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***Canadian Environmental Protection Act, 1999***  
**Federal Environmental Quality Guidelines**  
***Polybrominated Diphenyl Ethers (PBDEs)***

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## Introduction

Federal Environmental Quality Guidelines (FEQGs) provide benchmarks for the quality of the ambient environment. Where the FEQG is met there is low likelihood of adverse effects on the protected use (e.g., aquatic life or the wildlife that may consume them). They are based on the toxicological effects or hazards of specific substances or groups of substances and do not take into account analytical capability or socio-economic factors. FEQGs serve three functions: first, they can be an aid to prevent pollution by providing targets for acceptable environmental quality; second, they can assist in evaluating the significance of concentrations of chemical substances currently found in the environment (monitoring of water, sediment, and biological tissue); and third, they can serve as performance measures of the success of risk management activities. The use of FEQGs is voluntary unless prescribed in permits or other regulatory tools. Thus FEQGs, which apply to the ambient environment, are not effluent limits or “never-to-be-exceeded” values but may be used to derive effluent limits. The development of FEQGs is the responsibility of the Federal Minister of Environment under the *Canadian Environmental Protection Act, 1999*. The intent is to develop FEQGs as an adjunct to risk assessment/risk management of priority chemicals identified in the Chemicals Management Plan (CMP) or other federal initiatives. This factsheet describes the Federal Environmental Quality Guidelines for water, sediment and biological tissue to protect aquatic life and mammalian and avian consumers of aquatic life from adverse effects of polybrominated diphenyl ethers (PBDEs) present in some commercial products (Table 1).

**Table 1. Federal Environmental Quality Guidelines for Polybrominated Diphenyl Ethers (PBDEs)**

Homologue*	Congener	Water (ng/L)	Fish Tissue (ng/g ww)	Sediment ** (ng/g dw)	Wildlife Diet <sup>†</sup> (ng/g ww food source)	Bird Eggs (ng/g ww)
triBDE	total	46	120	44	–	–
tetraBDE	total	24	88	39	44	–
pentaBDE	total	0.2	1	0.4	3 (mammal) 13 (birds)	29 <sup>‡</sup>
pentaBDE	BDE-99	4	1	0.4	3	–
pentaBDE	BDE-100	0.2	1	0.4	–	–
hexaBDE	total	120	420	440	4	–
heptaBDE	total	17 <sup>  </sup>	–	–	64	–
octaBDE	total	17 <sup>  §</sup>	–	5600 <sup>§</sup>	63 <sup>§</sup>	–
nonaBDE	total	–	–	–	78	–

decaBDE	total	–	–	19 <sup>§#</sup>	9	–
<p>*FEQG for triBDE (tribromodiphenyl ether), tetraBDE (tetrabromodiphenyl ether), hexaBDE (hexabromodiphenyl ether), heptaBDE (heptabromodiphenyl ether), nonaBDE (nonabromodiphenyl ether) and decaBDE (decabromodiphenyl ether) are based on data for the congeners: BDE-28, BDE-47, BDE-153, BDE-183, BDE-206, and BDE-209, respectively unless otherwise noted.</p> <p>**Values normalized to 1% organic carbon.</p> <p>†Applies to mammalian wildlife unless otherwise noted.</p> <p>‡ Value based on the commercial PentaBDE formulation, DE-71, which contains mostly pentaBDE and some tetraBDE.</p> <p>  Values based on commercial OctaBDE mixture DE-79, which is composed mainly of heptaBDE and octaBDE (octabromodiphenyl ether).</p> <p>§Values adopted from Ecological Screening Assessment Report (Environment Canada 2006). Sediment guidelines for octaBDE and decaBDE were adapted from the SAR by being corrected for the sediment organic carbon in the actual tests, then normalised to 1% organic carbon instead of the 4% in the SAR.</p> <p>#Values based on commercial decaBDE mixture which is composed mainly of nonaBDE and decaBDE.</p>						

### Substance Identity

PBDEs are anthropogenic organobromine compounds comprising a class of substances with 209 possible congeners, each with 1–10 bromine atoms. PBDEs are used typically as commercial mixtures. The nomenclature used in this factsheet to distinguish commercial mixtures from congeners follows the convention used by the Government of Canada (2010) (see glossary at the end of this factsheet). That is, the three commercial mixtures are referred to as commercial PentaBDE (Pentabromodiphenyl Ether), commercial OctaBDE (Octabromodiphenyl Ether) and commercial DecaBDE (Decabromodiphenyl Ether). These commercial mixtures are comprised of a number of homologues (compounds which have the same base structure but which differ from each other by the number of bromine atoms in the molecule). Congeners, by contrast, are compounds within a homologue series having the same base structure as well as the identical number and type of atoms and differing only in the position of the bromine atoms in the molecule. The individual congeners are identified by BDE and the congener number (e.g., BDE-100). Commercial PentaBDE is predominantly a mixture of pentaBDE, tetraBDE and hexaBDE congeners, but may also contain trace levels of heptaBDE and triBDE (tribromodiphenyl ether) congeners. Similarly, commercial OctaBDE formulation mixture composed mainly of heptaBDE, octaBDE and hexaBDE congeners, but may also contain small amounts of nonaBDE congeners and decaBDE. Current formulations of commercial DecaBDE are almost completely composed of decaBDE and a very small amount of nonaBDE (Environment Canada 2006). The commercial PentaBDE and commercial OctaBDE formulations were banned by the European Union in 2004, and California and Hawaii in 2006. In Canada, the commercial PentaBDE formulation has been out of use since May 2003 (Environment Canada 2004). In December 2006, PBDEs were added to the “List of Toxic Substances” under CEPA 1999, and tetra-, penta- and hexaBDEs were identified as persistent, bioaccumulative, and inherently toxic (PBiT). Environment Canada (2006) also concluded that tetra-, penta- and hexaBDE congeners

meet the criteria for persistence and bioaccumulation, as defined by the *Persistence and Bioaccumulation Regulations* of CEPA 1999 (Government of Canada 2000), and their presence in the environment results primarily from human activity; therefore, they meet the conditions set out in CEPA 1999 for mandatory addition to the Virtual Elimination List. In July 2008, the manufacturing of all PBDEs and the use, sale, offer for sale and import of tetra-, penta- and hexaBDE congeners were prohibited (Environment Canada 2009). Environment Canada (2010) further evaluated decaBDE in view of the new published information on accumulation of decaBDE in biota and its potential transformation to persistent and bioaccumulative products. Overall, available data did not show that the decaBDE itself meets the numeric criteria for bioaccumulation as defined in the *Persistence and Bioaccumulation Regulations*.

### Uses

PBDEs have been used mainly as additive (not chemically bound) flame retardants in polymer resins and plastics and, to a lesser extent, in adhesives, sealants and coatings. When under production, it has been estimated that approximately 90% or more of commercial PentaBDE produced globally was used in polyurethane foams in office and residential furniture, automotive upholstery, sound insulation, and wood imitation products (WHO 1994; European Communities 2001; RPA Ltd. 2000). Most commercial OctaBDE produced globally was added to polymers (mainly acrylonitrile butadiene styrene) (ABS), which were then used to produce business cabinets, pipes and fittings, automotive parts, and appliances (WHO 1994; European Communities 2003). Most commercial DecaBDE has been used in high-impact polystyrene, computer and television casings, general electrical/electronic components, cables, and textile back coatings (OECD 1994; European Communities 2002).

Various initiatives since 2001 have resulted in significant changes in the production and use of the PBDEs in Canada and elsewhere. The *PBDEs Regulations* in Canada were made under CEPA 1999 and came into force on June 19, 2008. In addition to prohibiting the manufacture of PBDEs (i.e., tetra-, penta-, hexa-, hepta-, octa-, nona- and decaBDE congeners), the regulations also prohibit the manufacture, use, sale, offer for sale and import of mixtures, polymers, and resins containing those PBDEs that meet the criteria for virtual elimination (i.e., tetra-, penta- and hexaBDE congeners) under CEPA 1999. The only U.S. manufacturer of commercial PentaBDE and commercial OctaBDE (Great Lakes Chemical Corporation) ceased production of these products in late 2004 (Great Lakes Chemical Corporation 2005; U.S. EPA 2005). ICL Industrial Products (2005) also announced the complete termination of their production and sale of commercial OctaBDE by the end of 2004. In addition, the European Union has implemented a prohibition on the marketing and use of commercial PentaBDE and commercial OctaBDE. As a result of negotiations with the U.S. EPA, the two U.S. producers of commercial DecaBDE (Albemarle Corp. and Chemtura Corp.) and the largest U.S. importer, ICL Industrial Products Inc., announced commitments to end production, importation, and sales of commercial DecaBDE for most uses in the U.S. by December 31, 2012, and to end all uses by the end of 2013. In addition, these three companies have voluntarily committed to cease exports of commercial DecaBDE into Canada according to the same phase-out schedule. Even though production of commercial PentaBDE and commercial OctaBDE

have ceased globally and production of commercial DecaBDE is scheduled for phase-out, it is expected that various PBDE congeners may be present in the environment either due to their release prior to phase-out or due to items produced before phase-out remaining in use for some time.

### **Fate, behaviour and partitioning in the environment**

PBDE flame retardants are not chemically bound to host materials, and are susceptible to leaching (Rahman et al. 2001). The physical properties of PBDEs vary with the level of bromination. In general, PBDEs found in the commercial products (i.e., those having 4 or more bromine atoms) have molecular weights between 486 to 959 g/mol, a physical appearance ranging from viscous liquid to crystalline powder, vapour pressures between  $2.95 \times 10^{-9}$  and  $4.69 \times 10^{-5}$  Pa, water solubilities of <0.1 to 13.3 µg/L, log  $K_{ow}$  values between 6.27 to 9.97, and Henry's law constants from 11 to >44 Pa·m<sup>3</sup>/mol (Environment Canada 2006). Although the log  $K_{ow}$  of PBDEs and polychlorinated biphenyls (PCBs) have similar ranges, their BCFs (bioconcentration factor) greatly differ and this difference in behaviour is not understood.

With their low vapour pressures, very low water solubilities and high octanol/water partition coefficients (log  $K_{ow}$ ), it is expected that PBDEs will tend to bind to the organic fraction of particulate matter, soils and sediments. Computer modelling indicates that pentaBDEs would partition to the greatest extent to sediments, followed by soils, water, and air where they are subject to atmospheric transport and deposition. Lower brominated PBDEs (tetra- and pentaBDEs), however, are slightly more water soluble and can volatilize and adsorb onto particulate matter in the atmosphere (Gouin and Harner 2003). Although to a lesser degree than lower brominated congeners, decaBDE can also be subject to atmospheric transport and deposition (Noël et al. 2009). Overall, the occurrence of specific PBDEs in organic phases and/or biota is dependent on their degree of bromination (Strandberg et al. 2001; Harner and Shoeib 2002).

Predicted half-lives for atmospheric degradation of tetra-, penta- and hexaBDE in air are 7.1, 19.4 and 30.4 d, respectively (Environment of Canada 2006). With respect to biodegradation, tetra-, penta- and hexaBDE are predicted to be "recalcitrant" by the BIOWIN program. Using the EPIWIN program, estimated half-lives for pentaBDE are 600 days in aerobic sediment, 150 days in soil and 150 days in water (Palm 2001). decaBDE has been demonstrated to be susceptible to some biodegradation under certain anaerobic conditions using sludge inoculum (Gerecke et al. 2005).

Higher brominated PBDEs can be debrominated. For instance, heptaBDEs and pentaBDEs can be debrominated to hexaBDEs and tetraBDEs, respectively, in the gut of carp (*Cyprinus carpio*) (Stapleton et al. 2004a,b; Stapleton and Baker 2003). More specifically, BDE-183 (a heptaBDE congener) can be debrominated to BDE-154 (a hexaBDE congener), and BDE-99 (a pentaBDE congener) can be debrominated to BDE-47 (a tetraBDE congener). It has also been inferred by the presence of congeners not present in commercial mixtures (e.g., BDE-187 and -202), that decaBDE present in

herring gull eggs from the Great Lakes can be degraded to lower brominated congeners either in the maternal bird or in its food web (Gauthier et al. 2008).

PBDEs can bioaccumulate and biomagnify in the environment (Table 2). Bioaccumulation, biota-sediment accumulation, and biomagnification factors (BAFs, BSAFs, BMFs) were calculated for PBDE congeners using the available data (Environment Canada unpublished data). The BAFs ranged from 0.3 L/g ww for PeBDE-99 to 6.1 L/g ww for BDE-100, whereas the BSAFs ranged from 3.9 ng/g dw for BDE-153 to 15.8 ng/g dw for BDE-100. All BMFs calculated for PBDEs are greater than 10 between consecutive trophic levels (Table 2). Because the BMFs presented here are the maximum reported values that may span several trophic levels, they provide a protective estimate of biomagnification.

**Table 2. Selected bioaccumulation, biota-sediment accumulation and biomagnification factors for various PBDE congeners used to derive fish tissue and sediment quality guidelines (Environment Canada unpublished data)**

Homologue	Congener	BAF <sup>1,2</sup> (L/g ww)	BSAF <sup>1</sup> (ng/g dw)	BMF <sup>1,3</sup> (lw <sup>4</sup> )
triBDE	28	2.5	10.5	
tetraBDE	47	3.6	8.9	95.9
pentaBDE	99	0.3	13.0	25.2
pentaBDE	100	6.1	15.8	
hexaBDE	153	3.5	3.9	134.8

<sup>1</sup>geometric mean of available values; sediment values were adjusted to 1% organic carbon where possible.  
<sup>2</sup>based on data for mysterysnail (*Cipangopaludina chinensis*), prawn (*Macrobrachium nipponense*), mud carp (*Cirrhinus molitorella*), crucian carp (*Carassius auratus*), northern snakehead (*Ophicephalus argus*) and water snake (*Enhydis chinensis*).  
<sup>3</sup>trophic relationship for BMFs of BDE-153 and -99 is from fish to piscivorous mammal, and amphipod to piscivorous mammal for BDE-47.  
<sup>4</sup>lw refers to lipid weight. The values were lipid normalized because lipid content is a major factor in determining partitioning and body burden.

### Ambient concentrations

PBDEs have been detected in all environmental media in Canada. The use of PBDEs is declining because most commercial mixtures containing these chemicals have been either voluntarily phased-out by manufacturers or are subject to restrictions in Canada and other countries. The maximum ambient air concentration of PBDEs as a group measured was 1.3 ng/m<sup>3</sup> in Ontario (Gouin et al. 2002), 2.2 ng/m<sup>3</sup> in the Yukon (Alaee et al. 2001; Bidleman et al. 2001), and 0.031 ng/m<sup>3</sup> in British Columbia (Noël et al. 2009). In water, the total PBDE concentration were measured in the range of 0.004-0.013 ng/L in Lake Ontario and the most abundant congener groups were tetraBDE and pentaBDE (Luckey et al. 2002). In the rain water of BC the maximum PBDEs concentration recorded was 26.76 ng/L near an urban site (Noël et al. 2009). In the sediment, 2.7–91 µg/kg of PBDEs was detected in British Columbia (Rayne et al. 2003). In sewage sludge, the total PBDE level ranges from 1700–8200 µg/kg dw in southern Ontario (Kolic et al. 2004; La Guardia et al. 2001). A dramatic rise in biota concentrations is evident in the last two decades (Environment Canada 2006). The level of PBDEs in west coast crustaceans ranged from 4.2 to 480 µg/kg lipid, from 0.726 to 138 µg/kg ww in mountain whitefish muscle in the Columbia River, and from 27 to 50 µg/kg ww in trout from the Great

Lakes. PBDEs in eggs of heron, murre, fulmar, and herring gull ranged from 1.31 to 1544 µg/kg ww (Environment Canada 2006). British Columbia's farmed salmon had higher levels of PBDEs than wild salmon, with maximum concentrations of 2.9 ng/g ww for tetraBDE, 0.9 ng/g ww for pentaBDE, and 0.28 ng/g ww for hexaBDE (Easton et al. 2002). PBDEs have been identified in Canadian seals, whales and porpoises with the highest mean value of 665 ng/g ww identified in the blubber of female belugas from the St. Lawrence River (Lebeuf and Trottier 2001).

Gauthier et al. (2008) reported concentrations of PBDEs in herring gull eggs in the Laurentian Great Lakes (triBDE 1.2-7.1 ng/g ww; tetraBDE 91-274 ng/g ww; pentaBDE 147-708 ng/g ww; hexaBDE 51-174 ng/g ww; heptaBDE 6.4-17 ng/g ww; octaBDE 4-19 ng/g ww; and nonaBDE 1.9-8.5 ng/g ww. Peaking in 2006, it was also reported that BDE-209 (i.e., decaBDE) concentrations in herring gull eggs from the Great Lakes ranged from 4-20 ng/g ww, and from 1982 to 2006, the BDE-209 doubling times in gull eggs ranged from 2.1 to 3.0 years, whereas for octaBDEs and nonaBDEs, the mean doubling times ranged from 3 to 11 years and 2.4 to 5.3 years, respectively (Gauthier et al. 2008). In contrast, reported doubling times for tetra- to hexaBDEs were 4.9 to 8.7 years.

### Mode of action

PBDEs are endocrine disrupting chemicals (Darnerud et al. 2001) which inhibit the thyroid hormone transporter transthyretin thereby preventing effective thyroxine transport and inhibiting the induction of uridine diphosphoglucuronosyl transferase which would normally increase metabolism of thyroxine hormone (Zhou et al. 2002). This in turn, causes inhibition of the cholinergic nicotinic receptors in the brain and can lead to cognitive disorders. Kodavanti et al. (2005) attributed impaired neuronal development, memory and learning to changes at the cellular level to protein kinase C and calcium homeostasis and that the effect was similar to that of some PCBs. In a study by Ucán-Marín et al. (2010), lower but substantial binding affinity was reported for BDE-47 in competition with the major thyroid hormones (thyronine and thyroxine) on recombinant gull albumin and transthyretin, whereas a purported metabolite of BDE-47, 6-hydroxy-BDE-47 had higher binding affinities relative to these thyroid hormones.

Narcosis could also be a mode of action based on the similarity between chemical structure of PBDEs and petrochemicals but is expected to be orders of magnitude less sensitive than the targeted biochemical effects. For example, in dietary rat studies biochemical effects included increase in cholesterol level, liver and urine porphyrin level, liver cell damage, and necrosis (Great Lakes Chemical Corporation 1984). These effects are accompanied with histological effects, such as microscopic changes in the thyroid gland and liver (Great Lakes Chemical Corporation 1984) as well as changes in kidney cell cytoplasm (Norris et al. 1975). For invertebrates and plants no information exists on possible mode of action.

## Federal Environmental Quality Guidelines Derivation

### Federal Water Quality Guidelines

Federal Water Quality Guidelines (FWQGs) are benchmarks for aquatic ecosystems that are intended to protect all forms of aquatic life (vertebrates, invertebrates, and plants) from direct adverse effects for indefinite exposure periods via the water column (Table 1). FWQGs apply to both freshwater and marine environments unless it can be demonstrated that the toxicity differs significantly between these two environments (e.g., due to ionization).

Published PBDEs toxicity studies for aquatic organisms were evaluated for quality and completeness. Commercial PBDE formulations consist of a mixture of various homologues which differ in their relative toxicity. As a result, direct comparisons cannot be made among available toxicity studies. The composition of the commercial formulations may vary, but the congener distributions found in the environment will be a range of new/old releases of several potential commercial products. Also, commercial PBDE mixtures released into the aquatic environment will differ in their homologue distributions and the congener distributions will be a range of new/old releases of commercial products. Environmental concentrations will be affected by a wide array of processes, including potential biotransformation.



Chronic toxicity data in Environment Canada's (2006) Screening Assessment Report (SAR) along with more recent data were used for developing the guidelines for several PBDEs and associated homologues. The selection of toxicity endpoints followed the CCME (2007) and the SAR approaches. The acceptable toxicity data were all for invertebrates (Table 3). Very few fish toxicity data were available. They were not selected as critical toxicity values (CTV) because the values were higher or the studies were of unacceptable quality. The lowest toxicity endpoint for individual congeners was selected as the CTV. This value was divided by an application factor (AF) of 100 (10 to account for the extrapolation from laboratory to field conditions and inter- and intra-species variations in sensitivities, and 10 because PBDEs are persistent and bioaccumulative (Table 3).

### **Federal Fish Tissue Guidelines**

The Federal Fish Tissue Guidelines (FFTGs) are benchmarks for aquatic ecosystems that are intended to protect fish themselves from direct adverse effects (Table 1). FFTGs supplement water quality guidelines in that they provide a different metric to assess potential adverse effects. FFTGs apply to both freshwater and marine fish species, and specify the concentration of PBDE congeners found in whole body fish tissue (wet weight - ww) not expected to result in adverse effects (Table 1) to the fish themselves. They may not be appropriate to evaluate the impacts of PBDEs found in unrepresented aquatic biota (amphibians and plants in this case).

Currently, there exists no direct toxicity data on PBDEs based on fish tissue burdens. FFTGs were, therefore, based on fish tissue burdens estimated from PBDE concentrations in water and the degree to which fish are known to accumulate PBDE from water (i.e., bioaccumulation factors). Concentrations of PBDEs at and below their respective water quality guidelines are not expected to cause adverse effects in fish. By multiplying the WQG of a given congener by its corresponding BAF and rounding to two significant figures (Table 2), a tissue burden that is not expected to result in adverse effects in fish was estimated. This fish tissue burden is the FFTG. The BAF was the geometric mean of BAFs of species for that congener found in the literature (Environment Canada unpublished data). These BAFs were calculated from field sampling data on water concentration and aquatic biota levels, representing steady state bioaccumulation due to environmentally relevant levels of PBDEs. The use of field-based BAFs is a more realistic estimate of fish tissue burdens than laboratory based BAFs.

### **Federal Sediment Quality Guidelines**

The Federal Sediment Quality Guidelines (FSeQG) for the protection of aquatic life are intended to protect sediment dwelling animals as well as pelagic animals which bioaccumulate PBDEs from sediments (Table 1). FSeQGs apply to indefinite exposure periods to freshwater and marine sediments, and specify the concentration of PBDE congeners found in bulk sediment (dry weight) not expected to result in adverse effects. They may not be appropriate to evaluate the impacts of PBDEs in sediments to plants.

Environment Canada (2006) reviewed the sediment toxicity data for PBDEs and identified that the data are limited. In summary, the oligochaete, *Lumbriculus variegatus*, was more sensitive than the amphipod, *Hyalella azteca* and the midge, *Chironomus riparius* when exposed to commercial PentaBDE for 28-d (Great Lakes Chemical Corp. 2000a,b,c). The LOECs were 6.3, 13, and 50 mg/kg dw, respectively. For all species, the EC<sub>50</sub> for survival and reproduction was greater than the highest concentration tested (50 mg/kg dw). *Lumbriculus variegatus* was less sensitive to commercial OctaBDE than a commercial PentaBDE formulation showing no adverse effects when exposed for 28-days to concentrations of 1271 mg/kg dw, the highest concentration tested (Great Lakes Chemical Corporation 2001a,b). Similarly for a commercial DecaBDE formulation, *L. variegatus* did not show adverse effects when exposed for 28-d to concentrations of 4536 mg/kg dw, again the highest concentration tested (ACCBFRIP 2001 a,b). *Hyalella azteca* and *C. riparius* were not tested with commercial OctaBDE or DecaBDE formulations. Recently, BDE-153 and -154 were found to immediately and irreversibly change the structure of the anaerobic bacterial community cultures extracted from field collected sediments (Yen et al. 2009).

Data acceptable for the development of sediment quality guidelines are very limited and no new data from spiked sediment toxicity tests have been published since the SAR (Environment Canada 2006). FSeQGs for specific congeners were calculated from (unrounded) FFTGs derived herein, converted to dry weight assuming 75% moisture content, and divided by appropriate BSAFs reported in Table 2 [i.e.,  $\text{FSeQG} = (\text{FFTG} \div .25) \div \text{BSAF}$ ]. For commercial OctaBDE and DecaBDE products, the PNECs from the SAR (Environment Canada 2006) were adopted as the FSeQGs for these homologue groups, but adjusted to 1% organic carbon. Alternatively, benchmark values were also derived from corresponding water quality guidelines and a measure of the degree to which PBDEs partition from water to sediment; however these values were not selected as FSeQGs as they were higher than the FSeQG derived for specific congeners from FFTG (Table 1) and therefore may not be adequately protective.

**Table 3. Derivation of Federal Water Quality Guidelines (FWQG) for PBDE congeners**

Homologue	Congener	Species	Endpoint	CTV (µg/L)	AF*	Guideline (ng/L)	Reference
triBDE	28	<i>Acartia tonsa</i> (copepod)	5-d EC10 (larval development)	4.6	Lab to field = 10 P&B** = 10	46	Wollenberger et al. 2005
tetraBDE	47	<i>Acartia tonsa</i> (copepod)	5-d EC10 (larval development)	2.4	Lab to field = 10 P&B = 10	24	Wollenberger et al. 2005
pentaBDE	99	<i>Acartia tonsa</i> (copepod)	5-d EC10 (larval development)	0.39	Lab to field = 10 P&B = 10	3.9	Wollenberger et al. 2005
pentaBDE	100	<i>Acartia tonsa</i> (copepod)	5-d EC10 (larval development)	0.023	Lab to field = 10 P&B = 10	0.23	Wollenberger et al. 2005
hexaBDE	153	<i>Daphnia magna</i> (water flea)	21-d EC40 (lethality, reproduction, growth)	12	Lab to field = 10 P&B = 10	120	Nakari and Huhtala 2008
heptaBDE ***	-	<i>Daphnia magna</i> (water flea)	21-d NOEC/LOEC (survival, reproduction, growth)	1.7	Lab to field = 10 P&B = 10	170	CMABFRIP 1997
octaBDE***	-	<i>Daphnia magna</i> (water flea)	21-d NOEC/LOEC (survival, reproduction, growth)	1.7	Lab to field = 10 P&B = 10	170	CMABFRIP 1997

\* AF = Application Factor.

\*\* Persistent and bioaccumulative (P&B) as defined by the *Persistence and Bioaccumulation Regulations* of CEPA 1999 (Government of Canada 2000).

\*\*\* Substance tested was commercial OctaBDE which contains mainly heptaBDE and octaBDE congeners.

## Federal Wildlife Guidelines

The SAR (Environment Canada 2006) identified a complete lack of toxicity studies on wildlife (mammals and birds) as one of the uncertainties in their assessment. However a dietary residue guideline (CCME 1998) was developed based on rat and rabbit data, which was then extrapolated to mink. The resultant values were 0.0084 µg/g ww in food for pentaBDE congeners (based on toxicity studies in rat) and 0.06 µg/g ww in food for octaBDE congeners (based on toxicity studies in rabbit). Since that time there have been a series of results published on the effects of PBDEs on wildlife (described below).

The Federal Wildlife Dietary Guidelines (FWiDG) are intended to protect mammalian and avian consumers of aquatic biota. These are benchmarks of concentrations of toxic substances in aquatic biota (whole body, ww) which are consumed by terrestrial and semi-aquatic wildlife (Table 4). FWiDG for seven PBDE congeners were developed, following portions of the derivation processes outlined in CCME (1998) and SAR (Environment Canada 2006). These guidelines can be applied to the individual congener when known, or to the total concentration of the corresponding homologue. FWiDG may not be appropriate to extrapolate the impacts of these substances to other terrestrial consumers (e.g., reptiles). Since the minimum dataset requirements (CCME 1998) are not met, these guidelines should be considered to have low reliability and should be used with caution.

In addition to the diet-based guidelines, a guideline for the protection of avian wildlife was also developed based on toxicity studies conducted on bird eggs as described below.

### **Mammalian**

A number of toxicity studies (single-dose) were performed on rodents which identified behaviour and reproduction as very sensitive exposure endpoints (Table 4). Neurological malfunctioning can lead to cognitive disorders, altering the behaviour of mammals. Some PBDE-induced behavioural changes were altered activity patterns and capability to habituate (Eriksson et al. 2001), reduced cue-based performance in fear conditions (Taylor et al. 2003), and impaired spontaneous motor behaviour, learning, and memory (Viberg et al. 2002). Moreover, PBDEs effects in newborn mammals differ substantially from those demonstrated in exposed adults. Rodents receiving a single oral dose of PBDEs (dissolved in a mixture of egg lecithin, peanut oil and water) during a critical period of neonatal brain development displayed aberrant behaviour and activity patterns in adulthood that differed significantly from those seen in rodents which were not exposed. The observed adverse effects were irreversible and increased with age (Viberg et al. 2002).

There was a single study conducted on a mammalian wildlife species (Table 4). Martin et al. (2007) exposed ranch mink (*Mustela vison*) to wet diets containing 1, 5, or 10 µg/g (plus a control) of DE-71, a commercial PentaBDE formulation containing mainly penta- and tetraBDE congeners, for eight weeks. At 5 and 10 µg/g wet diet, systemic toxicity manifested as lower weight, and activation of the immune system (based on histology, blood chemistry, and challenge tests). EROD activity indicating phase 1 detoxification enzymes was measured in all treatments. It is difficult to include the 5 µg/g wet diet treatment because this group started as a 100 µg/g wet diet treatment but switched to the lower diet after 1 week due to food avoidance. Overall, the 1 µg/g wet diet would represent a no-observed-effect while the 10 µg/g wet diet showed a clear effect on growth. The MATC approach (geometric mean of NOAEL and LOAEL) is 3.16 µg/g wet diet. No behavioural or reproductive endpoints were examined. These latter endpoints proved very sensitive in rat studies based on single injections (Table 4).

**Table 4. Derivation of Federal Wildlife Dietary Guidelines (FWiDG) for mammalian and avian species.**

Homologue	Congener	Species and Reference	Endpoint	CTV (µg/kg bw)	AF	Guideline* (ng/g wet diet)
tetraBDE	47	Mouse ( <i>Mus musculus</i> ) <sup>a</sup>	Single dose LOAEL (Behavioural after 5 months)	10 500	Lab to field = 10; Rodent to wildlife = 10 acute to chronic = 10 FI:BW** = 0.24 (mink)	44
pentaBDE	99	Mouse ( <i>Mus musculus</i> ) <sup>a,b</sup>	Single dose LOAEL (Behavioural after 5 months)	800	Lab to field = 10 Rodent to wildlife = 10 acute to chronic = 10 FI:BW = 0.24 (mink)	3
PeBDE <sup>†</sup>	mixture	Mink ( <i>Mustela vison</i> ) <sup>c</sup>	reduced growth NOAEL 1 µg/g in diet LOAEL 10 µg/g diet	geomean 3.16 µg/g diet	100 (see text)	32
PeBDE <sup>†</sup>	mixture	American Kestrel ( <i>Falco sparverius</i> ) <sup>d,e</sup>	reproductive and behaviour effects	geomean (see text) 0.13 µg/g diet	Default = 10	13
hexaBDE	153	Mouse – NMRI ( <i>Mus musculus</i> ) <sup>f</sup>	Single dose LOAEL (Behavioural later in adult stage )	900	Lab to field = 10 Rodent to wildlife = 10 acute to chronic = 10 FI:BW = 0.24 (mink)	4
heptaBDE	183	Mouse – NMRI ( <i>Mus musculus</i> ) <sup>g</sup>	Single dose LOAEL (Behavioural after 2 months)	15 200	Lab to field = 10 Rodent to wildlife = 10 acute to chronic = 10	64
nonaBDE	206	Mouse – NMRI ( <i>Mus musculus</i> ) <sup>g</sup>	Single dose LOAEL (Behavioural after 2 months)	18 500	Lab to field = 10 Rodent to wildlife = 10 acute to chronic = 10 FI:BW = 0.24 (mink)	78
decaBDE	209	Mouse - NMRI ( <i>Mus musculus</i> ) <sup>h</sup>	Single dose LOAEL (Behavioural, Physiological later in adult	2220	Lab to field = 10 Rodent to wildlife = 10 acute to	9

Homologue	Congener	Species and Reference	Endpoint	CTV (µg/kg bw)	AF	Guideline* (ng/g wet diet)
			stage)		chronic = 10 FI:BW = 0.24 (mink)	

\* Guidelines can be calculated using other species-specific FI:BW. See CCME Protocol (1999) for a list of species-specific FI:BW ratios.

\*\* FI:BW ratio = Food intake: body weight ratios.

† Substance tested was commercial DE-71, a commercial PentaBDE formulation containing mainly penta-BDE and tetraBDE congeners.

References:<sup>a</sup> Eriksson et al. 2001; <sup>b</sup> Eriksson et al. 1998; <sup>c</sup> Martin et al. 2007; <sup>d</sup> Fernie et al. 2009a; <sup>e</sup> Fernie et al. 2009b; <sup>f</sup> Viberg et al. 2003a; <sup>g</sup> Viberg et al. 2006; <sup>h</sup> Viberg et al. 2003b.

## Avian

Based on the behavioural impairments in rodents exposed neonatally (Viberg et al. 2002), it was hypothesized that the developing bird embryo would be the most sensitive life stage. This is also the focus of the monitoring efforts and therefore the EQG was based on residues in eggs. The challenge has been to develop egg residue values that are surrogates for the most sensitive effects, whether it is in the adult (breeding behaviour, egg characteristics) or in the developing embryo (hatching/fledging success, growth). In both the laboratory and field studies physiological effects seen in the egg/embryo could be attributed to either parental or developmental factors. However, effects in adults were no more sensitive than the physiological/reproductive effects in embryos and young birds.

Two lines of evidence were used to develop environmental quality guidelines for the protection of avian wildlife. The first line of evidence used laboratory studies on captive birds (Fernie et al. 2005a,b, 2006, 2008, 2009a,b) with two different exposure regimes, both aimed at mimicking concentrations similar to those seen in gull eggs in populations from the Laurentian Great Lakes. In the first experiment, American kestrels eggs were exposed to pure PeBDE congeners; single dose, single exposure concentration injection into the air sac of the eggs on day 19 of the 28 day incubation period. Following hatch, chicks were variably dosed according to body weight by gavage once daily for a total of 29-d post hatch, resulting in whole body concentrations that were 86 ng/g ww or about 120-fold higher than the controls. There were no changes in hatching or fledging success, but PBDE-exposed chicks were larger and grew faster, an effect which the authors saw as potentially negative since the patterns were dissimilar from those of the control nestlings (Fernie et al. 2006). Physiological changes included impaired vitamin A homeostasis (declines in plasma and hepatic vitamin A homologues), alterations in some aspects of thyroid hormone metabolism (decreased plasma thyroxine but not T3), and for the female nestlings, evidence of oxidative stress at the cellular level (glutathione peroxidase and lipid peroxidation) (Fernie et al. 2005a). Immunological effects included an activation of cell-mediated immunity and a reduction in humoral-mediated systems (Fernie et al. 2005b).

In the second experiments (Fernie et al. 2008, 2009a,b), adult kestrels were exposed to two concentrations of a commercial PentaBDE formulation via the diet (0.3 and 1.5 µg/g ww of commercial mixture DE-71); hexabromocyclododecane (HBCD) was an

unanticipated artefact. Eggs laid by breeding pairs contained concentrations of PBDEs similar to those seen in the eggs of biota from the Laurentian Great Lakes over the period of 2000 to 2004. Effects were seen both in reproductive behaviour of adults (Ferne et al. 2008), and in eggs/chicks (Ferne et al. 2009b). Number of hatchlings and number of successful fledglings decreased at both dietary concentrations (effect in egg). Eggshell thickness declined about 8% in the high exposure group. Eggshell thinning was first documented as the result of dichlorodiphenyldichloroethylene (DDE) exposure. The danger point above which wild populations of American kestrels could not sustain themselves was 18% thinning or greater (Lincer 1975). The present study is the only study to associate eggshell thinning with PBDEs. In addition, the second generation of males from the diet DE-71 study (i.e., males exposed *in ovo* to DE-71), were paired with unexposed females, and these pairs produced fewer eggs and fewer fertile eggs (parental effect) than control pairs (Marteinson et al. 2010).

There were some complications with the Ferne et al. studies. In the first series of experiments involving exposure to pure PBDE congeners, the timing and/or methodology used to inject the eggs resulted in unexpectedly high embryonic mortality of the controls (~50% hatching success vs the expected >80% hatching success) (Ferne et al. 2006). In the second series of experiments involving the commercial mixture DE-71, HBCD was measured in the eggs but not the commercial DE-71 mixture used by Ferne et al. (2009a,b). Dose-responses were difficult to evaluate since there was often a single test concentration plus a control, and at best, two test concentrations. Responses to PBDE exposures appeared stimulatory at some doses and inhibitory at others. There are not sufficient dose-response studies firstly, to confirm that this is the case, and secondly to identify whether these responses could be considered as hormesis (low doses beneficial followed by detrimental effects at higher doses).

McKernan et al. (2009) dosed eggs of chicken, mallard and kestrel by single air-cell injection with up to five concentrations (plus control) of DE-71 ranging from 0.01 to 20 µg/egg. Absorption of the dose as well as toxic effects were followed from day 5 after laying until 1 day after hatch. Neither mallards nor chickens showed any lethal responses, but kestrels experienced a 55% decline in successful pipping (piercing the eggshell during hatching) and 45% decline in hatching success at 10 µg/egg (20 µg/egg gave identical responses). Control survival of 80% was in the expected range for captive kestrels. Given the 18% dose absorption from the air cell, an egg concentration of about 1800 ng/g ww was calculated for lethality. The authors stated that the observed lethal effects at 180 ng/g ww in eggs, though it was not statistically significant. Kestrels did not show any other effects: no EROD induction (hence no AhR-mediated toxicity), no thyroid response, and no changes in liver histology.

van den Steen et al. (2009) dosed European starlings with a single concentration of a mixture of 10 mostly pentaBDEs (congeners 28, 47, 49, 66, 85, 99, 100, 153, 154 and 183) via silastic tubing implants. PBDE concentrations in serum of adult females rose dramatically and the concentrations in eggs ranged from 130 to 220 ng/g ww. The authors indicated that fewer females in the treated group initiated egg laying but the effect was not significant. Eggs in the treated group were about 7% larger by volume and about 6%

heavier, both statistically significant. In general however, concentrations having an effect of <10% are typically considered “no effect” concentrations.

The second line of evidence correlates residues of PBDEs in bird eggs (mainly those of raptors) in the wild with detrimental effects. Osprey in Washington State all accumulated PBDE in eggs (Henny et al. 2009). The authors suggested that the concentrations in eggs, slightly above 1000 ng/g ww, were associated with decreased productivity (but did not observe any eggshell thinning). Johansson et al. (2009) correlated declining reproductive success in wild peregrine falcons in southern Sweden as PBDE concentrations increased. The birds also had residues of several other organohalogen contaminants. There was considerable scatter in the data and no clear threshold, but at ~1000 ng/g lw, nest productivity was 2.5 chicks per nest and at about 7000 ng/g lw in eggs, (~400 ng/g ww in eggs given 6.6% lipid), the average number of young per female dropped below 1.

Environmental quality guidelines for avian wildlife were developed for both diets and bird eggs. The derivation of the FWiDG for avian wildlife is based on the kestrel feeding studies of Fernie et al. (2008, 2009). Here, a dietary concentration LOAEL of 0.3 µg/g wet diet (this was the lowest concentration tested) resulted in reproductive and behavioural effects. Calculating a NOAEL as LOAEL / 5.6 (CCME 1993) and taking an MATC approach and applying an application factor of 10 would produce a value of 13 ng/g ww of diet (= 0.013 µg/g).

For the egg-based guideline, the value of 288 ng/g ww in eggs (Fernie et al. 2009) was chosen as the critical toxicity value for guideline development. This value is the sum of individual congeners, mostly 99, 153, and 100. The value is corroborated by the results of Johansson et al. (2009) of 400 ng/g ww in eggs from field studies. An application factor of 10 was applied to account for the lack of extensive dose-response data. Use of additional application factor(s) would lower the FEQG to background egg concentrations at which no detrimental effects were seen in laboratory (3.01 ng/g ww in kestrels (Fernie et al. 2009); <3.5 ng/g ww peregrines (Johansson et al. 2009). Nevertheless, the EQG has low reliability and should be used with caution.

### **Considerations in Guideline Implementation**

Environmental quality guidelines are presented here for various media for various PBDE congeners. The guidelines can be applied to the congener, when known, or to the total concentration of the corresponding homologous group. The congener-specific guidelines presented here are applicable given the approach to regulate and monitor PBDEs in Canada. While ideally the toxicity of the congeners and homologues would be inter-related through the use of toxic equivalents, such an interrelationship has not yet been developed by Environment Canada or any other jurisdiction, and there are not currently sufficient homologue-specific data to do this. With the toxic equivalents method, the measured concentrations for each congener are divided by their respective guideline and the sum of the fractions is taken. If the summed value is greater than 1 then the “guideline” is exceeded and additional analysis would be recommended. A second approach is similar, but uses the Water Quality Index in which the magnitude and



frequency of exceedences of the respective guideline is normalized to a percentage. High values are indicative of good quality (values well below FEQGs) whereas low percentages are indicative of poor quality, and again, follow up might be required. Both methods are useful for relative ranking of sites for additional consideration.

### Interactions with other contaminants

PBDEs exhibit additive toxicity with functionally isomorphic anthropogenic organic pollutants. Both PBDEs and DDE cause eggshell thinning (Fernie et al. 2009). PBDEs, dichlorodiphenyltrichloroethane (DDT), PCB, and HBCD all induce cancer via non-mutagenic mechanism (de Wit 2002). PBDEs also cause thyroid disruption, through the transthyretin binding mechanism of HBCD, TBBPA, and hydroxylated metabolites of PCBs (de Wit 2002). In studies by Ucán-Marín et al. (2009, 2010), lower but substantial binding affinity was reported for BDE-47 in competition with the major thyroid hormones (thyronine and thyroxine) on recombinant human and gull (avian) albumin and transthyretin, whereas a purported metabolite of BDE-47, 6-hydroxy-BDE-47 had higher binding affinities relative to these thyroid hormones. Comparatively, in vitro binding affinities of the PCB metabolite 4-hydroxy-CB187 were greater than both thyronine and thyroxine on both human and gull albumin and transthyretin. Furthermore, PBDEs, HBCD, and ortho-substituted and co-planar PCBs all affect neuron cell differentiation and lead to permanent aberrations in spontaneous motor behaviour in mammals (de Wit 2002). In fact, the additive toxicity of certain BFRs was demonstrated in a 26-day full life cycle study exposing copepods (*Nitocra spinipes*) to a mixture of BDE 28, HBCD, Tetrabromobisphenol A (TBBPA), Tetrabromobisphenol A hydroxyethyl ether (TBBPA OHEE), Tetrabromoethylcyclohexane (TBECH), and Tribromophenol (2,4,6BrPh). Significant increase in mortality was observed in mixtures consisting of these BFRs at their NOECs. At concentrations 5 times the NOEC values, all copepods died. Such observation raises the concern for addressing emergent toxicity of environmental complex mixtures.

PBDEs may function antagonistically with some organic pollutants. For example, PBDE-induced immunotoxic effects are in opposite direction of those demonstrated by PCBs seen on colonial waterbirds (Fernie et al. 2005). PBDEs also exert toxicity differently from other anthropogenic organic pollutants. For instance, while PBDEs are similar to ortho-substituted PCBs in chemical structure, there is contradictory evidence as to whether PBDEs act through the Ah receptor or not (Peters et al. 2006; Martin et al. 2007). PBDEs do not disrupt protein kinase activity and the immune system (Fernie et al. 2009).

### Data limitations

Throughout this factsheet it has been emphasized that there is a lack of toxicity data for media for which FEQGs were developed. Therefore, there is considerable uncertainty in the toxicity thresholds and thus conservative application factors have been used. To reduce uncertainty in these guidelines, long-term PBDE toxicity studies would be needed on species relevant to Canada. Should additional data become available, the FEQGs for PBDEs may be revised.

## References

ACCBFRIP (American Chemistry Council Brominated Flame Retardant Industry Panel). 2001a. Decabromodiphenyl ether: A prolonged sediment toxicity test with *Lumbriculus variegatus* using spiked sediment with 2% total organic carbon. Final report. Project No. 439A-113, Wildlife International, Ltd., February.

ACCBFRIP (American Chemistry Council Brominated Flame Retardant Industry Panel). 2001b. Decabromodiphenyl ether: A prolonged sediment toxicity test with *Lumbriculus variegatus* using spiked sediment with 5% total organic carbon. Final report. Project No. 439A-114, Wildlife International, Ltd., February.

Alaee, M., C. Cannon, D. Muir, P. Blanchard, K. Brice and P. Fellin. 2001. Spatial distribution and seasonal variation of PBDEs in Arctic and Great Lakes air. *Organohalogen Compd.* 52: 26–29.

Bidleman, T.F., M. Alaee and G. Stern. 2001. New persistent toxic chemicals in the environment. In: S. Kalhok (ed.), *Synopsis of research conducted under the 2000/2001 Northern Contaminants Program*. Department of Indian and Northern Affairs, Ottawa, Ontario. pp. 93–104.

CCME (Canadian Council of Ministers of the Environment). 1998. Protocol for the Derivation of Canadian Tissue Residue Guidelines for the Protection of Wildlife that Consume Aquatic. In: *Canadian Environmental Quality Guidelines*. Canadian Council of Ministers of the Environment, Winnipeg.

CCME (Canadian Council of Ministers of the Environment). 2007. A Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life. In: *Canadian Environmental Quality Guidelines, 1999*, Canadian Council of Ministers of the Environment, Winnipeg.

CMABFRIP. 1997. Octabromodiphenyl oxide (OBDPO): A flow-through life-cycle toxicity test with the cladoceran (*Daphnia magna*). Final Report. Wildlife International Ltd. Project Number: 439A-104, May 1997.

Darnerud, P., G. Eriksen, T. Johannesson, P. Larsen and M. Viluksela. 2001. Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. *Environmental Health Perspectives* 9 (suppl 1):49–68.

de Wit, C.A. 2002. An overview of brominated flame retardants in the environment. *Chemosphere* 46: 583–624.

Easton, M.D.L., D. Lusznjak and E. Von der Geest. 2002. Preliminary examination of contaminant loadings in farmed salmon, wild salmon and commercial salmon feed. *Chemosphere* 46: 1053–1074.

Environment Canada. 2004. PBDEs. CEPA Environmental Registry – Polybrominated Diphenyl Ethers (PBDEs). November 2009

([http://www.ec.gc.ca/CEPARegistry/documents/subs\\_list/PBDE\\_draft/PBDEfaq.cfm](http://www.ec.gc.ca/CEPARegistry/documents/subs_list/PBDE_draft/PBDEfaq.cfm))

Environment Canada. 2006. Supporting Working Document for the Environmental Screening Assessment of Polybrominated Diphenyl Ethers. Environment Canada, Gatineau, June 2006, 222pp

Environment Canada. 2009. Management of Toxic Substances. Substance Detail – Polybrominated Diphenyl Ethers. November 2009

([http://www.ec.gc.ca/TOXICS/EN/detail.cfm?par\\_substanceID=201&par\\_actn=s1](http://www.ec.gc.ca/TOXICS/EN/detail.cfm?par_substanceID=201&par_actn=s1))

Environment Canada. 2010. Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE): Bioaccumulation and Transformation. Environment Canada, Gatineau, August 2010, 147pp

Eriksson, P., E. Jakobsson and A. Fredriksson. 1998. Developmental neurotoxicity of brominated flame-retardants, polybrominated diphenyl ethers and tetrabromo-bis-phenol A. *Organohalogen Compounds* 35: 375–377.

Eriksson, J., E. Jakobsson, G. Marsh and Å. Bergman. 2001. Photo decomposition of brominated diphenyl ethers in methanol/water. Abstracts. The Second International Workshop on Brominated Flame Retardants. BFR 2001 Stockholm. May 14-16. Stockholm University, Sweden. p. 203-206.

European Communities. 2001. European Union risk assessment report. Diphenyl ether, pentabromo derivative (pentabromodiphenyl ether). CAS No.: 32534-81-9. EINECS No.: 251-084-2. Risk assessment. Final report, August 2000. United Kingdom on behalf of the European Union.

European Communities. 2002. European Union risk assessment report. Bis(pentabromophenyl) ether. CAS No.: 1163-19-5. EINECS No.: 214-604-9. Risk assessment. Final report, 2002. France and United Kingdom on behalf of the European Union.

European Communities. 2003. European Union risk assessment report. Diphenyl ether, octabromo derivative. CAS No: 32536-52-0. EINECS No.: 251-087-9. Risk assessment. Final report, 2003. France and United Kingdom on behalf of the European Union

Fernie, K.J., J.L. Shutt, G. Mayne, D. Hoffman, R.J. Letcher, K.G. Drouillard and I.J. Ritchie. 2005a. Exposure to polybrominated diphenyl ethers (PBDEs): changes in thyroid, vitamin A, glutathione homeostasis, and oxidative stress in American kestrels (*Falco, sparverius*). *Toxicol. Sci.* 88: 375-383.

Fernie, K.J., G. Mayne, J.L. Shutt, C. Pekarik, K.A. Grasman, R.J. Letcher and K. Drouillard. 2005b. Evidence of immunomodulation in nestling American kestrels (*Falco*

*sparverius*) exposed to environmentally relevant PBDEs. Environ. Pollut. 138: 485-493.

Fernie, K.J., J.L. Shutt, I.J. Ritchie, R.J. Letcher, K.G. Drouillard and D.M. Bird. 2006. Changes in the growth, but not the survival, of American kestrels (*Falco sparverius*) exposed to environmentally relevant polybrominated diphenyl ethers. J. Toxicol Environ. Health A 69: 1541-1554.

Fernie, K.J., J.L. Shutt, R.J. Letcher, I.J. Ritchie, K. Sullivan and D.M. Bird. 2008. Change in reproductive courtship behaviours of adult American kestrels (*Falco sparverius*) exposed to environmentally relevant levels of the polybrominated diphenyl ether mixture, DE-71. Toxicol. Sci. 102: 171-178.

Fernie, K.J., J.L. Shutt, G. Mayne, R.J. Letcher, I.J. Ritchie and D.M. Bird. 2009a. Environmentally relevant concentrations of DE-71 and HBCD alter eggshell thickness and reproductive success of American kestrels. Environ. Sci. Technol. 43: 2124-2130.

Fernie, K.J., S.C. Martenson, K. Sullivan, J.L. Shutt, R.J. Letcher, I.J. Ritchie and D.M. Bird. 2009b. The effects of brominated flame retardants (HBCD & PBDEs) on reproductive behaviour and physiology of American kestrels (*Falco sparverius*): conservation implications. poster presented at the 36<sup>e</sup> Annual Aquatic Toxicity Workshop. La Malbaie, Quebec.

Gauthier, L.T., C.E. Hebert, D.V. Chip Weseloh and R.J. Letcher. 2008. Dramatic changes in the temporal trends of polybrominated diphenyl ethers (PBDEs) in herring gull eggs from the Laurentian Great Lakes: 1982-2006. Environ. Sci. Technol. 42: 1524-1530.

Gerecke, A.C., P.C. Hartmann, N.V. Heeb, H.-P. E. Kohler, W. Giger, P. Schmid, M. Zennegg and M. Kohler. 2005. Anaerobic degradation of decabromodiphenyl ether. Environ. Sci. Technol. 39: 1078-1083.

Gouin, T., G.O. Thomas, I. Cousins, J. Barber, D. Mackay and K.C. Jones. 2002. Air-surface exchange of polybrominated diphenyl ethers and polychlorinated biphenyls. Environ. Sci. Technol. 36: 1426-1434. Supporting information available at [http://pubs.acs.org/subscribe/journals/esthag/supinfo/36/i07/es011105k/es011105k\\_s.pdf](http://pubs.acs.org/subscribe/journals/esthag/supinfo/36/i07/es011105k/es011105k_s.pdf).

Gouin, T. and T. Harner. 2003. Modelling the environmental fate of polybrominated diphenyl ethers. Environment International 29: 717-724.

Government of Canada. 2000. *Canadian Environmental Protection Act, 1999: Persistence and Bioaccumulation Regulations*, P.C. 2000-348, 29 March, 2000, SOR/2000-107. Available from: <http://www.gazette.gc.ca/archives/p2/2000/2000-03-29/pdf/g2-13407.pdf>

Government of Canada 2010. Final State of Science DecaPBDE. Available from : <http://www.gazette.gc.ca/>. Canada Gazette Vol 144. No 35. pp. 2318-2320.

Great Lakes Chemical Corporation. 1984. 90-day dietary study in rats with pentabromodiphenyl oxide (DE-71). Final report. Project No. WIL-12011, WIL Research Laboratories, Inc.

Great Lakes Chemical Corporation. 2000a. Pentabromodiphenyl oxide (PeBDPO): A prolonged sediment toxicity test with *Hyalella azteca* using spiked sediment. Wildlife International, Ltd. Project Number: 298A-111, April 2000.

Great Lakes Chemical Corporation. 2000b. Pentabromodiphenyl oxide (PeBDPO): A prolonged sediment toxicity test with *Chironomus riparius* using spiked sediment. Wildlife International, Ltd. Project Number: 298A-110, April 2000.

Great Lakes Chemical Corporation. 2000c. Pentabromodiphenyl oxide (PeBDPO): A prolonged sediment toxicity test with *Lumbriculus variegatus* using spiked sediment. Wildlife International, Ltd. Project Number: 298A-109, April 2000.

Great Lakes Chemical Corporation. 2001a. Octabromodiphenyl ether: A prolonged sediment toxicity test with *Lumbriculus variegatus* using spiked sediment with 2% total organic carbon. Final report. Project No. 298A-112, Wildlife International, Ltd., February.

Great Lakes Chemical Corporation. 2001b. Octabromodiphenyl ether: A prolonged sediment toxicity test with *Lumbriculus variegatus* using spiked sediment with 5% total organic carbon. Final report. Project No. 298A-113, Wildlife International, Ltd., February.

Great Lakes Chemical Corporation. 2005. Great Lakes Chemical Corporation Completes Phase-Out of Two Flame Retardants. Press Release. January 18, 2005 ([www.el.greatlakes.com/corp/common/jsp/index.jsp](http://www.el.greatlakes.com/corp/common/jsp/index.jsp)).

Harner, T. and M. Shoeib. 2002. Measurements of octanol-air partition coefficients for polybrominated diphenyl ethers (PBDEs): Predicting partitioning in the environment. J. Chem. Eng. Data 47: 228-232.

Henny, C.J., J.L. Kaiser, R.A. Grove, B.L. Johnson and R.L. Letcher. 2009. Polybrominated diphenyl ether flame retardants in eggs may reduce reproductive success of ospreys on Oregon and Washington, USA. Ecotoxicology 18: 802-813.

ICL Industrial Products. 2005. Termination of the production and sales of FR-1208 (Octabromodiphenyl oxide). Press Release. February 2, 2005. (<http://www.iclfr.com>).

Johansson, A.K., U. Sellstrom, P. Lindberg, A. Bignert and C.A. deWit. 2009. Polybrominated diphenyl ether congener patterns, hexabromocyclododecane, and brominated-biphenyl 153 in eggs of peregrine falcons (*Falco peregrinus*) breeding in Sweden. Environ. Toxicol. Chem. 28: 9-17.

- Kodavanti, P.R.S., T.R. Ward, G. Ludewig, L.W. Robertson and L.S. Birnbaum. 2005. Polybrominated diphenyl ether (PBDE) effects in rat neuronal cultures: 14C-PBDE accumulation, biological effects, and structure-activity relationships. *Toxicol. Sci.* 88: 181-192.
- Kolic, T.M., K.A. MacPherson, E.J. Reiner, T. Ho, S. Kleywegt, A. Dove and C. Marvin. 2004. Brominated diphenyl ether levels: a comparison of tributary sediments versus biosolid material. *Organohalogen Compounds* 66: 3830-3835.
- La Guardia, M.J., R.C. Hale, E. Harvey, T.M. Mainor and M.O. Gaylor. 2001. Polybrominated diphenyl ethers in land-applied sewage sludge (biosolids). Poster presented at the 22nd Annual Meeting of the Society of Environmental Toxicology and Chemistry, November 2001.
- Lebeuf, M. and S. Trottier. 2001. The relationship between age and levels of polybrominated diphenyl ethers in beluga whales from the St. Lawrence Estuary, Canada. *Organohalogen Compd.* 52: 22-25.
- Lincer, J.L. 1975. DDE-induced eggshell thinning in the American kestrel: A comparison of the field situation and laboratory results. *J. Appl. Ecol.* 12: 781-793.
- Luckey, F.J., B. Fowler and S. Litten. 2002. Establishing baseline levels of polybrominated diphenyl ethers in Lake Ontario surface waters. Unpublished manuscript dated 2002/03/01. New York State Department of Environmental Conservation, Division of Water, 50 Wolf Road, Albany, NY 12233-3508.
- Marteinson, S.C., D.M. Bird, J.L. Shutt, R.J. Letcher, J.I. Ritchie and K.J. Fernie. 2010. Multi-generational effects of PBDE exposure: Embryonic exposure of male American kestrels (*Falco sparverius*) to DE-71 alters reproductive success and behaviors. *Environ. Toxicol. Chem.* 29: 1740-1747.
- Martin, P. A., G.J. Mayne, S.J. Bursian, G. Tomy, V. Palace, C. Pekarik and J. Smits. 2007. Immunotoxicity of the commercial polybrominated diphenyl ether mixture De-71 in ranch mink (*Mustela vison*). *Environ. Toxicol. Chem.* 26: 988-997.
- McKernan, M.A., B.A. Rattner, R.C. Hale and M.A. Ottinger. 2009. Toxicity of polybrominated diphenyl ethers (DE-71) in chicken (*Gallus gallus*), mallard (*Anas platyrhynchos*) and American kestrel (*Falco sparverius*) embryos and hatchlings. *Environ. Toxicol. Chem.* 28: 1007-1017.
- Nakari, T. and S. Huhtala. 2008. Comparison of toxicity of congener-153 of PCB, PBB, and PBDE to *Daphnia magna*. *Ecotoxicology and Environmental Safety* 71: 514-518.
- Noël, M., N. Dangerfield, R.A.S. Hourston, W. Belzer, P. Shaw, M.B. Yunker and P.S. Ross. 2009. Do trans-Pacific air masses deliver PBDEs to coastal British Columbia,

Canada? Environmental Pollution 157: 3404-3412.

Norris J.M., R.J. Kociba, B.A. Schwetz, J.Q. Rose, C.G. Humiston, G.L. Jewett, P.J. Gehring and J.B. Mailhes. 1975. Toxicology of octabromobiphenyl and decabromodiphenyl oxide. Environ. Health Perspect. 11: 153–161.

OECD (Organisation for Economic Co-operation and Development). 1994. Selected brominated flame retardants. Risk Reduction Monograph OECD Environment Monograph Series No. 102, Paris. (<http://www.oecd.org/ehs/ehsmono/#RISK>).

Palm, A. 2001. The environmental fate of polybrominated diphenyl ethers in the Centre of Stockholm - assessment using a multimedia fugacity model. Master of Science Thesis, Umeå Universitet, Stockholm, Sweden. 58 pp.

Peters, A.K., J.T. Sanderson, A. Bergman and M. van den Berg. 2006. Antagonism of TCDD-induced ethoxyresorufin-O-deethylation activity by polybrominated diphenylethers (PBDEs) in primary cynomolgus monkey (*Macaca fascicularis*) hepatocytes. Toxicology Letters 164: 123–132.

Rahman, F., K.H. Langford, M.D. Scrimshaw and J.N. Lester. 2001. Polybrominated diphenyl ether (PBDE) flame retardants. Sci. Total Environ. 275: 1–17.

Rayne, S., M.G. Ikonou and B. Antcliffe. 2003. Rapidly increasing polybrominated diphenyl ether concentrations in the Columbia River system from 1992 to 2000. Environ. Sci. Technol. 37: 2847–2854.

RPA Ltd. (Risk Policy & Analysts Limited). 2000. Risk reduction strategy and analysis of advantages and drawbacks for pentabromodiphenyl ether. March 2000. Report prepared for the UK Department of the Environment, Transport and the Regions.

Stapleton, H.M. and J.E. Baker. 2003. Debromination of BDE congeners by the common carp (*Cyprinus carpio*). 5<sup>th</sup> Annual Workshop on Brominated Flame Retardants in the Environment, August 22-23, 2003. Boston, MA.

Stapleton, H.M., R.J. Letcher, J. Li and J.E. Baker. 2004a. Dietary accumulation of polybrominated diphenyl ethers by juvenile carp (*Cyprinus carpio*). Environ. Toxicol. Chem. 23: 1939-1946

Stapleton, H.M., R.J. Letcher and J.E. Baker. 2004b. Debromination of polybrominated diphenyl ethers BDE 99 and BDE 183 in the intestinal tract of the common carp (*Cyprinus carpio*). Environ. Sci. Technol. 38: 1054-1061

Strandberg, B., N.G. Dodder, I. Basu and R.A. Hites. 2001. Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air. Environ. Sci. Technol. 35: 1078–1083.

- Taylor, C. L., M. P. Latimer and P. Winn. 2003. Impaired delayed spatial win-shift behaviour on the eight arm radial maze following excitotoxic lesions of the medial prefrontal cortex in the rat. *Behavioural Brain Research* 147: 107-114.
- Ucán-Marín, F., A. Arukwe, A. Mortensen, G.W. Gabrielsen, G.A. Fox and R.J. Letcher. 2009. Recombinant transthyretin purification and competitive binding with organohalogen compounds in two gull species (*Larus argentatus* and *Larus hyperboreus*). *Toxicol. Sci.* 107: 440-450.
- Ucán-Marín, F., A. Arukwe, A. Mortensen, G.W. Gabrielsen and R.J. Letcher. 2010. Recombinant albumin transport protein from gull species (*Larus argentatus* and *hyperboreus*) and human: Chlorinated and brominated contaminant binding and thyroid hormones. *Environ. Sci. Technol.* 44: 497-504.
- U.S. EPA (United States Environmental Protection Agency). 2005. Polybrominated diphenylethers (PBDEs) Significant New Use Rule (SNUR) Questions and Answers. ([www.epa.gov/oppt/pbde/qanda.htm](http://www.epa.gov/oppt/pbde/qanda.htm)).
- van den Steen, E., M. Eens, A. Covaci, A.C. Dirtu, V.L.B. Jaspers, H. Neels and R. Pinxten. 2009. An exposure study with polybrominated diphenyl ethers (PBDEs) in female European starlings (*Sturnus vulgaris*): toxicokinetics and reproductive effects. *Environ. Pollut.* 157: 430-436.
- Viberg, H., A. Fredericksson, and P. Ericckson. 2002. Neonatal exposure to the brominated flame retardant 2,2',4,4',5-pentabromodiphenyl ether causes altered susceptibility in the cholinergic transmitter system in the adult mouse. *Toxicol. Sci.* 67: 104-107.
- Viberg, H., A. Fredriksson and P. Eriksson. 2003a. Neonatal exposure to polybrominated diphenyl ether (PBDE 153) disrupts spontaneous behaviour, impairs learning and memory, and decreases hippocampal cholinergic receptors in adult mice. *Toxicology and Applied Pharmacology* 192: 95-106.
- Viberg, H., A. Fredriksson, E. Jakobsson, and P. Eriksson. 2003b. Neurobehavioral derangements in adult mice receiving decabrominated diphenyl ether (PBDE 209) during a defined period of neonatal brain development. *Toxicol Sci.* 76: 112-120
- Viberg, H., N. Johansson, A. Fredriksson, J. Eriksson, G. Marsh and P. Eriksson. 2006. Neonatal exposure to higher brominated diphenyl ethers, hepta-, octa-, or nonabromodiphenyl ether, impairs spontaneous behavior and learning and memory functions of adult mice. *Toxicol. Sci.* 92: 211-218.
- WHO (World Health Organization). 1994. Brominated diphenyl ethers. *Environmental Health Criteria* 162, International Programme on Chemical Safety, WHO, Geneva.
- Whyte, J.J., R.E. Jung, C.J. Schmitt and D.E. Tilitt. 2000. Ethoxyresorufin-O-deethylase



(EROD) activity in fish as a biomarker of chemical exposure. *Crit. Rev. Toxicol.* 30: 347-570.

Wollenberger, L., L. Dinan and M. Breitholt. 2005. Brominated flame retardants: activities in a crustacean development test and in an ecdysteroid screening assay. *Environmental Toxicology and Chemistry* 24: 400-407.

Yen, J.H., W.C. Liao, W.C. Chen and Y.S. Wang. 2009. Interaction of polybrominated diphenyl ethers (PBDEs) with anaerobic mixed bacterial cultures isolated from river sediment. *Journal of Hazardous Materials* 165: 518-524.

Zhou, T., M.M. Taylor, M.J. DeVito and K.M. Crofton. 2002. Developmental exposure to brominated diphenyl ethers results in thyroid hormone disruption. *Toxicol. Sci.* 66: 105–116.

### List of Acronyms and Terms

AF - application factor

BAF - bioaccumulation factor

BCF - bioconcentration factor

BFR - brominated flame retardant

BMF - biomagnification factor

BrPh - tribromophenol

BSAF - biota-sediment accumulation factor

BW - body weight

CAS - Chemical Abstracts Service

CCME - Canadian Council of Ministers of the Environment

CEPA - Canadian Environmental Protection Act

Congener- a compound belonging to a homologue series differing only in the position of the bromine atoms in the molecule. For example, for the homologue tribromodiphenyl ether, the 3 bromine atoms can be positioned at carbons 1,2,3 or 1,2, 4, or 1,2,5, or 1,2,6, or 2,3,4, etc. resulting in up to a total of 24 possible congeners. The mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decabromo congeners can exist in 3, 12, 24, 42, 46, 42, 24, 12, 3, and 1 forms, respectively. There is only one congener of decaBDE as there is only one possible configuration for all 10 bromine atoms.

CTV - critical toxicity value

DW - dry weight

EROD - ethoxyresorufin-o-deethylase - (a catalytic measurement of cytochrome P4501A induction) is a biomarker in fish. A multitude of chemicals induce EROD activity in a variety of fish species, the most potent inducers being structural analogs of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (Whyte et al. 2000).

FEQG - Federal Environment Quality Guideline

FSeQG - Federal Sediment Quality Guideline

FWiDG - Federal Wildlife Dietary Guidelines

FFTG - Federal FishTissue Guideline

HBCD - hexabromocyclododecane

Homologue- a compound belonging to a series of substances, which have the same base structure and differing only by the number of repeating units, such as the number of bromine atoms in the molecule. For PBDEs the homologues are: monobromodiphenyl ether, dibromodiphenyl ether, tribromodiphenyl ether (with 3 bromine atoms), tetrabromodiphenyl ether (4Br), penta-bromodiphenyl ether (5 Br), hexabromodiphenyl ether (6 BR), heptabromodiphenylether (7 Br) , octa bromodiphenyl ether (8 Br), nonabromodiphenyl ether (9Br) and decabromodiphenyl ether.

LOAEL - lowest observable adverse effect level  
LW - lipid weight  
NOAEL - no observable adverse effect level  
PBDE - polybrominated diphenyl ether  
PBiT - (P)ersistent (B)ioaccumulative and (i)nherently (T)oxic  
PCB - polychlorinated biphenyl  
PNEC - probable no effect concentration  
RC - reference concentration  
SAR - Screening Assessment Report  
TDI - tolerable daily intake  
TBBPA - tetrabromobisphenol A  
TBBPA OHEE - tetrabromobisphenol A hydroxyethyl ether  
TBECH - tetrabromoethylcyclohexane  
UF - uncertainty factor  
WW - wet weight