic River: Effects on Reproduction and Survival. 6/10/03 Revised Final Report. submitted to Weston Solutions, Inc. <http://www.epa.gov/ne/ge/thesite/testofriver-reports.html>
- Kalamazoo - Final (Revised) Baseline Ecological Risk Assessment. Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site. 2003. prepared by CDM for Michigan Department of Environmental Quality., and Appendix D. Toxicity Reference Values (TRVs) for Mammals and Birds Based on Selected Aroclors. 3/6/03. memo from James Chapman, ecologist, USEPA Region 5 to Shari Kolak, RPM. USEPA Region 5, Chicago. http://www.michigan.gov/deq/0,1607,7-135-3311_4109_4217-84646-,00.html
- Sheboygan aquatic ERA - Sheboygan River and Harbor Aquatic Ecological Risk Assessment, 11/98, prepared by EVS and NOAA for USEPA Region 5. <http://response.restoration.noaa.gov/index.php> choose "Downloads" (under "Catalogs of?"), then "CPRD Publications and Technical Documents".
- Sheboygan terrestrial ERA - Sheboygan River and Harbor Floodplain Terrestrial Ecological Risk Assessment, Sheboygan, Wisconsin, 11/99, prepared by James Chapman, USEPA ecologist, for USEPA Region 5.
- Upper Green Bay - Focused Ecological Risk Assessment. Upper Green Bay Portion of the Fox River Site, Green Bay, WI. 2000. prepared by Mark Sprenger, Nancy Beckham and Karen Kracko, USEPA Environmental Response Team Center, NJ. Appendix C in Final Baseline Human Health and Ecological Risk Assessment, Lower Fox River and Green Bay, Wisconsin Remedial Investigation and Feasibility Study, 2002, Volume 2. <http://dnr.wi.gov/org/water/wm/foxriver/reportsanddocs.html> choose "All Risk Assessment Reports", then "Appendix C".

See Section 8 for all other references.

Appendix F

Coefficient of Variation

Appendix F1. Coefficient of Variation (CV), Neal's Landfill, Bloomington, IN						
Date	Location	Species	TEC	4-peak Aroclor	5-peak Aroclor	Sum of Congeners
8/01	1	CRC	0.15	0.09		
		GS	0.45	0.18		
	2	LS	0.97	0.41		
		WS	1.02	0.49		
	3S	CRC	0.12	0.06		
	4	BRH	0.12	0.13		
		LS	0.44	0.43		
		WS	0.78	0.11		
	5	BRH	0.25	0.61		
		LS	0.37	1.38		
	6	BRH	0.43			
		CP	0.49			
		LS	0.07			
11/02	0	CRC	0.13	0.08	0.08	
		WS	0.21 (1.72)	0.79 (0.91)	0.53 (0.67)	
	1	CRC	0.42	0.74	0.75	
	2	CRC	0.22	0.74	0.72	
		LS	0.49	0.89	0.88	
		WS	0.48	0.64	0.66	
5/03	1	CRC	0.25			0.22
		CF	0.16			0.49
	2	CRC	0.35			0.40
		LS	0.60			0.78
		WS	0.31			0.30
		CF	0.10			0.07
	3V	SRH	0.49			0.42

CV = SD / mean, see Appendices A7, B4, C7 for data

Species acronyms are defined in Appendices A1 and C1

11/02 Location 0 WS CV is for the reduced data set excluding the sample ID 15 outlier (Appendix B2 and Section 3.3.7). The second value in parenthesis is the CV for all of the white sucker data at Location 0.

Appendix G

Mink and Otter Liver PCB Toxicity Reference Values

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.							
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference
TEC No Observed Adverse Effect Concentration (NOAEC)							
Mink liver	I-TEQ NOAEC	pg/g	11 (Table 7), 18 (Table 8)	(5)	220 * 360 *	kit survival / body weight	Tillitt, et al. 96
Mink liver	WHO-TEQ NOAEC	pg/g	19	(5)	380	mandibular and maxillary squamous cell proliferation	Bursian, et al. 06b
Otter liver	I-TEQ EC ₁ (NOAEC)	ng/g pg/g	0.04 40	4.2	1 1000	hepatic retinoid (retinylpalmitate) concentration	Smit, et al. 96; Murk, et al. 98 as cited in Kannan, et al. 00
Mink liver	WHO-TEQ NOAEC	pg/g	56	(5)	1100 *	kit survival	Bursian, et al. 06b
Otter liver	I-TEQ EC ₁ (NOAEC)	ng/g pg/g	84 *	4.2	2 2000	hepatic vitamin A (retinol)	Murk, et al. 98
Mink liver, Hwy 48 / Richland Ck	WHO-TEQ	ng/g pg/g	0.077 77	2.6	3.01 3010		7/31/04
TEC Lowest Observed Adverse Effect Concentration (LOAEC)							
Mink liver	WHO-TEQ effect level	pg/g	22	(5)	440 *	EROD / ECOD induction	Bursian, et al. 03
Mink liver	S-TEQ threshold	pg/g	32	5	640	litter size	Kannan, et al. 00 derived from EC ₅₀ in Leonards, et al. 95

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.							
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference
Mink liver	WHO-TEQ effect range	pg/g	40-197	(5)	800-3900 *	mandibular and maxillary squamous cell proliferation	Bursian, et al. 06b
Mink liver	I-TEQ threshold	pg/g	60	5	1200	kit survival / body weight	Kannan et al. 00 derived from Tillitt et al. 96
Otter liver	I-TEQ LOAEC	ng/g pg/g	0.084 84	4.2	2 2000	hepatic retinoid concentration	Smit, et al. 96; Murk, et al. 98 as cited in Kannan, et al. 00
Mink liver, Hwy 48 / Richland Ck	WHO-TEQ	ng/g pg/g	0.077 77	2.6	3.01 3010		7/31/04
Mink liver	I-TEQ LOAEC	pg/g	207 (Table 8), 324 (Table 7)	(5)	4100 * 6500 *	kit survival / body weight	Tillitt, et al. 96
Mink liver	WHO-TEQ LOAEC	pg/g	210	(5)	4200 *	kit survival / body weight	Heaton, et al. 95; Tillitt, et al. 96 as cited in Bursian, et al. 03
Mink liver	WHO-TEQ range of effects	µg/kg pg/g	0.21-1.3 210-1300	(5)	4200-26000 *	mandibular and maxillary squamous cell proliferation	Beckett, et al. 05
Mink liver	WHO-TEQ LOAEC	pg/g	218	(5)	4400 *	kit survival	Bursian, et al. 06b
Otter liver	I-TEQ critical level (EC ₅₀)	ng/g pg/g	210	4.2	5 5000	hepatic vitamin A	Murk, et al. 98
Otter liver	TEQ field effect	ng/g pg/g	200 *	(4)	5 5000	disease incidence	Smit, et al. 96 cited in Kannan, et al. 00

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.								
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference	
Mink whole-body	S-TEQ EC ₅₀	pg/g	160	2-3	5300-8000 *	litter size	Leonards, et al. 95	
Mink whole-body	S-TEQ EC ₅₀	pg/g	200	2-3	6700-10000 *	kit survival	Leonards, et al. 95	
PCB No Observed Adverse Effect Concentration (NOAEC)								
Mink liver	PCB NOAEC	µg/g	0.09		2	kit survival, bodyweight	Heaton, et al. 95	
Mink liver	PCB NOAEC	µg/g	0.1-0.2	(5)	2.4 *	kit growth; cause of kit mortality	Wren, et al. 87	
Otter liver	PCB EC _i (NOAEC)	µg/g	0.17 *	4.2	4	hepatic vitamin A	Murk, et al. 98	
Mink liver	PCB NOAEC	ppb	300 (next lower treatment to LOAEC with $N \geq 5$)		6 *	kit bodyweight	Restum, et al. 98; Shipp et al. 98	
		µg/g	0.3	(5)				
Mink adipose	PCB NOAEC	ppm	0.4 * liver	(5)	8	kit survival	Homshaw, et al. 83	
Otter muscle	PCB NOAEC	ppm			8	increasing population	Roos, et al. 01	
Mink liver	PCB NOAEC	ppm	0.44 *	(5)	9	litter size	Kihlström, et al. 92	
Mink adipose	PCB NOAEC	ppm	0.7 * liver	(5)	14	kit production	Jensen, et al. 77	
Mink liver	PCB NOAEC	ng/g µg/g	727 0.727	(5)	15 *	mandibular and maxillary squamous cell proliferation	Bursian, et al. 06b	

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.								
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference	
Mink liver	Aroclor 1260 NOAEC	ppb µg/g	>750 >0.8	(5)	16 *	kit growth	Halbrook, et al. 99	
Mink liver, Hwy 48 / Richland Ck	PCB	ng/g µg/g	865 0.87	2.6	33700 34		7/31/04	
Mink whole-body	Aroclor 1260 NOAEC	ppb µg/g	>1800 >1.8	(2-3)	60-90 *	kit growth	Halbrook, et al. 99	
Mink liver	PCB NOAEC	µg/g	3.08	(5)	62 *	kit survival	Bursian, et al. 06b	
PCB Lowest Observed Adverse Effect Concentration (LOAEC)								
Mink liver	PCB max. observed effect	µg/g	0.18	1.8-4.2	4-10 *	baculum length	Harding, et al. 99	
Otter liver or muscle	PCB standard	ppm	0.25 * liver	(4.2)	6	vitamin A deficiency	Smit, et al. 96 cited in Mason 98	
Mink muscle	Clophen A50 LOAEC	µg/g	0.2-0.3 *	(2-3)	11	kit production / growth	Brunström, et al. 01	
Otter liver	PCB critical level (EC ₉₀)	µg/g	0.46 *	4.2	11	hepatic vitamin A	Murk, et al. 98	
Mink liver	PCB LOAEC	ppb µg/g	633 0.6	(5)	12 *	kit bodyweight	Restum, et al. 98; Shipp et al. 98	
Mink adipose	PCB LC ₁₀₀	ppm	0.65 * liver	(5)	13	kit survival	Hornshaw, et al. 83	
Mink liver	Metabolized A1260 LOAEC	ppm	1.2	(5)	24 *	kit survival	Platonow and Karstad 73	

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.							
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference
Mink muscle	Metabolized A1260 LOAEC	ppm	1.0	(2-3)	33-50 *	kit survival	Platonow and Karstad 73
Mink liver	PCB LOAEC	ng/g µg/g	1698 1.7	(5)	34 *	mandibular and maxillary squamous cell proliferation	Bursian, et al. 06b
Mink liver, Hwy 48 / Richland Ck	PCB	ng/g µg/g	865 0.87	2.6	33700 34		7/31/04
Mink whole-body	PCB EC ₅₀	µg/g	1.2	2-3	40-60	litter size	Leonards, et al. 95
Mink liver	PCB LOAEC	ng/g µg/g	2000 2	(5)	40 *	kit survival / growth	Wren, et al. 87 as cited by Poole, et al. 95
Mink liver	PCB LOAEC	µg/g	2.2	~5 *	45	kit survival / bodyweight	Heaton, et al. 95
Mink liver	PCB LOAEC	µg/g	3	(5)	60 *	kit growth / cause of kit mortality	Wren, et al. 87
Mink liver	PCB LOAEC	µg/g	3.13	(5)	63 *	kit survival	Bursian, et al. 06b
Mink liver	PCB range of effects	mg/kg	3-6	(5)	60-120 *	mandibular and maxillary squamous cell proliferation	Beckett, et al. 05
Otter	PCB LOAEC	µg/g	3.5 female			declining population	Henney, et al. 81 cited in Wren 91
Mink liver	PCB LOAEC	ppm	3.7 *	(5)	74	litter size	Kihström, et al. 92
Mink whole-body	PCB EC ₅₀	µg/g	2.4	2-3	80-120 *	kit survival	Leonards, et al. 95
Mink adipose	PCB LOAEC	ppm	4.3 * liver	(5)	86	kit production	Jensen, et al. 77

Appendix G1. PCB and TEC Tissue TRVs in Mink and Otter, and Analytical Results for a Road-kill Mink Collected from the Highway 48 Bridge Over Richland Creek, 7/31/04.							
Species / Tissue	TRV Basis	Units	Whole-tissue Concentration	% Lipid Content	Lipid-normalized Concentration	Toxicological Endpoint	Reference
Mink liver	Aroclor 1260 LOAEC	ppb µg/g	5396 5.4	(5)	110 *	kit growth	Halbrook, et al. 99
Mink whole-body	Aroclor 1260 LOAEC	ppb µg/g	6260 6.3	(2-3)	210-315 *	kit growth	Halbrook, et al. 99

For multiple lines within a single entry, the upper line has the units as in the cited paper, the lower line converted units.

* - calculated value not in the cited paper

() - not given in the original paper, assumed value used for lipid normalization.

EC_x - effective concentration, causes adverse effects in x % of test animals

ECOD - ethoxycoumarin-O-deethylase, one of the cytochrome P450/A1 subfamily isozymes (CYP1A1 isozymes)

EROD - ethoxycoumarin-O-deethylase, one of the cytochrome P450/A1 subfamily (CYP1A1 isozymes)

LC_x - lethal concentration, results in death of x % of test animals

mg/kg = µg/g

ppb = ng/g

ppm = µg/g

TEF sources: I-TEQ - Ahlborg, et al. 1992, 1994; S-TEQ - Safe 1993; WHO-TEQ - Van den Berg, et al. 1998

Threshold - geometric mean of NOAEC and LOAEC values

Appendix H

Modeled Accumulation of PCBs and TEC in Mink Livers

Appendix H1. Mean Fish Lipid Contents and Measured and Modeled Crayfish Lipid Contents, Neal's Landfill, Bloomington, IN

Reach	Species	Lipid Content (%)		CF/Fish Lipid Ratio
		2001	2003	2003
1	CRC	5.01	6.67	0.24
1	GS	3.36		
1	CF	1.96	1.59	
1	Mink diet	3.82	5.83	
2	CRC		3.29	0.54
2	LS	1.08	1.87	0.95
2	WS	4.17	5.37	0.33
2	CF	1.03	3.44	
2	Mink diet	2.36	3.50	
3S	CRC	2.98		
3V	SRH		4.47	
3	CF	0.72	1.48	
3	Mink diet	2.61	3.97	
4	BRH	3.00		
4	LS	0.95		
4	WS	1.99		
4	CF	0.90		
4	Mink diet	1.80		
5	BRH	2.72		
5	CP	2.53		
5	LS	1.09		
5	CF	1.03		
5	Mink diet	1.93		
6	BRH	2.02		
6	CP	1.40		
6	LS	2.19		
6	CF	2.08		
6	Mink diet	1.90		

2001 CF lipid = CRC lipid * 0.39 (mean of 2003 Reaches 1 and 2), or LS lipid * 0.95 (2003 Reach 2) (excluding CF16 outlier)

2003 Reach 3 CF lipid = SRH lipid * 0.33 (2003 Reach 2 CF/WS lipid ratio excluding CF16 outlier)

Mink diet lipid content = (0.835 * mean fish lipid) + (0.165 * CF lipid). The weighting factors are for 66% fish and 13% CF in diet with neutral terrestrial lipid contribution (terrestrial prey are assumed not to change the overall dietary lipid content)

Appendix H2. Modeled Accumulation of PCBs and TEC in Mink Livers, Neal's Landfill Site, Bloomington, IN.

Reach	Year	Dietary Basis	PCB Congeners in Mink Diet										189	PCBs in Diet mg/kg	Mink liver PCB µg/g	TEC pg/g
			77	81	105	114	118	123	126	156/157	167	169				
1	2001	Lipid	3248.76	170.75	7984.07	624.19	13047.00	423.38	43.74	369.20	107.35	1.11	6.51	228.19		
		Whole	124.12	6.52	305.04	23.85	498.47	16.18	1.67	14.11	4.10	0.04	0.25	8.72	22.41	2498.01
	2003	Lipid	873.03	59.37	1783.88	256.02	2813.80	263.57	15.77	148.30	63.43	U	5.62	217.55		
		Whole	50.94	3.46	104.09	14.94	164.18	15.38	0.92	8.65	3.70	U	0.33	12.69	32.62	1295.72
2	2001	Lipid	996.52	63.91	3284.00	249.81	5720.56	205.13	12.70	152.83	55.21	U	4.29	54.36		
		Whole	23.48	1.51	77.36	5.88	134.77	4.83	0.30	3.60	1.30	U	0.10	1.28	3.29	488.97
	2003	Lipid	333.32	18.37	1280.30	113.02	1939.21	101.31	4.94	65.53	23.94	U	2.28	49.51		
		Whole	11.66	0.64	44.77	3.95	67.81	3.54	0.17	2.29	0.84	U	0.08	1.73	4.45	280.13
1&2	2001	Whole	48.64	2.76	134.28	10.38	225.69	7.67	0.64	6.23	2.00	0.01	0.14	3.14	8.07	991.23
	2003	Whole	21.48	1.35	59.60	6.70	91.90	6.50	0.36	3.88	1.55	U	0.14	4.47	11.49	534.02
3S	2001	Lipid	326.10	18.30	629.23	52.70	1047.74	33.72	0.00	17.87	7.43	U	1.75	11.86		
	Whole	8.50	0.48	16.40	1.37	27.31	0.88	0.00	0.47	0.47	0.19	U	0.05	0.31	0.79	27.08
3V	2003	SRH	143.56	7.78	464.02	35.64	982.33	36.34	1.77	22.60	10.70	U	1.19	20.04		
		CF	90.25	4.06	212.35	15.58	548.23	19.24	0.96	10.79	6.34	U	0.57	8.42		
	Lipid	106.48	5.67	333.86	25.55	719.61	26.49	1.30	16.32	7.89	7.89	U	0.86	14.32		
		Whole	4.23	0.23	13.27	1.02	28.60	1.05	0.05	0.65	0.31	U	0.03	0.57	1.46	86.82
4	2001	Lipid	146.02	9.97	620.86	41.39	1187.88	41.84	0.00	36.71	17.96	U	1.20	24.19		
	Whole	2.63	0.18	11.18	0.75	21.39	0.75	0.00	0.66	0.66	0.32	U	0.02	0.44	1.12	21.24
5	2001	Lipid	22.51	0.56	126.07	8.41	242.20	8.92	U	13.44	6.67	U	0.85	5.86		
		Whole	0.44	0.01	2.44	0.16	4.68	0.17	U	0.26	0.13	U	0.02	0.11	0.29	5.30

Appendix H2. Modeled Accumulation of PCBs and TEC in Mink Livers, Neal's Landfill Site, Bloomington, IN.

Reach	Year	Dietary Basis	PCB Congeners in Mink Diet										PCBs in Diet		Mink liver		
			77	81	105	114	118	123	126	156/157	167	169	189	Diet	PCB	TEC	
			µg/kg													µg/g	pg/g
6	2001	Lipid	10.66	U	72.36	4.50	142.82	4.35	U	16.72	5.80	U	U	3.53			
		Whole	0.20	U	1.38	0.09	2.72	0.08	U	0.32	0.11	U	U	0.07	0.17	3.97	
2	2003	CF/WS	0.63	0.52	0.46	0.44	0.56	0.53	0.54	0.48	0.59	U	0.48	0.42			
		Diet-liver BMF	0.13	1.17	5.4	4.03	4.6	0.57	11.77	11.25	6.1	13.6	10	2.57			
		Mammal TEF	—	0.0001	—	0.0005	—	0.0001	—	0.1	0.0005	0.00001	0.01	0.0001			

Dietary Basis: Lipid - lipid normalized concentration (µg or mg/kg lipid), Whole - wet weight dietary concentration (µg or mg/kg wet weight diet and µg or pg/g ww diet)
 Lipid-normalized dietary concentrations calculated for mink dietary composition (Section 2.6) from fish and crayfish data in Appendices A4 and C4, except for 2003 Reach 3.
 Whole dietary concentration = Lipid-normalized dietary concentration * (dietary lipid content / 100)

See Appendix H1 for dietary lipid contents

Mink liver PCB = Whole diet PCB concentration * mink total PCB diet-to-liver BMF

Note: µg/g is equivalent to mg/kg

Mink liver TEC = Sum of whole diet congener concentration * respective mink congener diet-to-liver BMF * respective TEF * 1000 (to convert µg/kg to pg/g)
 CF - crayfish; SRH - shorthead redhorse; WS - white sucker

Diet-liver BMF - mink diet-to-liver biomagnification factor on a wet-weight basis (Tillitt, et al. 1996).

Mammal TEF - WHO dioxin toxic equivalency factor (Van den Berg, et al. 1998)

1&2 = (0.25 * Reach 1 PCB or TEC) + (0.75 * Reach 2 PCB or TEC). Represents combined foraging in Conard's Branch and Richland Creek (Section 2.6).

2003 Reach 3 lipid-normalized dietary concentrations includes modeled CF component calculated as Reach 3 SRH * 2003 Reach 2 CF/WS.

2003 Reach 2 CF/WS lipid-normalized congener and total PCB ratios calculated from Appendix C3 excluding CF16 outlier

Additional Consideration of Toxicity Reference Values Derived Through Meta-analysis

Addendum to the Focused Ecological Risk Assessment
PCBs and Mammalian and Avian Piscivores in Conard's Branch and Richland Creek

Neal's Landfill Site, Bloomington, Monroe County, Indiana

August 14, 2006

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Summary

Additional analysis of two sets of toxicity reference values (TRVs) was requested by Viacom, Inc. The results show that the TRVs for PCB dietary exposure to mink, and PCB ingestion dose to birds, are robust to data variability in the upper range of exposures, but not in the lower range of exposures. In other words, the “actual” TRVs are unlikely to be higher than the values used in the Focused Ecological Risk Assessment (FERA)—the analysis recommended by Viacom, Inc., resulted in no more than a 20 % increase in the calculated TRVs, and mostly less than 10 % changes related to variability in the upper range of exposures, but the “actual” TRVs might be lower than used in the FERA—the procedure resulted in 35 to 90 % decreases in calculated TRVs related to variability in the lower range of exposures.

This indicates that the “actual” values of the mink dietary no effect (500 µg total PCB/kg diet) and low effect (600 µg/kg) TRVs, and kingbird ingestion dose no effect (400 µg total PCB/kg_{BW}-d) and low effect (500 µg/kg_{BW}-d) TRVs (see FERA Table 10), are unlikely to be higher than the values used in the FERA, but the possibility that the “actual” values might be lower cannot be ruled out. Note that the kingfisher interpolated TRVs for PCB ingestion are one of two sets of PCB ingestion dose TRVs used in the FERA to represent uncertainty over the sensitivity of kingfisher to PCBs relative to the species tested for PCB effects.

Specific to mink, kit bodyweight is not a robust endpoint for an Aroclor-based TRV because the A1254 data set is sparse. Number of live kits per mated female is a robust endpoint for deriving an Aroclor-based TRV protective of mink as discussed above. Details of the procedure for adjusting the mink TRVs for continuous exposure over multiple breeding seasons or generations also are presented in this memo.

The Addendum supplements the discussion of PCB TRVs (FERA 4.2.2.1 and 4.2.2.3) and the associated uncertainties (FERA 6.3), and the comparison of the TRVs used in the FERA with the TRVs at recent Superfund sites (FERA Appendices E1 and E3).

Introduction and Results

Additional analysis of two sets of toxicity reference values (TRVs) was requested by Viacom, Inc. The TRVs in question are based on meta-analysis of multiple toxicity studies, a procedure used to derive 2 of the 8 sets of TRVs used in the focused ecological risk assessment (FERA)—mink dietary no effect (500 µg total PCB/kg diet) and low effect (600 µg/kg) TRVs, and kingbird ingestion dose no effect (400 µg total PCB/kg_{BW}-d) and low effect (500 µg/kg_{BW}-d) TRVs (FERA Table 10). The remaining 6 sets of TRVs in the FERA are derived through other approaches (FERA 4.2) and are not discussed in the Addendum.

The procedure, recommended by Dr. John Giesy, Entrix, consulting for Viacom, Inc., is to individually remove data points (treatment means) in turn from the combined data set to assess the sensitivity of the interpolated TRV to data variability. A published source for the procedure was not provided, hereafter referred to as the “Giesy procedure”. The outcome can reveal the general extent to which the regressions used for calculating the no effect and low effect TRVs are affected by variations in the underlying database. Modest changes in interpolated TRVs with individual data exclusions indicate that the TRV derivation is robust, that is, not overly dependent on the results of any single treatment. Modest changes also indicate that the separate studies combined together in the meta-

analysis are compatible with each other, and that a sufficiently close spacing of PCB exposures has been tested to reinforce the shape and location of the dose-response relationship in the vicinity of the selected effect levels that define the TRVs. Large changes in interpolated TRVs with individual data exclusions indicate possible shortcomings in one of these factors, for example, the results may be strongly affected by a single treatment, studies may have been inappropriately combined in the meta-analysis (incommensurable data), or the spacing of the PCB exposures of the combined studies may be too wide to reinforce the shape or location of the dose-response relationship around the selected effect levels.

The Giesy procedure is applied to the following data sets: ingestion of Aroclor 1248-amended feed by chicken and the effect on egg hatchability; and Aroclor 1254 dietary concentration and the effect on mink reproduction (number of live kits per mated female).

To combine separate studies together, the responses of the treatment groups are normalized to the respective control (unexposed) groups. This is accomplished by dividing the treatment response by the control response of the same study (Equation A1, Methods below). For example, if 40 % of eggs hatch to hens exposed to 2 ppm of a chemical, and 83 % of the eggs hatch to unexposed hens in the control group over the same time, the relative response of the 2 ppm treatment is 0.48 ($= 0.4 / 0.83$). The relative responses (RR) for the FERA TRVs based on meta-analysis are shown in Tables A3 and A4.

The combined results are shown in Figures A1 (egg hatchability) and A3 (mink reproduction). The TRV derivations are based on the dose-response relationships revealed by the combined data sets. Low effect is defined as a relative response of 75 % (a 25 % decrease in mean treatment response compared to the control response), shown as a horizontal line in Figures A1, A3 and A4, and no effect as a relative response of 90 % (a 10 % decrease in mean treatment response compared to the control response), not shown in the figures.

The TRVs are calculated by log-linear regression between the relative responses of the doses or dietary concentrations that bracket the selected effect level (90 % for no effect, 75 % for low effect) (Equation A2). Since only the data bracketing the effect level are used in the regression, only these data points need be included in the Giesy procedure, that is, individual exclusion of more distal data points has no effect on the log-linear regression. In an additional scenario for the hatchability TRV, two adjacent data points are simultaneously excluded because they could have been averaged together and then used as a single value for interpolating TRVs (the two data points were kept distinct in the original FERA TRV derivation).

The preceding discussion does not mean that distal data points are irrelevant. As part of the meta-analysis procedure, combined data sets that did not form interpretable dose-response relationships were entirely dropped from further consideration. An example is PCB dose to chicken and the effect on egg production (Figure A2). The behavior of the data set as a whole (all the data points) determined whether or not a particular toxicological endpoint was considered for TRV derivation, which served to exclude from further consideration unresponsive or inconsistent endpoints as well as inappropriate combinations of incompatible data.

The Giesy procedure shows asymmetric effects that indicate much less variability in the upper range of the interpolated TRVs than in the lower range. Exclusion of the higher of the data points used in the TRV derivations results in less than a 10 % change in egg hatchability TRVs (Table A1), and a 20 % or less change in

mink reproductive TRVs (Table A2). This indicates that the upper portions of the dose-response relationships are well characterized (adequate dose spacing and consistent trends across treatments and studies).

The upper range of the hatchability TRVs are particularly robust. Two of the egg hatchability studies included treatments at the same ingestion dose (0.67 mg PCB/kg_{bw}-d) with similar relative responses (0.42 – Lillie, et al. 1975, and 0.55 – Scott 1977). Simultaneously excluding both of these data changes the TRVs by less than 5 % (Table A1).

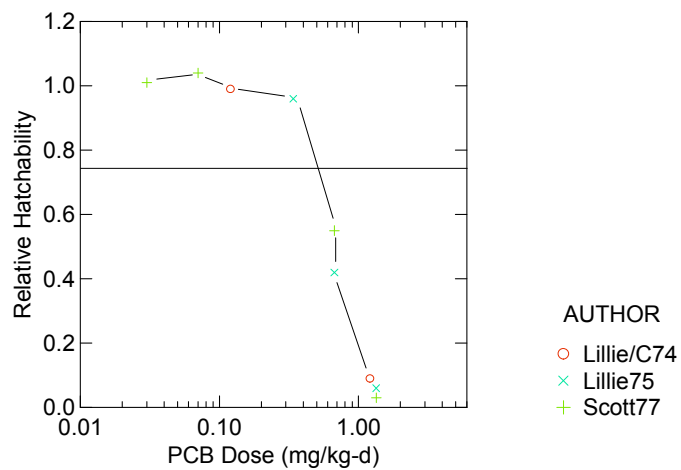
In contrast, exclusion of the lower of the data points used in the TRV derivations results in 35 to 55 % decreases in egg hatchability TRVs (Table A1), and 60 to 90 % decreases in mink reproductive TRVs (Table A2). This indicates that the lower portions of the dose-response relationships are less well characterized than the upper portions.

Poor characterization of the lower portion of the dose-response relationship is readily apparent for the mink reproductive studies—there have been no experimental exposures to less than 1 mg A1254/kg diet (Figure A3). Wren, et al. (1987) reported a control dietary concentration of 0.02 mg PCB/kg, which is 2 orders of magnitude lower than the lowest dietary concentration tested. Interpolation over 2 orders of magnitude exposure, as in the first data exclusion scenario (W1) in Table A2, is considered highly uncertain, and was not accepted for TRV derivation for the FERA (see Methods below).

Uncertainty over the lower range does not carry over to the upper range of the TRVs, which, for example, are well established for mink exposed to Aroclor 1254—reproduction is effectively suppressed in all treatments at or above 2 mg A1254/kg diet (exposure over a single breeding season). Similarly, the upper estimate of the no effect TRV, about 1 mg A1254/kg diet (exposure over a single breeding season), is robust (not likely to be higher because a small 2-fold increment is associated with catastrophic effects), but the possibility of a lower “true” no effect TRV cannot be ruled out.

The mink reproductive low effect TRV is supported by a similar TRV based on kit bodyweight at birth. However, the underlying data for the kit bodyweight-based TRVs are sparse, and the TRV calculations are affected greatly by data exclusions (not shown). Two treatments bracket the low effect target relative response of 0.75, but there are no other experimental treatments for higher or lower dietary concentrations of Aroclor 1254, other than a reported control concentration 2 orders of magnitude lower than the lowest treatment concentration (Figure A4). This means that the mink kit bodyweight low effect TRV is not robust, that is, the value fluctuates excessively with individual data exclusions (not shown because it would either involve interpolation over an unacceptably large concentration range, or would require extrapolation beyond the reduced data set, which would disqualify the derivation from the FERA TRV process⁵⁸).

⁵⁸ Only interpolation within the available data set was allowed for TRV derivation (see Methods below).



o Lillie, et al. 1974 and Cecil, et al. 1974 (reporting the same study)

x Lillie, et al. 1975

+ Scott 1977

Figure A1. Hatchability, Aroclor 1248 Dose to Chicken Hens

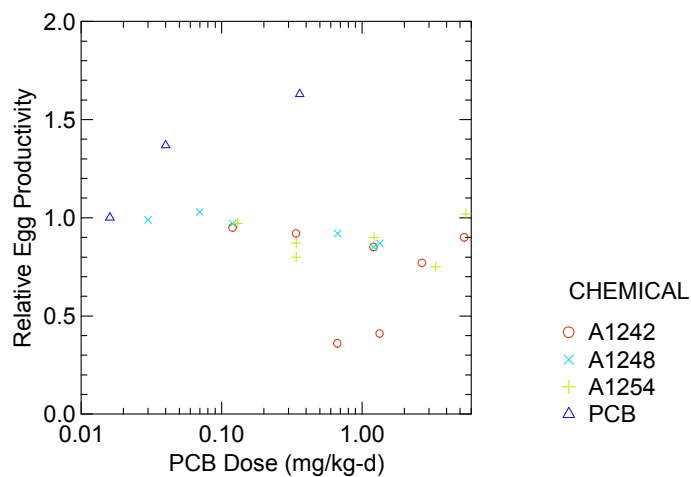


Figure A2. Egg Productivity, PCB Dose to Chicken Hens

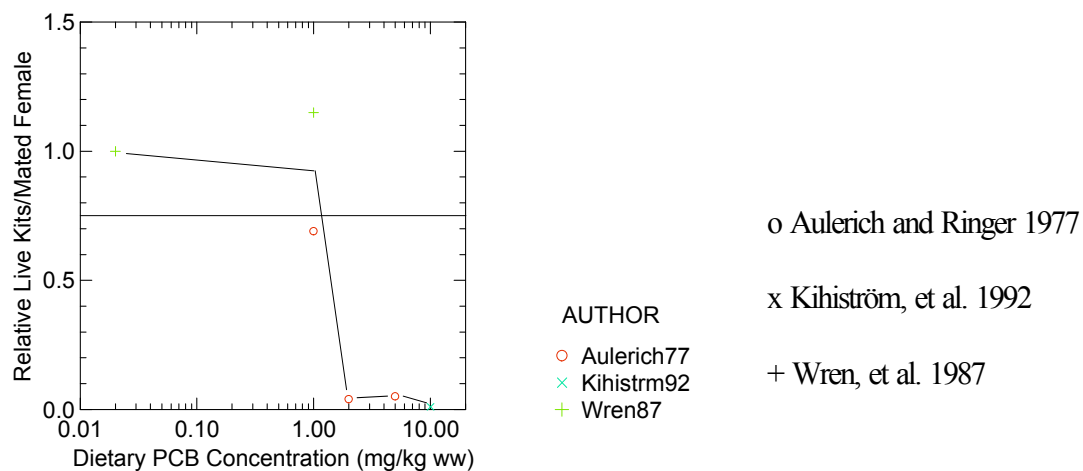


Figure A3. Live Kits per Mated Female Mink Exposed to Commercial Aroclor 1254 for 1 Breeding Season

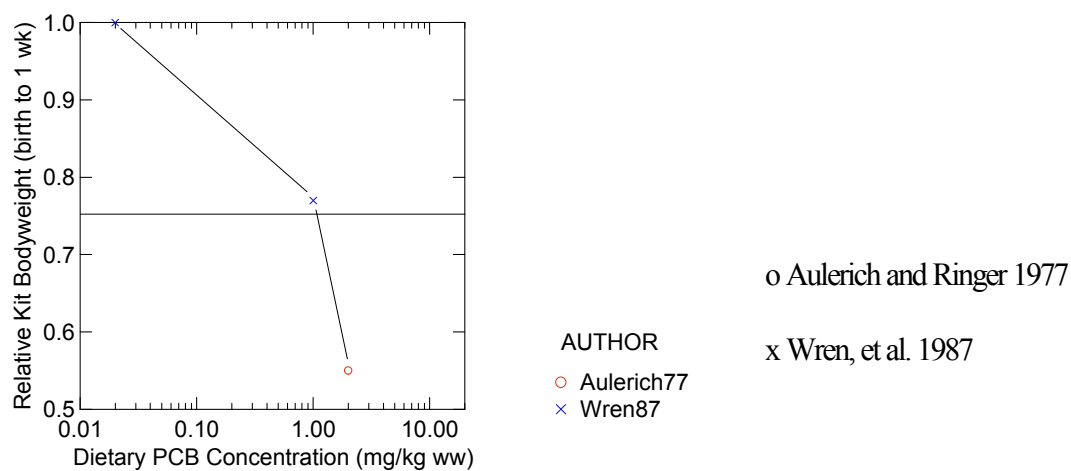


Figure A4. Mink Kit Bodyweight at Birth, Maternal Exposure to Commercial Aroclor 1254 for 1 Breeding Season

Table A1. Log-Linear Interpolation of PCB Toxicity Reference Values (TRV) for Chicken

Excluded Treatment	Treatment dose <		Treatment dose >			Target	TRV	Percent Change in TRV	Interpolation Points (Author initial and treatment PCB dose in mg/kg-d)
	Control	TRV	TRV	RR	RR				
	RR	dose	RR	dose	RR				
	M _i	D _j	M _j	D _{j+1}	M _{j+1}				
ratio	mg/kg-d	ratio	mg/kg-d	ratio	ratio	mg/kg-d	%		
LOAEL TRV - hatchability, exposed to A1248									
original	1	0.34	0.96	0.67	0.55	0.75	0.48		L34 – S.67
L.34	1	0.12	0.99	0.67	0.55	0.75	0.31	–35	LC.12 – S.67
S.67	1	0.34	0.96	0.67	0.42	0.75	0.44	–8	L.34 – L.67
S.67 L.67	1	0.34	0.96	1.21	0.09	0.75	0.46	–4	L.34 – LC1.21
NOAEL TRV - hatchability, exposed to A1248									
original	1	0.34	0.96	0.67	0.55	0.9	0.38		L34 – S.67
L.34	1	0.12	0.99	0.67	0.55	0.9	0.17	–55	LC.12 – S.67
S.67	1	0.34	0.96	0.67	0.42	0.9	0.37	–3	L.34 – L.67
S.67, L.67	1	0.34	0.96	1.21	0.09	0.9	0.37	–3	L.34 – LC1.21

Notes for Table A1.

Excluded Treatment - datum excluded from interpolation of TRV to assess the influence of study variability on TRV derivation, designated as author initial and treatment PCB concentration in ppm

RR - relative response = treatment response / control response

TRV - toxicity reference value for PCB dose (D) (mg/kg_{bw}-d)

$$\text{Log}_{10} \text{TRV} = \text{Log}_{10} D_j + (((M_i * P) - M_j) * ((\text{Log}_{10} D_{j+1} - \text{Log}_{10} D_j) / (M_{j+1} - M_j)))$$

$$\text{TRV} = 10^{\text{Log}_{10} \text{TRV}}$$

Where M_i is the control relative response (1.0 by definition because the response data is normalized to controls), D_j is the dose of the treatment that produced a relative response (M_j) greater than P (the TRV relative response), and D_{j+1} is the dose of the treatment that produced a relative response (M_{j+1}) less than P. Log_{10} is the logarithm to the base 10.

Interpolation Points - sources of data for the log-linear interpolation are separated by the symbol –.

Author: L - Lillie, et al. (1975); LC - Lillie, et al. (1974) and Cecil, et al. (1974) (reporting the same study); S - Scott (1977)

Table A2. Log-Linear Interpolation of PCB Toxicity Reference Values (TRV) for Mink

Excluded Treatment	Treatment conc < TRV		Treatment conc > TRV			Target RR	TRV	Percent Change in TRV	Interpolation Points (Author initial and treatment PCB concentration in ppm)
	Control RR		RR		RR				
	M _i	C _j	M _j	C _{j+1}	M _{j+1}				
	ratio	ppm	ratio	ppm	ratio	ratio	ppm	%	
Low Effect TRV - live kits per mated female, exposed to A1254 in a single breeding season									
original	1	1	0.92	2	0.04	0.75	1.14		W1/A1 – A2
W1	1	0.02	1	1	0.69	0.75	0.47	– 59	W0.02 – A1
A1	1	1	1.15	2	0.04	0.75	1.28	+12	W1 – A2
A2	1	1	0.9	5	0.05	0.75	1.37	+20	W1/A1 – A5
No Effect TRV - live kits per mated female, exposed to A1254 in a single breeding season									
original	1	1	0.92	2	0.04	0.9	1.02		W1/A1 – A2
W1	1	0.02	1	1	0.69	0.9	0.07	– 93	W0.02 – A1
A1	1	1	1.15	2	0.04	0.9	1.17	+15	W1 – A2
A2	1	1	0.9	5	0.05	0.9	1.04	+2	W1/A1 – A5

Notes for Table A2.

Excluded Treatment - datum excluded from interpolation of TRV to assess the influence of study variability on TRV derivation, designated as author initial and treatment PCB concentration in ppm

conc - dietary concentration of PCBs (mg/kg wet weight (ww))

RR - relative response = treatment response / control response

TRV - toxicity reference value for dietary PCBs (mg/kg wet weight (ww))

$$\text{Log}_{10} \text{TRV} = \text{Log}_{10} C_j + ((M_i * P) - M_j) * ((\text{Log}_{10} C_{j+1} - \text{Log}_{10} C_j) / (M_{j+1} - M_j))$$

$$\text{TRV} = 10^{\text{Log}_{10} \text{TRV}}$$

Where M_i is the control relative response (1.0 by definition because the response data is normalized to controls), C_j is the dietary concentration of the treatment that produced a relative response (M_j) greater than P (the TRV relative response), and C_{j+1} is the dietary concentration of the treatment that produced a relative response (M_{j+1}) less than P. Log₁₀ is the logarithm to the base 10.

Interpolation Points - sources of data for the log-linear interpolation are separated by the symbol –. Two values averaged together to calculate one of the interpolation points are shown by the symbol /.

Author: A – Aulerich and Ringer (1977); W – Wren, et al. (1987).

Table A3. White Leghorn Chicken Aroclor 1248 Toxicity Studies							
Study	Exposure			Relative Response Compared to Control			
	Exposure Duration	Dose to Hen	Egg Conc (whole ww)	Egg Productivity	Hatchability	Chick BW	Chick Survival
	week	mg/kg-d	mg/kg	————— ratio —————			
Lillie, et al 1974 and Cecil, et al 1974 (reporting the same study)	9	0.12		0.97 0-9 wk	0.99 0-9 wk	0.94 6-9 wk	0.99
		1.21	10 9 wk	0.85 0-9 wk	0.13 0-9 wk; 0.09 8 wk	0.67 6-9 wk	0.44
Lillie, et al 1975.	8	0.34			0.96 0-8 wk		
		0.67			0.75 0-8 wk; 0.42 8 wk		
		1.34			0.24 0-8 wk; 0.06 8 wk		

Table A3. White Leghorn Chicken Aroclor 1248 Toxicity Studies							
Study	Exposure		Relative Response Compared to Control				
	Exposure Duration	Dose to Hen	Egg Conc (whole ww)	Egg Productivity	Hatchability	Chick BW	Chick Survival
	week	mg/kg-d	mg/kg	ratio			
Scott 1977	8	0.03	0.16 4 wk; 0.22 8 wk	0.99 8 wk	1.01 4 wk; 1.01 8 wk		
		0.07	0.33 4 wk; 0.41 8 wk	1.03 8 wk	0.98 4 wk; 1.04 8 wk		
		0.67	2.2 4 wk; 3 8 wk	0.92 8 wk	0.73 4 wk; 0.55 8 wk		
		1.34	4.5 4 wk; 7 8 wk	0.87 8 wk	0.03 4 wk; 0.03 8 wk		

Notes for Table A3.

Conc - concentration

ww - wet weight

wk - week

Relative Response Compared to Control = treatment response / control response

Dose: Calculated from experimental data when available. Generic calculation based on a white leghorn hen food ingestion rate of 0.067 kg feed/kg_{BW}-d (Medway and Kare 1959 cited in USEPA 1995).

Table A4. Mink Aroclor 1254 Toxicity Studies										
Study	Exposure			Relative Response Compared to Control						
	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time	
	month	mg/kg diet	mg/kg	ratio						
Aulerich and Ringer, 1977	4.2	1		0.8	0.90	0.86	0.69			
	9.7	2		0.29	0.24	0.14	0.04	0.55 birth		0 4 wk
	4.2	5		0.25	0.50	0.20	0.05			
Wren, et al. 1987	6.1	1 (control 0.02)	2.8 liver (control 0.09)	0.99	1.09	1.16	1.15	0.77 1 wk; 0.75 3 wk; 0.71 5 wk		1.00 5 wk nearly all due to starvation (control 75 % due to trauma or infection, but none to starvation)
Kihström, et al. 1992	3	10	74 fat 1.3 muscle	0.34	0.66	0	0			

Notes for Table A4.

Conc - concentration

♀ - female

BW - bodyweight

Relative Response Compared to Control = treatment response / control response

Methods

The method for meta-analysis is an adaptation of the procedures used by Leonards, et al. (1995) to derive mink tissue toxicity reference values (TRVs) from multiple toxicological studies (see FERA Appendix G for the Leonards, et al. (1995) TRVs). The same control-normalization procedure is used to calculate relative responses, which allows multiple studies to be combined in a single database. Other examples of this normalization approach for meta-analysis of ecotoxicological studies include Isnard, et al. (2001), Tanaka and Nakanishi (2001), and Calabrese (2005). In an extension of Leonards, et al. (1995), commercial PCB products are separately assessed, for example, each of the Aroclors are separately considered, and studies using field-exposed prey are separated from those using Aroclor product-amended feed. Also, Leonards, et al. (1995) modeled mink tissue uptake of PCBs, but the FERA focuses on dietary PCB concentration. An important distinction is Leonards, et al. (1995) based their TRVs on pronounced effects (50 % decreases in litter size or kit survival), while the FERA TRVs are based on the onset of effects (10 to 25 % decreases in reproduction measured as live kits per mated female) consistent with regulatory practice (FERA 1.2 and 4.2.1). Leonards, et al. (1995) fitted dose-response equations to the meta-analytical data, while the FERA TRVs are calculated through linear interpolation.

Linear Interpolation

The effluent toxicity testing guidance in the water program (e.g., Klemm, et al. 1994; Chapman, et al. 1995) is modified for deriving PCB TRVs from multiple mink or chicken studies. The guidance recommends linear interpolation between the treatments showing effects that bracket the chosen effect level. The linear interpolation method avoids the complications associated with selection of the appropriate regression model by focusing on the mean dose-response trend in the region surrounding the chosen effect level. Confidence intervals are then calculated through a bootstrap method. The method assumes monotonicity, that is, that the mean response decreases as the test concentration increases, and data are smoothed (adjusted) if this pattern is violated.

The linear interpolation method was developed for deriving TRVs from the results of individual toxicity studies. However, for the present effort, the results of multiple studies are combined to better reveal the shape of the dose-response relationship for PCBs. This is necessary because most of the individual PCB toxicity studies tested a limited number of doses. Interpolation is strictly implemented for this effort—no extrapolations beyond the empirical data range are performed.

The first modification is to normalize the data so multiple studies can be compared on a common basis. The reason for combining research results is to better define the shape of the dose-response relationship compared to that shown by the relatively low number of doses tested in any single experiment. Normalization is accomplished by dividing each mean treatment response by the respective mean control response. Examples of this normalization procedure for combining multiple toxicity studies include Leonards, et al. (1995), Isnard, et al. (2001), Tanaka and Nakanishi (2001) and Calabrese (2005). The normalized responses are termed “relative response” (RR) for this effort, but have been described as “response ratio” in other meta-analyses (Hedges, et al. 1999).

$$RR = \text{treatment response} / \text{control response of the same study}$$

[A1]

The relative responses are plotted on semi-log graphs (base 10 logarithm dose or concentration vs. relative response). The plots showing interpretable dose-response relationships are used to derive the no- and low-effect TRVs by a linear interpolation between the treatments that bracket the effect level of concern. The plots showing obviously inconsistent dose-response relationships, either because there is no relationship or because the combined studies are incompatible for some reason, are excluded for TRV derivation, for example, egg production in PCB-exposed chicken (Figure A2).

The second modification is interpolation is only performed when the selected effect size falls within the steep linear portion of the dose-response plot. There are two purposes: 1) the linear interpolation method is applicable to linear responses, but will over- or underestimate for nonlinear portions of the dose-response relationship; and 2) this avoids interpolation over excessively large exposure gradients for which the shape of the dose-response relationship is poorly known. The practical result is that most of the interpolations are performed between relatively small gradients in exposure values. The majority of the TRV interpolations for mink occur between treatments that differ in dietary concentrations by 2-fold or less. Interpolation is not performed for the TRV for A1254 and kit survival, for example, because there is a 100-fold difference between the dietary concentrations of the treatments that bracket the target low-effect response. Many of the bird TRVs are interpolated between small gradients (2- or 3-fold for A1242 or A1248 dose and hatchability). Interpolations are not performed for greater than 10-fold differences in treatment doses.

A third modification is log-linear interpolation (Equation 2) is used since it gives a better fit within the linear portion of the data plots compared to the linear interpolation in the guidance:

$$\begin{aligned} \text{Log}_{10} \text{TRV} &= \text{Log}_{10} C_j + (((M_1 * P) - M_j) * ((\text{Log}_{10} C_{j+1} - \text{Log}_{10} C_j) / (M_{j+1} - M_j))) \\ \text{TRV} &= 10^{\text{Log}_{10} \text{TRV}} \end{aligned} \quad [\text{A2}]$$

where TRV is the interpolated toxicity reference value, P is the chosen effect size (FERA 4.2.1), M_1 is the control relative response (1.0 by definition because the response data is normalized to controls), C_j is the dietary concentration of the treatment that produced a relative response (M_j) greater than P, and C_{j+1} is the dietary concentration of the treatment that produced a relative response (M_{j+1}) less than P. The symbols used in Equation A2 are the same as the ones in the USEPA guidance for effluent toxicity testing. Equation A2 is used for interpolating TRVs on the basis of PCB concentration in mink diet. A similar equation is used for interpolating TRVs on the basis of bodyweight-normalized dose to chicken, where C is replaced by D for dose.

A fourth modification is data are not smoothed when treatment responses exceed control responses (relative responses > 1) to allow for hormesis (enhanced response at very low doses). All three of the commercial PCB products tested in mink feeding studies show possible hormetic effects on the number of live kits per mated female (Aroclors 1242 and 1254, and Clophen A50). The same modification to the linear interpolation method to allow for potential hormesis was made in a recent comparison of techniques for calculating effective doses (Isnard, et al. 2001).

A fifth modification is the procedure for deriving confidence intervals is not implemented since the only available data from the published mink and chicken studies are the treatment means (the underlying data for the individual

replicates were not presented for any of the studies). The bootstrapping method for generating confidence intervals for the linear interpolation method requires the full replicate data.

Effect Size

Effect size is the decrease in response of animals or plants exposed to a chemical compared to unexposed controls that is selected as the level of concern for assessing risk. The selected effect sizes for this effort are not based on receptor-specific life history/population models. The effect sizes used in this effort are chosen for pragmatic reasons—to minimize model dependence, approximate the power of well-designed toxicity studies, and maintain general consistency in approach with other regulatory uses of toxicity test data. In short, to select a low effect size that is expected to be detectable in a well-designed study, and is reasonably consistent with prior Agency practice. The very steep PCB dose-response plots make the question of the appropriate low effect level somewhat moot, since there is a small range between no-effect and total-effects levels.

A reasonably detectable effect size consistent with Agency practices in other programs would fall between 20 and 25 %. The higher of these values is chosen for this effort to ensure that the low effect size represents a non-trivial departure from the control response (equivalent to 75 % relative response).

The no effect size is set at 10 % (relative response of 90 %). Similar to the rationale for the choice of low effect size, 10 % is chosen for no effect size because it is unlikely to be identified as a LOAEL in a reasonably well-designed toxicity study, is lower than the *de minimis* effect-level identified in a review of regulatory decision-making, but is at the minimum size so that the calculated TRV is not strongly model-dependent (various regression techniques will likely give similar values).

The question of the appropriate value for the low effect size is made somewhat moot by the very steep dose-response plots for PCBs. For example, the A1248 oral dose to hens associated with complete hatch failure (1 mg/kg-d) is less than 3 times greater than the dose showing no effect (0.4 mg/kg-d) (Figure A1). The same is true for mink endpoints. Live kit production is completely suppressed at a dietary concentration of 2 mg/kg A1254, but the no effect level may be near 1 mg/kg (exposure over a single breeding season) (Figure A3). With such steep dose-response curves, refinements of the effect level will produce only relatively small changes in the interpolated TRVs.

Study Selection

Study results are selected according to the following criteria: 1) studies published in journals (gray literature⁵⁹ excluded), 2) primary sources (secondary sources⁶⁰ excluded), 3) matched control and treatment responses, 4) continuous PCB exposure up to or through the initiation of breeding (responses following cessation of exposure are

⁵⁹ Gray literature refers to studies not published in journals or books, or abstracts of results that provide insufficient information on methods and data. Examples of gray literature include meeting abstracts, government reports, master's or doctoral theses, unpublished research notes, and prepublication drafts.

⁶⁰ Primary sources are to the original publications reporting research results. Secondary sources are review articles, compilations, or other summaries of previously published work.

excluded if sufficient time elapsed to allow depuration⁶¹ to occur prior to breeding), and 5) treatment responses individually reported by dose and Aroclor (aggregated responses based on combinations of exposure levels or combinations of Aroclors are excluded). The individual Aroclor constraint is not applied to studies with field-contaminated prey. Statistical significance is not a criterion for selection of treatments within a study since the objective is to develop dose-response relationships over the full gradient tested (treatments that do not differ from the control response are as important for delineating the dose-response relationship as the treatments that do differ). When response data are reported for more than one exposure time, data for later exposure periods take precedence over earlier exposure periods or data averaged over the entire exposure period. Data are taken from text, tables, or figures so long as the selection criteria are met.

Only studies in which the test animals were exposed to commercial PCB products are used for calculating TRVs. Studies performed with field-contaminated prey are not directly used for calculating TRVs (to avoid possible confounding effects of contaminants not occurring in PCB products), but are included to contribute to the weight-of-evidence for response trends (e.g., evidence of hormesis), to contribute to the estimation of the proportional change in mink responses when the exposure duration increases from one breeding season to two breeding seasons or generations, and for overall comparison with Aroclor studies. Aroclor and field contamination studies are plotted separately for mink, but since only one chicken study is included with field-contaminated feed, it is plotted on the same graph with chicken Aroclor studies to conserve space (the field-contaminated study is shown as “PCB” in Figure A2).

It is not feasible to exactly match the exposure durations between studies. Exposure durations range from 6 to 14 wk for chicken feeding studies, with most between 6 and 9 wk. For mink, the studies are segregated by the number of breeding seasons exposure was maintained (the results of exposure over 2 breeding seasons or 2 generations are analyzed separately from 1-season results). The data show no obvious effects due to the range in exposure durations (other than the 1-season vs. 2-season or 2-generation results for mink which are therefore disaggregated).

The exposure route for all of the mink and chicken studies used for deriving the FERA TRVs is through contaminated diet.

Toxicity Endpoints

Data for the following reproductive and growth endpoints were collected from a review of mink PCB studies: whelping frequency (number of female mink giving birth per number mated), total kits (live and stillborn at birth) per whelped female, live kits per whelped female (at birth), live kits per mated female (at birth), kit bodyweight, and kit survival. Since the effects of the first three endpoints are integrated in the number of live kits per mated female, TRVs are not separately calculated for whelping frequency or for total or live kits per whelped female. Kit bodyweight and survival are reported for various times following birth as given in the original studies. TRVs are calculated for kit bodyweight at birth, but not for later times, because the database for later times is smaller than for

⁶¹ Depuration is the elimination of chemicals from an animal after the cessation of exposure, through metabolic conversion and/or excretion.

bodyweight at birth. Kit survival was reported for 4 to 6 weeks following birth in the studies used for TRV derivation.

For chicken PCB studies, the toxicity endpoints include egg productivity, egg fertility, hatchability, chick bodyweight, chick survival, and chick deformity.

Data Conversions

The mink dietary PCB concentrations are as given in the original studies when available. Two studies expressed the exposure in terms of daily ingestion (mg PCB/mink/d), instead of dietary concentration (Brunström, et al. 2001; Kihiström, et al. 1992). The dietary concentration is calculated by dividing the daily PCB ingestion by the daily food ingestion reported in each study. For some of the study results, the reported data are converted to make them consistent with the toxicity endpoints assessed in this effort. For example, if the number of live kits per mated female is not given in the original study, it is calculated by multiplying the number of live kits per whelped female by the fraction of females whelped of those mated.

The chicken dietary PCB concentrations are converted to bodyweight-normalized doses by multiplying by the food ingestion rate reported in the study, or by a default leghorn hen food ingestion rate of 0.067 kg feed/kg_{BW}-d (Medway and Kare 1959).

Adjustment for Long-term Effects in Mink

An additional modification was made for the mink TRVs only. Two mink feeding studies, one performed with Clophen A50-supplemented feed and one with field-contaminated prey, reported the reproductive effects of PCBs associated with exposures over both one and two breeding seasons, and the latter study also reported the reproductive effects in two generations of exposed females. Both studies showed increasingly adverse effects in the second year or generation of continuous exposure. Since only single-season exposures have been reported for commercial Aroclor feeding studies, TRVs protective for long-term occupancy of a site by female mink are calculated by multiplying the single-season Aroclor TRVs by the mean ratio of the Clophen A50 and field-contamination TRVs for exposure over two breeding seasons or generations divided by the corresponding TRVs for single-season exposure in the same studies.

The effects of one and two breeding seasons exposure to Clophen A50 on the number of live kits per mated female are shown in Figure A5⁶², and the effects of exposure to field-contaminated prey are shown in Figure A6 for one and two breeding seasons duration and through two generations exposure. Kit bodyweight and kit survival show similar, but less pronounced, responses as number of live kits per mated female (not shown as figures, see Table A5). The relative responses of the various treatments are shown in Table A6.

⁶² A similar figure is given in Calabrese (2005) (his Figure 4-12), although Kihiström, et al. (1992) is not cited for the 10 mg/kg treatment in the on-line bibliography. The Figure 4-12 control concentration is shown as an order of magnitude lower than reported by Brunström, et al. (2001), and the Brunström, et al. (2001) control is instead shown as one of the treatments. Calabrese (2005) also did not identify the solid line in his Figure 4-12 as first year results and the dotted line as second year results.

The results of the Clophen A50 study by Brunström, et al. (2001) are combined with a single high-exposure, one-breeding season exposure Clophen A50 treatment in a study by Kihiström, et al. (1992). All the remaining data in Figure A5 are reported by Brunström, et al. (2001).

TRV calculations are shown in Table A5, along with the ratios of the TRVs for continuous PCB exposure over 2 breeding seasons or 2 generations divided by the respective TRVs for PCB exposure over a single breeding season. The number of live kits per mated female is the most responsive endpoint to the PCB exposure duration—with 2 breeding seasons or 2 generations exposure, the low effect TRVs decrease to approximately 30 to 40 % of the values of the single breeding season exposure TRVs. Kit bodyweight or kit survival are less affected, with multiple seasons or generations exposure TRVs ranging from 40 to 90 % of the single breeding season exposure TRVs.

Although the FERA mink TRVs are based on number of live kits per mated female, the adjustment for long-term exposure is based on the mean ratio (0.5) for all three endpoints (live kits per mated female, kit bodyweight, and kit survival). This was done to make full use of the available information on the effect of PCB exposures through multiple breeding seasons and generations on mink responses.

Studies of other mammals besides mink that show similar increases in toxicity with exposures to PCBs or dioxin over multiple breeding seasons or generations are discussed in FERA 4.2.2.2. See FERA Appendix E1 for comparisons with mink PCB TRVs at other Superfund sites—the adjusted mink low effect TRV in the FERA is similar to that at the Fox River Superfund Site (which was externally peer-reviewed), and is less conservative (higher) than at the Hudson River Superfund site (also externally peer-reviewed). The adjusted mink TRVs in the FERA are lower than the NOAEC and LOAEC TRVs based on a mink feeding study performed for the Housatonic River Superfund site (Appendix E1), but are consistent with the confidence intervals of the LC_{10} and LC_{20} calculated by probit analysis. The Housatonic study was not carried through 2 breeding seasons or generations, so may underestimate the severity of long-term exposures (see FERA 4.2.2.1).

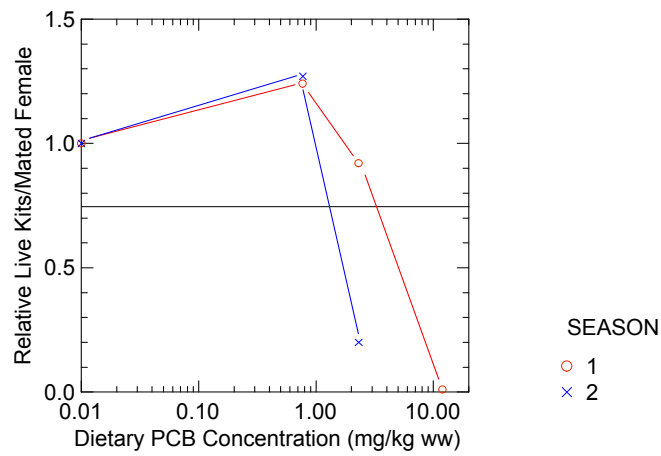


Figure A5. Live Kits per Mated Female Mink Exposed to Commercial Clophen A50 for Single and Multiple Breeding Seasons (Brunström, et al. 2001; Kihlström, et al. 1992)

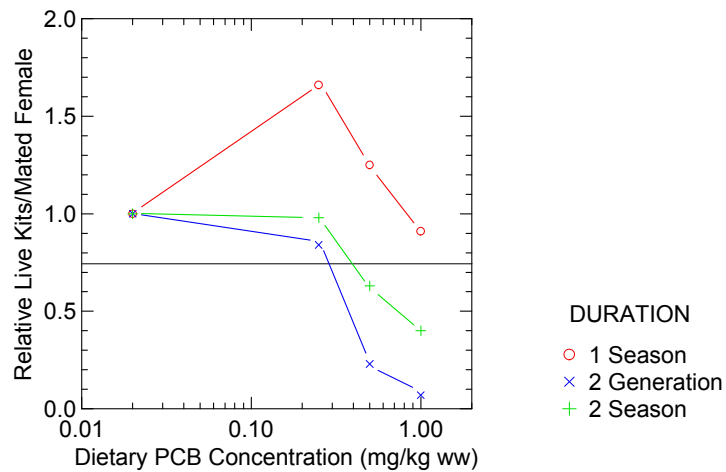


Figure A6. Live Kits per Mated Female Mink Exposed to Field-contaminated Fish for Single and Multiple Breeding Seasons or Generations (Restum, et al. 1998)

Table A5. Comparison of 1 Breeding Season Exposure to PCBs versus 2 Breeding Seasons or Generations Continuous Exposure in Mink

Chemical or Field study author	Response	Exposure Duration	Control RR	Treatment conc < TRV		Treatment conc > TRV		Target		P TRV	Effect level	Study
				conc	RR	conc	RR	RR				
				M _i ratio	C _j ppm	M _j ratio	C _{j+1} ppm	M _{j+1} ratio	ratio			
A50	live kit/ mated ♀	1 season	1	2.31	0.92	12	0	0.75	3.13	low effect	Brunstm01, Kihistm92	
		1 season	1	2.31	0.92	12	0	0.9	2.39	no effect	Brunstm01, Kihistm92	
		2 season	1	0.77	1.27	2.31	0.2	0.75	1.31	low effect	Brunstm01	
		2 season	1	0.77	1.27	2.31	0.2	0.9	1.13	no effect	Brunstm01	
	Ratio 2 season / 1 season		0.47 no effect									
	Ratio 2 season / 1 season		0.42 low effect									
	Restum	live kit/ mated ♀	1 season	1	1	0.91			0.75	>1.00	low effect	Restum98
2 season			1	0.25	0.98	0.5	0.63	0.75	0.39	low effect	Restum98	
2 generation			1	0.25	0.84	0.5	0.23	0.75	0.28	low effect	Restum98	
Ratio 2 season / 1 season		<0.39 low effect										
Ratio 2 generation / 1 season		<0.28 low effect										
Restum		kit bodywt	1 season	1	1	0.77			0.75	1.00	low effect	Restum98
	2 season		1	0.5	0.79	1	0.74	0.75	0.87	low effect	Restum98	
	2 generation		1	0.25	0.87	0.5	0.69	0.75	0.40	low effect	Restum98	
	Ratio 2 season / 1 season		0.87 low effect									
	Ratio 2 generation / 1 season		0.40 low effect									
	Restum	kit survival	1 season	1	0.25	0.93	0.5	0.72	0.75	0.45	low effect	Restum98
2 season			1	0.25	0.95	0.75	0.11	0.75	0.32	low effect	Restum98	
2 generation			1	0.25	0.8	0.5	0.18	0.75	0.26	low effect	Restum98	
Ratio 2 season / 1 season		0.72 low effect										
Ratio 2 generation / 1 season		0.58 low effect										
Mean ratio 2 season or 2 generation / 1 season		0.52 low effect (all studies)										

Notes for Table A5.

bodywt - bodyweight

Kit bodyweight is for birth to 1 week age.

Study - Brunstm01–Brunström, et al. (2001); Kihistm92–Kihiström, et al. (1992); Restum98–Restum, et al. (1998).

A1254 live kit/mated 1 season M_j of 0.92 is the mean of 1.15 (Wren87) and 0.69 (Aulerich77) both at 1 mg/kg dietary concentration.Restum kit survival 2 season M_j of 0.11 at C_j of 0.75 are the means of 0.05 and 0.16 (M_j) at 0.5 and 1.0 (C_j), respectively.

See notes to Table A2.

Table A6. Mink PCB Toxicity Studies with Exposures Over 1 Breeding Season and 2 Breeding Seasons or 2 Generations										
Study	Exposure				Relative Response Compared to Control					
	PCB & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
Brunström, et al. 2001	Clophen A50; product	6	0.77 (control 0.01)		0.96	1.20	1.30	1.24	0.99 birth	
			2.31		0.97	1.04	0.95	0.92	0.82 birth	
		18	0.77	11 lipid muscle (control <1)	0.95	1.22	1.34	1.27	0.90 birth 0.69 2 wk 0.67 5 wk	0.49 2 wk
Kihström, et al. 1992	Clophen A50; product	3	2.31	54	0.42	0.80	0.45	0.20	0.75 birth	0 2 wk
			12	181 fat 4.0 muscle	0.11	0.12	0	0		

Table A6. Mink PCB Toxicity Studies with Exposures Over 1 Breeding Season and 2 Breeding Seasons or 2 Generations										
Study	Exposure				Relative Response Compared to Control					
	PCB & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
		month	mg/kg diet	mg/kg	ratio					
Restum, et al. 1998, Shipp, et al. 1998, and Tillitt, et al. 1996 (reporting the same study)	PCB - sum of Aroclors 1242, 1248, 1254, and 1260; Field - Saginaw Bay carp	6 month (P ₁ 1992)	0.25 mg/kg (control 0.02 mg/kg)		1.36	1.16	1.19	1.66	0.93-0.94 birth 0.75-0.89 3 wk 0.75-0.85 6 wk	1.06 3 wk 0.93 6 wk
			0.5 mg/kg		1.35	1.02	0.91	1.25	0.84-0.87 birth 0.67-0.75 3 wk 0.65-0.68 6 wk	0.81 3 wk 0.72 6 wk
			1.0 mg/kg		1.16	1.02	0.77	0.91	0.75-0.79 birth 0.51-0.59 3 wk 0.35-0.49 6 wk	0.32 3 wk 0.32 6 wk
		16 month (P ₁ 1993)	0.25 mg/kg	0.98 mg/kg liver (control 0.07 mg/kg)	1.02	0.95	0.96	0.98	0.88-1.09 birth 0.87-0.91 3 wk 0.92 6 wk	0.99 3 wk 0.95 6 wk

Table A6. Mink PCB Toxicity Studies with Exposures Over 1 Breeding Season and 2 Breeding Seasons or 2 Generations										
Study	Exposure				Relative Response Compared to Control					
	PCB & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
		month	mg/kg diet	mg/kg	0.78	0.92	0.80	0.63	0.77-0.81 birth 0.65-0.67 3 wk 0.93 6wk	0.62 3 wk 0.05 6 wk
			0.5 mg/kg	0.89 mg/kg liver	0.66	0.63	0.59	0.40	0.73-0.74 birth 0.50-0.59 3 wk 0.60-0.66 6 wk	0.15 3 wk 0.16 6 wk
			1.0 mg/kg	1.57 mg/kg liver						
		12 month F ₁ of 6-month exposed parents (F ₁ -1 1993)	0.25 mg/kg	0.63 mg/kg liver (control 0.02 mg/kg)	0.85	1.05	0.96	0.84	0.87 birth 1.03-1.10 3 wk 0.89-0.95 6 wk	0.76 3 wk 0.80 6 wk
			0.5 mg/kg	0.96 mg/kg liver	0.76	0.88	0.31	0.23	0.64-0.73 birth 0.42 3 wk 0.54 6 wk	0.16 3 wk 0.18 6 wk

Table A6. Mink PCB Toxicity Studies with Exposures Over 1 Breeding Season and 2 Breeding Seasons or 2 Generations										
Study	Exposure			Relative Response Compared to Control						
	PCB & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
		month	mg/kg diet	mg/kg	ratio					
			1.0 mg/kg	1.47	0.63	0.53	0.09	0.07	0.51-0.60 birth	0 3 wk

Notes for Table A6.

Relative Response Compared to Control = treatment response / control response

Source: product is commercial product mixed with food; field is field-contaminated biota prepared as food

Exposure Duration additional information for the Restum, et al. 1998 study:

P₁ - parental generation

F₁ - offspring of P₁

Date - calendar year in which reproductive effects were assessed for a particular treatment

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