Canadian Tissue Residue Guidelines for the Protection of Wildlife Consumers of Aquatic Biota

POLYCHLORINATED BIPHENYLS (PCBs)

Polychlorinated biphenyls (PCBs) is the generic term for a group of 209 structurally similar compounds (congeners) that contain up to 10 chlorine atoms in a biphenyl ring (Tanabe 1988). Commercially, PCBs were produced worldwide as complex mixtures of chlorobiphenyls under a variety of trade names, including Askarel, Clophen, Kanechlor, and Phenoclor. All PCBs manufactured in North America were manufactured by Monsanto Co. under the trade name Aroclor. Typically, the numerical part of the name provides information about the composition of the PCB mixture. For example, Aroclor 1254 contains 54% chlorine by weight. These technical formulations were used most extensively in closed electrical systems as dielectric fluids. Other applications included plasticizers, heat transfer fluids, hydraulic fluids, lubricants, wax extenders, and special adhesives (CCREM 1986). In Canada, PCBs are currently used only in closed electrical equipment manufactured before 1980 (Strachan 1988).

Approximately 1.2×10^6 t of PCBs were produced worldwide between 1929 and 1977, the time commercial production was banned in North America. Of this total, more than half (635 000 t) was produced in North America. Though never manufactured in Canada, approximately 40 000 t were imported into the country from the United States (CCREM 1986). At present, only 24 300 t of this amount are accounted for, either in storage for disposal or in active use, mostly in electrical transformers. Thus, 40% of all PCBs imported to Canada are unaccounted for and are assumed to have been released to the environment (CCREM 1987).

PCBs continue to threaten wildlife due to their persistence and ubiquitous distribution. They also continue to enter Canada through long-range transport and leakage from improper disposal containers and aging transformers. The purpose of the Canadian tissue residue guidelines (TRGs) is to provide benchmark levels of PCBs in aquatic life above which their mammalian and avian predators may be at risk of consuming PCB concentrations known to result in adverse affects.

Development of a PCB TRG requires consideration of many factors. As previously mentioned, PCB congeners vary in chemical and physical properties, the most important of which for biota are the octanol–water partition coefficient (K_{ow}), rate of metabolism, and toxicity. The K_{ow} estimates a chemical's ability to partition into the lipid fraction in an organism. PCBs have log K_{ow}s ranging from 4.3 to 8.26, with a bell-shaped lipid partition curve (Hawker and Connell 1989). High-K_{ow}s partition into lipid less than expected because their large molecular size impedes passage across biological membranes (Shaw and Connell 1984; Gobas et al. 1986). Therefore, those PCBs with mid-K_{ow}s are most likely to accumulate in aquatic organisms and their predators. Some PCBs have a chemical structure conducive to metabolism (e.g., PCB 77) (see Table 2 for the IUPAC nomenclature) and are thus not expected to accumulate, while others (e.g., PCB 157) may have elimination half-lives of >1000 d (Niimi and Oliver 1983; Tillitt et al. 1996). Combined, differences in rates of uptake, elimination, and metabolism of the various chemical components among species translate into PCB profiles in organisms that are greatly different from the technical PCB mixtures originally released into the environment, a phenomenon termed biological weathering. Abiotic chemical weathering, due to differences in solubility and rates of volatilization, degradation, and hydrolysis of the individual components of the mixture, also alters the PCB characterization in sediment and water, and thus the quality and quantity of PCBs available for biological uptake. The result is that certain PCBs may be preferentially retained as they move through the trophic levels, while others are lost (Jones et al. 1993). In general, PCBs that are persistent in environmental samples are also the most toxic. Notably, environmental samples with comparable levels of total PCB concentrations may differ widely in toxic potential depending on the profile of individual congeners in the sample. Toxicities of individual congeners vary by a factor of 10 000 (Ahlborg et al. 1994).

Table 1. Canadian tissue residue guideline for PCBs for the protection of wildlife consumers of aquatic biota (Environment Canada 1998).

Compound	Guideline value (ng TEQ·kg^{-1} diet ww)*	
PCBs	mammalian	0.79
	avian	2.4

*TEQ is total dioxin toxic equivalents. See text for details.

Toxicity

Mode of Action

Non-*ortho* and mono-*ortho* substituted congeners (i.e., co-planar PCBs) have a similar mode of action as 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (TCDD). These compounds are potent ligands and activators of the arylhydrocarbon (*Ah*) receptor, but linkages between enzyme induction and PCB-specific organ toxicity are unclear (Brouwer 1991). Some hydroxy (-OH) metabolites of coplanar PCBs disrupt the homeostasis of hepatic vitamin A, an important vitamin in tissue development, reproduction, and resistance to disease (Smit et al. 1996). The toxic mode of action for non-coplanar PCBs is not well understood.

Mammalian Toxicity

Although acute exposure to PCBs is generally not regarded as a serious problem, various adverse effects have been recorded in laboratory tests. In mammals, symptoms of acute PCB toxicity include, but are not limited to, anorexia, weight loss, lethargy, internal bleeding, liver enlargement, porphyria, reproductive impairments, and death. From the data examined, it is apparent that the various manufactured PCB products exhibit variable acute toxicities. For example, Fishbein (1974) reported that the acute oral LD_{50}s of formulated PCBs in rats range from 3980 $mg \cdot kg^{-1}$ bw for Aroclor 1221 to 11 000 $mg \cdot kg^{-1}$ bw for Aroclor 1248. The 14-d LD_{50}s for Aroclor 1242 and Aroclor 1254 in mink are similar: >3000 and 4000 $mg \cdot kg^{-1}$ bw, respectively (Aulerich and Ringer 1977).

Wildlife most commonly endure chronic, low-level exposure to PCBs. In laboratory studies, chronic exposure to PCBs elicits various adverse responses in mammalian receptors, ranging from acne to death. Toxic effects of numerous mammalian species were examined, including mink, monkeys, pigs, ferrets, and rats. In general, mink and monkeys proved to be the most sensitive species to chronic dietary exposure to PCBs. Death may occur at relatively low doses during long-term exposure. For example, a mortality rate of 67% was observed in mink *(Mustela vison)* fed 1.5 mg Aroclor 1242·kg^{-1} bw per day for 247 d; a mortality rate of 71% was observed in mink fed 1.5 mg Aroclor 1254·kg^{-1} for 281 d (Aulerich and Ringer 1977; Bleavins et al. 1980). Changes in liver weight, total body weight, and growth rate are sublethal responses that are commonly reported for chronic exposure to PCBs. Increased liver weights were observed in female rabbits within 28 d of consuming 10 mg Aroclor 1254·kg^{-1} bw per day (Villeneuve et al. 1971). Female mink dosed with 2.5 mg Aroclor 1254·kg^{-1} diet (0.4 $mg \cdot kg^{-1}$ bw per day) for 88–102 d had significantly greater liver weights than control animals, though body weight gain did not significantly vary between the two groups (Aulerich et al. 1985). Significant weight loss was reported for male and female mink dosed with 7.4 $mg \cdot kg^{-1}$ diet (0.8 $mg \cdot kg^{-1}$ bw per day) and 26.3 $mg \cdot kg^{-1}$ diet (4.1 $mg \cdot kg^{-1}$ bw per day), respectively, for 28 d with Aroclor 1254 (Hornshaw et al. 1986). Rhesus monkeys *(Macaca mulatta)* are very sensitive to nonlethal effects of PCBs. Two months of ingesting 0.1–0.2 mg Aroclor 1248·kg^{-1} bw per day resulted in hair loss, acne, and swelling of the eyelids (Barsotti et al. 1976).

Reproductive and development impairment are the most sensitive endpoints in mammals, with mink and monkeys again being the most sensitive. Complete reproductive failure in mink has been observed when dosed with 5–40 $mg \cdot kg^{-1}$ diet of Aroclor 1242 or Aroclor 1254 (Aulerich and Ringer 1977; Jensen et al. 1977; Bleavins et al. 1980; Aulerich et al. 1985). Ferrets are more tolerant than mink to Aroclor 1242, with reproductive failure occurring at 20 $mg \cdot kg^{-1}$ diet (Bleavins et al. 1980). Administration of 0.025 $mg \cdot kg^{-1}$ bw per day of Clophen A-60 for 400 of 489 d significantly reduced the number of female mink that whelped in the second year, but not the total number of kits per female (den Boer 1984). A high dose level of 2.025 $mg \cdot kg^{-1}$ bw per day for 51 d resulted in 40% mortality, and all females failed to reproduce (den Boer 1984). Although male fertility, whelping success, and fecundity were not impacted, growth of kits nursed for 3–5 weeks by mink fed diets containing 1 $mg \cdot kg^{-1}$ of Aroclor 1254 (0.15 $mg \cdot kg^{-1}$ bw per day) was significantly reduced relative to controls (Wren et al. 1987).

Female rhesus monkeys fed diets containing 2.5 or 5.0 $mg \cdot kg^{-1}$ of Aroclor 1248 6 months before conception, during gestation, and 3 months after delivery (15 months total) had higher abortion rates and greater early infant mortality than the control group (Allen and Barsotti 1976; Barsotti et al. 1976). Infants had low birth weights, were small in stature, and had hyperpigmentation of the skin. Female rhesus monkeys dosed with 0.08 mg Aroclor 1254·kg^{-1} bw per day for 3 years before breeding, with doses continuing through gestation until infants were 7 weeks old, showed a significantly greater incidence of fetal mortality (Arnold et al. 1995). Lower conception rates were observed at doses of 0.02, 0.04, and 0.08 $mg \cdot kg^{-1}$ bw per day (Arnold et al. 1995). Pregnant cynomolgus monkeys *(M. fascicularis)* administered a dose of 0.1 $mg \cdot kg^{-1}$ bw per day of Aroclor 1254 for 3 d per week for 238 d experienced 100% fetal mortality (Truelove et al. 1982).

A few novel studies have tried to account for weathering of PCB technical mixtures by incorporating meat (e.g., fish) contaminated with a known amount of PCBs into the diets of test organisms. Although studies such as these provide insight into the overall toxicity of natural food sources, they were not considered for guideline derivation because of the presence of contaminants other than PCBs in the diet items. Mink fed diets containing contaminated carp from the Great Lakes, giving an Aroclor 1254 concentration of 1.5 $mg \cdot kg^{-1}$ in the feed, failed to reproduce (Hornshaw et al. 1983). Mink fed diets containing perch (0.69 mg $PCBs \cdot kg^{-1}$ diet), sucker (0.63 mg $PCBs \cdot kg^{-1}$ diet), and whitefish (0.48 mg $PCBs \cdot kg^{-1}$ diet) for 7 months exhibited impaired reproductive performance and/or kit survival compared to controls (Hornshaw et al. 1983). Only mink on alewife diets (0.21 mg $PCB \cdot kg^{-1}$) reproduced as well as the controls.

In a similar study, mink were fed diets containing 0, 10, 20, or 40% carp (with known amounts of PCBs) from Lake Michigan before and throughout the reproductive period (12 weeks total). Mink consumed, on average, 0.004, 0.13, 0.26, and 0.32 mg $PCBs \cdot kg^{-1}$ bw per day, respectively (Heaton et al. 1995). Percent kit survival to 6 weeks (weaning) was 85, 28, 11.5, and 0% for the 0, 10, 20, and 40% carp diets, respectively. There was a significant inverse dose-dependent response between weights of kits and proportion of carp in the maternal diet, with 20 and 40% carp diet groups significantly different from the control. Relative organ weights of kits whelped and nursed by treated females were generally less than those of the control group. Reproductive success of common seals *(Phoca vitulina)* fed diets containing fish from the Wadden Sea (1.5 mg $PCB \cdot d^{-1}$) for 2 years was significantly impaired compared to those fed fish from the Atlantic (0.22 mg $PCBs \cdot d^{-1}$) (Reijinders 1986; Brouwer et al. 1989).

The ability of PCBs to promote carcinogenesis appears to be generally restricted to the liver. For example, weanling Sherman rats fed 5 $mg \cdot kg^{-1}$ bw per day of Aroclor 1260 for a period of 21 months had increased incidences of neoplastic nodules, liver lesions, and hepatocellular carcinomas compared to the control group (Kimbrough et al. 1975). In contrast, short-term exposure to PCBs may have a negative effect on cancer promotion (Kerkvliet and Kimeldorf 1977).

Limited information was located on the effects of PCBs on mammalian immune systems. Nevertheless, available data suggest that PCBs have the potential to compromise immuno-competence. Marked thymus atrophy was observed in rabbits fed 0.18 mg Aroclor 1254 $\cdot kg^{-1}$ bw per day for 8 weeks (Street and Sharma 1975). Pregnant cynomolgus monkeys administered 0.1 or 0.4 $mg \cdot kg^{-1}$ bw per day of Aroclor 1254 for approximately 11 months exhibited a reduced response to sheep erythrocyte antigens (Truelove et al. 1982).

Avian Toxicity

It appears that birds are slightly less sensitive than mammals to the effects of PCBs. Nonetheless, the types of effects that are associated with acute and chronic exposures to PCBs are similar, including lethality, reduced growth rates, liver enlargement, and reproductive impairment.

Acute and subacute toxicities are similar among various PCB technical mixtures. For example, 5-d LC_{50}s for ring-necked pheasants *(Phasianus colchicus)* are 1312, 1091, and 1260 $mg \cdot kg^{-1}$ diet for Aroclor 1248, Aroclor 1254, and Aroclor 1260, respectively (Heath et al. 1972). Decreased survival and growth are frequently observed in avian species chronically exposed to relatively high levels of PCBs. For example, ring-necked pheasants fed 20 mg Aroclor 1254 $\cdot d^{-1}$ died, on average, within 46 d, whereas all but one of those receiving placebos survived until the end of the experiment (8 months) (Dahlgren et al. 1972). Growth rates are reduced in white leghorn *(Gallus domesticus)* cockerels fed 12 mg Aroclor 1242 $\cdot kg^{-1}$ bw per day for 21 d (Flick et al. 1965).

PCBs adversely affect avian reproduction most commonly by reducing egg productivity, egg hatchability, and chick growth rates, though teratogenic effects have also been observed. Of the avian species tested, chickens have been shown to be very sensitive to the effects of chronic exposure to technical mixtures of PCBs. For example, eggs from white leghorn hens fed diets containing 20, 40, or 80 $mg \cdot kg^{-1}$ of Aroclor 1242 had significantly reduced hatchability (60, 23, and 0%, respectively) within 2 weeks of treatment (Britton and Huston 1973). Assuming that chickens consume approximately 6% of their body weight in food each day, this dietary concentration corresponds to an Aroclor 1242 consumption rate of 0.6 $mg \cdot kg^{-1}$ bw per day (CCME 1993). Similar results are reported by Lillie et al. (1974), wherein white leghorn egg production and hatchability were reduced by a maternal diet containing 1.2 $mg \cdot kg^{-1}$ of either Aroclor 1242, Aroclor 1248, or Aroclor 1254, but not by 0.12 $mg \cdot kg^{-1}$.

In a relatively recent study, white leghorn chickens were fed diets containing contaminated carp from Lake Michigan with PCB residues of 0.3 (control), 0.8 (low dose), or 6.6 mg PCBs·kg^{-1} diet (high dose). Deformity rates in embryos and chicks were 17, 24, and 40% in control, low, and high doses, respectively. Edema of the head/neck was the most common teratogenic effect (64%), followed by abdominal edema (15%) and foot/leg deformities (14%) (Summer et al. 1996). Results from this study must be viewed with caution as ≥75% of control and low dose and 15% of high dose hens had fatty liver hemorrhagic syndrome (FLHS).

Several authors have linked embryo deformities in field-collected eggs to PCB exposure (Kubiak et al. 1989; Tillitt et al. 1992; Yamashita et al. 1993; Giesy et al. 1994a [re-evaluated in Ludwig et al. 1996]). Deformity rates of double-crested cormorant *(Phalacrocorax auritus)* and caspian tern *(Hydroprogne caspia)* embryos from eggs collected from several locations in the Great Lakes from 1986 to 1991 averaged 8.6 and 11.4%, respectively. Rates were significantly correlated to total PCB and TEQ concentrations in the eggs. (TEQs are discussed in the following section.) The most common deformities were subcutaneous edema, gastroschisis,[†] and bill defects (Ludwig et al. 1996). The rate and extent of gross brain asymmetry in cormorant chicks collected as eggs from Lake Ontario (highly contaminated) and Crofton, British Columbia (moderately contaminated), were significantly greater than in birds from Lost Mountain Lake, Saskatchewan (minimally contaminated) (Henshel et al. 1997). Concentrations of TEQs (PCBs and dioxins/furans) or 2,3,7,8-TCDD could account for more of the variability in asymmetry measurements than could concentrations of individual PCB congeners.

Despite reducing egg hatchability, PCB technical mixtures seem to have little effect on the physical characteristics of eggs. For example, maternal dietary concentrations up to 80 mg·kg^{-1} for Aroclor 1242 and 20 mg·kg^{-1} for Aroclor 1248 and Aroclor 1254 do not alter white leghorn egg weight, eggshell thickness, or eggshell weight (Britton and Huston 1973; Lillie et al. 1974).

Toxic Equivalency Factors

Toxic equivalency factors (TEFs) have been developed to compare toxicities of environmental samples with different congener makeups (Van den Berg et al. 1998; Table 2). Contaminants are assigned TEFs based on their ability to induce a response in the cytochrome enzyme system relative to the most potent inducer, 2,3,7,8-TCDD. Within a sample, individual chemical concentrations are multiplied by their respective TEFs, and all products are summed to give a value expressed in toxic equivalency units (TEQs). This method takes into consideration the unique concentrations and toxicities of the individual components within a chemical mixture. This method, however, is unable to account for nonadditive interactions among different chemicals that are known to occur. It may be difficult to extrapolate TEFs between species due to differences in toxicokinetics. Furthermore, TEFs are currently available for only a few select PCB congeners. Thus the influence of many congeners, most notably non-coplanar PCBs, may go ignored. Despite these limitations, the use of TEQs improves correlations between PCB contamination and observed adverse effects (Giesy et al. 1994a, 1994b; Leonards et al. 1995). For this reason, TEQs form the basis of the PCB TRG.

Tissue Residue Guideline Derivation

Table 2. IUPAC numbering and toxic equivalency factors (TEFs) for selected PCB congeners (Environment Canada 1998).

		TEF[*]	
IUPAC No.	**Structure**	**Mammalian**	**Avian**
PCB 77	3,3',4,4'	0.000 1	0.05
PCB 81	3,4,4',5	0.000 1	0.1
PCB 126	3,3',4,4',5	0.1	0.1
PCB 169	3,3',4,4',5,5'	0.01	0.001
PCB 105	2,3,3',4,4'	0.000 1	0.000 1
PCB 114	2,3,4,4',5	0.000 5	0.000 1
PCB 118	2,3',4,4',5	0.000 1	0.000 01
PCB 123	2',3,4,4',5	0.000 1	0.000 01
PCB 156	2,3,3',4,4',5	0.000 5	0.000 1
PCB 157	2,3,3',4,4',5'	0.000 5	0.000 1
PCB 167	2,3',4,4',5,5'	0.000 01	0.000 01
PCB 189	2,3,3',4,4',5,5'	0.000 1	0.000 01

[*]1998 WHO TEF values (Van den Berg et al. 1998); see text for details.

The PCB TRG for the protection of wildlife that consume aquatic biota was developed according to the CCME protocol (CCME 1998).

[†]A syndrome characterized by an enlarged yolk sac attachment, organs outside the body, and a thin or absent body wall.

Guideline Derivation for PCBs

To develop the PCB TRG, concentrations of commercial mixtures (usually Aroclor formulations) in food were first converted to TEQs for acceptable toxicity studies. This conversion was done using published information about the chemical composition of the Aroclor mixtures and 1998 WHO TEFs (Duinker et al. 1988; Schulz et al. 1989; van den Berg et al. 1998). These latest TEFs differentiate between mammalian and avian receptors (Table 2). PCB congeners incorporated into the TEQ estimate are 77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, and 189. Table 3 lists the TEQ conversion factors derived for common commercial PCB mixtures.

Table 3. Toxic equivalent conversion factors for some PCB commercial mixtures (Environment Canada 1998).

	Conversion factor (ng TEQ·mg^{-1} product)*	
Mixture	**Mammalian TEFs**	**Avian TEFs**
Aroclor 1242	5.1	234.6
Aroclor 1248	12.8	251.3
Aroclor 1254	30.1	44.5
Aroclor 1260	11.3	25.5
Clophen A50	97.3	365.1
Clophen A60	470.7	476.9

* TEQ is total dioxin toxic equivalents calculated using 1998 WHO TEF values (Van den Berg et al. 1988); see text for details.

From daily diet doses of PCB-based TEQs, a TDI was calculated for each study as the geometric mean of the LOAEL and NOAEL. According to the protocol (CCME 1998), when the NOAEL is not determined, it may be estimated by dividing the LOAEL by 5.6. There is no evidence to suggest that this relationship does not hold for PCB toxicity data (Environment Canada 1998). For the purposes of deriving a national value, the mammalian and avian RCs must be as inclusive as possible to accommodate all species and regions in Canada. Therefore, they are based on the highest mammalian and avian food intake:body weight ratios (FI:BW) known for Canadian wildlife, namely 0.24 for female mink and 0.94 for Wilson's storm petrel (CCME 1998). These RC values apply to freshwater, marine, and estuarine systems. It is recognized that use of the highest FI:BW ratio may not always be appropriate (e.g., in areas where Wilson's storm petrel is not found). For this reason, RCs for a suite of mammalian and avian receptors have been calculated (Environment Canada 1998).

Mammalian Reference Concentration

The lowest RC for mammalian species (0.79 ng TEQ·kg^{-1} diet) is derived from a study in which male and female mink were fed diets containing 0 or 1 mg Aroclor 1254·kg^{-1} diet for 185 d. Although male fertility, whelping success, and fecundity were not affected, growth of kits nursed for 3 and 5 weeks by mink fed diets containing 1 mg·kg^{-1} of Aroclor 1254 was reduced significantly by ~25–30% compared to controls (Wren et al. 1987). Assuming an FI:bw ratio of 0.15 mg·kg^{-1} bw for captive mink, 1 mg Aroclor 1254·kg^{-1} diet corresponds to a dietary consumption rate of 0.15 mg Aroclor 1254·kg^{-1} bw per day. On a TEQ basis, this concentration is 4.5 ng TEQ·kg^{-1} bw per day and represents the LOAEL. The NOAEL (0.8 ng TEQ·kg^{-1} bw per day) was calculated by dividing the LOAEL by 5.6. The geometric mean of the LOAEL and the NOAEL divided by an uncertainty factor of 10 gives a TDI of 0.19 ng TEQ·kg^{-1} bw per day. An uncertainty factor of 10 was chosen to accommodate differences in interspecies sensitivities to PCBs. An RC of 0.79 ng TEQ·kg^{-1} diet ww was obtained by dividing the TDI for mink by the highest FI:bw ratio for wild mammals (0.24) (CCME 1998).

Avian Reference Concentration

The most sensitive results from an acceptable study found that white leghorn chicks from hens fed 0.12 mg Aroclor 1254·kg^{-1} bw per day experienced a 10% reduction in growth rate (Lillie et al. 1974). This dose corresponds to a LOAEL of 5.3 ng TEQ·kg^{-1} bw per day, assuming Aroclor 1254 contains 44.55 ng TEQ·mg^{-1} product. A NOAEL of 0.95 ng TEQ·kg^{-1} bw per day was calculated by dividing the LOAEL by 5.6 (CCME 1998). A TDI of 2.3 ng TEQ·kg^{-1} bw per day was derived from the geometric mean of the LOAEL and NOAEL. No uncertainty factor was incorporated as evidence indicates that white leghorn chickens may be inherently 10–1000 times more sensitive to TEQ exposure than wildlife species (Kennedy et al. 1996). For example, calculated TDIs as high as 211 ng TEQ·kg^{-1} bw per day (100-fold greater than the critical study) resulted in only minor effects in American kestrel and Japanese quail (Elliott et al. 1990, 1991; Hoffman et al. 1996; see Environment Canada 1998). Also, consultations with avian experts from the Canadian Wildlife Service corroborated the fact that the leghorn chicken is a particularly sensitive species and perhaps not fully representative of all avian species (L. Brownlee,

G. Fox, S.W. Kennedy, and R.J. Norstrom 1998, Canadian Wildlife Service, Environment Canada, Hull, Quebec, pers. com.). Dividing the lowest TDI (2.3 ng TEQ·kg^{-1} bw per day) by the highest FI:bw for wild birds (0.94) results in an avian RC of 2.4 ng TEQ·kg^{-1} diet ww.

PCB Tissue Residue Guideline

The lowest RC between the mammalian and avian values, 0.79 ng TEQ·kg^{-1} diet ww, is adopted as the PCB TRG. The guideline refers to the TEQ concentration due to PCBs found in an aquatic organism on a wet weight basis that is not expected to result in adverse effects on wildlife consuming these aquatic organisms (Environment Canada 1998). The assumption is that by capturing the dioxin-like toxicity of the non- and mono-*ortho*-PCBs (i.e., coplanar congeners), the TRG will also be protective of the non-coplanar congeners.

In environments where both PCBs and polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) contribute significantly to the TEQ concentration in aquatic prey items, the reader is advised to consider also the Canadian TRG for PCDDs and PCDFs, which is currently under development. It is recommended that the lower value between the PCB and PCDD/PCDF TRGs take precedence as the total TEQ concentration (i.e., PCB and PCDD/PCDF TEQs combined) in aquatic biota to protect wildlife consumers against the common mode of action shared by these chemicals.

TEQ Levels in the Canadian Environment

TEQs for several species of aquatic organisms based on 1998 WHO mammalian TEFs range from 0 to 264 ng·kg^{-1} ww. Almost 70% of the values fall below the guideline of 0.79 ng TEQ·kg^{-1}. Using avian TEFs, approximately 73% of the values fall below this guideline value, with a maximum of 446 ng·kg^{-1} ww. In general, invertebrates and fish from the Great Lakes are most contaminated, while fish from Alberta and bivalves from the Atlantic coast contain very low levels of TEQs (Environment Canada 1998).

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