Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE)

Bioaccumulation and Transformation

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Summary

In July 2006, the final decision on the screening assessment of substances – polybrominated diphenyl ethers (PBDEs) – was published by the Minister of the Environment and the Minister of Health in the *Canada Gazette*, Part I. It was concluded that PBDEs—i.e., tetrabromodiphenyl ether (tetraBDE), pentabromodiphenyl ether (pentaBDE), hexabromodiphenyl ether (hexaBDE), heptabromodiphenyl ether (heptaBDE), octabromodiphenyl ether (octaBDE), nonabromodiphenyl ether (nonaBDE) and decabromodiphenyl ether (decaBDE)—which are found in commercial PentaBDE, OctaBDE and DecaBDE technical formulations, are entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity, and thus meet the criteria set out in paragraph 64(a) of the Canadian Environmental Protection Act, 1999 (CEPA 1999). In addition, it was concluded that all seven PBDEs homologues met criteria for persistence, but only tetra- to hexaBDE met the criteria for bioaccumulation as defined in the *Persistence and Bioaccumulation Regulations*. The analysis also noted that the higher brominated diphenyl ethers, and decaBDE in particular, could accumulate to some degree in biota and debrominate to bioaccumulative and persistent transformation products.

Since the completion of the Ecological Screening Assessment, a large amount of new information has been published regarding the accumulation of decaBDE in biota and the potential transformation of decaBDE to persistent and bioaccumulative products. The purpose of the present report is to provide an updated analysis of bioaccumulation and transformation of decaBDE, by summarizing evidence considered in the original Screening Assessment, and then examining the related new science published up to August 25, 2009.

Overall, available data do not show that the decaBDE itself meets the numeric criteria for bioaccumulation as defined in the *Persistence and Bioaccumulation Regulations*. With regard to bioaccumulation, potential factors such as low assimilation efficiency and/or metabolic transformation appear to be important determinants of accumulation in organisms. Nevertheless, some recent studies have shown concentrations of decaBDE to be increasing steadily in some wildlife species and there are a few equivocal reports of biomagnification factors (BMFs) exceeding 1. In some cases, such as in the tissues of kestrel, sparrowhawk, peregrine falcon, glaucous gull, red fox, shark, harbour porpoise and whitebeaked dolphin, measured concentrations of decaBDE in tissues are interpreted as high. While trophic magnification or bioaccumulation is a potential explanation for these high concentrations, it is also very possible that some biota are exposed to very high exposure concentrations of decaBDE by consuming contaminated refuse and/or inhabiting decaBDE hotspots close to industrialized areas.

This report also considers it reasonable to conclude that decaBDE may contribute to the formation of lesser-brominated PBDEs and other metabolic products in organisms—potentially those that are bioaccumulative. Although there is some uncertainty, the evaluation found evidence that fish and mammals appear to have some capacity to metabolically break down decaBDE. In fish, decaBDE appears to form hepta- to nonaBDEs, and potentially penta- and hexaBDEs. In mammals, debromination of decaBDE down to heptaBDEs has been observed. In both fish and mammals, formation of lower brominated PBDEs appears to be very low and only a fraction (typically on the order of a few percent) of the total amount of decaBDE administered to the organism. However, some rodent studies have made inferences, based on mass balance evaluations, that rates of transformation may be higher, with one study suggesting that approximately 45% of the

total dose of decaBDE was unaccounted for and may have been metabolized to other compounds (such as hydroxylated and hydroxymethoxylated PBDEs) and/or bound as inextricable residues.

The evaluation of transformation in the environment identified numerous laboratory studies that provide evidence that decaBDE may break down in the environment, particularly as a result of photodegradation and biodegradation. Studies of photodegradation of decaBDE sorbed to solids in aqueous and dry systems have demonstrated transformation of decaBDE to tri- to nonaBDEs, tri- to octabrominated dibenzofurans (octaBDFs) and unidentified products. While relevant to the environment, the actual fraction of decaBDE exposed to sunlight adsorbed to atmospheric and aquatic particulates, or solids (anthropogenic or natural), would be a small fraction of the total amount of decaBDE in the environment. Biodegradation studies have also shown potential breakdown of decaBDE mainly to nona-, octa- and heptaBDEs, while transformation to triBDEs has also been shown under enhanced laboratory conditions. Overall, biodegradation appears to occur at a much slower rate than that of phototransformation, with half-lives in the range of several years to several decades. The photolytic half-life of decaBDE adsorbed to house dust and exposed to sunlight has been reported to range from approximately 1–2 months (assuming 8 hours of sunlight per day).

Modelling of bioaccumulation factors (BAFs) and BMFs was conducted to estimate whether transformation products of decaBDE resulting from processes in organisms and in the general environment could be bioaccumulative. The evaluation found that many of the identified transformation products could be bioaccumulative (i.e., have BAFs in excess of 5000) and some could biomagnify in food chains. The analysis also indicated potential transformation of decaBDE to products (i.e., tetra- to hexaBDEs) that have been established as bioaccumulative based on empirical evidence.

While laboratory studies on the transformation of decaBDE support a conclusion that transformation to lower BDEs and BDFs should be occurring in the environment, the phenomenon has not been conclusively shown through monitoring studies to occur in the environment. This suggests that the process of environmental transformation may be very slow and subtle, and possibly may have relevance to a small fraction of the total decaBDE reservoir in the environment. Evidence of transformation may be shielded by existing patterns of PBDEs in the environment which are dominated by congeners found in the commercial products. The fact that fewer studies have historically measured octa- and nonaBDE congeners in environmental samples makes the elucidation of debromination patterns of decaBDE in the environment challenging.

While this report has focused on decaBDE, its analyses and conclusions provide useful inferences on alternative flame retardants with similar chemical structures and use patterns, such as decabromodiphenyl ethane (decaBD ethane). DecaBDE and decaBD ethane have only minor structural differences relating to the bond between their aromatic rings and, thus, these substances may have similarities in physical and chemical properties, persistence, transformation patterns, and accumulation in organisms. Based on the similarity in properties between decaBDE and decaBD ethane, the presence of decaBD ethane in Canadian wildlife, and the potential for decaBD ethane to be used as a large-scale replacement for decaBDE, there is also a need to further understand the potential risks from decaBD ethane in the environment and its capacity to accumulate in wildlife and transform to potentially bioaccumulative products. Understanding the risk from alternatives generally will help to ensure that substitutions of flame retardants are made on an informed basis.

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List of Acronyms

Acronym	Description
B	bioaccumulative
BAF–QSAR	bioaccumulation factor – quantitative structure-activity relationship
BAF	bioaccumulation factor
BCF	bioconcentration factor
BDE	brominated dipheynyl ether
BDE209	congener form of decaBDE
BDF	brominated dibenzofuran
BDL	below detection limit
BFR	brominated flame retardant
BMF	biomagnification factor
bw	body weight
bw/d	body weight per day
CEPA 1999	*Canadian Environmental Protection Act, 1999*
CF	confidence factor
Ci/mol	curies/mol
cm	centimetre
CYP1A	cytochrome P4501A
d	day or days
DE	diphenyl ether
decaBDE	decabromodiphenyl ether
DL	detection limit
dw	dry weight
ECNI	electron capture negative ionization
E_D	dietary assimilation efficiency
EI	electron ionization
EVA	Experimental Value Adjustment
FID	flame ionization detector
g	grams
GC	gas chromatography
GC/MS	gas chromatography / mass spectrometry
GC–ECD	gas chromatography – electron capture detection
GI	gastrointestinal
h	hour or hours
heptaBDE	heptabromodiphenyl ether
hexaBDE	hexabromodiphenyl ether
HIPS	high-impact polystyrene
HPLC	high-performance liquid chromatography
HRGC	high-resolution mass spectrometry
HRMS	high-resolution mass spectrometry
k_2	elimination rate constant
k_E	fecal egestion rate constant
k_G	growth rate constant

kg/d	kilograms per day
k_M	metabolic rate constant
K_{oa}	octanol-air partition coefficient
KOAWIN	model for prediction of octanol-air partition coefficient
K_{ow}	octanol-water partition coefficient
k_T	total elimination rate
L	litre
Lipid	lipid weight
LRMS	low-resolution mass spectrometry
M	molarity, as the number of moles of a solute in 1 litre of solution
MDL	method detection limit
mg/d	milligrams per day
mg/kg	milligrams per kilogram
MITI	Ministry of International Trade and Industry (Japan)
mL	millilitre
mmol/kg	millimole per kilogram
mPa	millipascal
MMP	Molecular Modelling Pro
n	number of samples
ng/d	nanogram per day
ng/g	nanogram per gram
nm	nanometre
NMR	nuclear magnetic resonance
nonaBDE	nonabromodiphenyl ether
OC	organic carbon
octaBDE	octabromodiphenyl ether
P	persistent
PBDD	polybrominated dibenzo-*p*-dioxin
PBDE	polybrominated diphenyl ether
PBDF	polybrominated dibenzofuran
PCB	polychlorinated biphenyl
pentaBDE	pentabromodiphenyl ether
pg/g	picogram per gram
QSAR	quantitative structure-activity relationship
QSPR	quantitative structure-property relationships
sediment BSAFs	sediment biota-sediment accumulation factors
SIM	selected ion monitoring
soil BSAFs	soil biota-soil accumulation factors
SRM	standard reference material
TCE	trichlorethane
tetraBDE	tetrabromodiphenyl ether

THF	tetrahydrofuran
TMF	trophic magnification factor
TTR	transthyretin
µg	micrograms
µg/kg	micrograms per kilogram
µg/L	micrograms per litre
µL	microlitres
µm	micrometer
µmol/kg	micromole per kilogram
UK	United Kingdom
US	United States
UV	ultraviolet
v/v	volume per volume
W	Watts
w/w	weight per weight
ww	wet weight
WWTP	wastewater treatment plant

1 Introduction

The purpose of this report is to provide an updated analysis of the bioaccumulation and environmental transformation of decabromodiphenyl ether (decaBDE), to be considered in the context of the information and analyses already published in the final screening assessment on polybrominated diphenyl ethers (PBDEs) (Canada 2006). This evaluation is considered a state of the science review. While this report does not critique individual studies, it considers the reliability of individual studies when forming a weight of evidence for persistence, bioaccumulation or inherent toxicity to non-human biota. This report considers materials published up to August 25, 2009.

1.1 Background

In July 2006, the ministers of Environment and Health published their final screening assessment on PBDEs (Canada 2006). The environmental screening assessment examined various supporting information and developed conclusions based on a weight-of-evidence approach as required under subsection 76.1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999). As the term "screening assessment" implies, this was not an exhaustive review of all available data, but rather, it presented the most critical studies and lines of evidence supporting the conclusions (e.g., relating to persistence, bioaccumulation, inherent toxicity, risk quotients and long-range transport to remote regions like the Arctic). The PBDE screening assessment concluded that PBDEs—tetrabromodiphenyl ether (tetraBDE), pentabromodiphenyl ether (pentaBDE), hexabromodiphenyl ether (hexaBDE), heptabromodiphenyl ether (heptaBDE), octabromodiphenyl ether (octaBDE), nonabromodiphenyl ether (nonaBDE) and decaBDE—which are found in commercial PentaBDE, OctaBDE and DecaBDE technical formulations, are entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity and thus meet the criterion under paragraph 64(a) of CEPA 1999. In addition, it was concluded that all of the PBDEs assessed met criteria for persistence, but only tetra- to hexaBDEs met the criterion for bioaccumulation as identified in the *Persistence and Bioaccumulation Regulations* (Canada 2000) under CEPA 1999. It also noted that higher brominated PBDEs, and decaBDE in particular, could accumulate to some degree in biota and debrominate to bioaccumulative and persistent transformation products.

Information obtained as of October 2004 was considered for inclusion into the environmental screening assessment (Environment Canada 2006a; Environment Canada 2006b), while information received between November 2004 and October 2005 was reviewed but not generally added to the assessment, as these studies supported the conclusions of the draft assessment published for public comment in 2004. Since 2004, a large amount of information has been and continues to be published on PBDEs and on the issues of decaBDE bioaccumulation and transformation. This report examines the new data concerning decaBDE and examines the development of lines of evidence respecting this substance's bioaccumulation and transformation. In addition, a Notice of Objection, dated February 14, 2007, was submitted to the Minister of the Environment by

the Sierra Legal Defence Fund (now known as Ecojustice Canada) on behalf of the David Suzuki Foundation, Environmental Defence, and the Canadian Environmental Law Association, requesting the establishment of a Board of Review to clarify the Canadian regulatory approach on PBDEs and to recommend changes to the proposed Polybrominated Diphenyl Ethers Regulations. In order to consider information and issues raised in the Notice of Objection and to inform a decision respecting the formation of a Board of Review, this detailed evaluation was prepared to consider the current science respecting the bioaccumulation and transformation of decaBDE to bioaccumulative products. This report considers published information up to August 25, 2009, identified in original literature and review documents. In addition to retrieving the references from a literature database search, direct contacts were made with researchers, academics, industry and other government agencies to obtain relevant information on decaBDE.

The analysis presented herein summarizes relevant information on bioaccumulation and transformation examined in the PBDE screening assessment (Canada 2006; Environment Canada 2006b), and examines how the new science builds upon existing lines of evidence respecting bioaccumulation and transformation. Ecological considerations rather than those relating to human health are presented. While the screening assessment of PBDEs (Health Canada 2004) concluded that worst-case estimates of Canadians' exposure to PBDEs were much lower than the levels of exposure which caused health effects in laboratory animals, in light of uncertainties regarding the available database, Health Canada supports Environment Canada's actions to limit the use of PBDEs so that levels do not reach a point where they could potentially harm the health of Canadians. The evaluation does not consider decaBDE accumulation in humans; however, consideration is given to laboratory rodent studies that are used in human health risk assessment since such studies also provide insight into impacts relating to mammalian wildlife.

This state of science report has undergone substantial internal and external written peer review/consultation. While external comments were taken into consideration, the final contents remain the responsibility of Environment Canada. Additionally, the draft of this evaluation was subject to a 60-day public comment period. The critical information and considerations upon which the evaluation is based are summarized below.

1.1.1 Composition of Commercial DecaBDE

In this evaluation, information on both the congener form of decaBDE (BDE209) and the commercial product also known as Decabromodiphenyl Ether (DecaBDE) are considered. Since nona- and octaBDEs are also found in DecaBDE formulations, these congeners are considered if appropriate.

According to the World Health Organization (WHO 1994), current manufactured formulations of DecaBDE typically contain
- decaBDE, 97–98% w/w, and
- other PBDEs (mainly nonaBDE), 0.3–3.0 w/w.

Older commercial DecaBDE products contained a higher proportion of lower brominated PBDEs (mainly nonaBDE and octaBDE isomers) than more recent formulations. For

instance, FR-300-BA, produced in the 1970s and no longer commercially available, contained 77.4% decaBDE, 21.8% nonaBDE and 0.8% octaBDE (Norris et al. 1975).

La Guardia et al. (2006) analyzed compositions of the currently manufactured DecaBDE product, Saytex 102E, and compared it with that of Bromkal 82-ODE, which has not been manufactured for more than a decade (Table 1-1). They found that Saytex 102E and Bromkal 82-ODE contained 96.8% and 91.6% BDE209, respectively. Both formulations contained nonaBDEs, with BDE206 in the highest quantity, followed by BDE207 then -208. In addition, Bromkal 82-ODE contained 0.56% octaBDEs, with BDE196, -203 and -197 identified. OctaBDEs were not identified in the Saytex 102E product.

Commercial OctaBDE products (no longer in production) also contained decaBDE (La Guardia et al. 2006). For instance, Bromkal 79-8DE contained 49.6% decaBDE as well as significant nonaBDE (Table 1-1). In comparison, DE-79 contained lesser amounts of deca- and nonaBDE (i.e., 1.31 and 13.07%, respectively).

Table 1-1: Concentrations (%, w/w) of PBDEs in selected commercial Octa- and DecaBDE products (La Guardia et al. 2006)

PBDE Congener	Commercial OctaBDE Products		Commercial DecaBDE Products	
	DE-79	Bromkal 79-8DE	Saytex 102E	Bromkal 82-ODE
BDE154	1.07	0.04	nd[2]	nd
BDE144	0.1	0.12	nd	nd
hexaBDE[1]	< 0.02	nd	nd	nd
BDE153	8.66	0.15	nd	nd
BDE139	nd	nd	nd	nd
BDE140	< 0.02	nd	nd	nd
BDE138	0.62	nd	nd	nd
BDE184	< 0.02	< 0.02	nd	nd
heptaBDE[1]	< 0.02	nd	nd	nd
BDE175/183	42	12.6	nd	nd
BDE191	< 0.02	nd	nd	nd
BDE180	1.7	nd	nd	nd
BDE171	1.81	0.17	nd	nd
BDE201	0.78	< 0.02	nd	nd
BDE197	22.2	10.5	nd	0.03
BDE203	4.4	8.14	nd	0.07
BDE196	10.5	3.12	nd	0.46
BDE194	< 0.02	nd	nd	nd
octaBDE[1]	< 0.02	nd	nd	nd
BDE208	0.19	< 0.02	nd	0.07
BDE207	11.5	11.2	0.24	4.1
BDE206	1.38	7.66	2.19	5.13
BDE 209	1.31	49.6	96.8	91.6

[1] Categorized only based on degree of bromination
[2] nd = not detected

1.2 Definitions and Rationale

For the purposes of this document, it is important to define and distinguish between bioaccumulation, bioconcentration and biomagnification. The term "bioaccumulation" has been recently summarized by Arnot and Gobas (2006):

> *Bioaccumulation is a process in which a chemical substance is absorbed in an organism via all routes of exposure as typically occurs in the natural environment, i.e., dietary and ambient environment sources. Bioaccumulation is the net result of competing processes of chemical uptake and elimination including the diet, respiratory exchange, fecal egestion, metabolic transformation of the "parent" compound and growth dilution… Growth dilution is considered a "pseudo-elimination" route since the chemical is not actually eliminated by the organism but the concentration can be diluted by an increase in the volume of tissue.*

Thus, it is reasonable to expect that if a chemical is detectable in organism tissues, it is there as the result of the process of bioaccumulation. Bioaccumulation can be of concern because when organisms take up chemicals, the likelihood that those organisms will be harmed generally increases with the amount accumulated. In addition, as bioaccumulation increases, there is increased likelihood that contaminated organisms will cause indirect harm to predators that consume them. Chemicals that are highly bioaccumulative are of particular concern because they tend to biomagnify, i.e., increase in concentration from prey to predator across several trophic levels. Thus, relatively low ambient concentrations of highly bioaccumulative substances have the potential to cause both direct toxicity and indirect toxicity due to biomagnification.

It is important to distinguish between the process of "bioaccumulation" and the term "bioaccumulative" as used in the *Persistence and Bioaccumulation Regulations* (Canada 2000), a regulation made under CEPA 1999 and as described in Canada's Toxic Substances Management Policy (Canada 1995a, 1995b). According to the *Persistence and Bioaccumulation Regulations*:

> *A substance is bioaccumulative*
> *(a) when its bioaccumulation factor is equal to or greater than 5000;*
> *(b) if its bioaccumulation factor cannot be determined in accordance with a method referred to in section 5, when its bioconcentration factor is equal to or greater than 5000; and*
> *(c) if neither its bioaccumulation factor nor its bioconcentration factor can be determined in accordance with a method referred to in section 5, when the logarithm of its octanol-water partition coefficient is equal to or greater than 5.*

The Regulations go on to indicate that "[t]he determination of persistence and bioaccumulation…must be made…taking into account the intrinsic properties of the substance, the ecosystem under consideration and the conditions in the environment." Thus, determining whether these criteria are met involves professional judgement which considers the intrinsic properties of the substance and ecosystem under consideration.

These criteria were first proposed as part of the federal Toxic Substances Management Policy (Canada 1995a, 1995b) and were intended to address lipophilic substances with the potential to bioaccumulate and biomagnify in aquatic organisms to levels causing effects at the top of the food web. Application of a criterion of 5000 for bioaccumulation

factors (BAFs) or bioconcentration factors (BCFs), or 5 for log K_{ow} (octanol-water partition coefficient), are recommended in the Policy. Substances that undergo significant bioaccumulation and/or biomagnification are considered "bioaccumulative." As noted above, professional judgement is required in the application of these criteria. Notably, the Policy indicates that log K_{ow} should be used with caution in predicting the bioconcentration and bioaccumulation potential of organic substances (Canada 1995b). For instance, many substances can be metabolized and this can function to decrease bioaccumulation potential of the parent compound, and thus many substances with a very high log K_{ow} (e.g., >5) can have very low bioaccumulation/bioconcentration potential.

Based on the definition in CEPA 1999 and its regulations which have their basis in the Toxic Substances Management Policy, the term "bioaccumulative" is not synonymous with the term "bioaccumulation." Rather, "bioaccumulation" is the process which may lead to a substance being considered "bioaccumulative." Evidence that a substance is both persistent and bioaccumulative when combined with evidence of toxicity and release or expected release into the environment provides a significant indication of its potential to cause ecological harm (Environment Canada 2007).

Gobas and Morrison (2000) provide a concise definition of the process of "biomagnification" as it is currently understood:

> *This is the process in which the chemical concentration in an organism achieves a level that exceeds that in the organism's diet, due to dietary absorption. The extent of chemical biomagnification in an organism is best determined under laboratory conditions, where organisms are administered diets containing a known concentration of chemical, and there is no change in chemical uptake through other exposure routes (e.g., respiratory surface, dermis).*
>
> *Biomagnification also can be determined under field conditions, based on chemical concentrations in the organism and its diet. Biomagnification factors derived under controlled laboratory conditions, which exclude uptake through routes other than the diet, are different from those determined under field conditions, because field-based biomagnification factors are inevitably the result of chemical uptake by all routes of chemical uptake, rather than dietary absorption alone.*

The terms "trophic magnification," "food web magnification" and "food web biomagnification" are often used interchangeably to refer to the same phenomenon. This is the phenomenon whereby chemical concentrations in organisms increase with trophic level, resulting in higher concentrations in predators than in prey (i.e., biomagnification through successive trophic levels). Gobas and Morrison (2000) describe "food-chain" bioaccumulation of neutral organic substances as

> *…the process in which chemical concentrations in organisms increase with each step in the food-chain, resulting in chemical concentrations in predators that are greater than those in their prey. Because concentrations of many hydrophobic chemicals in organisms increase as the lipid content of the organism increases, the occurrence of food-chain bioaccumulation is detected best by comparing chemical concentrations in predators and prey on a lipid weight basis. An increase in lipid-based concentrations in organisms with increasing trophic level indicates food-chain bioaccumulation.*

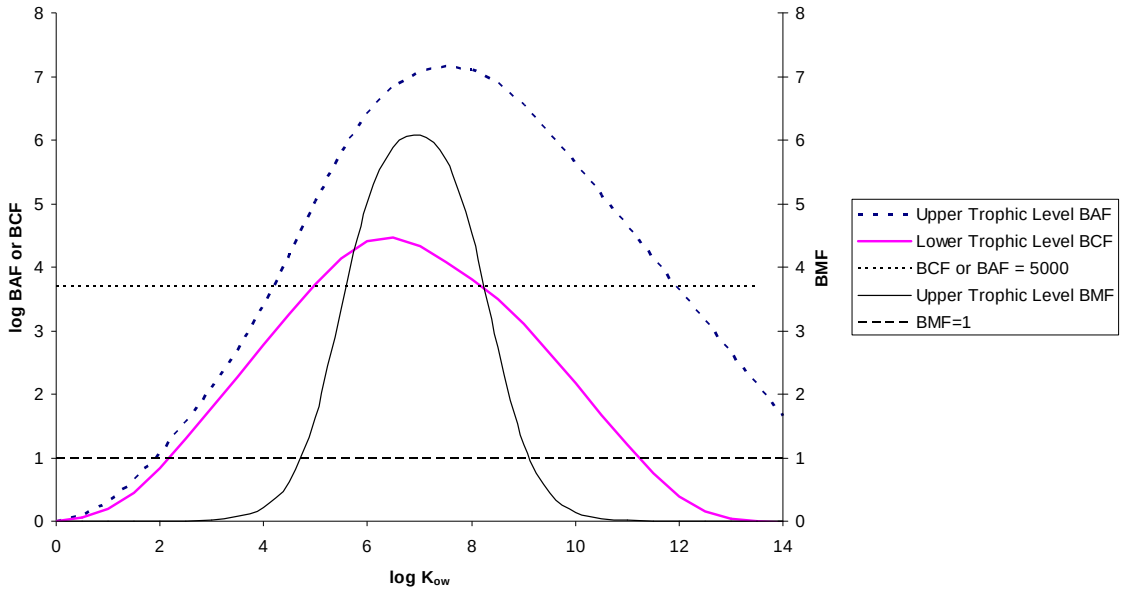
Potential indicators of biomagnification or trophic magnification include field-measured biomagnification factors (BMFs—the ratio in concentration between predator and prey) for known predator-prey relationships, or trophic magnification factors (TMFs—sometimes called food web magnification factors) which examine the incremental magnification with each trophic level for the food web as a whole. TMFs also represent the average ratio of concentrations between predator and prey through an entire food web. A BMF or TMF exceeding 1 indicates that biomagnification or trophic magnification is occurring. The use of BMFs and TMFs are generally thought to be more appropriate for terrestrial and marine mammals which breathe air rather than water. Note that in laboratory feeding studies, dietary BAFs are often estimated by representing the relationship between the test organisms and their diet (i.e., synonymous with the definition of a BMF). For the purposes of this report, dietary BAFs are referred to as BMFs.

The BAF criterion of 5000 as identified in the *Persistence and Bioaccumulation Regulations* denotes chemicals which are highly bioaccumulative at the organism level and may also suggest the potential for biomagnification or trophic magnification. It should be noted that BAFs measured or estimated from total water concentrations are dependent on the quantity and quality of organic carbon and particulate matter in the water column. The available fraction of a chemical in water determines the uptake potential of a substance and subsequently the bioaccumulation potential. The BAF–QSAR model of Arnot and Gobas (2003) provides bioaccumulation predictions for a generic aquatic food web (with lower, middle and upper trophic levels) based on the current understanding of bioaccumulation processes in aquatic organisms, accounting for the available fraction in water. Figure 1-1 illustrates the model-predicted relationship for hydrophobic, non-metabolized substances between log K_{ow}, BAF, BCF and BMF for aquatic lower and upper trophic levels in the model. The model predictions suggest that as log K_{ow} increases toward and above 4–5, the BAF increases toward and above 5000. Also, in this range of log K_{ow}, the BMF begins to significantly exceed 1, indicating that biomagnification may occur.

The underlying mechanism for this phenomenon is described by Kelly et al. (2004). For non-metabolized chemicals in aquatic organisms, the BMF results from competing processes of chemical uptake (respiratory and dietary) and chemical elimination (respiratory and gastrointestinal), which depend on organism physiology and chemical hydrophobicity. For aquatic organisms, respiratory elimination is significant for less hydrophobic chemicals, and elimination processes override gastrointestinal absorption processes. However, as hydrophobicity increases (i.e., log $K_{ow} > 5$), respiratory elimination slows and gastrointestinal absorption becomes significant, resulting in biomagnification (i.e., a BMF exceeding 1). At very high log K_{ow}, dietary chemical absorption efficiency tends to decline because chemicals are too strongly sorbed to the food matrix to be absorbed into the organism, resulting in a decline in both the BAF and the BMF (Arnot and Gobas 2003, 2004). Regardless of log K_{ow}, when sufficient, chemical metabolism often sustains the total elimination rate at a high enough level to prevent chemical biomagnification, even for higher log K_{ow} substances (Arnot and Gobas 2003; Kelly et al. 2004).

This relationship between log K_{ow}, BAF and BMF for non-metabolized substances, which is based on current knowledge of bioaccumulation and biomagnification in aquatic organisms, supports the use of a BAF criterion of 5000 to indicate when bioaccumulation may occur. Note that the BCF is a less conservative endpoint since it does not increase as quickly with log K_{ow} as the BAF. However, a lower trophic level BCF of 5000 generally corresponds to a log K_{ow} of 5, which is consistent with the log K_{ow} criterion of the *Persistence and Bioaccumulation Regulations*.

Figure 1-1: The relationship between BAF, BCF and BMF for upper and lower trophic levels predicted by the BAF–QSAR model for hydrophobic, non-metabolized substances



Two additional measures which can also provide evidence of biomagnification include biota-sediment accumulation factors (sediment BSAFs) and biota-soil accumulation factors (soil BSAFs). The BSAF represents the steady-state concentration ratio between an organism and soil/sediment, on a lipid-normalized and organic-carbon-normalized basis. Assuming equal chemical sorptive capacities between lipid and organic carbon, sediment BSAFs of 1 are expected when organisms are in equilibrium with sediments (i.e., in the absence of food web biomagnification or trophic dilution). However, given the observed difference in sorptive capacity between lipid and organic carbon (see Seth et al. 1999), BSAFs up to approximately 3 are still consistent with equilibrium conditions. Alternatively, the American Society for Testing Materials (ASTM 1997) recommends a "cut-off" value of 1.7 to represent equilibrium conditions. Thus, sediment BSAFs that exceed approximately 1.7 to 3 suggest that food web biomagnification or an alternative magnification process is increasing organism chemical concentrations above equilibrium. Although there are currently no guidelines for evaluating soil BSAFs, a similar range would likely be appropriate to assess whether a soil BSAF value provides evidence that food web biomagnification or an alternative magnification process is increasing organism chemical concentrations above equilibrium.

The evaluation of bioaccumulation data in this report mainly relies on a combination of measured and model-predicted BAFs, BCFs, BMFs and TMFs, and in a few cases, measured sediment and soil BSAFs. Concentrations of decaBDE in organisms are also considered subjectively; however, without quantification of exposure concentrations, definite conclusions respecting the capacity for decaBDE to bioaccumulate cannot be made from biota concentrations alone. Consideration is also given to chemical uptake and elimination and metabolism. An organism's metabolic capacity can reduce the bioaccumulation potential of a substance; however, this metabolic capacity can also result in the formation of bioaccumulative transformation products. Full evaluation of BAFs, BCFs, BMFs and TMFs would consider the bioaccumulation potential of both the parent substance and its metabolic product(s). Further discussion on metabolic transformation is found later in this report.

2 Evidence for Bioaccumulation

The evaluation of new lines of evidence conducted in this section relies on the definitions of bioaccumulative and biomagnification summarized in the preceding section. Most importantly, the evaluation of whether these new lines of evidence indicate that decaBDE is bioaccumulative relies primarily on ratio-based methods for bioaccumulation assessment. From the definitions in Section 1.2, it can be argued that the degree of chemical accumulation in an organism is best characterized by a ratio comparing the concentration of the subject substance in an organism to the appropriate exposure concentration in the organism's environment (i.e., to enable quantification of BCFs, BAFs, BMFs, TMFs or BSAFs).

Other evidence, such as the presence of high concentrations in top predators, is sometimes useful as a supporting line of evidence for ratio-based measures of bioaccumulation. However, it is recognized that the characterization of biota chemical concentrations as "high" is subjective and is based on professional judgement, and thus prone to alternative interpretation.

The evidence provided by studies quantifying chemical residues in tissues is often confounded by various factors. For instance, in many monitoring and field studies, particularly those showing unusually high concentrations in predator organisms, there is a lack of knowledge regarding levels of exposure. In such instances, quantification of the BAF or BMF is impossible. Although a high concentration of a chemical in a top predator could be due to trophic magnification, it could also be due to high geographically localized exposure to a particular chemical, as would be the case if the organism was inhabiting or scavenging from waste dumps. It is also possible that uptake routes other than the food chain can be occurring, such as inhalation, or direct ingestion of plastic. Further, the analysis of decaBDE can be challenging, affected by a number of critical factors that may contribute to the overall accuracy of reported levels in the environmental samples. For instance, de Boer and Wells (2006) note that particular attention is required during sampling and analysis to avoid issues relevant to

- degradation due to sunlight, or UV light;

- poor solubility;
- high background concentrations/potential for contamination and frequent low concentrations in biota; and
- thermal degradation.

In addition, Covaci et al. (2007) describe analytical issues relevant to gas chromatography (GC) set-up affecting the precision of congener determination. They discuss a number of chromatographic interferences that hamper good quality data, particularly in relation to di-, tetra- and hexaBDEs. Apart from using adequate standards, Björkland et al. (2003) also highlighted the need to optimize several other parameters relevant to GC / mass spectrometry (MS), such as ionization energy, moderating gas pressure, ion source temperature and analyzer temperature.

The analysis of decaBDE in environmental samples has apparently improved over the past decade, as demonstrated by a recent inter-laboratory study undertaken to validate and harmonize the analytical methodology (Leonards and Duffek 2008). In total, ten routine laboratories participated in this evaluation of decaBDE in dust and sediment, including nine laboratories from Europe and one from Canada. Overall, the evaluation found acceptable accuracy in results from all laboratories when special attention was focused on quality assurance / quality control (QA/QC) procedures.

The next section summarizes the data respecting decaBDE biota concentrations considered in the PBDE screening assessment report (Canada 2006; Environment Canada 2006b). This summary is followed by an analysis of new data available after 2004 and an interpretation of their significance in relation to bioaccumulation and biomagnification of decaBDE.

2.1 Biota Concentration Data

2.1.1 Information Evaluated in the Screening Assessment

- Norstrom et al. (2002) did not detect decaBDE in herring gull (*Larus argentatus*) eggs from the Great Lakes.
- Lichota et al. (2004) determined a total PBDE concentration (67% of which was decaBDE) of 0.777 µg/kg lipid measured in fat from Vancouver Island marmot (*Marmota vancouverensis*).
- European Communities (2002) and Law et al. (2003) summarized the results of many analyses for decaBDE in fish and marine mammals from Europe and elsewhere. It was noted that decaBDE was detected only occasionally, at a concentration close to the detection limit of the method used.
- DecaBDE (i.e., BDE209) was detected once at a concentration of 1.4 µg/kg wet weight (ww) in mussels from Japan (sampling conducted from 1981 to 1985) (Watanabe et al. 1987).
- Dodder et al. (2002) analyzed decaBDE concentrations in freshwater fish from the northeastern United States and found that all concentrations of decaBDE were below the analytical detection limit (ranged from 1.4 to 1.6 µg/kg ww).

- Ikonomou et al. (2002a) conducted sampling from ringed seal (*Phoca hispida*) blubber between 1981 and 2000, but none of the samples contained decaBDE concentrations in excess of the procedural blanks.
- Ikonomou et al. (2000, 2002a, 2002b) analyzed decaBDE in marine biota collected from the British Columbia coast and Holman Island, Northwest Territories, between 1981 and 2000. The levels of decaBDE were equivalent to those of the procedural blanks.
- DecaBDE (i.e., BDE209) was detected in 18 of 21 analyzed eggs of peregrine falcons (*Falco peregrinus*) from Sweden, at concentrations from 28 to 430 µg/kg lipid (Lindberg et al. 2004).
- A study by de Boer et al. (2001) reported on sampling conducted in 1999 in the North Sea and the Tees estuary, UK, of marine mammals—harbour seal (*Phoca vitulina*), harbour porpoise (*Phocoena phocoena*), white-beaked dolphin (*Lagenorhynchus albirostris*) and bottlenose dolphin (*Tursiops truncatus*)—for levels of BDE209 in liver, muscle and blubber tissues. Most samples showed concentrations of BDE209 below detection limits (ranged from approximately 0.8 to 9.0 µg/kg lipid); however, a few samples showed elevated levels in specific tissues (e.g., up to 26 µg/kg lipid in the blubber of harbour seal, up to 160 µg/kg lipid in the liver of harbour porpoise, and up to 318 µg/kg lipid in white-beaked dolphin).
- Sampling was conducted (de Boer et al. 2004) to determine the occurrence of decaBDE in liver, muscle tissue and eggs of high trophic level bird species from the United Kingdom and the Netherlands. Levels of decaBDE were detected in 10 of 28 liver samples (range < 1.5 to 181 µg/kg lipid), 14 of 28 muscle samples (range < 4.2 to 563 µg/kg lipid) and 25 of 68 eggs (range < 1.8 to 412 µg/kg lipid). Concentrations in Swedish peregrine falcon (*Falco peregrinus*) eggs, which were re-analyzed in the study, were all within 30% of those originally determined by Lindberg et al. (2004). Highest concentrations of decaBDE were measured in muscle tissue samples collected from United Kingdom heron (*Ardea cinerea*) and peregrine falcon (*Falco peregrinus*), and in eggs from Swedish peregrine falcon.

Further details on these studies are provided in Appendix A and in the *Supporting Working Document for the Ecological Screening Assessment of Polybrominated Diphenyl Ethers* (Environment Canada 2006b).

2.1.2 New Biota Concentration Data

Recently, a large number of studies showing tissue concentrations of decaBDE in various global biota have been published. These studies are discussed below and are interpreted with reference to bioaccumulation and biomagnification as defined in earlier sections of this report.

Voorspoels et al. (2006a) reported decaBDE concentrations in red fox (*Vulpes vulpes*) from Belgium. Samples of liver (n=30), muscle (n=33) and adipose tissue (n=27) were collected from foxes which were being processed for diagnostic screening of rabies infection. Concentrations of decaBDE, detected in less than half of the samples of each tissue type, ranged from < 9.1 to 760 ng/g lipid for liver, from < 3.9 to 290 ng/g lipid in muscle and from < 3.7 to 200 ng/g lipid for adipose tissue. The authors concluded that

these data confirmed "unambiguously" that decaBDE does "bioaccumulate" in red fox. However, in a follow-up paper by Voorspoels et al. (2007), decaBDE was not detected in the prey species of fox—wood mice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*). Thus, for this food chain, biomagnification was not identified. However, the authors noted that the amount of tissue that was available for analysis was low due to the small size of the rodents and this caused some elevated limits of quantification, ranging from 7.3 to 17 ng/g lipid for BDE209.

Voorspoels et al. (2006b) reported decaBDE concentrations in birds of prey from Belgium which were found dead or severely injured and had to be euthanized. Bird species included the common buzzard (*Buteo buteo*), sparrowhawk (*Accipiter nisus*), long-eared owl (*Asio otus*), barn owl (*Tyto alba*) and tawny owl (*Stryx aluco*). Tissues analyzed from each species included brain, fat, liver, muscle and adipose tissue. Levels of decaBDE were detected in nearly all serum samples (2–58 ng/g lipid for all species combined) and in some liver samples (below detection limit (BDL)—190 ng/g lipid for all species combined), but not in any other tissues. Based on these findings, the authors concluded that either the exposure to decaBDE was low, or that this congener was poorly accumulated. In the follow-up paper by Voorspoels et al. (2007), decaBDE was not detected in prey species of buzzard (wood mice, *Apodemus sylvaticus*; and bank voles, *Clethrionomys glareolus*) or sparrowhawk (great tit, *Parus major*), meaning that biomagnification could not be assessed.

Jaspers et al. (2006) analyzed liver and muscle samples from seven species of aquatic birds and terrestrial predatory birds from Belgium—grey heron (*Ardea cinerea*) and great crested grebe (*Podiceps cristatus*), both aquatic, and common buzzard (*Buteo buteo*), kestrel (*Falco tinnunculus*), sparrowhawk (*Accipiter nisus*), long-eared owl (*Asio otus*) and barn howl (*Tyto alba*), all terrestrial. DecaBDE (i.e., BDE209) was detected in one of two muscle samples from barn owl at 68 ng/g lipid and not detected in muscle tissue of any other species (detection limit (DL) = 1–2.25 ng/g lipid). DecaBDE (BDE209) was not detected in liver of heron, grebe or buzzard. The range of decaBDE concentrations in liver of the remaining species was 52–85 ng/g lipid. However, decaBDE was only detected in a few samples from the terrestrial birds, but not in the aquatic birds. The authors suggested that terrestrial bird species might be more exposed to decaBDE than aquatic species.

Christensen et al. (2005) reported decaBDE residues in muscle and fat from coastal (n=6) and interior (n=6) grizzly bears (*Ursus arctos horribilis*) in British Columbia, Canada. DecaBDE concentrations (i.e., of BDE209) ranged from approximately 1 to 2.77 μg/kg lipid in coastal bears and from approximately 0.5 to 41.71 μg/kg lipid in interior bears (concentrations read from graph), suggesting a higher exposure to decaBDE for bears feeding in interior environments where diets are predominantly terrestrial in nature. The authors also used a "bioaccumulation slope" method to characterize the observed bioaccumulation of decaBDE in coastal and interior bears. This analysis is evaluated further in Section 2.2.

Verreault et al. (2004, 2005) analyzed decaBDE in two Norwegian arctic top predators, glaucous gull (*Larus hyperboreus*) and polar bear (*Ursus maritimus*), inhabiting Svalbard and Bjoroya, Norway. In gull plasma from Svalbard (n=27), decaBDE ranged from < 0.05 to 0.33 ng/g ww while egg concentrations (n=20) ranged from below detection to 170 ng/g lipid. In Bjoroya, gull plasma (n=89) and egg (n=4) concentrations of 410 ng/g lipid and 23–53 ng/g lipid, respectively, were determined. In polar bear plasma from Svalbard (n=15), decaBDE was detected in only one sample, at a concentration of 0.1 ng/g ww. The authors claim that decaBDE was "bioaccumulative" to a limited degree. However, this claim was based solely on the occurrence of decaBDE in tissues without an evaluation of whether the concentrations are high relative to the environment or prey.

Vorkamp et al. (2005) reported concentrations of decaBDE in eggs of peregrine falcon (*Falco peregrinus*) from southwestern Greenland collected from 1986 to 2003. Although decaBDE was detected in all samples (n=37) at concentrations ranging from 3.8 to 250 ng/g lipid, high concentrations were only found in two eggs from 1995 and 2002 and the median concentration was 11 ng/g lipid. The authors noted a significantly increasing temporal trend in decaBDE concentration in the falcon tissues. They also concluded that their results provided evidence of some uptake and bioaccumulation of decaBDE (ruling out the idea of a size threshold for membrane permeation of decaBDE). The authors characterized decaBDE concentrations as low in falcon eggs.

Sørmo et al. (2006) reported analyses of decaBDE in biota from Svalbard, Norway. DecaBDE (BDE209) was not detected in two calanoid copepod species (method detection limit (MDL) = 0.012–1.3 ng/g lipid) but was detected at a concentration of 7.22 ng/g lipid in ice amphipod (*Gammarus wilkitzkii*). The decaBDE concentration in polar cod (*Boreogadus saida*; n=7) ranged from 0.05 to 0.42 ng/g lipid with a mean of 0.2 ng/g lipid. DecaBDE (i.e., BDE209) was detected in only 1 of 6 samples of ringed seal (*Pusa hispida*) blubber, at a concentration of 0.02 ng/g lipid (MDL = 0.014–0.75 ng/g lipid). Polar bear (*Ursus maritimus*) adipose tissue (n=4) contained 0.03–0.16 ng/g lipid of decaBDE with an average concentration of 0.09 ng/g lipid. DeWit et al. (2006) summarized additional data from Muir et al. (2006) and Skaare (2004) for polar bears in Svalbard. The BDE209 congener (decaBDE) was not detected in the Muir et al. (2006) study (MDL = 1 ng/g lipid) and was 1 ng/g lipid in the Skaare (2004) study.

Chen et al. (2007) reported measurements of decaBDE concentrations in birds of prey from northern China including samples of muscle, liver and kidney from common kestrel (*Falco tinnunculus*; n=6), sparrowhawk (*Accipiter nisus*; n=11), Japanese sparrowhawk (*Accipiter gularis*; n=6), little owl (*Athene noctua*; n=6), scops owl (*Otus sunia*; n=6), long-eared owl (*Asio otus*; n=6), upland buzzard (*Buteo hemilasius*; n=3) and common buzzard (*Buteo buteo*; n=3). The common kestrel had the highest mean concentrations of decaBDE in muscle (2150 ng/g lipid), liver (2870 ng/g lipid) and kidney (483 ng/g lipid), followed by scops owl (mean range of 59–537 ng/g lipid in muscle, liver and kidney), and sparrowhawk (mean range of 83–249 ng/g lipid in muscle, liver and kidney). The concentration of decaBDE in tissues from the other species did not exceed 150 ng/g lipid. The authors concluded that decaBDE was especially elevated compared to other published reports and that this may be related to significant production, usage or disposal

of decaBDE-containing products in China. They also suggested that exposure to decaBDE could be higher in terrestrial food chains than in aquatic food chains.

Lam et al. (2007) assessed PBDEs levels in eggs of birds of southern China. The researchers examined eggs (n=5, for each species) of little egret (*Egretta garzetta*), black-crowned night heron (*Nycticorax nycticorax*), Chinese pond heron (*Ardeola bacchus*) and cattle egret (*Bubulcus ibi*) from Hong Kong, Ziamen and Quanzhou. Levels of decaBDE ranged from < 0.5 (in black-crowned night heron from Quanzhou) to 99 ± 130 ng/g lipid (in Chinese pond heron from Xiamen). The higher abundance of BDE209 in birds from Xiamen appeared to correspond with high production of electronics in Xiamen. Exposure concentrations were not determined in this study.

Johnson-Restropo et al. (2005) reported the findings of a monitoring study of marine fish—teleosts, Atlantic stingray (*Dasyatis sabina*) and sharks (*Rhizoprionodon terraenovae* and *Carcharhinus leucas*)—and dolphins (*Tursiops truncatus*) of coastal Florida. BDE209 (decaBDE) was not detected (MDL=0.022 ng/g lipid) in muscle samples from silver perch (*Bairdiella chrysoura*), striped mullet (*Mugil cephalus*), spotted seatrout (*Cynoscion nebulosus*) and red drum (*Sciaenops ocellatus*), and also not detected (MDL=0.022 ng/g lipid) in blubber samples from bottlenose dolphin. The measured concentrations of decaBDE in hardhead catfish (*Arius felis*) and Atlantic stingray were reported to be 4.5 ng/g lipid and 0.1 ng/g lipid, respectively, while for sharks, the decaBDE concentration ranged from 16.9 ng/g lipid in spiny dogfish (*Squalus acanthias*) to 778 ng/g lipid in bull shark (*Carcharhinus leucas*). DecaBDE (i.e., BDE209) was the most abundant congener in sharks, suggesting either that there was preferential exposure to decaBDE or that preferential accumulation of decaBDE was occurring in shark species.

Gauthier et al. (2008) conducted monitoring of bird eggs and reported on temporal trends (1982–2006) for PBDEs, and most notably, for BDE209 in pooled samples of herring gull (*Larus argentatus*) eggs from seven colonies on lakes Superior, Michigan, Huron and Ontario, and on the Detroit and Niagara rivers. BDE209 concentrations in 2006 egg pools ranged from 4.5 to 20 ng/g ww and composed 0.6–4.5% of the total PBDE concentrations measured. The authors noted mean doubling times for BDE209 concentrations in eggs of 2.1–3.0 years. For octa- and nonaBDEs, the mean doubling time was determined to be 3–11 years and 2.4–5.3 years, respectively. Based on this rapid increase in concentrations over time, they indicate that the potential for decaBDE to bioaccumulate appears to have been previously underestimated, but they did not quantify exposure concentrations. The researchers also suggested that congener patterns in egg samples suggested meta-position metabolic debromination of BDE209 and -207 to BDE197.

Shaw et al. (2007) reported decaBDE concentrations in blubber of harbour seals (*Phoca vitulina concolor*) of the northwest Atlantic coast from 1991 to 2005. The BDE209 congener (decaBDE) was detected in 2 of 4 samples at concentrations up to 7.4 ng/g lipid.

DeWit et al. (2006) summarized decaBDE levels in Arctic biota from many additional studies:

- concentrations of 0.025 to 0.12 ng/g ww in moss from Norway (Schlabach et al. 2002);
- 0.5, 0.1 and 0.8 ng/g lipid in grouse (*Lagopus sp.*), lynx (*Lynx lynx*) and moose (*Alces alces*) liver, respectively, from Norway (Mariussen et al. 2004);
- detectable (but not quantified) amounts in eggs of birds of prey from northern Norway (Herzke et al. 2005); and
- 3.6–33 ng/g lipid in the muscle tissue of blue mussels (*Mytilus edullis*) and 0.98–0.99 ng/g lipid in the liver of Atlantic cod (*Gadus morhua*) from Norway (Fjeld et al. 2004).

Verreault et al. (2007) monitored decaBDE in egg yolk and plasma of male and female glaucous gulls (*Larus hyperboreus*) from the Norwegian Arctic. DecaBDE (i.e., BDE209) was "virtually non-detectable" in egg yolk and plasma, but the authors speculated that the presence of detectable concentrations of octa- and nonaBDEs in the samples suggested accumulation and subsequent *in vivo* debromination of decaBDE. Although the detection limit for decaBDE was not reported, detection limits for hepta- to nonaBDEs were reported to be 0.16 ng/g ww and are presumably the same for decaBDE.

Ismail et al. (2009) evaluated concentrations of BDE209 along with other flame retardants in archived Lake Ontario, Canada, lake trout (*Salvelinus namaycush*) samples collected between 1979 and 2004. Concentrations of BDE209 were consistently detected in lake trout samples throughout the study period and these concentrations ranged from 2.3 to 12 ng/g lipid (0.27 to 1.3 ng/g ww). In contrast to the other PBDE congeners, a large increase (approximately fourfold) in lake trout BDE209 occurred between 1998 and 2004. The study also found a significant increase in BDE209 concentrations between 1979 and 2004, with an overall doubling time of 19 years ($p < 0.05$).

Guo et al. (2008) conducted an analysis of PBDE concentrations and compositional profiles in tissues of freshwater and marine wild fish, and farmed fish in the Pearl River Delta (PRD) of China. BDE209 was detected in 70 of the total 187 samples, with a range of 0.39 to 59.9 ng/g dry weight (dw), in skin, gills, gastrointestinal tract, liver and muscle samples. However, the authors suggested that this detection frequency was likely underestimated due to the much higher analytical detection limit for BDE209 (10 ng/g) than that for other lower brominated PBDE congeners (0.2 ng/g). The highest and lowest BDE209 ratios (to sum of total analyzed PBDEs) were found in skin and liver, respectively (mean/maximum of 48.0/99.2% in skin and 8.2/83.3% in liver). The highest and lowest BDE209 concentrations occurred in skin and liver, with the median levels of 95.5 and 2.54 ng/g lipid (based on pooled tissue samples of skin, gills, gastrointestinal tract, liver and muscles for all fish), respectively. The authors proposed that this may be attributed to the fact that the liver is a primary tissue for biotransformation of organic compounds and that BDE209 has a low half-life time in fish. Guo et al. (2008) suggested that BDE209 could be accumulated in gills and gastrointestinal tract since the lipid-normalized BDE209 concentrations in gills and gastrointestinal tract from their

study were actually slightly higher than those in liver and muscle. The study concluded that BDE209 is accumulative in fish tissues under natural environments, and speculated that BDE209 may bioaccumulate significantly, particularly in biota from areas heavily polluted with decaBDE, such as the Pearl River Delta.

Potter et al. (2009) measured PBDE levels in 23 peregrine falcon (*Falco peregrinus*) eggs, obtained between 1993 and 2002 from 13 nests covering 11 locations in the Chesapeake Bay region of the United States. The mean BDE209 contribution to total PBDEs measured in egg samples was 5.9% and ranged from 0% for eggs in or near undeveloped land to 18.6% for eggs from a densely populated area. The highest BDE209 burden found in study eggs was 48.2 ng/g ww from an urban highway bridge site. The order of PBDE congener dominance (from most to least dominant) in the peregrine falcon eggs analyzed typically was BDE 153 > BDE 99 > BDE 100 > BDE 154 > BDE 209 > BDE 183 > BDE 197 > BDE 47 > BDE 207. The authors suggested that the source of the higher brominated congener signature in peregrine falcon eggs remains unresolved, and that the answer may relate to the bird's biotransformation capabilities.

BDE209 was detected in all 114 eggs in a peregrine falcon (*Falco peregrinus*) egg study in the northeastern United States (Chen et al. 2008). Eggs were collected from 1996 to 2006 (excluding 1997 and 1998). Concentrations ranged from 1.4 to 420 ng/g ww for BDE209. The authors reported that the median concentration of 26 ng/g ww (or 480 ng/g lipid) was much higher than observed in concentrations of European birds (e.g., Greenland peregrine eggs, Belgian buzzards and sparrowhawks), but comparable to those in Chinese kestrels. BDE209 concentrations and their contribution to the total PBDE concentrations were significantly higher in urban than in rural eggs ($p < 0.005$). In contrast to the other PBDE congeners, BDE209 concentrations were shown to increase significantly ($r = 0.348$, $p < 0.005$), with a doubling time of 5 years. In addition to BDE209, eight nona- and octaBDE congeners were frequently detected in this study. Together with BDE209, they constituted 16–57% of the total PBDE concentration determined in urban eggs and 4.9–53% in rural eggs.

Kunisue et al. (2008) analyzed the spatial trend of PBDE levels and congener patterns in avian species by using stored historical tissue samples taken from open sea, Japanese coastal and inland birds. PBDEs were detected in all the avian pectoral muscle samples analyzed in this study; however, BDE209 was not detected in two of the three open-water bird samples, one of two coastal bird samples, and one of four inland bird samples. Japanese coastal and inland birds accumulated higher PBDE levels than open sea birds. In addition, accumulation of higher brominated congeners such as BDE209 was predominant in the omnivorous species that lived closest to areas inhabited by humans. For example, the mean concentration of BDE209 in jungle crow (*Corvus macrorhynchos*) samples from 1998 was 440 ng/g lipid. In the jungle crow, BDE153, BDE183 and BDE209 were the predominant congeners, and relatively higher proportions of octa- and nona-BDE congeners were also found compared with other birds.

Additional studies are also described in Section 2.2. These studies contained sufficient information for conducting a ratio-based evaluation of bioaccumulation or biomagnification.

2.1.3 Synthesis of Biota Concentration Data

The detected concentrations of decaBDE in a wide range of biota provide confirmation that this substance can accumulate, at least to some degree, in organisms. However, these data provide little evidence that decaBDE may be bioaccumulative. The main shortcoming is that the data fail to compare the measured concentrations with either those determined in the environment in which the biota reside (e.g., as a BAF or BCF) or the prey that the biota consumes (e.g., as a BMF or TMF).

In the absence of a ratio-based assessment of whether decaBDE is bioaccumulative or biomagnifying, it is still possible to subjectively judge whether the observed concentrations in top predators appear high, indicating potential trophic magnification.

Concentrations of decaBDE have been observed to be relatively high (e.g., exceeding 100 ng/g lipid) in some top predator species, including

- birds of prey, especially kestrel, sparrowhawk and owl from China; peregrine falcon, sparrowhawk and kestrel in the UK and Sweden; peregrine falcon in Greenland; and buzzard in Belgium;
- red fox in Belgium;
- sharks of coastal Florida;
- marine mammals such as harbour porpoise and white-beaked dolphin; and
- some marine bird species, including heron from Sweden and glaucous gulls from Byoroya and Svalbard, Norway.

Of these, the Greenland peregrine falcon results and the coastal Florida shark results may provide the strongest evidence that trophic magnification might be occurring, given the potential remoteness of the sites of sampling. However, Vorkamp et al. (2005) also note that the peregrine falcon (*Falco perigrinus*) subspecies of Greenland from which samples were taken migrates to Central and South America in the winter, following a route along the Atlantic coast of North America. As a result, there is a potential that the organisms were exposed to locationally specific contaminated environments that may explain their relatively high loading of decaBDE. Although high decaBDE concentrations are observed in Florida sharks, similarly high concentrations are not observed in teleost fish species from the same ecosystem, which would likely make up the prey species for shark. While trophic magnification is a potential explanation for this difference, it is also possible that the sharks are consuming contaminated refuse.

For the remaining results, several factors tend to confound the findings with respect to whether decaBDE is bioaccumulative. These include

- the potential that the sampled species inhabit decaBDE hotspots close to industrialized areas; and

- the large number of non-detects within the same studies, and species in which relatively high concentrations were observed.

While there are some high BDE209 concentrations in some biota, overall the data from surveillance studies directly contradict a generalized conclusion that the concentration of decaBDE is relatively high in top predators. The surveillance data show

- relatively low and often non-detected concentrations of decaBDE in marine fish species, even though some of these species such as salmon or cod could be considered top predators within their respective food webs (see Appendix A for summary of these data);
- relatively low and often non-detected concentrations of decaBDE in mammalian predators such as grizzly bear, polar bear and lynx which feed near the top of their respective food webs;
- relatively low and often non-detected concentrations of decaBDE in marine mammals which feed near the top of their respective food webs;
- relatively low and often non-detected concentrations of decaBDE in marine/aquatic birds which feed at the top of their respective food webs; and
- relatively low and often non-detected concentrations of decaBDE in birds of prey, often in the same studies as those where relatively high concentrations were also observed.

Generally speaking, it appears that the higher concentrations of decaBDE in biota are anomalies when the broader dataset of decaBDE concentrations is considered. Thus, the evidence based on concentrations in top predators fails to provide a strong indication that decaBDE is bioaccumulative or biomagnifying in food webs.

2.2 Bioconcentration, Bioaccumulation and Biomagnification Data

This section first summarizes the data respecting decaBDE bioaccumulation considered in the PBDE screening assessment report (Environment Canada 2006a; Environment Canada 2006b), then examines new data available after 2004 and its significance.

2.2.1 Information Evaluated in the Screening Assessment

- The Japanese Ministry of International Trade and Industry (MITI 1992) determined that the BCF for carp ranged from < 5 to < 50 in their 6-week bioconcentration study (recalculated to < 3000 by European Communities (2002)). Given the exceptionally low water solubility limit of decaBDE it is not expected that this substance will be appreciably taken up from the water phase by aquatic organisms.
- Stapleton et al. (2004) exposed juvenile common carp (*Cyprinus carpio*) to decaBDE (> 98% purity as reported by Cambridge Isotope Laboratories) amended in food on a daily basis for 60 days (d) (940 ng/d/fish) followed by a 40-d period in which fish were fed clean food. DecaBDE (i.e., BDE209) was not detected in whole fish tissues during the exposure or depuration periods (detection limit of approximately 1 µg/kg ww);

however, several peaks were observed in the chromatograms of the exposed fish that were not observed in the control fish, suggesting transformation of decaBDE. Their results suggested that at least 0.44% of decaBDE was bioavailable in the form of its metabolites. This value could be higher if other metabolites were present which were not determined in these studies.

- Kierkegaard et al. (1999) dosed juvenile rainbow trout (*Oncorhynchus mykiss*) with food contaminated with Dow FR-300 (composition not determined in this study, but reported as 77.4% decaBDE, 21.8% nonaBDE and 0.8% octaBDE by Norris et al. 1973, 1974). A small proportion of the test material was taken up during the 120-d exposure phase of the experiment, amounting to about 0.02–0.13% based on the muscle concentrations of the total hexa- to decaBDEs present, or approximately 0.005% based only on the decaBDE concentrations in muscle and the mean dietary dose of Dow FR-300. The authors did not report the decaBDE concentration in food, meaning that a dietary BAF cannot be estimated from the study data.
- Metabolism studies using rats (Norris et al. 1973, 1974; El Dareer et al. 1987) suggested that decaBDE has a very low bioaccumulation potential in mammalian species. For instance, Norris et al. (1973, 1974) dosed male and female rats with 1.0 mg of ^{14}C-labelled DecaBDE as a suspension in corn oil and found that around 90.6% of the administered ^{14}C-labelled DecaBDE was excreted in the feces within 24 hours (h), and by 48 h, all of the administered chemical had been excreted. Tissue accumulation studies in which rats were fed diets of decaBDE at a rate of 0.1 mg/kg body weight per day showed that bromine contents in various tissues were not significantly greater than those of the controls. The bromine content of the adipose tissue of decaBDE-dosed rats was found to be significantly increased at the $p < 0.03$ level but not at the $p < 0.01$ level when compared with the controls (Norris et al. 1973, 1974).

The bioaccumulation data from these studies are also summarized in Appendix B. Additional studies were also reviewed in the screening assessment but their findings did not allow estimates of bioaccumulation parameters. Rather, they are more relevant to the evaluation of metabolite formation, and are discussed in Section 3.

2.2.2 New Bioaccumulation Data

The bioaccumulation data from studies published after 2004 are discussed in this section and summarized in Appendix B.

Studies on Aquatic Species

Stapleton et al. (2006) exposed 45 juvenile rainbow trout (*Oncorhynchus mykiss*) to spiked food containing decaBDE (purity reported as 98.7%, no further characterization of test material for impurities was undertaken) for a period of 5 months. The 45 fish were randomly distributed to three experimental tanks while an additional 15 fish that were fed non-spiked food were kept in a separate tank. The concentration of decaBDE in the spiked food was 940 ng/g and the fish (average weight 91.2 g) were fed at a rate of 1% of their body weight per day. One fish from each tank was sacrificed for analysis at 9 time points throughout the 5-month exposure period. Blood was sampled from individual fish at the initiation of exposure and at three time points during the final 3 months of the

study. Samples of blood serum, intestine, liver and carcass at each sampling time were each analyzed for decaBDE and lower brominated BDE congeners.

The net uptake of decaBDE during the experiment was estimated at 3.2% based on the total burden of hepta- through decaBDE congeners present in the carcass, or 3.7% if the liver was included in the calculation. The decaBDE concentration on the final day of exposure was highest in the liver (342 ng/g ww) followed by the intestine (~60 ng/g ww, read from graph), serum (26–40 ng/g ww) and carcass (5.3 ng/g ww). The detection limit in this study was 1 ng/g ww. Several hepta-, octa- and nonaBDE congeners were also accumulated, potentially, as a result of decaBDE debromination (fish were dosed only with decaBDE, and background decaBDE concentrations in the control fish ranged from < 0.5 ng/g to 0.5 ng/g). These debromination results are discussed further in Section 3.1.

Using concentration data from day 112, the reported lipid content of the test fish, and the known decaBDE concentration/lipid content in food reported by Stapleton et al. (2006), it is possible to estimate BMFs for decaBDE on its own and for the combined burden of decaBDE plus debrominated congeners. Table 2-1 summarizes the calculations.

Table 2-1: BMFs estimated from the decaBDE rainbow trout feeding study by Stapleton et al. (2006; and personal communication from HM Stapleton to Environment Canada, January 2008; unreferenced)

	Food[1]	Carcass[1]	Serum[1]	Liver[1]
DecaBDE concentration (ng/g lipid)	9307	204	3300[2]	11 958
Fraction of decaBDE in total BDE burden	n/a	0.25	0.68	0.92
Total BDE concentration (ng/g lipid)[3]	n/a	831.3	4853	16 163
DecaBDE BMF	n/a	0.02	0.35	1.28
Total BDE BMF	n/a	0.09	0.52	1.74

[1]Lipid contents: food – 10.1%; carcass – 4.5%; serum – 1% (assumed); liver – 2.3%.
[2]Median of reported values (26 and 40 ng/g ww) divided by estimated lipid content.
[3]Inferred by dividing decaBDE by the fraction of decaBDE in total BDE burden.

It is expected that the higher lipid content in food (10.1% lipid) compared with carcass and liver (4.5% and 2.3%, respectively) may cause the ww BMFs to underestimate the bioaccumulation potential of decaBDE, and as a result, lipid-weight BMFs were calculated. For decaBDE on its own, the BMFs ranged from 0.02 (carcass) to 1.28 (liver) whereas for the total BDE burden, the BMFs ranged from 0.09 to 1.74.

Based on the findings of other studies (refer to Section 3), it is possible that decaBDE could be transformed to other transformation products not analyzed in this study. The Stapleton et al. (2006) study did not include these potential products as analytes. If they are formed in rainbow trout, then the reported net uptake of neutral BDEs only would underestimate the actual total uptake of decaBDE. If metabolites other than hepta-, octa-, and nonaBDEs were being formed and persisting in the fish, then the BMFs calculated above would underestimate the total accumulation potential of decaBDE-related compounds.

Tomy et al. (2004) studied the uptake by juvenile lake trout (*Salvelinus namaycush*) of twelve tetra- to heptaBDEs (Wellington Laboratories, all purities > 96%) plus DecaBDE (technical grade, Great Lakes Chemical Corp., purity not provided) from spiked commercial fish food. Test fish were exposed to spiked food for 56 d followed by a 112-d elimination period. Seventy fish each were exposed to low and high concentrations (measured in food) of technical-grade DecaBDE, and a non-exposed control group was also monitored (concentrations measured in food). Significant uptake of decaBDE was observed for both the low- and high-exposure treatments. For the low-exposure treatment, depuration of chemical during the elimination phase was non-detectable (slope not significant) and the absorption efficiency, half-life and BMF were not estimated. For the high-exposure treatment, the absorption efficiency was estimated at 5.2% (Tomy et al. 2004; and personal communication from G Tomy to Environment Canada, July 2009, unreferenced) with a half-life of 26 ± 5 d and a BMF[1] of 0.3. Although lower brominated PBDE congeners appeared to be bioformed in the fish, it was not possible to include the debrominated congeners in the BMF estimates because similar congeners were also present in the spiked food or potentially present as impurities in the technical-grade DecaBDE used in the study. In addition, the study contained other uncertainties such as the use of fiberglass aquaria which may have resulted in some adsorption of test material.

Ciparis and Hale (2005) examined the bioavailability and accumulation of multiple PBDEs, including decaBDE, from sediments and biosolids to the aquatic oligochaete, *Lumbriculus variegatus*. Oligochaetes were exposed to either composted biosolids containing 1600 ng/g total PBDEs or artificial sediments spiked with technical Penta- and DecaBDE formulations (1300 ng/g total PBDEs). The experimental protocol included a 28-d uptake phase followed by a 21-d elimination phase. Following solvent extraction, clean-up in a size exclusion column, and further purification using solid-phase extraction columns, decaBDE was quantified from substrates and tissues on a gas chromatography (GC) device equipped with a halogen-selective electrolytic conductivity detector with MDLs of 190 ng/g and 20 ng/g for tissues and substrates, respectively. Although significant accumulation of lower brominated PBDEs (especially BDE47 and BDE99) was observed with both biosolids and spiked sediments, uptake of decaBDE was minimal and it was not possible to estimate steady-state sediment BSAFs or kinetic parameters for decaBDE accumulation. The authors speculated that the bioavailability of decaBDE was limited by its high log K_{ow} (suggesting that desorption from sediment particles is minimal) and large molecular size, which may impede its transport across cell membranes.

Burreau et al. (2004, 2006) reported the results for three separate food web monitoring programs for PCBs and PBDEs. Burreau et al. (2004) sampled perch (*Perca fluviatilis*; n=120, 33 individuals), roach (*Rutilus rutilus*; n=23, 8 individuals) and pike (*Esox lucius*; n=51, 25 individuals) in the Lumparn estuary in the Åland archipelago in the Baltic Sea

[1] BMF = $\alpha F/k_d$ where α is the absorption efficiency, F is the feeding rate on a lipid basis and k_d is the total elimination rate constant

and analyzed muscle tissue composites from each species.[2] Burreau et al. (2006) described monitoring studies for the Baltic Sea and the Atlantic Ocean (south of Iceland). The Baltic Sea study was conducted in 1998 and sampled zooplankton (Calanoid crustacea; n=3 net tows), sprat (*Sprattus sprattus*; n=6), herring (*Clupea harengus*; n=5) and Atlantic salmon (*Salmo salar*; n=10), while the Atlantic Ocean study was conducted in 1999 and sampled zooplankton (*Calanoid sp*.; n=10), small herring (*Clupea harengus*; n=6), large herring (*Clupea harengus*; n=10) and Atlantic salmon (n=10). The detection limits ranged from 140 to 148 pg/g. Analytical detection limits ranged from 14 to 14.8 pg/g ww. Determinations of decaBDE were blank-corrected. Table 2-2 provides a summary of median observed concentrations in the Baltic Sea and North Atlantic Ocean biota samples.

Table 2-2: Summary of observed concentrations of decaBDE in freshwater and marine biota from the Lumparn Estuary, Baltic Sea and North Atlantic Ocean (Burreau et al. 2004, 2006)

Location	Species	Median concentration (ng/g lipid)	Number of samples with decaBDE detected / Number of samples analyzed
Lumparn Estuary	Roach *Rutilus rutilus*	48	3/8
Lumparn Estuary	Perch *Perca fluviatilis*	1.3	12/33
Lumparn Estuary	Pike *Esox lucius*	1.7	4/25
Baltic Sea	Zooplankton *Calanoid sp*.	2.1	3/3
Baltic Sea	Sprat *Sprattus sprattus*	0.082	3/6
Baltic Sea	Herring *Clupea harengus*	0.24	4/5
Baltic Sea	Atlantic salmon *Salmo salar*	0.41	3/10
North Atlantic Ocean	small herring *Clupea harengus*	0.31	6/6
North Atlantic Ocean	large herring *Clupea harengus*	0.039	3/6
North Atlantic Ocean	Atlantic salmon *Salmo salar*	not detected	0/10

To examine the potential for food web biomagnification of decaBDE in the biota data for each Baltic Sea food web (roach-perch-pike; zooplankton-sprat-herring-salmon), the authors conducted an analysis of trophic magnification. This involved a regression of lipid-normalized concentration vs. $\delta^{15}N$ according to the following model:

[2] The disparity between the n of samples and the n of analysis is explained by the fact that only the samples in which the congener was detected are reported.

$$C = A \bullet e^{(B \bullet \delta 15N)}$$

Where C is the biota concentration (lipid-normalized), A is a constant representing $\delta^{15}N$ at the base of the food chain and B represents the "biomagnification power" of the substance. A positive B-value indicates biomagnification while a negative B-value indicates trophic dilution of chemical concentrations. B is similar to a TMF except that a TMF is expressed on an arithmetic, rather than logarithmic basis, and the TMF is based on a regression with trophic level (estimated from $\delta^{15}N$) rather than the $\delta^{15}N$ content itself. The B-values for both food webs were not significantly different from zero, indicating that biomagnification of decaBDE did not appear to be occurring in these food webs. Failure to detect decaBDE in salmon from the Atlantic Ocean precluded a similar analysis for this food web.

Using the reported concentration data, it is also possible to estimate lipid-normalized BMFs for specific predator-prey combinations; these are summarized in Table 2-3. BMFs range from 0.03 to 5, depending on the predator-prey combination, suggesting that biomagnification was taking place in some predator-prey combinations. However, it is important to consider that the exact feeding relationships for these food webs are unknown, resulting in considerable uncertainty in these BMF estimates.

Table 2-3: Estimated BMFs for decaBDE in sampled biota from Lumparn Estuary, Baltic Sea and Atlantic Ocean pelagic food webs reported by Burreau et al. (2004, 2006)

Location	Predator/Prey	BMF (lipid-normalized)
Lumparn Estuary	perch/roach *Perca fluviatilis / Rutilus rutilus*	0.03
	pike/roach *Esox lucius /Rutilus rutilus*	0.04
	pike/perch *Esox lucius / Perca fluviatilis*	1.31
Baltic Sea	sprat/zooplankton *Sprattus sprattus* / Calanoid crustacea	0.04
	herring/sprat *Clupea harengus / Sprattus sprattus*	2.93
	herring/zooplankton *Clupea harengus / Calanoid sp.*	0.11
	salmon/sprat *Salmo salar / Sprattus sprattus*	5.00
	salmon/herring *Salmo salar /Clupea harengus*	1.71
Atlantic Ocean	large herring/small herring *Clupea harengus / Clupea harengus*	0.13

In evaluating the Burreau et al. (2004, 2006) studies, the United Kingdom (2007a) cautions that the relatively high levels of decaBDE in procedural blanks and low concentrations of decaBDE in biota samples create uncertainty in the overall

biomagnification analysis. Currently, this appears to be a common issue with field studies of decaBDE in biota.

Shaw et al. (2009) studied the bioaccumulation of PBDEs in northwest Atlantic marine food webs. To evaluate the transfer of PBDEs from prey to predator, the study compared PBDEs measured previously in harbour seal blubber with whole fish samples of seven species of fishes comprising the major prey of harbour seals (*Phoca vitulina concolor*). Eighty-seven individual fish (> 35 cm) were collected off the coast of Maine during the May–June 2006 Gulf of Maine Trawl Survey of commercial groundfish stocks. Species included silver hake (*Merluccius bilinearis*, n=10), white hake (*Urophycis tenuis*, n=17), Atlantic herring (*Clupea harengus*, n=20), American plaice (*Hippoglossides platessoides*, n=10), alewife (*Alosa pseudoharengus*, n=10), and winter flounder (*Pseudopleuronectes americanus*, n=10). Atlantic mackerel (*Scomber scombrus*, n=10) were caught by hook and line from the same area during June 2006. Whole fish were transported to the laboratory on ice where standard length and weight were recorded, then frozen and stored at −40°C prior to shipment to the analytical laboratory. Fish whole-body samples were pooled and homogenized into 17 composites prior to analysis. For extraction, the fish sample (~1.5 g of lipids, between 5 and 100 g tissue) was homogenized and mixed with sodium sulphate. After addition of the internal PBDE standards, a mixture of cyclohexane and dichloromethane was applied to the column for extraction of PBDEs along with other lipophilic compounds and fat. The extract was washed, dried, and after solvent evaporation, gravimetric lipid determination was performed. The final extract was evaporated by a stream of nitrogen to a final volume of 50 µL containing C^{13} labelled BDE139 as recovery standard. The measurements were performed by high-resolution gas chromatography / high resolution mass spectrometry (HRGC/HRMS).

Total PBDE concentrations in fish ranged from 18.3 to 81.5 ng/g, lipid (overall mean 62 ± 34 ng/g, lipid), compared with total PBDE concentrations in harbour seal samples of 80 to 25 720 ng/g lipid (overall mean 2403 ± 5406 ng/g, lipid—analyzed for earlier study). BDE209 was detected in 35% of the fish samples and in 25% of the harbour seal blubber samples. BDE209 concentrations ranged from non-detect (0.2 ng/g lipid) to 4 ng/g lipid in fish, and from 1.1 to 7.6 ng/g lipid in seal blubber (mean value=1.2 ng/g, lipid). The similarity between fish and seal BDE209 concentrations was in contrast to the total PBDE concentrations, which were two orders of magnitude higher in the harbour seals than in fish. For BDE209, BMFs from fish to seals ranged from 0.67 (American plaice) to 0.75 (Atlantic mackerel) to 1.3 (white hake), which the authors suggested represented low biomagnifications potential. This contrasted with BMFs for the other PBDEs, which averaged from 17 to 76.5, indicating high biomagnification in this marine food web. The authors suggested that the presence of higher brominated congeners, including BDE209, at measurable levels in fish and seal tissue, along with high biomagnification of BDE153, -155, and -154, suggests recent exposure to the octa- and decaBDE formulations in this U.S. coastal marine food web.

Law et al. (2006) conducted a field study of the trophic magnification of decaBDE in a pelagic food web of Lake Winnipeg. Samples of fish, plankton, mussels, sediment and water were collected from the south basin of the lake near Gimli, Manitoba. Muscle

tissue from multiple fish species were collected between 2000 and 2002, including walleye (*Stizostedion vitreum*; n=5), whitefish (*Coregonus clupeaformis*; n=5), emerald shiner (*Notropis atherinoides*; n=5), burbot (*Lota lota*; n=5), white sucker *Catostomus commersoni*; n=5) and goldeye (*Hiodon alosoides*; n=3). Samples of net plankton (n=5; zooplankton and phytoplankton combined) were collected using horizontal tows with 160-µm nets (precise date not indicated in article). Mussels (*Lampsilis radiata*; n=5, muscle tissue retained for analysis) were collected by divers in 2002. Sediment grab samples were collected at 4 locations with only the surficial 2 cm of sediment retained. Water was sampled in 2004 using a Teflon column packed with XAD-2 absorbent. Each XAD-2 column was used to sample six 54-L samples from 324 L of water collected. Samples were pulled through an inline glass-fibre filter (1-µm pore size) and then onto a XAD-2 column.

All samples were analyzed for decaBDE (and several other chemicals) using GC/MS, with additional analyses of organic carbon (OC) for sediments and lipid and $\delta^{15}N$ for biota. The $\delta^{15}N$ measurements were used to estimate trophic position. The detection limit of the analytical method used was 0.1 µg/kg for biota and sediment samples, and 15 pg/L for water. The decaBDE concentration, lipid and OC contents, and estimated trophic level of biota are summarized in Table 2-4.

Using the trophic levels estimated from $\delta^{15}N$ data, the rank order of the trophic levels in the pelagic food web was estimated to be mussel $\Rightarrow$ zooplankton, whitefish $\Rightarrow$ goldeye, white sucker $\Rightarrow$ burbot, walleye (top predators). A regression of the lipid-normalized concentration of decaBDE vs. trophic level was used to estimate a TMF of 3.6[3] (r^2=0.46 p=0.0001) for decaBDE in the pelagic food web. Predator-prey BMFs (on a lipid-normalized basis) were also calculated using the biota dataset. The estimated BMFs for decaBDE ranged from 0.1 to 34, depending on the predator-prey combination.

While estimated TMFs and BMFs are intended to provide a real-world indication of trophic magnification and biomagnification of decaBDE in an aquatic food web, it is important to consider some of the uncertainties associated with this study.

Many of the concentrations of decaBDE in the Law et al. (2007) study were near the detection limit, increasing uncertainty in these determinations and raising the possibility of false positives. In addition, biomagnification was identified using lipid-normalized data; however, certain tissues were characterized by very low lipid concentrations (e.g., walleye, burbot and mussel muscle). Such low lipid contents result in extremely uncertain concentrations expressed on a lipid weight basis. When biomagnification is evaluated in this study on the basis of ww concentrations, biomagnification is not shown to occur.

There is further uncertainty respecting the appropriateness of lipid-normalization for decaBDE. This substance has been suggested to bind protein in some situations, although based on chemical structure, protein binding is not expected. It is possible that decaBDE is subject to non-specific binding in blood plasma (i.e., lipids) (e.g., Han et al. 2007). As

[3] Note that this is the corrected value published in Law et al. (2007).

a result of this binding, preferential accumulation in liver could occur, but this has not been established definitively.

Table 2-4: Analytical results from Law et al. (2006) for decaBDE, lipid content, organic carbon content and $\delta^{15}N$ in water, sediments and biota from Lake Winnipeg

Sample	$\delta^{15}N$	Trophic position	Mean lipid[1] or organic carbon[2] content	Average decaBDE concentration (ng/g lipid[1]; ng/g dry weight (dw)[2] or pg/L[3])
Water (dissolved phase)	n/a	n/a	n/a	<15 pg/L
Sediment	n/a	n/a	2%	0.63
Walleye (*Stizostedion vitreum*) muscle	17.8	2.4	1.15%	24.7
Whitefish (*Coregonus clupeaformis*) muscle	12.0	0.8	8.78%	3.6
Mussel (*Lampsilis radiata*) muscle	9.5	-	0.32%	50.8
Zooplankton (*Calanoid sp.*)	9.7	1.00	13.67%	1.2
Emerald shiner (*Notropis atherinoides*) muscle	16.0	1.9	3.18%	40.3
Goldeye (*Hiodon alosoides*) muscle	16.1	1.95	2.34%	41.6
White sucker (*Catostomus commersoni*) muscle	15.2	1.7	2.27%	12.0
Burbot (*Lota lota*) muscle	16.6	2.2	0.33%	98.7

n/a – not applicable
[1]For biota samples
[2]For sediment samples
[3]For water samples

The United Kingdom (2007a) also highlighted the following issues regarding the Law et al. (2006) study:

- There is some uncertainty as to how well the $\delta^{15}N$ analysis characterizes trophic levels. The resulting trophic structure does not necessarily match that which would be expected based on species size and feeding characteristics (e.g., emerald shiner has a higher estimated trophic level than whitefish but based on size/life history, it would be expected to feed lower in the food web).
- There is uncertainty as to how well the samples represent the food web, since sample sizes were small in some cases, and they were collected at varying times between 2000 and 2002.
- Information regarding the actual feeding relationships of the analyzed species is lacking. It is unclear whether the species examined feed primarily on one another or whether other species that were not sampled could also be an important component of the food web.

In a study of eastern Canadian Arctic marine food webs, Tomy et al. (2008) examined the extent of trophic transfer of seven PBDE congeners, including BDE209. PBDEs were analyzed in the blubber of the beluga whale (*Delphinapterus leucas*, n=5), narwhal (*Monodon monoceros*, n=5) and walrus (*Odobenus rosmarus*, n=5). Whole-organism homogenates of Arctic cod (*Boreogadus saida*, n=8), shrimp (*Pandalus borealis* and *Hymenodora glacialis*, n=5), clams (*Mya truncate* and *Serripes groenlandica*, n=5), deepwater redfish (*Sebastes mentella*, n=5) and mixed zooplankton (n=5) were analyzed. Samples were collected in various parts of the Canadian eastern Arctic between 1996 and 2002, and archived. Marine mammal blubber was extracted using a ball mill shaker with anhydrous sodium sulphate and hexane:dichloromethane (DCM). Each cell was spiked with recovery internal standards, then shaken and left to stand 2–4 h before centrifuging, then decanted. The extraction was repeated twice, combining the decanted extracts. Fish, shrimp and clam tissues were homogenized with dry ice in a laboratory blender, then stored overnight in a –20°C freezer to allow for sublimation of the CO_2. Thawed tissue was weighed and mixed with pelleted diatomaceous earth (Hydromatrix) then added to a cell along with the recovery internal standards RIS and extracted using an accelerated solvent extractor (ASE 300). Zooplankton samples were weighed frozen and homogenized by directly mixing with Hydromatrix prior to accelerated solvent extraction. Void space was filled with sand. After extraction, anhydrous sodium sulphate was added to the collection bottles to remove water. Extracts were reduced in volume and filtered. Lipid content was determined gravimetrically in an aliquot of extract, while lipid was removed from the remainder of the extract by gel permeation chromatography. After volume reduction, samples were further cleaned using Florisil according to Law et al. (2006). The BDE fraction was reduced in volume to 200 µL and instrument performance internal standards (10 µL of 2 ng/µL aldrin) were added. PBDEs in tissue samples were analyzed by gas chromatography electron capture negative ion mass spectrometry (GC-ECNIMS). PBDEs were detected in selected ion monitoring (SIM) mode using the [Br]- ions (*m*/*z* 79, 81) and an external standard solution containing the BDE-mix (36 BDE congeners) and BDE-209 for quantification. Procedural and instrument blanks were used, and all PBDE samples were blank-corrected. The relative trophic level of the organisms

was determined by stable isotopes ($\delta^{15}N$), analyzed at the University of Georgia. BMF was determined as the trophic level adjusted ratio of the concentration in the predator tissue to that of the concentration in the prey tissue.

The total concentration of the seven congeners analyzed ranged from 0.4 ng/g lipid in walrus to 72.9 ng/g lipid in zooplankton. BDE209 mean concentrations ranged from non-detect (walrus, beluga and narwhal) to 18.7 lipid ng/g (zooplankton). BDE209 was found to contribute significantly to the body burden of total PBDEs in the lower trophic level organisms: 60% in redfish and 75% in arctic cod, for example. Conversely, in the upper trophic level organisms like beluga and narwhal, BDE209 accounted for less than 2% of the total BDE burden, whereas BDE47 accounts for over 40% in these samples. The authors suggested that elevated BDE209 levels in lower trophic level organisms may reflect greater exposure to this compound through zooplankton, and limited metabolic capabilities with respect to BDE209. BDE209 lipid-adjusted BMFs for individual predator–prey relationships were as follows: beluga (blubber):cod (whole body) < 1; beluga (blubber):redfish (whole body) < 1; and cod (whole body):zooplankton (whole body) < 1. BMFs were not calculated for narwhal:redfish and narwhal:cod. A statistically significant TMF of 0.3 was estimated for BDE209 ($p=0.002$; correlation coefficient $R^2=0.25$). The authors concluded that BDE209 concentrations decreased with trophic level, suggesting metabolic depletion of this congener or reduced assimilation up the food web. The authors suggested that these results are consistent with other studies that have shown that BDE209 is not an abundant congener in higher trophic level organisms. The authors speculated that this could be due to an enhanced BDE209 metabolic capability among higher trophic level organisms.

In a follow-up to their eastern Canadian Arctic food web study, Tomy et al. (2009) conducted a study of western Canadian Arctic marine food webs to examine the trophodynamics of PBDEs. PBDEs were measured in beluga whale (*Delphinapterus leucas*), ringed seal (*Phoca hispida*), Arctic cod (*Boreogadus saida*), Pacific herring (*Clupea pallasi*), Arctic cisco (*Coregonus autumnalis*), pelagic amphipod (*Themisto libellula*) and Arctic copepod (*Calanus hyperboreus*). The animals selected were from the sample archived repository at Fisheries and Oceans Canada. The brominated compounds were measured in the blubber of ringed seal and beluga, in the whole organism minus liver for the pelagic fish, and pooled composites for the invertebrates. Extraction methods were as per Tomy et al. (2008) (see above study). PBDEs were analyzed by high-resolution GC-ECNIMS. BDE209 was quantified by isotope dilution using $^{13}C_{12}$-BDE-209 and the *m/z* values of 486.6/488.6 and 494.6/496.6 for quantification and confirmation for the native and isotope internal standard, respectively. Stable isotopes of nitrogen, expressed as $\delta^{15}N$, were analyzed at the stable isotope laboratory at the University of Winnipeg (Manitoba, Canada). BMFs were determined as per Tomy et al. (2008) (see above study).

The relative trophic level status of the studied individual organisms was established using stable isotopes of $\delta^{15}N$. This analysis indicated the following food web: beluga whale > ringed seal > Arctic cod > Pacific herring and Arctic cisco > pelagic amphipod > Arctic copepod. The total concentration of the seven congeners analyzed (BDE47, -85, -99,

-100, -153, -154 and -209) ranged from 2.6 ng/g lipid in ringed seals to 205.4 ng/g lipid in Arctic cod. The median concentrations of BDE209 ranged from 0.04 ng/g lipid in the calanus (arctic copepod) to 7.23 ng/g lipid in Arctic cisco. The rank order of median BDE209 concentrations was: cisco > Arctic cod > herring > beluga and ringed seal > themisto > calanus. The researchers calculated lipid-normalized BMFs for BDE209 as follows: ringed seal (blubber):cod (liver) = 0.3; beluga (blubber):cod (liver) = 0.3; beluga (blubber):herring (liver) = 0.9; beluga (blubber):cisco (liver) = 0.03; cod (liver):calanus (whole body) = 12.7; cod (liver):themisto (whole body) = 4.8. The results suggested that depletion of BDE209 is taking place in the higher trophic level animals but that biomagnification may be occurring in the lower trophic level species. Although biomagnification was apparent in the lower food chain, there is some uncertainty in the results. For instance, it appears that the organisms included in the study were collected at different times and locations. None of the PBDEs showed a statistically significant positive relationship with trophic level, and no statistically significant TMF was found for BDE209.

Wu et al. (2009) evaluated the biomagnification of PBDEs, including BDE209, in a highly contaminated freshwater food web from southern China. Wild aquatic species representing different trophic levels were sampled in 2006 from a reservoir surrounded by several e-waste recycling workshops. Two top predator species, water snake (*Enhydris chinensis*) and northern snakehead (*Channa argus*), and their prey, mud carp (Cirrhinus molitorella), common carp (*Cyprins carpio*), crucian carp (*Carassius auratus*), and prawn (*Macrobrachium nipponense*), were sampled. Chinese mysterysnail (*Cipangopaludina chinensi*) was also collected by hand from the shallow water around the reservoir. Samples of small organisms (e.g., Chinese mysterysnail, prawn and mud carp) were pooled. The body weight and length of samples were measured, then stored at -20°C until further treatment. For chemical analysis, samples were thawed and whole-body homogenized, after which two sub-samples were taken from each specimen: one for PBDEs determination, and one for nitrogen stable isotope analysis. Samples were ground with ashed anhydrous sodium sulphate, spiked with surrogate standards, and extracted with hexane/acetone (1/1, v/v) for 48 hours. Lipids (gravimetrical method) were determined on an aliquot of the extract. PBDEs were analyzed by GC/MS ECNI mode and operated in SIM mode. Recoveries of the spiking blanks ranged from 76.9 to 105.2% for PBDEs. Relative standard deviations (RSD) of sample triplicates were less than 24% for BDE209. Stable isotopes of nitrogen, expressed as $\delta^{15}N$, were analyzed by a flash EA 112 series elemental analyzer interfaced with a Finigan MAF ConFlo 111 isotope ratio mass spectrometer.

TMFs were determined as per Tomy et al. (2004) using lipid-normalized concentrations and the trophic levels of the food web components. The TMF values ranged from 0.26 to 4.47 for PBDEs. For BDE209, the TMF was 0.26, suggesting trophic dilution, although the result was only marginally statistically significant at p=0.053. The authors suggest that different PBDE levels in the organisms, different environmental conditions (e.g., warmer water temperature), and the different food web composition could be a factor when comparing the lower TMF results of this study to northern food web studies. No significant correlation between TMFs and log K_{OW} was found for PBDEs, and the authors

suggested that other factors (e.g., metabolism) might play a more important role in PBDEs transfer in the food web.

Yu et al. (2009) analyzed concentrations of 10 PBDE congeners in a Pearl River Estuary food web of the Pearl River Delta region of southern China, to understand the accumulation behaviour of these substances. Two hundred and fifty-four biota samples (four species of invertebrates and ten species of fish) were collected from the Pearl River Estuary between 2005 and 2007. These species included sand swimming crab (*Ovalipes punctatus*, Samoan crab (*Scylla serrata*), ark shell (*Tegillarca granosa)*, oncomelania (*Oncomelania hupensischiui*), common mullet (*Mugil cephalus)*, red eelgoby (*Odontamblyopus rubicundus)*, robust tonguefish (*Cynoglossus robustus)*, slimy spinefoot (*Siganus canaliculatus*), silver sillago (*Sillago sihama*), pompano (*Psenopsis anomala)*, Japanese eel (*Anguilla japonica)*, flatheadfish (*Platycephalus indicus)*, large yellow croaker (*Pseudosiaena crocea)*, and Bombay duck (*Harpodon nehereus)*. One hundred and twenty-four individual or composite samples were analyzed for PBDEs. Samples were homogenized, extracted and analyzed by GC/MS ECNI and operated in SIM mode. The concentrations of PBDEs in organisms varied from 6.2 to 208 ng/g lipid weight. However, BDE209 was detected in only 18% of samples, ranging from non-detect to 1.6 ng/g lipid. Because of its low presence, BDE209 was excluded from further analysis of the report and no TMF was calculated for this congener.

In a study of *in vivo* and environmental debromination of decaBDE, La Guardia et al. (2007) monitored decaBDE concentrations in sediments and aquatic organisms in the receiving environment of a wastewater treatment plant (WWTP) located in Roxboro, North Carolina. All samples were extracted and purified using size-exclusion chromatography and then analyzed for PBDEs using GC/MS in electron capture negative ionization (ECNI) mode and electron ionization (EI) mode. Further study details are provided in Sections 3.1.2 and 3.2.1. In samples collected in 2002, decaBDE was detected in both sediments and tissues of sunfish (*Lepomis gibbosus*) and crayfish (*Cambarus puncticambarus sp. c*) collected immediately downstream of the WWTP outfall. The reported 2002 concentrations from this location in sediments, sunfish and crayfish were 1 630 000 μg/kg organic carbon (OC), 2880 μg/kg lipid and 21 600 μg/kg lipid, respectively. The much higher concentration in crayfish was attributed to the sediment-association of this species and the authors speculated that crayfish could form a link for the transfer of decaBDE from sediments to pelagic organisms.

Based on the La Guardia et al. (2007) results, it is possible to estimate sediment BSAFs of 0.0018 for sunfish and 0.013 for crayfish. These are well below values suggested to potentially indicate biomagnification (i.e., ~1.7 to 3; refer to Section 1.2). It is possible that a combination of low sediment bioavailability and/or metabolic transformation could be limiting the bioaccumulation and biomagnification of decaBDE in this system.

Wang et al. (2007) examined water, sediment and aquatic species collected from a small lake in Beijing, China, which receives effluent discharged from a large WWTP. Samples were homogenized, extracted and analyzed using HRGC/HRMS using EI ion source. The researchers found that average accumulations of 12 PBDEs (total, tri- to heptaBDEs) and

BDE209 were 6.33 and 237.01 μg/kg dw in sediments. BDE209 concentrations in lake water and effluent were below the analytical detection limit (not given for water; 1 μg/kg (ww or dw unknown) for sediment and biota). High concentrations of BDE209 were determined for lichen (1572 μg/kg dw), march brown (*Limnodrilus hoffmeisteri*; 11.37 μg/kg dw), coccid (114 μg/kg dw) and the zooplankton *Monia rectirostris, Monia micrur* and *Monia macrocopa* (151.9 μg/kg dw). Average concentrations in common carp (*Cyprinus carpio*), Java tilapia (*Tilapia nilotica*), leather catfish (*Silurus meridionalis*), crusian carp (*Carassius auratus*) and Chinese softshell turtle (*Chinemys reevesii*) were much lower, ranging from below detection to 19.32 μg/kg dw. Bioconcentration/bioaccumulation for BDE209 was not identified. In addition, the authors found no obvious biomagnification of PBDEs when they analyzed the relationship between PBDE concentrations and organism trophic level.

Xiang et al. (2007) sampled biota and sediment samples for PBDEs, including BDE209, from the Pearl River Estuary of China. In sediments they found that BDE209 was the dominant congener, ranging from 792 to 4137 ng/g OC in sediment samples (median 1372 ng/g OC). With respect to biota, they found non-detectable and measurable BDE209 concentrations in all biota species. Concentrations ranged up to 532.3 ng/g lipid in large yellow croaker (*Pseudosiaena crocea*; n=13, median=117.4 ng/g lipid), 623.5 ng/g lipid in silvery pomfret (*Platycephalus argenteus*; n=10, median=24.4 ng/g lipid), 38.4 ng/g lipid in flathead fish (*Platycephalus indicus*; n=17, median=0.0 ng/g lipid), 373.4 ng/g lipid in robust tongue fish (*Cynoglossus robustus*; n=8, median=0.0 ng/g lipid), 150.4 ng/g lipid in Bombay duck (*Harpodon nehereus*; n=9, median=0.0 ng/g lipid), 555.5 ng/g lipid in jinga shrimp (*Metapenaeus affinis*; n=10, median=0.0 ng/g lipid), 405.3 ng/g lipid in greasy-back shrimp (*Metapenaeus crocea*; n=10, median=30.3 ng/g lipid), and 88.5 ng/g lipid in mantis shrimp (*Oratosquilla oratoria*; n=9, median=42.47 ng/g lipid). The study notes that the high BDE209 concentrations in biota apparently resulted from elevated concentrations of BDE209 in local sediments. However, sediment BSAFs were calculated to range from 0 to 0.04 for BDE209 and trophic magnification was not deemed to be occurring based on the data shown in this study.

Eljarrat et al. (2007) reported the results of fish (n=29), sediment (n=6) and effluent (n=3) sampling conducted in November 2005 from the River Vero in Spain. They found high BDE209 concentrations in sediments (up to 12 459 ng/g dw) and fish—barbel (*Barbus graellsii*) and carp (*Cyprinus carpio*), from non-detectable to 707 ng/g lipid—downstream of an industrial park containing industries producing textiles and epoxy resins and involved with polymide polymerization. Using concentration measured in sediments and fish, the authors calculated sediment BSAFs for BDE209 of 0.0011 to 0.0013, thus suggesting that bioaccumulation was not occurring based on these data.

DeBruyn et al. (2009) studied marine horse mussels (*Modiolus modiolus*) and sediment collected off the coast of Vancouver Island, British Columbia, Canada near the city of Victoria to evaluate and compare patterns of PBDE and PCB bioaccumulation. Samples were collected from 14 stations within 800 metres of a municipal outfall, and from three reference locations. At each station, three surface sediment samples (0–2 cm) and 15

randomly selected mussels (> 50 mm length) were collected. Mussels were measured for shell length and width, total weight, tissue weight, age and sex, prior to compositing and homogenizing tissue samples for chemical analysis by station. PBDEs were analyzed using HRGC-HRMS. Methods for PBDE determinations and QA/QC procedures were as per the U.S. Environmental Protection Agency (U.S. EPA) Methods 1 668A (21) and 1 614 (22), with some modifications. Sediment BSAFs were calculated (lipid and organic carbon normalized) for BDE209.

Sediment BDE209 concentrations were measured at all stations, ranging from 232 pg/g dw to 2550 pg/g dw. BDE209 was the predominant congener in sediment near the municipal wastewater. Although sediment BDE209 concentrations were highest near the wastewater outfall (2550 pg/g dw), the congener exhibited a gradient of decreasing predominance with proximity to the outfall, from approximately 80% of total PBDEs at reference locations to approximately 40% at the outfall station and in wastewater. BDE209 concentrations in mussel tissue ranged from "not detected" to 5305 pg/g dw (wastewater outfall station), and were above the detection limit at 7 of 17 stations. BSAFs for BDE209 were calculated using sediment and mussel tissue concentrations from the sampling stations. At 10 sites, BSAFs could not be calculated, as mussel tissue BDE209 concentrations were below the limits of quantification. For the remaining sites, sediment BSAFs were calculated as: 1.48 (outfall), 0.52 (200 m), 1.59 (400 m), 0.97 (400 m), 3.53 (reference), 0.94 (reference), and 1.18 (reference). The results generally suggest that BDE209 was not accumulating appreciably in the mussels at any site except for the reference location.

Riva et al. (2007) studied the effect of BDE209 on freshwater bivalve zebra mussels (*Dreissena polymorpha*) under laboratory conditions. The primary objective of the study was to investigate the potential genotoxicity of BDE209. For this study, several hundred mussels were sampled at a depth of 4–5 m from the Italian subalpine great lakes. Mussels (still attached to rocks) were transferred to the laboratory, where they were maintained in glass tanks at a 12-hour light/dark photoperiod, with constant temperature (20°C) and oxygen (> 90% saturation), and fed a suspension of *Pseudokirchneriella subcapitata.* Approximately 150 acclimated mussels per BDE209 concentration were selected for exposure tests. DecaBDE (98% purity) was dissolved in isooctane/toluene (9:1 v:v) mixture, dissolved in dimethylsulfoxide (DMSO), and then added to water for final nominal concentrations of 0.1, 2 and 10 ug/L. Exposure assay aquaria were screened against direct sunlight to avoid photodegradation. Water was changed daily and mussels were fed 2 hours prior to water renewal. Control groups of mussels receiving either freshwater or a solvent blank (DMSO) were also monitored.

The mussels reached a relatively constant concentration of decaBDE in their tissues after 48 hours of exposure, for each treatment concentration. The United Kingdom (2008) interpreted the graphical representations of tissue concentrations and suggested a BCF for decaBDE in mussels on the order of 1000 l/kg or above. Riva et al. (2007) also suggested that there was also evidence for the presence of lower brominated congeners in the mussels after 168 hours exposure. These were not determined quantitatively but they

were interpreted to have consistent GC analysis peaks with three heptaBDEs, three octaBDEs and three nonaBDEs.

Nyholm et al. (2008) used zebrafish (*Danio rerio*) to study the extent of transfer of 11 structurally diverse brominated flame retardants (BFRs) from females to their eggs. The tested BFRS included the PBDEs BDE28, BDE183 and BDE209. The tested BDE209 compound was synthesized at the Department of Environmental Chemistry at Stockholm University, and labelled standards (e.g., ^{13}C) BDE209 were purchased (Cambridge Isotope laboratories—purity of PBDEs not provided). The adult fish were exposed to BFRs via their feed. The mixture of 11 BFRs in ethanol was added to freeze-dried chironomids, giving nominal concentrations of 1 and 100 nmol/g of each molecular species on a dry weight basis; ethanol was allowed to evaporate. Twenty-three males and 23 females were used for each dose level. Zebrafish were fed daily at ~2% of their body weight and sampled after 0, 3, 7, 14, 28, 35 and 42 days, 24 hours post-feeding. Two fish from each sampling period were pooled for analysis. Eggs were collected directly after spawning on days 0, 2–3, 6–7, 13–14, 27–28, 34–36, and 41–42. Samples were extracted and analyzed by GC/MS.

Average lipid contents of the fish and eggs were 3.36% and 0.47%, respectively. Female zebrafish exposed to high-dose feed (100 nmol/g) had approximately an order of magnitude greater BDE209 concentrations (3.8 to 9.6 nmol/g lipid) than those exposed to low-dose feed (0.17 to 0.97 nmol/g lipid). Maximum BDE209 concentrations in high-dose female zebrafish were measured after 28 d of exposure (9.6 nmol/g lipid), while maximum BDE209 in the low-dose fish was measured after day 42 (0.97 nmol/g lipid).

Egg concentrations from fish exposed to high-dose feed (100 nmol/g) were also approximately an order of magnitude greater in BDE209 concentrations (3.4 to 11 nmol/g lipid) in comparison to those of fish exposed to low-dose feed (0.46 to 2.2 nmol/g lipid). Maximum BDE209 concentrations in high-dose eggs were measured after 28 d of exposure (11 nmol/g lipid), while maximum BDE209 in the low-dose fish was measured after day 14 (2.2 nmol/g lipid). Eggs to fish ratios were calculated by dividing the BDE209 concentration at each point in time by the concentration measured in the fish. Egg/fish ratios were significantly ($p < 0.05$) higher than 1 for BDE209 in both exposure groups, with a higher ratio (i.e., higher BDE209 transfer) for the low-dose group. The authors suggested that the high egg/fish ratio may be influenced by the BFRs binding to the lipoproteins needed for egg production, or that the metabolic systems in eggs have a lower capacity for transformation than those in fish.

Marine Mammals and Terrestrial Species

Huwe and Smith (2007a, 2007b) examined the dietary accumulation, debromination and elimination of decaBDE in rats. Sprague-Dawley rats (n=26) were dosed with a commercial DecaBDE formulation (DE-83R 98.5% purity; 0.3 μg/g of diet) for a 21-d exposure period, which was followed by a 21-d elimination period. Following the 21-d exposure period, rats were sacrificed in groups of three for tissue analysis (liver, gastrointestinal tract, plasma, and remaining carcass) on days 0, 3, 7, 10, 14 and 21 of the

elimination phase. Control rats were also sacrificed on these days to determine background PDBE concentrations (n=3 on day 0 and n=1 on all other days), and background values were subtracted from the PBDE determinations in the exposure group. Feces were collected from dosed rats daily during the exposure phase and pooled for analysis. Rat feed, feces and tissues were analyzed for a suite of PBDEs, including hepta- to decaBDE congeners.

Based on the analytical results, it was estimated that only 5% (or 3.6 μg) of the total decaBDE dose was retained in rat tissues following the 21-d dosing period, while approximately 50% was excreted to feces during this time. In addition to decaBDE, the authors concluded that one nonaBDE congener (BDE207) and two octaBDE congeners (BDE201 and BDE197) were derived from the uptake of decaBDE. However, the total burden of BDE207, -201 and -197 accounted for only 3% of the total decaBDE dose, and 45% of the dosed decaBDE was unaccounted for in rat tissues and feces. The authors speculated that the formation of bound and/or hydroxylated metabolites which were not included in their analysis was a likely explanation for the incomplete mass balance of decaBDE.

Based on the observed carcass concentration of decaBDE a "BCF" (analogous to a BMF[4]) of 0.05 (on a ww basis, justified by the fact that the percent of lipid in food and carcass were similar) was calculated. It is not known whether decaBDE concentrations achieved steady state in rat tissues during the 21-d exposure period and, as a result, it is uncertain how well the reported BMF represents the potential steady-state value. It is also likely that the BMF based on a full accounting of decaBDE plus neutral, bound and or hydroxylated metabolites could be higher, but the concentrations of bound and hydroxylated metabolites were not reported. The study reported the half-life of decaBDE in rat tissue based on the observed elimination of decaBDE during the 21-d elimination period. First-order half-lives for decaBDE ranged from 3.9 d in plasma to 8.6 d in carcass. For the liver and plasma, second-order decay equations were found to represent the data well, with relatively rapid distribution phase half-lives of 0.7 and 1.2 d, respectively, but longer elimination phase half-lives of 20.2 and 75.9 d, suggesting potential persistence of decaBDE in rat tissues following chronic dosing. However, the authors cautioned that there was a high level of uncertainty in the second-order estimates.

Huwe et al. (2008) conducted a study of 29 male Sprague-Dawley rats to determine and compare the adsorption, distribution and excretion of PBDEs administered for 21 d as either a dust reference material or as a corn oil solution. The dust reference material (NIST Standard Reference Material 2585), containing a characterized and homogeneous composition of PBDEs, was mixed with rat chow. The corn oil solution contained commercial PBDE products DE-71, DE-79 and DE-83 (purity not given). These products were first dissolved in toluene and then mixed into a corn oil mixture. Daily doses were administered to the rats of either 1 or 6 μg/kg body weight (bw) of the dust/food or corn oil mixtures. The rats were randomly divided into five groups: controls (4 rats), low oil (4 rats), high oil (13 rats), low dust (4 rats) and high dust (4 rats). Most rats were dosed over

[4] The authors used the term "BCF"; however, given that this value was based on a comparison between rat tissue and food concentrations, it is actually analogous to a BMF.

a period of 21 d, and then were killed 24 hours after their last feeding. To assess whether rats were approaching a steady-state body burden, three groups of rats (n=3) from the high-oil-dose group were killed on days 3, 7 and 14 after dosing began. Sampling was conducted of the feed and oil mixtures, feces and tissues (epididymal fat, liver, kidney, brain, gastrointestinal tract and remaining carcass) for 15 PBDEs (BDE28/38, -47, -85, -99, -100, -138, -153, -154, -183, -186, -197, -203, -206, -207 and -209).

To determine whether steady-state body burdens were reached, only epididymal fat was sampled and analyzed. This analysis showed that all major tri- to octaBDEs had reached or were approaching steady state after 14 d, with no statistically significant differences between PBDE concentrations on days 14 and 21.

Retention of PBDEs in the body of rats was congener dependent and ranged from 4.0 to 4.8% of the dose for BDE209, and 10.1 to 22.6% for nonaBDEs, to approximately 69 to 78% for BDE47, -100, and -153, but did not generally differ between the dust and oil treatment groups. The study did not consistently detect nona- and decaBDEs in the adipose tissues above that of the controls. Urine contained less than 0.3% of any congener. Fecal excretion was the major route of elimination and was described in the study as that component of the dose not adsorbed. Fecal excretion was found to reach a steady state by day 2, with no statistically significant differences between mean concentrations in feces on days 2, 11 or 20. Excretion of BDE209 was approximately 68%, and ranged from 55.5 to 91.7% for the nonaBDEs. The amount of the BDE209 dose not adsorbed or excreted as parent compound ranged from 28% to 31.9%. Metabolic transformation products could have accounted for some portion of these percentages. Derived BCFs (analogous to BMFs as defined in this study) for adipose tissues were inversely related to the degree of bromination, and ranged from 7 to 24 for tri- to hexaBDE, 1 to 6 for hepta- to nonaBDEs and < 1 for decaBDE. For the liver tissues, BCFs for all PBDEs were below 1 except for BDE206 (BCF=2.4), and -207 (BCF=1.09). Hepatic Cyp2B1 and 2B2 mRNA expression increased in rats receiving the higher PBDE doses, suggesting potential effects of metabolic activity. The use of PBDE mixtures in this study made it impossible to conclusively determine whether metabolic debromination had occurred, although some of the findings are suggestive that metabolic transformation may have taken place (e.g., higher BMFs for some congeners like BDE206, and the detection of hydroxylated tetra- to hexaBDEs in feces).

Kierkegaard et al. (2007) reported the findings of a 3-month feeding study with dairy cows. The study was originally undertaken to measure the long-term mass balance of PCBs; however, archived samples from two cows and feed were subsequently analyzed for a range of PBDEs. Over the 13-week study period, the cows were kept indoors and milk and feces were sampled once per week. The feed consisted of silage, concentrate and a mineral supplement which was not deliberately spiked with PBDEs. As a result, the PBDE concentration in food generally represented “background” contamination levels.

The milk and feces samples were pooled according to the following scheme to obtain a series of 5 composite samples representing discrete portions of the 13-week study period: three 3-week composites and two 2-week composites. In addition, one of the cows was

slaughtered at the end of the 13-week study, and samples of adipose tissue from 6 fat compartments as well as tissues from liver, kidney, heart and leg muscle were collected for analysis. Silage samples were retained for analysis at three intervals during the 13-week study, and concentrate and mineral samples were analyzed once during the study. All samples/composites of feed, milk, feces and tissue were analyzed for hepta- to nonaBDEs using HRMS and were analyzed for decaBDE using LRMS in negative chemical ionization mode (limit of quantification = 0.4 to 150 pg/g lipid or dw).

DecaBDE (i.e., BDE209) was the dominant congener in all matrices except milk, suggesting that milk levels were influenced more by the existing burden of PBDEs in storage tissue rather than uptake from food. In addition, PBDE levels were higher in the adipose storage tissues than in organ tissues. Based on these observations, the authors proposed that the cows were in a state of PBDE elimination rather than accumulation and that the observed PBDE concentrations may have been influenced by exposure to PBDEs prior to initiation of the 13-week experiment. Although a mass-balance analysis of input and output fluxes of PBDEs was attempted, it was largely unsuccessful due to a large increase in octa-, nona- and decaBDE congeners in the second silage sample relative to the first and third samples. It was unclear whether this second sample accurately represented the cows' exposure since the increase of PBDEs in feces did not appear to coincide with the greater increase in decaBDE concentration in feed. PBDE concentrations in concentrate and mineral supplements were much lower than in the silage and did not likely affect the overall mass balance conducted on the PBDEs in this study.

The Government of the United Kingdom (United Kingdom 2007a) conducted a critical analysis of the Kierkegaard et al. (2007) study and calculated dietary accumulation factors for a cow from silage based on either adipose tissue or whole body and the average silage concentration or end silage concentration. They concluded that the high variation in silage PBDE concentration led to considerable uncertainty in the study results with respect to bioaccumulation. To estimate accumulation factors and BMFs on a lipid-normalized basis, they assumed a 4% lipid content in silage. Table 2-5 summarizes the calculated accumulation factors and BMFs for nona- and decaBDE congeners from the Kierkegaard et al. (2007) study.

The calculated BMFs and accumulation factors were well below 1 for decaBDE and only exceeded 1 for BDE207. For BDE207, it is uncertain whether the estimated values represent direct accumulation of BDE207 from food, or accumulation combined with bioformation as a result of the debromination of decaBDE. The United Kingdom (2007a) study proposed that, for chemicals such as decaBDE which undergo transformation once accumulated in organisms, BMF estimates should be based on the total burden of parent chemical and the metabolites resulting from accumulation and transformation of the parent chemical. In the case of the Kierkegaard et al. (2007) study, it is difficult to do so since the lower brominated PBDEs were also present in the feed and their presence in tissue could thus have resulted from both accumulation and bioformation. Furthermore, based on studies with rats, it is likely that alternative transformation pathways are also present in mammals which result in the presence of polar metabolites, bound residues and

water-soluble residues. For a full accounting of the total chemical burden related to the accumulation of decaBDE, these would also have to be quantified.

Table 2-5: Summary of accumulation factor calculations by United Kingdom (2007a) using the findings of Kierkegaard et al. (2007) for one cow

Parameter	Congener			
	BDE206	**BDE207**	**BDE208**	**BDE209**
Concentration Data				
Mean concentration in silage (ng/kg lipid)[1]	4150	2583	1626	98 750
Silage concentration over last feeding period (ng/kg lipid)[1]	625	450	220	12 000
Mean adipose tissue concentration (ng/kg lipid)	552	1867	155	3700
Mean concentration in organs/muscle (ng/kg lipid)	239	740	49	2378
Estimated mean whole body concentration in cow (ng/kg lipid)	286	909	65	2576
Derived Accumulation Factors for Adipose Tissue				
Ratio of mean adipose concentration (ng/kg lipid) to mean silage concentration (ng/kg lipid)	0.13	0.72	0.095	0.037
Ratio of mean adipose concentration (ng/kg lipid) to silage concentration over last feeding period (ng/kg lipid)	0.88	4.1	0.70	0.31
Derived Accumulation Factors for Whole Body				
BMF based on estimated whole body concentration (ng/kg lipid) to mean silage concentration (ng/kg lipid)	0.069	0.35	0.040	0.026
BMF based on estimated whole body concentration (ng/kg lipid) to silage concentration over last time period (ng/kg lipid)	0.46	2.0	0.30	0.21

[1] Assumed lipid content of 4%.

Thomas et al. (2005) examined the absorption of decaBDE from diet by three captive juvenile grey seals (*Halichoerus grypus*). The captive seals were fed a constant diet of herring for 3 months prior to the initiation of the 3-month study (6 months total time). During the 3-month study, feeding with herring continued (1–2.5 kg/d), with all fish obtained from a single batch caught in the North Sea. The second month of the study

involved a decaBDE exposure phase with the diet supplemented with 12 μg decaBDE per day, dissolved in a cod liver oil capsule. For the final month of the study, the diet was supplemented with the cod liver oil capsule only to measure elimination of accumulated decaBDE. Fish, blood and feces samples were collected and analyzed for PBDEs on a weekly basis throughout the 3-month period, while blubber biopsies were taken and analyzed for PBDEs 3 times during the study (beginning, 3 d after initiation of exposure phase, and end, after 29 d on a decaBDE-free diet).

The blood decaBDE concentration increased from non-detectable at the start of the exposure phase (day 28) to a maximum of approximately 1000 ng/g lipid (value read from graph) between 5 and 11 d after the end of the exposure phase. Concentrations of decaBDE in blubber ranged from non-detectable to 3.9 ng/g lipid on day 30 (3 d into the exposure phase) and from 3.4 to 7.4 ng/g lipid on day 83 (after 29 d on a decaBDE-free diet). The percentage of total ingested decaBDE estimated to be in blubber on day 30 ranged from 36 to 68% (for one of the three seals, decaBDE was not detected in blubber) and on day 83, from 11 to 15%.

During the elimination phase, the decaBDE half-lives in blood were estimated to be between 8.5 and 13 d, most likely due to a combination of metabolic transformation/elimination and transfer to blubber. The authors suggested that once stored in blubber, decaBDE was unlikely to be metabolized.

The calculation results indicate a relatively high (i.e., exceeding 1) BMF for blood and relatively low BMF for blubber. The relatively high blood-based BMF suggests that significant magnification could be occurring from the diet to blood and related tissues, indicating relatively high bioaccumulation potential. The blood BMFs reported by Thomas et al. (2005) may be somewhat uncertain because of other non-lipid constituents (proteins) that can be attributed to the overall sorptive capacity of blood, thus making blood lipid normalization less accurate. The lower BMFs for blubber could be explained by the large storage capacity of fat tissues for lipophilic chemicals—it is possible that only a small fraction of the potential steady-state concentration in blubber was reached during the 26-d exposure phase.

Based on the mass balance of measured input flux (in consumed diet) and output flux (in feces) of decaBDE, the authors determined an average absorption efficiency of 89% for decaBDE. The authors suggested that the high absorption efficiency called into question the theories regarding molecular size thresholds for chemical absorption. The relatively high apparent absorption efficiency was attributed to the following factors:

- Large fat reserves (i.e., blubber) which provide a reservoir for lipophilic chemicals.
- High food absorption efficiency in homeothermic carnivores such as seals, which creates strong a fugacity gradient in the gut, resulting in greater chemical uptake.
- Possible incomplete collection of feces; however, it was estimated that over a 24-hour period, not more than 10% of feces was lost.
- Degradation of decaBDE in the gut. The lower brominated PBDE congeners remained constant in feces despite the supplement with decaBDE during the second month, suggesting that degradation was not significant.

Table 2-6: Estimated BMFs from the Thomas et al. (2005) feeding study with captive juvenile grey seals (*Halichoerus grypus*)

Parameter	Value	Units
Daily dose of decaBDE	12	μg/day
Fish feeding rate	1–2.5	kg/day
Exposure concentration	4.8–12 48–120	ng/g ww ng/g lipid
Maximum blood concentration (approximate)	1000	ng/g lipid
Blood BMF	**8.3–20.8**	**g/g lipid**
Median blubber concentration – Day 30	3	ng/g lipid
Median blubber concentration – Day 83	5.3	ng/g lipid
Blubber BMF – Day 30	**0.025–0.063**	**g/g lipid**
Blubber BMF – Day 83	**0.044–0.11**	**g/g lipid**

An additional explanation for the high apparent absorption efficiency which was not discussed by the authors is the potential formation of bound metabolites and/or the formation and subsequent excretion of phenolic and/or methoxylated metabolites. Recent studies with Sprague-Dawley rats (Mörck et al. 2003; Huwe and Smith 2007a, 2007b; Riu et al. 2008) have indicated that the formation of phenolic and methoxylated metabolites may be an important transformation pathway for accumulated decaBDE. Thus, if phenolic or methoxylated metabolites were present but not analyzed in the feces, it is possible that the output flux was underestimated, resulting in an overestimate of the absorption efficiency of decaBDE. Another potential uncertainty relates to an inconsistency in identified dosage rate. While the high proportion of total ingested decaBDE estimated to be present in blubber on day 30 and 83 (i.e., up to 68%) indicates significant decaBDE adsorption, there is some uncertainty in the overall findings of the study.

By using the reported decaBDE concentrations in blood and blubber in conjunction with an estimated decaBDE concentration in food, it is possible to estimate blood-based and blubber-based BMFs for the Thomas et al. (2005) exposure study. Unfortunately, the authors did not report the lipid content of the herring consumed by the seals; however, based on values reported in the literature (e.g., Iverson et al. 2002; Jensen et al. 2007) a value of 10% lipid was used as a reasonable approximation for the BMF estimates summarized in Table 2-6. Note that the blood concentrations do not appear to have reached steady state during the exposure phase and it is likely that this also applies to the blubber concentrations. Furthermore, the blubber concentrations at day 30 and day 83 are unlikely to represent maximum accumulation of decaBDE (maximum accumulation would be expected at or after the end of the exposure phase (at day 54). Thus, the blood-based and blubber-based BMFs underestimate the steady-state BMF to an unknown degree.

Sellström et al. (2005) analyzed decaBDE in soil and earthworm (species not identified), samples collected in 2000 from three research stations (with reference plots and sewage sludge amended plots) and two farms (reference and amended/flooded soils) in Sweden. Soil decaBDE concentrations at the various sites ranged from 0.015 to 22 000 ng/g dw, and organic carbon content (based on loss-on-ignition data) ranged from 2.12 to 7.22%. Concentrations of decaBDE in worms ranged from 0.99 to 52 000 ng/g lipid. Using these data, Sellström et al. (2005) calculated site-specific soil BSAFs for co-occurring worm and soil samples. The estimated BSAFs ranged from 0.04 to 0.7 and averaged 0.3. Based on these results, the authors concluded that decaBDE was bioavailable in soils. However, in this study, decaBDE was not shown to be bioaccumulating in earthworms. The authors did not observe any evidence of photolytic debromination in soils.

The soil BSAFs determined by Sellström et al. (2005) were all below the range that might provide evidence of decaBDE biomagnification (i.e., 1.7 to 3; refer to Section 1.2).

As part of their study of grizzly bears from British Columbia, Christensen et al. (2005) conducted a bioaccumulation analysis for decaBDE. Their method involved the estimation of a "bioaccumulation slope" based on a comparison of decaBDE concentration with the proportion of meat in the diet. The rationale was that if trophic magnification was causing an increase in decaBDE concentrations in prey above that in consumed vegetation, then the concentration of decaBDE in bears would increase as the proportion of prey-derived meat in their diet increased. A positive bioaccumulation slope indicates trophic magnification while a negative bioaccumulation slope indicates that trophic dilution was taking place. The authors indicated that the bioaccumulation slope for decaBDE was negative (albeit not significantly different from 0) but did not provide the value. The lack of a significant positive bioaccumulation slope for decaBDE suggests that decaBDE was not undergoing trophic magnification in the studied grizzly bear food webs based on a diet of meat. The study did not examine the potential significance of decaBDE exposure via consumed vegetation or inhaled air.

In their study of decaBDE in the marine food web of Svalbard, Norway, Sørmo et al. (2006) also attempted to estimate BMFs for predator and prey species. Unfortunately, the high number of non-detected concentrations precluded the estimation of BMFs for polar bear / ringed seal or ringed seal / polar cod combinations. The estimated BMFs for polar cod / ice amphipod based on the mean concentrations in each species (except for ice amphipod for which there was only one sample) were 0.1 (wet weight basis) and 0.03 (lipid weight basis), indicating no evidence of biomagnification for this predator-prey combination.

2.2.3 Model Predictions

Published models exist for predicting bioaccumulation in aquatic food webs and biomagnification in terrestrial mammals. The BAF–QSAR model described by Arnot and Gobas (2003) has generic applicability to the Canadian environment, and a modified version of this model was applied during the Government of Canada's categorization of its Domestic Substances List. This model predicts both BAFs and BCFs for three representative fish trophic levels (low, middle and upper) in a generic aquatic food web

based on a standard set of conditions found in the Canadian environment. For the terrestrial environment, Gobas et al. (2003) describe a terrestrial biomagnification model for adult male wolves (*Canis lupus*) based on the work of Kelly and Gobas (2003). It predicts BMFs for wolves based on chemical log K_{oa} (logarithm of octanol-air coefficient), chemical log K_{ow}, and a set of parameters describing the lichen-caribou-wolf food web of Bathurst Inlet in the Canadian Arctic. Both of these models incorporate a metabolic rate constant as part of chemical elimination, allowing for metabolism correction of BAF, BCF and BMF predictions based on field or laboratory observations. For both models, however, the default assumption is zero metabolism.

BAF and BCF predictions for fish were made for decaBDE using the BAF–QSAR model. Environment Canada's review of log K_{ow} values for decaBDE revealed that a log K_{ow} of 8.7 reported by Wania and Dugani (2003) is considered to represent the most reliable value available. For further rationale regarding the selection of log K_{ow} values, refer to Appendix C. Two prediction scenarios were conducted: the first with no correction made for metabolic transformation, and the second corrected for metabolism based on the laboratory observations of Tomy et al. (2004). Tomy et al. determined a half-life of 26 d in juvenile lake trout fed a diet containing decaBDE, which is consistent with a total elimination rate (k_T) of approximately 0.027/d. The Tomy et al. (2004) value was used to derive an *in vivo*-based metabolic rate constant (k_M) according to the method of Arnot et al. (2008b). In this method, when k_T is available, k_M is derived according to the following equation:

$$k_M = k_T - (k_2 + k_E + k_G)$$

where:

k_M = the metabolic rate constant (1/d);
k_2 = the elimination rate constant (parameterized using data from Arnot et al. 2008a);
k_E = fecal egestion rate constant (parameterized using data from Arnot et al. 2008a); and
k_G = growth rate constant (parameterized using data from Arnot et al. 2008a).

The method of Arnot et al. (2008b) provides for the estimation of confidence factors (CFs) for the k_M to account for error associated with the *in vivo* data (i.e., measurement variability, parameter estimation uncertainty and model error). A CF of ±3.0 was calculated for the available BMF data.

Because metabolic potential can be related to body weight and temperature (e.g., Hu and Layton 2001; Nichols et al. 2006), the k_M was further normalized to 15°C and then corrected for the body weight of the middle trophic level fish in the Arnot-Gobas model (184 g) (Arnot et al. 2008a). The middle trophic level fish was used to represent overall model output and is most representative of fish weight likely to be consumed by an avian or terrestrial piscivore. After normalization routines, the k_M ranges from 0.02 to 0.17 with a median value of 0.06.

The BAF and BCF predictions for the middle trophic level fish for decaBDE are summarized in Table 2-7. All predicted BCF values are below 5000, which is expected given that uptake and elimination via the gills (which BCF accounts for) is limited and only important for substances with a log K_{ow} of approximately less than 4.5.

Table 2-7: BAF and BCF predictions for decaBDE using the Arnot-Gobas kinetic model (v1.11)

k_M (metabolism-corrected; days)	Log K_{ow} used	Arnot-Gobas BCF	Arnot-Gobas BAF	Half-life (days)
1.93E-02 (2.5%)	8.7	251	**161 618**	99
0.058 (average)	8.7	90	**29 386**	35
0.17 (97.5%)	8.7	31	4056	12
0 (no metabolism)	8.7	2570	**2 630 268**	795

Note: Bolded values exceed the BAF/BCF criterion of 5000.

The predicted BAF, when corrected for metabolic transformation, ranges from 4056 to 161 618 depending on the rate of metabolism. The predicted BAF for an average k_M, which can be said to represent the typical fish metabolic potential in the Canadian environment, was calculated to be 29 386. When a default of no metabolism is used in the model, the BAF is several orders of magnitude higher than the BAF calculated with average metabolic rate potential. These results demonstrate the influence of both chemical partitioning behaviour (i.e., log K_{ow}) and metabolic transformation on the bioaccumulation potential of decaBDE.

The metabolism-corrected BAFs probably provide the best estimate of the bioaccumulation potential of decaBDE since metabolic transformation of decaBDE has been demonstrated or inferred in most laboratory studies. It is important to note that these corrected BAFs could underestimate the total chemical bioaccumulation related to decaBDE since they are based on parent chemical only and do not account for the additional presence of metabolites in tissues.

BMF predictions for wolves were made using a spreadsheet version of the Gobas et al. (2003) model. The model can be re-parameterized for dietary assimilation efficiency (E_D) and k_M, the default settings of which are 90% and 0%, respectively. In addition to the range of potential log K_{ow} values described for the BAF–QSAR model in Appendix C, two log K_{oa} estimates were available: 15.27 (Tittlemier et al. 2002) and 18.423 (predicted by the QSAR model, KOAWIN). However, the BMF predictions do not vary significantly for log K_{oa} or log K_{ow} values in these ranges, and systemic variation of log K_{oa} and log K_{ow} made little difference in the predicted output. Four prediction scenarios were conducted: the first with no correction for metabolic transformation, and the remaining three corrected for metabolism based on the laboratory observations of Huwe and Smith (2007a, 2007b). They determined a range of potential half-lives for decaBDE in rats based on a combination of first-order and second-order approximations to elimination data for carcass, liver and blood plasma. For carcass, a first-order half-life of 8.6 d was found to best represent the elimination data. For blood plasma and liver, second-order models (i.e., with distribution and elimination phases) provided the better

fit, with elimination phase half-lives (representing slower elimination from residual body stores) of 75.9 and 20.2 d, respectively. These half-lives were used to infer k_M values using a similar method as with the BAF–QSAR model, resulting in k_M ranging from 0.0086 to 0.08/d. Because these rates are based on rodent exposures, they also must be scaled to the body weight of the wolf (e.g., Hu and Layton 2001). This results in k_M values of 0.004 to 0.03 assuming a rat weight of 0.25 kg and the wolf model body weight of 80 kg (Hu and Layton 2001; Arnot et al. 2008b).

The terrestrial BMF predictions for decaBDE are summarized in Table 2-8. An E_D of ~56% was calculated as outlined in Kelly et al. (2004) using the E_D model for humans as the best estimator for carnivores. In the absence of a correction for metabolism, the predicted BMF was 89 and, when corrected for metabolism BMF predictions, ranged from 0.5 to 3.5 depending on the k_M used in the model (Table 2-8). These metabolism-corrected and assimilation-efficiency-corrected predictions are within the range of experimental BMF values reported for decaBDE for terrestrial and aquatic receptors. The metabolism-corrected BMFs probably provide the best estimate of the biomagnification potential of decaBDE since metabolic transformation of decaBDE has been demonstrated or inferred in most laboratory and captive feeding studies reviewed from the open literature. Thus, corrected BMFs suggest a lack of or low level of biomagnification largely as a result of metabolism of decaBDE.

It is important to note that the corrected BMFs could underestimate the total chemical biomagnification related to decaBDE since they are based on parent chemical only and do not account for the additional presence of metabolites in tissues. Thus, if all metabolites were included in the BMF calculations, it is possible that all predicted BMFs might be higher.

Table 2-8: Wolf BMF predictions for decaBDE made using the terrestrial biomagnification model of Gobas et al. (2003)

k_M (wolf BW normalized) (days)	Log K_{ow} used	Log K_{oa} used	Gobas BMF	Half-life (days)
0.004 (based on half-life of 79.5 d decaBDE in plasma)[1]	8.7	15.2	3.5	173
0.03 (based on half-life of 8.6 d decaBDE in carcass)[1]	8.7	15.2	0.5	23
0 (no metabolism)	8.7	15.2	89	4119

Note
[1]Half-lives observed by Huwe and Smith (2007a, 2007b).

2.3 Weight-of-Evidence Analysis

2.3.1 Summary of Evidence

This section provides a summary of evidence which is currently available regarding the bioaccumulation and biomagnification of decaBDE. It is intended to synthesize the existing state of the science regarding whether decaBDE is "bioaccumulative" or may biomagnify in food chains. Unequivocal evidence supporting the conclusion that decaBDE is bioaccumulative and meets the criteria for bioaccumulation under the *Persistence and Bioaccumulation Regulations*, or is biomagnifying in food chains, was not available. Additional considerations and interpretations on the capacity for decaBDE to bioaccumulate and/or biomagnify are also summarized in a third section.

1. ***Equivocal***[5] *evidence with respect to whether decaBDE has significant potential to bioaccumulate or biomagnify in the environment:*

- The findings of Tomy et al (2009) which found BMFs of 12.7 and 4.8 for the cod:calanus and cod:themisto feeding relationships, suggesting that biomagnification may be occurring at these lower trophic levels in a western Arctic marine food web. However, BMFs for other feeding relationships within the same food chain were below 1 for higher trophic-level feeding relationships and the calculated TMF was also below 1. The study also has some uncertainty since organisms were sampled at different times and in different locations.
- DeBruyn et al. (2009) calculated decaBDE sediment BSAFs for seventeen sites off the coast of British Columbia, Canada. At 10 sites, BSAFs could not be calculated as mussel tissue BDE209 concentrations were below the limits of quantification. For the remaining sites, all calculated BSAFs were low (i.e., ≤ 1.48), except at one reference site where the sediment BSAFs was calculated as 3.53, which suggests that, at this location, decaBDE may have biomagnified in the mussels.
- The toxicity study by Riva et al. (2007), which is based on the interpretation of the United Kingdom (2008), suggested accumulation of decaBDE in zebra mussel potentially up to or exceeding 1000 l/kg (lipid). However, the study was conducted as a genotoxicity study and many study attributes relevant for an evaluation of bioconcentration were lacking (e.g., measured concentrations in water, precise concentrations in organism). Further, exposure concentrations may have exceeded the water solubility limit of decaBDE.
- A feeding study with cows by Kierkegaard et al. (2007) found that all cow-silage BMFs were less than 1. However, a full accounting of parent decaBDE plus metabolites was not conducted. There was significant uncertainty in the results of this study due to the high variability of decaBDE concentrations in the silage feed.
- The findings of a Lake Winnipeg pelagic food web study by Law et al. (2006) led to an estimated TMF of 3.6 for a Lake Winnipeg food web, with many BMFs exceeding 1. These observations were based on parent decaBDE only. If both decaBDE plus metabolites were considered, it is possible that the TMF and BMFs

[5] Studies cited here are considered by Environment Canada to provide uncertain evidence for decaBDE to bioaccumulate or biomagnify in the environment.

might be higher. However, this study had a number of limitations that make the results less certain.

- A feeding study by Stapleton et al. (2006) using juvenile rainbow trout found tissue-specific BMFs which ranged up to 1.74 for decaBDE and neutral metabolites. Other metabolites which were not measured may have also been present. However, BMFs based on serum and carcass data did not exceed 1.
- Two studies by Burreau et al. (2004, 2006) on food webs of the Baltic Sea evaluated biomagnification in two Baltic Sea food webs and found that biomagnification of decaBDE did not appear to be occurring. Failure to detect decaBDE in salmon from the Atlantic Ocean precluded a similar analysis for this food web. In addition, the relatively high levels of decaBDE in procedural blanks and low concentrations of decaBDE in biota samples created uncertainty in the overall biomagnification analysis.
- In a feeding study using captive seals by Thomas et al. (2005), blood-serum-based BMFs exceeded 1 but blubber BMFs were well below 1. Blubber concentrations are unlikely to have reached steady state. The steady-state BMFs for blubber could be higher. However, this study reported an unusually high calculated dietary assimilation efficiency of 89%, which would not be expected for decaBDE.
- Although high concentrations of decaBDE have occasionally been observed in birds of prey (especially in China and Europe), red fox, shark, marine mammals and a few marine bird species, these high observed concentrations are confounded by the potential that the sampled species inhabit decaBDE hotspots close to industrialized areas of Europe and China. Thus, if environmental exposures were very high, organisms could still achieve very high concentrations in their tissues even though BAFs or BMFs were very low.
- Relatively high concentrations of decaBDE were observed in peregrine falcons in Greenland.
- Using the BAF–QSAR model, metabolism-, weight- and temperature-normalized aquatic BAF predictions range from approximately 4000 to 162 000 and reflect the uncertainty associated with estimating metabolic rates in fish. The average BAF corrected for metabolism was calculated to be approximately 30 000. If the BAF–QSAR model also accounted for the body burden of metabolites, then the BAF estimates could potentially be higher than the range predicted for the parent compound.
- Using the Gobas et al. (2003) wolf model, metabolism-corrected terrestrial BMF predictions can range from slightly above to slightly below 1 depending on the rate of metabolism assumed in the wolf model. The moderately high dietary efficiency of 56% contributes to a BMF greater than 1 when the lower metabolism rate constant is used. It should be noted that it is difficult to compare modelled BMF with any of the available measured BMFs because none of them are reported for the wolf or other terrestrial carnivore food web. Any comparisons involve uncertainties due to different species with different metabolic rates, assimilation efficiencies and body weights.

2. *Evidence that **does not support**[6] the conclusion that decaBDE has significant potential to bioaccumulate or biomagnify in the environment:*

- A feeding study by Huwe and Smith (2007a, 2007b) using rats determined a BMF of 0.05, while a study by Huwe et al. (2008) obtained a derived BMF of less than 1. A full accounting of parent decaBDE and metabolites could have increased the BMF estimates.
- Calculated sediment BSAFs determined by La Guardia et al. (2007), Xiang et al. (2007) and Eljarrat et al. (2007) were well below thresholds indicative of biomagnification above equilibrium conditions.
- A study by Sørmo et al. (2006) for a marine food web in Svalbard, Norway, identified only very low concentrations and a very low BMF (0.03) for the polar cod / ice amphipod predator-prey relationship. Other BMFs could not be calculated because of the high frequency of samples in which decaBDE was not detected, suggesting a low potential for biomagnification in general in this food web.
- A study by Sellström et al. (2005) of the accumulation of decaBDE in earthworms from soil found that soil BSAFs were well below the threshold range (>1.7 to 3) that would indicate biomagnification above equilibrium conditions.
- Relatively low and often non-detected concentrations of decaBDE were found for a wide range of middle to top predators including some marine fish, mammalian predators (grizzly bear, polar bear and lynx), marine mammals, marine/aquatic birds and birds of prey (multiple studies, often the same ones as those where relatively high concentrations were also observed).
- There was minimal observed uptake of decaBDE by *Lumbriculus variegatus* exposed to decaBDE in biosolids and artificial spiked sediments (Ciparis and Hale 2005).
- A study by Christensen et al. (2005) on grizzly bears suggested that biomagnification is not occurring.
- A feeding study by Tomy et al. (2004) using juvenile lake trout determined a BMF of 0.3, using the kinetic method based on parent decaBDE only. If metabolites were included in the BMF estimate, it may have been higher.

3. Additional considerations and evidence:

- A BCF study by MITI (1992) resulted in an estimated BCF < 3000. Note that this BCF value was based on the estimated water solubility of decaBDE and the detection limit in tissues, and therefore is highly uncertain.
- There was low chemical uptake efficiency (i.e., at least 0.44%) and evidence of metabolism in carp (Stapleton et al. 2004).
- There was low chemical uptake efficiency (i.e., from at least 0.02 to 3.2%) and evidence of metabolism in rainbow trout (Stapleton et al. 2006; Kierkegaard et al. 1999).

[6] Studies cited here indicate that decaBDE does not have the potential to bioaccumulate or biomagnify in the environment; these studies include both reliable and less certain evidence.

- Metabolism studies using rats (Norris et al. 1973, 1974; El Dareer et al. 1987) suggested that decaBDE has low retention (generally less than 5%), low bioaccumulation potential in mammalian species, and rapid excretion.
- Very low chemical assimilation efficiencies in fish suggest that assimilation efficiency in carnivorous mammals may be lower than predicted by the E_D model for humans. However, the predicted E_D value is based on a relationship using multiple halogenated organics and so is appropriate for modelling decaBDE. Fish do show a significantly lower E_D than mammals at a log K_{ow} of 8.7 (conservatively estimated at 56% vs. ~0%) using the relationships summarized in Kelly et al. 2004). Thus, high assimilation of halogenated aromatics by mammals would contribute to a potential for higher BMF values in mammals. However, there is little evidence from terrestrial food web studies to substantiate this. BMF values could be offset by a high rate of gut metabolism to lower brominated and hydroxylated bio-transformation products, which would limit the E_D vs. model predictions of E_D.

2.3.2 Conclusion Respecting Bioaccumulation

The existing evidence for the bioaccumulation of decaBDE does not support a conclusion of "bioaccumulative" as defined in the current *Persistence and Bioaccumulation Regulations* under CEPA 1999. While most available data show that decaBDE has limited potential to bioaccumulate or biomagnify in the environment, some evidence suggests a higher BAF than previously considered for decaBDE, and some new data suggest possible biomagnification. The modelling undertaken to support this evaluation, however, shows uncertainty associated with metabolism in fish, as model-predicted aquatic BAFs range from below the 5000 criterion to well above 5000. Predicted terrestrial carnivore BMF values also range from below 1 to greater than 1 depending on the rate of metabolism assumed. Although less relevant than BAF or BMF, experimental BCF measures are below the 5000 criterion. The substance is shown to be increasing in concentrations in some wildlife species, and some data suggest that decaBDE has reached concentrations in some organisms interpreted to be high. DecaBDE (i.e., BDE209) is also considered to be contributing to the bioaccumulation potential of total PBDEs as a result of metabolism to lower brominated forms (to be discussed later in this report).

3 Evidence of Transformation

This section examines the transformation of decaBDE to determine whether there is concern respecting its transformation to bioaccumulative products in organisms (i.e., *in vivo* by metabolism) and in the environment (i.e., by abiotic and biodegradation processes). The evidence provided by studies quantifying transformation may be confounded by the following limitations:

(a) Unknown or uncharacterized purity of the parent compound, and the presence (often unquantified) of lower brominated PBDEs in the materials used (e.g., studies typically used decaBDE with a purity of approximately ≥98%). There is a potential that lower brominated PBDE congeners may be present as impurities in the food used in *in vivo* transformation studies. Some of the lower congeners may have higher accumulation factors than decaBDE. Thus, even if these impurities

were present in the food (or the decaBDE test substance itself) at concentrations below the detection limit, they could still accumulate to measurable concentrations in the organism during the course of the experiment.

(b) Analytical challenges, which could influence the accuracy of the results (see Section 2).

(c) Use of laboratory studies to make inferences respecting processes and rates of transformation in the environment.

These issues are discussed in more detail later in this section.

3.1 In Vivo Transformation

3.1.1 Information Evaluated in the Screening Assessment for PBDEs

The following summarizes the evidence regarding debromination of decaBDE which was available during and prior to 2004 and considered in the screening assessment for PBDEs (Environment Canada 2006b).

In a decaBDE feeding study with juvenile carp, Stapleton and Baker (2003) and Stapleton et al. (2004) found that seven transformed products of decaBDE accumulated in the whole fish and liver tissues over the exposure period. These were identified as penta- to octaBDEs with only BDE154 and -155 (i.e., hexaBDEs) being positively identified (personal communications from HM Stapleton to Environment Canada, July 2009; unreferenced). The unidentified hepta- and octaBDE congeners have recently been determined to be BDE188, -202 and -197 (Stapleton et al. 2006). It also appeared that higher brominated PBDEs, such as octaBDE, may be further debrominating to lower brominated PBDE homologues. By comparing the structure of decaBDE with those of its identified degradation products, Stapleton and Baker (2003) suggested that this debromination may be due to the action of deiodinase enzymes which normally act on thyroid hormone. The bioavailability of decaBDE (parent chemical plus metabolites) was estimated at 0.44%. This value could be higher if other metabolites were present (not determined in these studies). It should be noted that the alternative explanation provided by Stapleton and Baker (2003) for the transformation of decaBDE, i.e., that it could have also been due to the action of intestinal microflora, has recently been refuted by Benedict et al. (2007), whose study confirmed that carp intestinal flora cannot debrominate PBDEs.

In their feeding study with juvenile rainbow trout, Kierkegaard et al. (1999) detected several hexa-, hepta-, octa- and nonaBDEs in liver and muscle tissues of the treated fish and some of these congeners were not measured in the food. It was not possible to determine in this study whether their presence was a result of decaBDE metabolism or the efficient adsorption of congeners initially present in trace amounts in the food.

Metabolism studies using rats (Norris et al. 1973, 1974; El Dareer et al. 1987) suggest that decaBDE has low bioaccumulation potential in mammalian species. For instance, Norris et al. (1973, 1974) dosed male and female rats with 1.0 mg of ^{14}C-labelled decaBDE as a suspension in corn oil and found that approximately 90.6% of the administered ^{14}C-labelled decaBDE was excreted in the feces within 24 h, and by 48 h,

all of the administered chemical had been excreted. Tissue accumulation studies in which rats were fed diets of decaBDE at a rate of 0.1 mg/kg body weight (bw)/d showed that bromine contents in various tissues were not significantly greater than those of the controls. The bromine content of the adipose tissue of decaBDE-dosed rats was found to be significantly increased at the $p<0.03$ level but not at the $p<0.01$ level when compared with the controls (Norris et al. 1973, 1974). Further discussion (beyond that presented in Canada 2006 or Environment Canada 2006b) respecting the El Dareer et al. (1987) study is provided below (also cited as NTP 1986).

The National Toxicology Program (NTP 1986) and El Dareer et al. (1987) evaluated the adsorption of ^{14}C-labelled decaBDE (97.9 to 99.2% pure) using F344/N rats. DecaBDE (BDE209) was administered through diet (total food consumption was 50 000 mg/d, on days 1–7 and 9–11 amended with unradiolabelled decaBDE and on day 8 with radiolabelled decaBDE). The total dose of decaBDE ranged from 3718 to 3826 mg/kg/d. The results showed that after 72 hours of exposure, from 91.3 ± 4% to 101 ± 4% of the radioactivity was recovered in the excreted feces. In a further study, the researchers fed both unlabelled and radiolabelled decaBDE (~22–25 mg/kg bw) in food (total food consumption 48 000 mg/d). The rats were fed with unradiolabelled decaBDE in food on days 1–7 and 9, or 9–10, or 9–11, and radiolabelled decaBDE in food on day 8. They determined that 82.5 to 86.4% of the radiolabelled decaBDE was recovered from the feces, and total recovery ranged from 83.2 to 89.3%. Excretion in urine accounted for about 0.01% of the administered dose of decaBDE. They detected trace levels of radioactivity in most tissues, with the highest levels found in liver, kidney, lung, skin and adipose tissue. The authors continued with an intravenous study in which rats were injected with 1.07 mg/kg ^{14}C-decaBDE and were sacrificed 72 hours after dosing. The results revealed that 74% of the dose was found in the feces and gut contents, suggesting that biliary excretion was important. The authors determined that the excreted material was mainly unchanged decaBDE (up to 40%) and three metabolites (unidentified) (up to 49.8%). In a final study, biliary excretion over 4 hours was evaluated after intravenous administration of 0.9 mg/kg of ^{14}C-decaBBDE to rats. The authors found that 7.17% of the dose appeared in the bile after 4 hours. The researchers indicated that one unidentified metabolite was observed in the bile.

Mörck et al. (2003) conducted a feeding study to investigate possible distribution pathways and metabolites of decaBDE in rats. This study involved orally dosing rats with radiolabelled decaBDE. The decaBDE substance used in the study was synthesized by a multi-step process involving the bromination of ^{14}C-labelled phenol ultimately giving ^{14}C-decaBDE (purity determined at > 98%). Before administration, the radiolabelled decaBDE was diluted in unlabelled decaBDE synthesized using the same process (also with a purity of > 98%). This study used the radioactivity associated with non-extractable residues, lipid-bound residues, water-soluble residues, phenolic metabolites and neutral metabolites/parent compound to make inferences about the uptake and metabolic transformation of decaBDE. A total of 8 “conventional” rats and 2 bile-duct-cannulated rats were dosed orally by gavage with 3 mmol/kg, 15 Ci/mol radiolabelled decaBDE in a dosing vehicle (lutrol/soya phospholipone) designed to maximize decaBDE solubility and absorption. Feces were collected for analysis from the conventional and cannulated rats at

24-hour intervals, while bile was collected and analyzed from the cannulated rats at 4, 12, 24, 48 and 72 hours post-dosing. The conventional rats were sacrificed at day 3 post-dose (n=4) and day 7 post-dose (n=3) for analysis of tissues.

Radioactivity related to decaBDE was present in the feces, bile and tissues of exposed rats as a combination of non-extractable residues, lipid-bound residues, water-soluble residues, phenolic metabolites and neutral metabolites/parent compound, indicating the potential presence of parent decaBDE and debrominated metabolites, as well as conjugated metabolites and metabolites bound to lipid or other tissues. In conventional rats, phenolic metabolites increased from ~8% to ~14% in the three days post-dose while neutral metabolites/parent compound decreased from ~29% to ~17% in the three days post-dose. Bile contained predominantly lipid-bound residues (decreasing from ~60% to ~11% in the three days post-dose) and water soluble residues (increasing from ~9% to ~62% in the three days post-dose).

The study found that approximately 90% of the single oral dose was excreted via feces. By totalling the amount of ^{14}C collected in urine and feces and subtracting that from 100%, the authors estimated that 9% remained in the body (i.e., as parent compound and metabolites). The highest concentrations of radioactivity (on a fresh weight basis) were found in adrenals, kidney, heart and liver after both 3 and 7 d. On a lipid weight basis, plasma and liver had the highest levels; levels in other tissues were low. The high absorption (relative to previous studies) of decaBDE was likely due to the use of the lutrol/soya phospholipone carrier. Ten percent of the dose excreted in feces came from bile. Of the total radioactivity excreted in feces, it was inferred that approximately 65% of the dose was eliminated as metabolites. In this study, metabolites were those characterized as non-extractable, water-soluble, lipid-bound, phenolic metabolites and parent compound/neutral metabolites. Thus, there is some uncertainty that the 65% included some proportion of bound parent compound or metabolites.

To characterize decaBDE metabolites, the authors further manipulated and analyzed the phenolic and neutral metabolites / parent compound fractions. Detection was conducted using GC/MS (ECNI). Based on their findings, the authors inferred, although they did not positively determine, the presence of debrominated, phenolic and methoxylated metabolites. Non-transformed decaBDE dominated the neutral metabolites / parent compound fraction of both feces and tissues, but small amounts (< 0.5%) of three nona-brominated metabolites were also present. Analysis of the phenolic fraction of feces and tissues also inferred the presence of hydroxylated and hydroxyl-methoxylated (i.e., guiacol-type) substances with five to seven bromine atoms which the researchers inferred were penta- to heptaBDEs.

From the metabolite characterization, the authors speculated that the potential sequence of events in metabolic transformation of decaBDE would involve debromination followed by hydroxylation of the exposed benzene ring to form an ortho catechol either directly via an arene oxide or by monohydroxylation followed by secondary oxidation of the resulting phenol. The resulting ortho catechol would then be methylated, potentially by the action of catechol-O-methyltransferase to form the observed guaiacol

(hydroxymethoxylated) metabolites. The initial debromination step would have to remove least 2 bromines from the hydroxylated/methoxylated ring but appeared to also remove bromines from the other ring.

The authors also speculated that cytochrome P450 enzymes might be responsible for the large proportion bound to residues present in the small intestine. The action of cytochrome P450 following debromination is consistent with the formation of phenols and catechols, as well as the formation of reactive intermediates that bind covalently with macromolecules, which would explain the high level of bound residues in the small intestine.

3.1.2 New Evidence of Debromination *in Vivo*

Fish

Stapleton et al. (2006) exposed 45 juvenile rainbow trout to spiked food containing decaBDE (Cambridge Isotopes Laboratories, > 98% pure) for a period of 5 months. The debrominated products accounted for approximately 73% of the total PBDE burden in the carcasses. These products were identified as primarily BDE208 (nonaBDE) and BDE202 and -201 (octaBDEs) with a small fraction of BDE188 (heptaBDE). NonaBDEs (primarily BDE207 and -208) accounted for 26% of the burden in serum with only minor amounts of octaBDEs present, and untransformed decaBDE accounting for the remainder (approximately 68%). In the liver, the burden was primarily decaBDE with only a small fraction of lower brominated PBDEs (primarily nonaBDEs). The predominance of BDE202 as a product of decaBDE debromination was similar between rainbow trout (observed here) and carp from a previous study, and has been viewed as a potential marker for transformation as this congener has apparently not been measured in the commercial DecaBDE or OctaBDE products. The researchers also found that concentrations of the hepta- and octaBDE congeners increased in concentration over the duration of the exposure. Based on the total body burden of hepta- to decaBDE, uptake of decaBDE was estimated at 3.2% of total dose. Since no measurement was made of other metabolites, the researchers suggest that the estimate of uptake could be higher.

Stapleton et al. (2006) also conducted an *in vitro* study using rainbow trout liver microsome fractions to confirm the metabolic capacity of rainbow trout tissues and potential metabolites. This was partnered with an *in vitro* study using carp liver microsome fractions that was also undertaken to confirm the metabolic capacity of carp demonstrated by Stapleton et al. (2004). In this study, liver microsomes were prepared from three fish and incubated for 1 and 24 h at 25°C. Analysis was conducted for PBDE congeners; however, recovery was low for the 1 h incubation (< 60%) in comparison to the 24 h incubation (> 80%). The study also had a control consisting of a boiled microsome fraction. Both carp and rainbow trout liver microsomes were observed to transform decaBDE even after the 1-h incubation. The carp liver microsomes debrominated decaBDE to hexa-, hepta-, octa- and nonaBDEs, although very little production of the nonaBDE congeners was observed (mirroring the *in vivo* experiment) in both 1- and 24-h incubations. After 24 h, the carp microsomes transformed 65% of the decaBDE mass (30% was transformed to hexaBDE congeners). The liver microsomes of

rainbow trout transformed as much as 22% of decaBDE to octa- and nonaBDEs in the 24-h incubation. The controls showed no transformation of decaBDE.

The authors concluded that their results supported the hypothesis that deiodinase enzymes were catalyzing debromination of decaBDE; however, they also cautioned that it was not possible to rule out the concurrent or alternative action of P450 enzymes. Stapleton's work shows that removal of bromine atoms occurs preferentially from the meta- or para-substituted positions (personal communication from HM Stapleton to Environment Canada, January 2008; unreferenced).

Tomy et al. (2004) studied the uptake, by juvenile lake trout, of 12 tetra- to heptaBDEs plus decaBDE from spiked commercial fish food. Three lower brominated PBDE congeners (unknown penta- and hexaBDE, and BDE140) appeared to be bioformed in the exposed fish. The authors hypothesized that debromination of decaBDE was a potential explanation since they were not specifically dosed to the organisms. However, it should be noted that each of the PBDE congeners dosed in this study had purities of > 96%, and thus there is uncertainty regarding whether these congeners were in the dosed material as impurities. They suggested that the structural similarity of BDEs to thyroxine (T4) could mean that deiodinase enzymes were debrominating higher brominated PBDEs to lower brominated PBDEs. The authors concluded that the degree biotransformation, especially for decaBDE, was likely to vary considerably between species, leading to high potential interspecies variability in bioaccumulation.

La Guardia et al. (2007) examined the potential for *in vivo* debromination of decaBDE in aquatic organisms inhabiting the receiving environment of a WWTP located in Roxboro, North Carolina, which, based on releases reported by industry to the U.S. EPA's Toxics Release Inventory, was determined to receive wastewater from a large plastics manufacturing facility. The PBDE congener profile was tracked from the WWTP effluent to the receiving environment sediments and to biota in order to evaluate whether significant debromination was occurring. In 2002, samples of wastewater sludge, sediments and biota (sunfish, creek chub and crayfish) were collected. Then in fall 2005, samples of wastewater sludge, sediments and biota (sunfish only) were collected. Aquatic biota sampling involved the use of minnow traps at a location 15 m downstream of the WWTP outfall. All samples were extracted and purified using size-exclusion chromatography analyzed for PBDEs GC/MS in ECNI mode and EI mode.

A total of 23 PBDE congeners were detected in the biota samples. Of these, decaBDE was only detected in 2002 samples of sunfish (2880 µg/kg lipid) and crayfish (21 600 µg/kg lipid). The much higher concentration in crayfish was attributed to the sediment-association of this species and the authors speculated that crayfish could form a link from sediments to pelagic organisms. The authors also speculated that the lack of detected decaBDE concentrations in chub could be due to an enhanced ability of this species to metabolize decaBDE. Chub are closely related to carp, which have previously been demonstrated to have an enhanced capability to debrominate decaBDE (Stapleton et al. 2004). Two octa- and three heptaBDE congeners which were not detected in sludge and sediments were found in chub tissues, suggesting bioformation of these homologues.

Based on these findings, the authors concluded that decaBDE is bioavailable in natural environments and could undergo metabolic debromination in the field, resulting in bioformation of lower brominated PBDEs.

Lebeuf et al. (2006) examined the effects of decaBDE and PCB126 on hepatic concentrations of PBDEs and methoxy-PBDEs in Atlantic tomcod. The decaBDE used in their experiment (DE-83R, Great Lakes Chemical Corp.) consisted of greater than 96% BDE209, with BDE153, -183 and -203 detected in amounts of between 0.00024 and 0.034% on a mass basis. Further characterization revealed that as many as seven nonspecified PBDEs were qualitatively detected (including four heptaBDEs and three octaBDEs), in addition to the three nonaBDEs. The fish used in the study were captured in the St. Lawrence estuary in 2001 and were acclimated to laboratory conditions for about 6 months. The fish were fed twice a week with capelin until the beginning of April, and then with rainbow smelts until the end of the study. After the acclimation period, 200 fish were randomly distributed in groups of 25 fish and placed into eight 500-L fibreglass tanks. On day 0, fish from half the tanks were anaesthetized in water. Half of these fish (i.e., composing 4 tanks) were injected interaperitoneally with a dose of PCB126, and the other half were injected with corn oil alone. The PCB126 was injected to evaluate the impact of cytochrome P4501A (CYP1A) induction on the biotransformation of injected PBDEs contained in the decaBDE commercial product administered to the fish. The fish from two of the four tanks that had received PCB126 and the fish from two of the four tanks that had received corn oil alone were injected with decaBDE (dose 400 ng/g fish; fish from the remaining tanks received a dose of corn oil alone) after 21 d.

Fish were sampled and analyzed for decaBDE and potential transformation products after seven weeks following decaBDE administration. The study found BDE209, -208, -207, -206, -203 and three unidentified octaBDEs in the liver of the fish. All these congeners were essentially absent in the control fish. These were also measured in the decaBDE product administered in the experiment, and thus, their presence cannot be attributed exclusively to biotransformation. Also, the presence of these substances could have been due to thermal degradation of the decaBDE product during analysis, or due to transformation in the tank due to fecal egestion of decaBDE. Despite an increase in EROD activity in the liver of tomcod dosed with PCB126 and decaBDE compared to decaBDE alone, no further increases of PBDE hepatic concentrations were observed. However, depleted concentrations of BDE17 and 6-methoxy-BDE47 (both were measured in the control fish) were found in the fish injected with decaBDE compared to control fish. The researchers attributed this to activated hepatic metabolic enzymes other than CYP1A. Fish with the injected PCB126 showed an even more significant depletion of BDE17 than those with decaBDE treatment and significantly lower concentrations of BDE203. While the fish in this study only exhibited limited capacity to metabolize decaBDE, the study demonstrates the importance of methodologies to evaluate the potential sources of substances detected in organisms following administration of decaBDE products.

The finding of Lebeuf et al. (2006) that CYP1A appears to play a limited role in the transformation of PBDEs is consistent with that of Valters et al. (2005), who found

limited evidence of cytochrome P450 metabolism in common carp plasma, as hydroxylated PBDEs represented only 0.8% of the fraction of the total PBDE compound. Benedict at al. (2007) also concluded (at least based on their study using BDE99) that it is doubtful that CYPs are responsible for debromination of BDE99. In their evaluation of BDE99 debromination in carp microflora and microsomes, they concluded that an endogenous process that occurs with approximately equal activities in the intestine and liver microsomes appears responsible for debromination of BDE99 to BDE47. They also showed that this process can be inhibited by the presence of reverse thyronine. The exact metabolic mechanism could not be fully elucidated from their study, but they noted that their work suggests that debromination may involve thyroid hormone deiodinases.

Nyholm et al. (2009) used zebrafish (*Danio rerio*) as a vertebrate model organism to study the uptake, metabolite formation and elimination of 11 structurally diverse BFRs, including BDE28, BDE183 and BDE209 (purity not reported). Solutions containing a mixture of BFRs were made from stock solutions of individual BFRs in iso-octane or tetrahydrofuran, and evaporated almost to dryness. The residues were taken up in ethanol. Resulting mixed BFR solutions were added to a chironomid feed, giving nominal concentrations of 1 and 100 nmol/g of each molecular species on a dry weight basis. A control feed was made with ethanol only. Zebrafish were fed with BFR-treated or control chironomid feed for a 42-d exposure period and then with untreated feed for a 14-d elimination period. Samples were extracted and analyzed by GC/MS.

Zebrafish exposed to high-dose feed (100 nmol/g) for 42 d had detectable BDE209 concentrations after the 2-week elimination period, but concentrations were tenfold lower than those of BDE 28 (which was determined to have the highest concentration among BFRs analyzed). Uptake efficiency was lowest for BDE209 (< 1%) compared to other BFRs studied, and elimination half life was estimated at 6.5 d. No relationship was found between the log K_{ow} values of the BFRs and the levels determined in fish. The study detected several compounds in fish tissues that may have been metabolites of the dosed BFRs. Notably, several hexaBDEs were tentatively identified by full-scan GC/MS and these compounds may have been debromination products of BDE183 or BDE209; however, one cannot discount the possibility that they were impurities in the material dosed to the organisms. No further discussion of potential debromination of BDE209 was mentioned in this study.

Terrestrial Organisms

Sandholm et al. (2003) conducted a study of the bioavailability, absorption and metabolic transformation of decaBDE in Sprague-Dawley rats. A total of 36 male rats weighing 200–220 g each were dosed with decaBDE at a dosage rate of 2 µmol/kg and the bioavailability, elimination and metabolite formation in plasma were monitored over a 6-d period. One subset of 18 rats was dosed orally by gavage while a second subset of 18 rats was dosed intravenously. Blood plasma was monitored at regular intervals (at 1, 3, 6, 24, 48, 72, 96, 120 and 144 h) over the 6-d monitoring period. In addition, plasma samples collected from a separate 7-d single-dose study with radiolabelled decaBDE (Mörck et al. 2003) were analyzed to determine the total radioactivity associated with neutral and phenolic fractions in plasma of rats exposed to decaBDE.

The oral bioavailability was estimated to be approximately 26%, although the sampling methodology may have resulted in an artificially high determination of bioavailability. Oral bioavailability was defined as the fraction of administered parent compound reaching systemic circulation. The authors indicated that the 26% level was much higher than that reported in early studies with decaBDE (i.e., by Norris et al. 1975; NTP 1986; El Dareer et al. 1987). Based on the analysis of plasma samples from the 7-d study with radiolabelled decaBDE, the phenolic radioactivity in plasma at days 3 and 7 was 4 times that of the neutral fraction, suggesting significant oxidative metabolism of neutral decaBDE, debrominated metabolites, and higher exposure to phenolic metabolites than to neutral parent/metabolite compounds. The presence of phenolic metabolites compared with neutral compounds indicated that the adsorption of BDE209 would have been greater than their calculated level of 26%. The elimination of decaBDE from plasma was multi-phased with terminal (i.e., longest) half-lives of 51 h (oral exposure) and 58 h (intravenous exposure).

The neutral compounds in plasma were identified as decaBDE (> 99.5%) as well as three nonaBDEs (< 0.5%). In addition to the neutral compounds, 13 phenolic metabolites were determined in plasma with the major metabolites inferred to consist of a hydroxyl-octaBDE, a hydroxyl-nonaBDE and a guaiacol-type hydroxymethoxy-hexaBDE. Based on the observed parent and metabolite composition, the following general sequence for metabolic transformation of decaBDE was proposed:

1. Reductive debromination of decaBDE
2. Subsequent oxidation to phenolic metabolites
3. Formation of guaiacol-type metabolites via an arene oxide and dihydrodiol, or via two sequential oxidation steps followed by methylation

The authors also described how hydroxyl-octaBDE and hydroxyl-nonaBDE could have affinity for transthyretin (TTR), which normally functions as a transport protein for thyroxine in plasma. The binding to TTR could explain the high fraction of phenolic metabolites observed in plasma and may cause the phenolic metabolites to persist as bound residues in plasma. The binding of phenolic metabolites could disrupt thyroxine transport in blood, resulting in adverse hormonal effects. This behaviour was reported by the authors to be similar to that reported for PCB metabolites.

Hakk and Letcher (2003) summarized the findings of Orn and Klasson-Wehler (1998), Hakk et al. (2002) and Mörck and Klasson-Wehler (2001) with respect to the fecal and biliary metabolites formed by rats dosed with decaBDE. They concluded that decaBDE underwent oxidative debromination to guiacols (hydroxyl-methoxylated BDEs), hydroxylated BDEs and nonaBDEs. Additional discussions and conclusions by Hakk and Letcher (2003) included the following points:

- A likely metabolic pathway between decaBDE and hydroxylated or hydroxymethoxylated BDEs is debromination as a first step, followed by the formation of an arene oxide, and subsequent hydroxylation and/or methoxylation.

- PBDE mixtures are inducers of Phase I and Phase II metabolism, suggesting that these metabolic pathways are also important for PBDEs.
- Hydroxylated BDEs are likely to compete with thyroxine for binding to TTR.
- Guiacol metabolites could further oxidize to quinones, which are highly reactive and would bind to cellular macromolecules. Thus, bound residues may make up a significant proportion of metabolite body burdens.

Huwe and Smith (2007a, 2007b) examined the dietary accumulation, debromination and elimination of decaBDE in rats. The burden in body tissues and feces of one nonaBDE congener (BDE207) and two octaBDE congeners (BDE201 and BDE197) were higher than could be explained by the total dose of each congener associated with their background concentrations in rat feed. The authors concluded that the elevated burdens were the result of reductive debromination of decaBDE. The formation of BDE197 and -207 from BDE209 results from meta debromination(s), while para and meta debrominations are responsible for BDE201 formation. The authors proposed that the action of deiodinase enzymes may be involved in the meta debromination of decaBDE. These deiodinase enzymes are normally involved with the meta dehalogenation of the thyroid hormone thyroxine and are present in many tissues of the body.

However, the total burden of BDE207, -201 and -197 accounted for only 1% of the total decaBDE dose, suggesting that either debromination was not the primary metabolic pathway or that the debrominated products rapidly underwent further metabolism. Overall, 45% of the dosed decaBDE was unaccounted for in rat tissues and feces and the authors speculated that the formation of bound and/or hydroxylated metabolites that were not included in their analysis was a likely explanation for the incomplete mass balance of decaBDE.

Kierkegaard et al. (2007) reported the findings of a 3-month feeding study with two dairy cows. The congener profile in adipose and organ tissue differed from the profile in silage and it appeared that BDE207, -197, -196 and -182 accounted for a much higher proportion of the total burden in tissue than in silage. Tracing of potential degradation of decaBDE during extraction and cleanup with radiolabelled standards ruled out photolytic debromination as a source of these hepta- to nonaBDEs during extraction/cleanup. The authors also discounted higher dietary absorption of these congeners as an explanation for the observed differences between feed and tissues since enhanced absorption was not apparent for similar congeners (i.e., BDE183 compared to BDE182). Therefore, it was concluded that the observed increase of BDE207, -197, -196 and -182 was likely due to reductive debromination of decaBDE. The authors proposed the following two potential transformation pathways for decaBDE:

1. Ortho debromination to BDE206, followed by meta debromination to BDE196, followed by meta debromination to BDE182
2. Meta debromination to BDE207, followed by meta debromination to BDE197

In a study by Riu et al. (2008), Wistar rats were dosed orally with 99.8% pure [^{14}C]-radiolabelled decaBDE dispersed in peanut oil, gestational days 16 to 19. Urine and feces

were collected daily over this 4-d period. The animals were sacrificed on day 20 of gestation (24 h after the last dose of ^{14}C-decaBDE) and the amounts of radioactivity, corresponding decaBDE levels, and metabolic profiles were determined in various organs and tissues. Extraction and analytical methods were developed to achieve the radio-chromatographic separation of decaBDE metabolites formed *in vivo*.

More than 19% of the 4-d administered dose was recovered in the body (tissues + carcass) of the rats, demonstrating relatively high absorption of decaBDE as well as efficient distribution to tissues. The fecal route was the main excretion pathway, accounting for approximately 66.3% of the administered dose, whereas urinary excretion was very low (accounting for approximately 0.11% of the total dose). In feces extracts, approximately 97% of the radiolabel was found to be unchanged decaBDE. Characterization of the metabolites in feces and tissues, conducted using liquid chromatography/mass spectrometry (LC/MS) nuclear magnetic resonance (NMR), identified all 3 nonaBDEs, an unidentified octaBDE and a hydroxylated octaBDE derivative. Together (excreta + tissues + carcass), metabolites accounted for approximately 7% of the total radioactivity administered to rats. In addition, a very limited amount of an additional metabolite was also detected (not enough for structural identification); however, it could not be ruled out that the compound corresponded to a hydroxylated heptaBDE. Additional unidentified polar metabolites were detected in urine, with similar compounds found in the contents of the intestine, but not the feces.

Riu et al. (2008) found decaBDE residues to be highest in the adrenals, ovaries, liver, kidney and heart, and their calculated values were significantly higher than those of plasma. Liver was reported as the target tissue for decaBDE (11 mg/kg, corresponding to 6.5% of the administered dose). However, the highest residual levels were found in endocrine glands: the adrenals (33 mg/kg) and ovaries (16 mg/kg). Both decaBDE and its metabolites were reported to cross the placental barrier in rats following oral administration (approximately 0.5% of the total dose was found in the fetuses).

Van den Steen et al. (2007) investigated the accumulation in tissues and debromination of BDE209 in European starlings (*Sturnus vulgaris*), using silastic tube implants as modes of exposure. The implants were inserted under the skin by a small incision along the ribs. The implants contained BDE209 (purity not given, but sourced from Wellington Laboratories, Guelph, Canada) dissolved in iso-octane and mixed in peanut oil. The iso-octane was evaporated off by heating the oil mixture to a constant weight. After preparation, there were no detectable levels of other PBDE congeners in the oil solution. In total, seven adult male starlings were used in the study, with four receiving an implanted dose of 46.8 ± 2.2 μg BDE209, and with three (the control group) receiving an implant with only peanut oil. During the 76-d exposure period, blood samples were taken every 3–7 d. The birds were then were euthanized and pectoral muscle and liver were excised. Blanks consisting of water instead of blood were included in each sample batch and their PBDE concentrations were subtracted from values determined for biotic samples.

Before implantation, BDE209 concentrations in blood were below the limit of quantitation (i.e., 0.8 and 0.5 µg/L for BDE209 and other BDEs, respectively). Accumulated BDE209 peaked on day 10 (16 ± 4.1 µg/L) in the blood of the exposed starlings. After this peak, there was a decline to 3.3 ± 0.4 µg/L in blood at the end of the 76-d exposure period, which suggested elimination. Tissue concentrations were below the limit of quantitation in the control group (5.6 and 2.9 µg/kg lipid in muscle and liver, respectively). In the exposed group, the muscle concentrations were about two times those in liver (i.e., 461 and 340 µg/kg lipid in muscle, compared with 269 and 337 µg/kg lipid in liver). Their study found that various PBDEs were present in both the control and exposed group muscle and liver tissues, with the differences being most pronounced for the nona- (BDE206, -207, and -208) and octaBDEs (BDE196 and -197). The octaBDEs BDE203 and -205 did not differ much between the groups. The authors concluded that this study provided evidence for the transformation of BDE209 to lower brominated PBDEs in songbirds. The significance of these findings is difficult to interpret since the composition of the test material was not provided, and thus the accumulation of nona- and octaBDEs from the test material cannot be ruled out.

Chen et al. (2008) measured BDE209 in all 114 eggs in a peregrine falcon (*Falco peregrinus*) egg study in the northeastern United States. Eggs were collected between 1996 and 2006 (excluding 1997 and 1998), and concentrations ranged from 1.4 to 420 ng/g ww. The authors noted that in addition to BDE209, eight nona- and octaBDE congeners frequently detected in their peregrine falcon study have rarely been reported in wildlife. Together with BDE209, these congeners composed 16–57% of total PBDEs in urban eggs and 4.9–53% in rural eggs. The researchers noted that the concentration of BDE201 was not in proportion with this congener's presence in the OctaBDE commercial mixture and this was suggestive that it was formed *in vivo* from higher brominated PBDEs. In addition, they detected BDE202 in 107 of 114 eggs. Since BDE202 is not known to be found in OctaBDE or DecaBDE mixtures, they also speculated that its presence could be attributed to metabolic transformation of higher brominated PBDEs.

3.1.3 Conceptual Models of Metabolism *in Vivo*

This section summarizes pathways of metabolism based on those proposed in reviewed studies. This analysis respects that some studies contain uncertainties (e.g., potential impurities in the test substance administered to organisms, lack of chemical specificity in analyses of metabolites). Based on the reviewed literature, the amount of decaBDE which apparently may have been transformed to lesser-brominated PBDEs like nona-, octa- and heptaBDEs *in vivo* appears to be generally low, on the order of a small percent of the total decaBDE dosed. However, some studies have made inferences respecting higher levels of metabolism (potentially up to 45 to 65% of the total dose of decaBDE), which could be a result of metabolic transformation products including hydroxylated and hydroxymethoxylated PBDEs and/or the formation of bound residues.

Fish

Based on currently available studies and information reviewed in this report by Stapleton et al. (2004, 2006), personal communications from HM Stapleton to Environment Canada (January 2008 and July 2009) (unreferenced), Kierkegaard et al. (1999) and Tomy et al. (2004) (respecting the uncertainties in these latter two studies), the following pathways for metabolic transformation of decaBDE are deduced:

1. DecaBDE (i.e., BDE209) is debrominated in fish as a first step in metabolism, producing at least debrominated hepta- to nonaBDEs, but also potentially penta- and hexaBDEs.
2. Thyroid hormone deiodinase enzymes which normally remove iodine from thyroxine appear to be involved in this debromination pathway.
3. Bromine has been observed to be preferentially removed from the meta and para positions.

Mammals

According to the discussion and findings of Sandholm et al. (2003), Mörck et al. (2003), Hakk and Letcher (2003), Huwe and Smith (2007a, 2007b) and Kierkegaard et al. (2007) the following pathways of transformation are deduced:

1. Reductive debromination to nona-, octa- and heptaBDEs is the likely first step in the metabolism of decaBDE.
2. Similar to fish, debromination may be the result of action by deiodinase enzymes.
3. The debrominated neutral metabolites then appear to undergo hydroxylation to potentially form phenols or catechols, potentially via an arene oxide. This could involve the action of cytochrome P450 enzymes.
4. The potentially formed hydroxylated BDEs may compete with thyroxine for binding to TTR, a thyroxine transport protein present in blood serum.
5. The catechols may then be methylated, potentially by the action of catechol-O-methyltransferase, to form the observed guaicols.
6. The guiacol metabolites could further oxidize to quinones, which are highly reactive and would bind to cellular macromolecules.
7. The reactive intermediates would also be subject to rapid conjugation via Phase II metabolic processes, leading to water-soluble metabolites which would be excreted via bile and feces, as was observed in conventional and cannulated rats.

3.1.4 Implications for Bioaccumulation Assessment

The existing evidence for the debromination and metabolism of decaBDE suggests metabolic pathways which lead to the formation of the following:

1. Lower brominated PBDEs (down to heptaBDE congeners in mammals and potentially down to pentaBDE congeners in fish)
2. Possibly hydroxylated BDEs (only shown in mammals)
3. Possibly hydroxymethoxylated BDEs (only shown in mammals)
4. Unknown products (potential formation in both mammals and fish)

In order to evaluate whether these metabolites have the potential to bioaccumulate, or biomagnify in food webs, model predictions were made using the BAF–QSAR model and the terrestrial biomagnifications model of Gobas et al. (2003). It is acknowledged that in some cases some of the lower brominated PBDEs may have field or laboratory measurements of BAFs or BMFs. However, field and laboratory bioaccumulation data for the bioformed hydroxylated and hydroxymethoxylated BDE metabolites are generally lacking, and application of the models for all types of metabolites was seen as a consistent way to inform our knowledge regarding the bioaccumulation and biomagnification potential of these metabolites. The PBDE screening assessment supporting working document (Environment Canada 2006b) provides a summary of the measured aquatic BCFs and BAFs for the PentaBDE and OctaBDE commercial products. These studies showed that BCFs and BAFs exceed 5000 for tetra-, penta- and hexaBDEs (this is consistent with the findings of the modelling exercise conducted for this review).

For making BAF predictions with the BAF–QSAR model, the log K_{ow} of each metabolite was based on reported measurements wherever possible. In the absence of reported measurements, log K_{ow} was estimated using the Experimental Value Adjustment (EVA) method in the KOWWIN estimation program. The EVA uses a reference logK_{ow} from another structurally close substance (in this case decaBDE) and corrects predictions for metabolites based on structural differences with decaBDE. For each metabolite, two prediction scenarios were conducted: the first with no correction for metabolic transformation, and the second corrected for metabolism based on the laboratory observations of Stapleton et al. (2004) and Tomy et al. (2004). Where it was not possible to estimate k_M for lower brominated PBDEs from laboratory data, reasonable approximations were made that took into consideration rates of metabolism for similar congeners. For hydroxylated and hydroxymethoxylated BDEs, there was no information that would allow for estimation of k_M, and a value of 0.026/day (i.e., equal to that estimated for decaBDE) normalized to the body weight of the model's middle trophic level fish at 15^{o}C (~0.02/day), was chosen for illustrative purposes. These metabolites are expected to undergo further metabolism; however, the actual rates of metabolism are unknown. The chosen value and resulting BAFs represent reasonable hypothetical predictions. Further details of log K_{ow} and k_M estimates are provided in Appendix D.

Figure 3-1 provides frequency histograms of the pooled BAF predictions for all potential metabolites and for the middle trophic level. In the absence of metabolism, the BAFs of all metabolites are predicted to exceed 5000. However, with consideration given to metabolic transformation (a more realistic scenario), the predicted BAFs range from 295 to approximately 6 000 000. Based on the fit of a normal distribution to the corrected BAF histogram, it is estimated that approximately 74% of the metabolite BAF predictions exceed 5000. The metabolite groups illustrated to exceed the BAF of 5000 include hydroxymethoxy-penta- to nonaBDEs, hydroxy-hexaBDEs, and penta- to octaBDEs. The remaining 26% of BAF predictions that are below 5000 consist of hydroxyl-octa- to nonaBDEs and nonaBDEs. The large proportion of high BAFs, even when metabolic transformation is considered, suggests that many decaBDE metabolites could potentially be bioaccumulative.

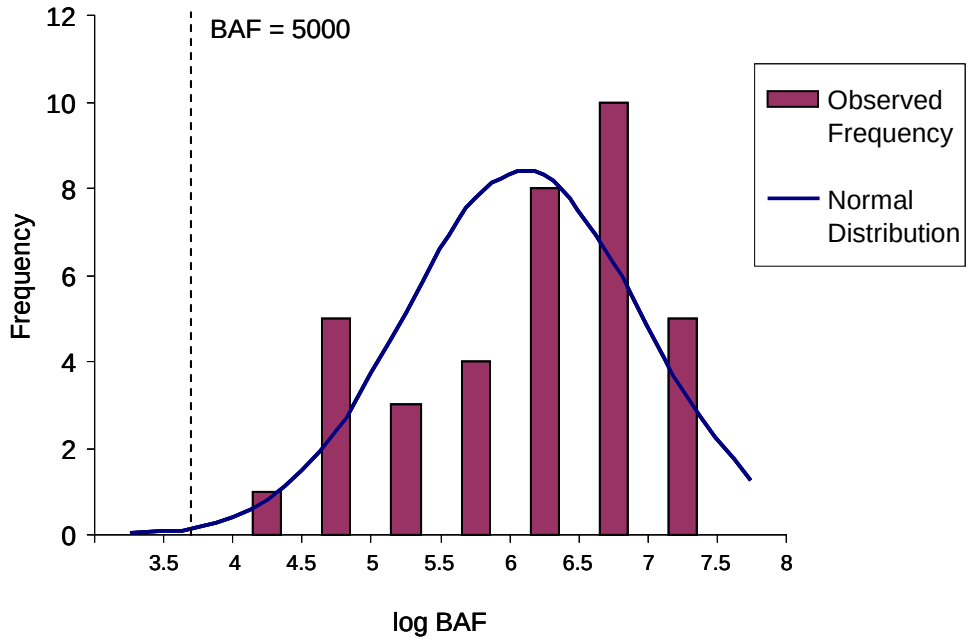
For the hydroxylated and hydroxymethoxy metabolites the actual rate of metabolism is unknown and therefore the BAF predictions are uncertain. If the secondary transformation of these metabolites occurred much faster than approximately 0.02/d then it is possible that the BAFs would be much lower. At the same time, it is possible that in certain species the enzymes responsible for Phase II metabolism might be less developed or lacking, resulting in persistence of these metabolites in tissues and much higher BAFs that approach those of the non-corrected values.

For the lower brominated PBDE metabolites, the metabolism-corrected BAF predictions are considered to have higher certainty than the predictions for the substituted forms since the k_M values chosen for the model were based on laboratory observations in most cases (the one exception was for nonaBDEs, which were assumed to be metabolized at a rate based on that determined for decaBDE). It should be noted that the half-lives chosen for model inputs were often relatively short (metabolism is relatively fast) compared to the maximum half-lives observed in lab studies (refer to Appendix D). Therefore the predicted BAFs are less conservative (i.e., lower) than if the longest observed half-lives were chosen. Despite the use of less conservative half-lives, the predicted BAFs for penta- to octaBDEs exceeded 5000.

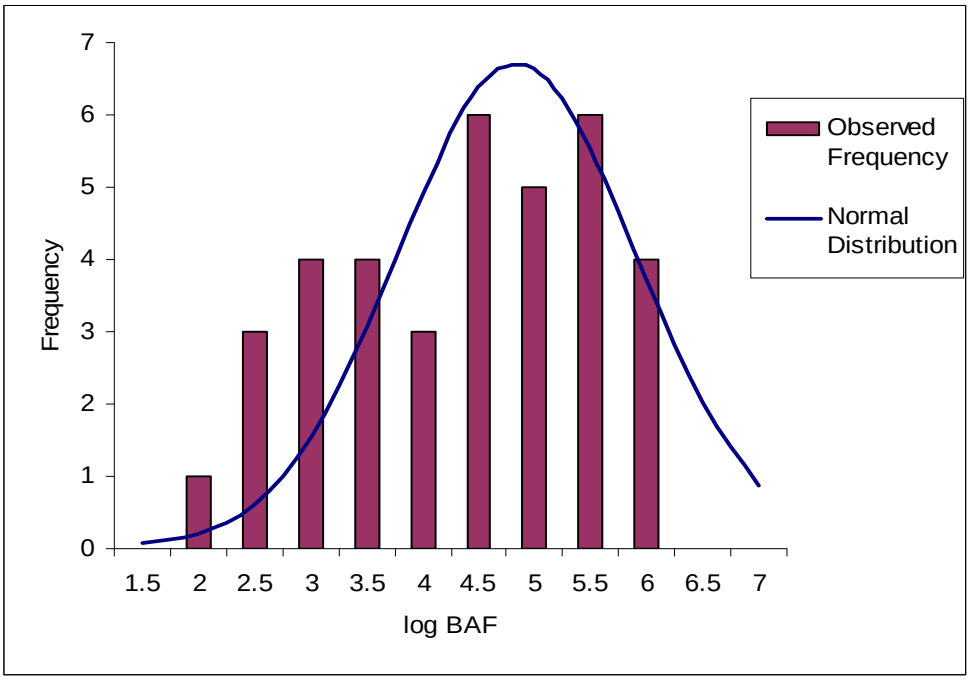
It is acknowledged that decaBDE metabolites are formed in tissues and accumulation in tissue from water-phase exposure (as quantified by the BAF) may be of limited relevance for decaBDE. However, these BAF predictions are still useful for providing a perspective on the bioaccumulation potential of the metabolite chemicals. The model results raise concerns that many of the metabolites formed in fish as a result of the accumulation and metabolism of decaBDE are potentially bioaccumulative and may be prone to biomagnification in food webs, potentially resulting in increased exposure and risk to upper trophic level organisms.

Figure 3-1: Combined frequency distributions of predicted BAFs for metabolites of decaBDE (predictions for middle trophic level)

(a) No Correction for Metabolism



(b) Corrected for Metabolism



BMF predictions for decaBDE metabolites in wolves were made using a spreadsheet version of the Gobas et al. (2003) model. Log K_{ow} values were chosen based on the rationale described in Appendix D, whereas log K_{oa} for each metabolite was estimated using KOAWIN and by inputting corrected logK_{ow} values first. Two prediction scenarios were modelled: the first did not consider metabolic transformation, while the second considered metabolism based on the laboratory observations of Huwe and Smith (2007a, 2007b). Where it was not possible to estimate k_M for lower brominated PBDEs from laboratory data, reasonable approximations were made by considering rates of metabolism for similar congeners. For hydroxylated and hydroxymethoxylated BDEs, there was no information which would allow for the estimation of k_M and thus a value of 0.004/d was selected for illustrative purposes. This value corresponds to the longest observed half-life for decaBDE, nonaBDEs and octaBDEs derived by Huwe and Smith (2007a, 2007b), normalized to the body weight of wolf used in the Gobas model. The hydroxylated and hydroxymethoxylated BDE metabolites are expected to undergo further metabolism; however, the actual rates are unknown.

Calculation of BMFs for the BDEs were corrected for dietary assimilation efficiency according to the E_D vs. log K_{ow} relationship described in Kelly et al. (2004) for humans as a representative homeotherm. In the absence of metabolism, the BMFs of all metabolites are predicted to be very high, with BMFs ranging from ~4 to ~89 for chemicals within the ranges of log K_{ow} and log K_{oa} estimated for decaBDE metabolites. However, with consideration given to metabolic transformation (a more realistic scenario), the predicted BMFs are lower, ranging from 5 to 6. All metabolites' BMF values were > 1, primarily due to a relatively high dietary assimilation efficiency assumed for these substances.

In mammals, the actual rate of metabolism is unknown for the hydroxylated and hydroxymethoxy metabolites and therefore the BMF predictions are uncertain and may be high. If the secondary transformation of these metabolites occurs much faster than 0.012/d then it is possible that the BMF would be lower and possibly not exceed 1. At the same time, it is possible that in certain species, the enzymes responsible for Phase II metabolism might be less developed or lacking, resulting in persistence of these metabolites and much higher BMFs that approach the BMF values estimated without consideration given to metabolic transformation.

Because these decaBDE metabolites are formed in tissues, the BMF, which quantifies the potential for chemical transfer from prey tissues to predator tissues, is seen as an accurate indicator of bioaccumulation and biomagnification potential. The predicted BMFs, which exceed 1 for all metabolites, raise concerns that the metabolites formed in mammals as a result of the accumulation and metabolism of decaBDE have the potential to biomagnify in food webs, potentially resulting in increased exposure and risk to upper trophic level organisms.

3.2 Debromination in the Environment

Abiotic Degradation

The following bullets summarize the evidence regarding abiotic debromination of decaBDE which was considered in the screening assessment for PBDEs (Environment Canada 2006a, Environment Canada 2006b).

- Norris et al. (1973, 1974) exposed solid decaBDE (98% decaBDE and 2% nonaBDE) to sunlight in water. The bromine concentration in water increased during the experiment, suggesting that photodegradation was occurring. However, it was not possible to determine the relevance of this study to photolysis in the environment.

- Watanabe and Tatsukawa (1987) carried out photolysis experiments with decaBDE (97% decaBDE and 3% nonaBDE) in a mixture of hexane, benzene and acetone (8:1:1). After 16 h of exposure to ultraviolet (UV) light, decaBDE was found to debrominate primarily to tri- to octaBDEs, but brominated furans with 1 to 6 bromine atoms were also formed. Under sunlight, similar patterns of transformation were determined.

- Söderström et al. (2004) undertook photodegradation studies in which decaBDE (Dow FR-300 BA; exact composition was not provided, but contained traces of octa- and nonaBDEs) was dissolved in toluene and applied as a thin layer to silica gel, sand, soil or sediment. The toluene was evaporated off in the dark. Exposure was to natural sunlight and to UV light generated by three mercury lamps. Commercial DecaBDE applied to solid matrices was observed to transform following a pathway of reductive debromination with products including primarily hexa- to nona-BDEs with some occurrence of penta and tetra products (i.e., BDEs 47, -100, -99, -119). Only trace amounts of lower brominated PBDEs were formed. Photodegradation occurred fastest in toluene and silica gel (estimated half-life of less than 15 min), followed by sand (half-life of 12–13 h), sediment (half-life of 30–60 h) and soil (half-life of 150–200 h). In addition to lower brominated PBDEs, tetra- and pentabrominated dibenzofurans (tetra- and pentaBDFs) were detected as phototransformation products of decaBDE adsorbed to sand, sediment and soil. Based on the relative amounts of debromination products in the samples, the authors concluded that the origin of BDE47, -99, -100, -153 and -183, which are common in environmental samples, was "probably primarily from emissions of technical PentaBDE products and possibly from other degradation pathways of decaBDE."

- Jafvert and Hua (2001) conducted multiple photodegradation studies of decaBDE (98% purity) on hydrated surfaces of glass and silica sand particles, humic acid-coated silica particles, and glass surfaces in contact with aqueous solutions. The studies were conducted under simulated and natural sunlight. The amount of decaBDE remaining after light exposure for 60–72 h ranged from 29 to 88% depending on the type of surface, test conditions and light used. The presence of unidentified compounds was inferred in some of the treatments. Further analysis of

the unidentified compounds was largely inconclusive, although there was some evidence of the formation of hexa- to nonaBDEs.

- Hua et al. (2003) precipitated decaBDE onto surfaces (quartz glass, silica particles and humic acid-coated silica particles), hydrated them with reagent-grade water, and irradiated the system with artificial sunlight or with natural sunlight. After 60–72 h of irradiation, 29–56% of the initial decaBDE remained on the quartz surface, depending on the type of light and wavelength used. Addition of humic acid slowed the rate of decaBDE decay. Small amounts of nona- and octaBDEs were likely formed as degradation by-products. BDE47 and -99 were not formed at detectable levels of the analytical method used (high-performance liquid chromatography, HPLC).

- Palm et al. (2003) investigated the photodegradation of decaBDE dispersed in toluene, dichloromethane or a solvent mixture of hexane:benzene:acetone (8:1:1) and then irradiated with filtered (300 nm) artificial xenon lamps. A half-life of approximately 30 minutes was determined for all the test systems used. Reductive debromination was found to occur, first with three isomers of nonaBDE formed, then six isomers of octaBDE, then to several isomers of heptaBDE, and finally to trace amounts of hexaBDE. Seventy-five percent of decaBDE degradation followed a pathway of debromination, while the products of the remaining 25% could not be determined. In a follow-up study conducted under natural sunlight over 2 d, the researchers found complete disappearance of decaBDE, with nona- to pentaBDEs formed. A similar pattern was observed when decaBDE was dispersed in tetrahydrofuran (THF) and exposed to artificial sunlight for 84 h; however, PBDEs with fewer than 5 bromine atoms per molecule were also formed.

- Palm et al. (2003) also investigated the photochemical transformation of decaBDE in an aerosol smog chamber in which decaBDE was adsorbed to silicon dioxide at a concentration of about 1% by weight. The particles were suspended in water, atomized, dried and dispersed as an aerosol in a smog chamber. The conditions used were assumed to maximize the degradation potential of decaBDE. Although the conditions may not replicate atmospheric transport, the particle size used (diameter of approximately 1µm in the aerosol) can be considered environmentally relevant. When the aerosol was exposed to simulated sunlight (fluorescent lamps) and/or hydroxyl radicals, the rate of degradation of decaBDE was just barely measurable, at a rate of $< 6 \times 10^{13}$ cm^3 $molecule^{-1}s^{-1}$ for reaction with hydroxyl radicals. The products of reaction were not determined.

- Palm et al. (2004) provided early results of photolysis experiments with decaBDE adsorbed on pyrogenic silicon dioxide as a carrier material in aqueous suspension. The half-life of decaBDE exposed to UV light in aqueous suspension was reported as 8.8 h, which was much slower than in non-aqueous solvents. Polybrominated dibenzofurans (PBDFs) were confirmed as short-lived intermediates in this study. There was no information available on the other transformation products that were formed.

- Keum and Li (2005) investigated the debromination of decaBDE in aqueous solution when in contact with the reducing agents zerovalent iron, iron sulphide and sodium sulphide. In the experiments with zerovalent iron, decaBDE was rapidly transformed to lower brominated PBDEs. Approximately 90% of the parent was converted to mono- to hexaBDE congeners after 40 d. During the initial reaction period (up to 5 d), decaBDE was predominantly transformed into hexa- and heptaBDEs, but tetra- and pentaBDEs were predominant after 14 d. The results demonstrated reductive debromination. The experiments with iron sulphide and sodium sulphide also showed transformation of decaBDE to lower brominated PBDEs, but the rate was slower than that found in the presence of the zerovalent iron. A similar profile of transformation products was found to that determined in the experiment using zerovalent iron. The Government of the United Kingdom (United Kingdom 2005) points out that the actual conditions used in the Keum and Li (2005) experiment are not directly related to the environment and that concentrations of sulfides present in the environment are a few orders of magnitude lower than those used in this study. Thus, in the environment, other chemical parameters could influence reaction rate. Nevertheless, similar reactants to those used in this study (e.g., iron-bearing minerals and sulphide ions) are present in sediments and soils under anaerobic conditions, although it is uncertain whether environmental conditions are appropriate to activate similar reactions under natural conditions.

Biodegradation

The following bullets summarize the evidence regarding biodegradation of decaBDE that was considered in the screening assessment for PBDEs (Canada 2006; Environment Canada 2006b):

- The biodegradation of decaBDE was reported by MITI (1992). Under aerobic conditions using activated sludge inoculum, no degradation was detected after two weeks of exposure as measured by biological oxygen demand.

- The Chemical Manufacturers Association Brominated Flame Retardant Industry Panel (CMABFRIP 2001) investigated the anaerobic biodegradation of ^{14}C-labelled decaBDE in a sediment-water system over 32 weeks. The decaBDE tested was a mixture of unlabelled substance (97.4% decaBDE, 2.5% nonaBDE and 0.04% octaBDE) with ^{14}C-labelled decaBDE (radiolabelled decaBDE with a purity of 96%). Overall, decaBDE was found to be stable under the conditions tested. The study determined that less than 1% of the total radioactivity was found as $^{14}CO_2$ and $^{14}CH_4$, indicating that essentially no mineralization had occurred.

- Gerecke et al. (2005) undertook experiments under anaerobic conditions using sewage sludge as inoculum in glass bottles (100 mL) filled with a 1-cm layer of glass beads. These containers were then spiked with technical DecaBDE (98% purity, 10.0 nmol/sample) and incubated at 37 ±1°C in the dark for up to 238 d. The DecaBDE contained trace amounts of nonaBDEs (i.e., BDE 206 at approximately 2% on a molar basis, and BDEs 207 and 208 at approximately 0.04% each on a molar basis). No octaBDEs were detected in the sample (detection limit was

0.005 nmol/sample). To stimulate degradation, primers (i.e., 4-bromobenzoic acid, 2,6-dibromobiphenyl, tetrabromobisphenol A, hexabromocyclododecane and decabromobiphenyl) were added in the amount of 9–11 nmol/sample. Two other experiments were carried out with two nonaBDE congeners to determine the fate of these potential decaBDE degradation products. Starch and yeast (50 g) were added immediately before the bottle was filled with 20 mL of freshly collected digested sewage sludge. The bottles were tightly capped and incubated at 37 ±1°C for up to 238 d in the dark. Analysis by GC/HRMS indicated that decaBDE decreased by 30% within 238 d in the experiments with the primers (i.e., from 11.2 to 7.9 nmol/bottle) and this corresponded to a pseudo-first-order degradation rate constant of 1 x 10^{-3} d^{-1}. Without the primers, the rate constant was 50% lower. DecaBDE (BDE209) was observed to degrade to 2 nonaBDEs and 6 octaBDEs. This was indicative of reductive debromination. All three nonaBDE congeners in the separate bottles were also found to undergo reductive debromination. The mass balance between the loss of decaBDE and the formation of transformation products illustrated that 3 nmol disappeared, while only 0.5 mol of transformation products were identified. The researchers noted that this may have resulted from the formation of unidentified transformation products or bound (non-extractable) decaBDE residues, or from imprecisions in the analytical procedure used. The study demonstrated that the debromination of decaBDE proceeded most readily by the loss of bromine from the para and meta positions, as shown by the formation of BDE208 or -207.

3.2.1 New Evidence of Debromination in the Environment

Abiotic Degradation

Bezares-Cruz et al. (2004) investigated the photodegradation of decaBDE (98% pure obtained from Great Lakes Chemical Company) dissolved in hexane and exposed to natural sunlight. DecaBDE (i.e., BDE209) at a concentration of 2-5 μM in hexane degraded rapidly to lower brominated PBDE congeners in the presence of sunlight, with up to 99% reduction in the decaBDE concentration in 30 minutes (exposure to summer sunlight). First-order degradation rate constants were estimated at 1.86×10^{-3}/s in July sunlight (half-life = 6.2 minutes) and 1.11×10^{-3}/s in October sunlight (half-life = 10.4 minutes). Unfortunately, the use of hexane as a solvent in this study makes it difficult to extrapolate these results to a natural setting. The authors concluded that photodegradation in nature would be limited by sorption of decaBDE to particulates, light attenuation by humic materials and lower concentrations of hydrogen donor chemicals—which may also be less favourable hydrogen donors in the aquatic environment. Stapleton (2006a) also concluded that "degradation of decaBDE dissolved in water (or organic solvents) is not expected to be of environmental relevance" and this is supported by the fact that decaBDE has extremely low water solubility. Since this study is not applicable to natural settings, the results were not considered further in the assessment of whether decaBDE is undergoing significant debromination to lower brominated PBDE congeners in the environment.

The photodegradation of decaBDE in an 80:20 methanol/water mixture, in pure methanol, in tetrahydrofuran and in water/humic acids mixtures exposed to artificial

ultra-violet (UV) light was investigated by Eriksson et al. (2004). The decaBDE used in the experiments had a purity of approximately 98%. When exposed to UV light, decaBDE dissolved in the organic solvents or solvent:water mixture degraded rapidly, with degradation rate constants of approximately 4×10^{-4}/s in the methanol/water mixture (half-life ~ 0.5 h), 6.5×10^{-4}/s in methanol (half-life ~ 0.3 h) and 8.3×10^{-4}/s in pure tetrahydrofuran (half-life ~ 0.23 h). The use of organic solvents and artificial conditions not representative of natural settings makes it difficult to extrapolate these results to the environment. The results of this study were therefore not considered relevant to environmental conditions.

The water / humic acid mixture tested by Eriksson et al. (2004) can potentially provide a better simulation of potential photodegradation in a natural setting since humic acids are often present in aquatic systems and could play a role in photodegradation. To prepare the solution for this experiment, 20 mL of a saturated solution of decaBDE in ethanol was combined with 10 mL of ethanol containing 50 mg of humic acid. Approximately 10 mL of ethanol was evaporated using a nitrogen stream, and the remaining solution was combined with 2 L of water. The solution was then heated and maintained at 80°C for 1 hour under a constant flow of nitrogen. The solution was cooled to room temperature and then transferred to cylindrical reaction vessels for the irradiation experiments. The Government of the United Kingdom (United Kingdom 2007a) estimated a final humic substances concentration for this experiment of approximately 25 mg/L and concluded that traces of ethanol may have been present since it was unclear how much ethanol would have been lost by heating at 80°C. The experiments were replicated between 2 and 5 times but it is not clear how many replicates were conducted specifically for the water / humic acid treatment.

The rate of degradation of decaBDE in water with humic acid was 3×10^{-5}/s (half-life ~ 6.4 h). Although the products were not presented in detail in the article, they were described as being almost identical to those determined for the methanol/water experiment which produced a range of lower brominated PBDEs (including 3 nonaBDEs, at least 7 octaBDEs, 8 heptaBDEs and small amounts of hexaBDEs), mono- to pentabromodibenzofurans (mono- to pentaBDFs), and possibly brominated-methoxylated-dibenzofurans. One key difference with the water / humic acid experiment was the formation of a higher proportion of pentaBDFs. Because both water and humic acid are naturally occurring substances, it is possible that similar reactions could occur in the natural environment. However, the actual environmental rates and degree of debromination are uncertain since this experiment used artificial light and it is uncertain what fraction of decaBDE in the environment might be associated with humic acids, as opposed to particulates. Although the authors also reported results for the water-only treatment, there was a high level of difficulty with this experiment due to the extremely low water solubility of decaBDE. As a consequence, the findings of the water-only experiment were highly uncertain.

In their study respecting the effect of sewage sludge application on concentrations of PBDEs in soils and earthworms, Sellström et al. (2005) provide a brief discussion of an investigation of photolytic debromination of BDE209 amended in soil. The BDE209 was

amended with soil from Björketorp, placed in glass test tubes and exposed to artifical UV light on a "rocking/rolling" apparatus for 0, 7, 14 and 21 d. Controls were also exposed to the same agitation, but were protected from the light. Ultimately, soils showed no evidence of photolytic breakdown. The authors concluded that soil appears to encapsulate and shield contaminants so that they are less likely to break down when exposed to sunlight.

Hagberg et al. (2006) observed the formation of PBDF congeners as a result of the photolytic decomposition of decaBDE in toluene. DecaBDE (i.e., BDE209) was dissolved in toluene to a concentration of $2x10^6$ µg/L and then irradiated with UV radiation from a fluorescent tube with or without filtering to generate UV-A (320–400 nm), UV-AB (280–400 nm) or UV-ABC (250–400 nm) light. The light exposures were conducted in petri dishes for 2, 4, 8 and 16 h (parallel exposures) and a dark control was also conducted. All samples were subjected to a serial clean-up technique to separate PBDFs from PBDEs, and identification and quantification of PBDFs were made using HRGC/HRMS. The analysis technique was able to identify PBDFs with 6 or fewer bromines; hepta- and octaBDFs were not included in the analysis scheme. After decaBDE:toluene solution was irradiated with UV-A, UV-AB or UV-ABC, 27 mono- to hexaBDFs were detected, with the majority of products being tetra- to hexaBDFs. The PBDFs formed accounted for 0.31% (UV-A), 0.35% (UV-AB) and 1.2% (UV-ABC) of the initial amount of decaBDE on a molar basis and the authors suggested a trend toward greater transformation (but similar transformation products) at lower wavelengths. The authors also concluded that the observed mono- to hexaBDFs were likely the result of stepwise debromination of higher PBDFs since mono-to hexaBDEs were not detected in any of the irradiated solutions. While these findings demonstrate the possibility of decaBDE transformation to PBDFs, they are considered to have low applicability to the natural environment.

Barcellos da Rosa et al. (2003) studied the photolysis of decaBDE dissolved in toluene. DecaBDE (BDE209, Sigma-Aldrich, 98% purity) was dissolved in toluene at a concentration of 0.31 mM and exposed to light from a 500-watt (W) high-pressure xenon lamp. Following light exposure, samples of the decaBDE:toluene solution were analyzed by GC – flame ionization detector (FID) to identify and quantify decaBDE and debrominated congeners down to hexaBDEs. The decaBDE congener was observed to undergo exponential decay with a photolysis rate constant of $3x10^{-4}$/s. Several debrominated congeners were observed in the decaBDE:toluene solutions and the degradation of decaBDE was inferred to proceed via sequential debromination to nona-, octa- and heptaBDEs. The authors concluded that while their results demonstrated the potential for and pathway of photolytic debromination, further work to confirm the environmental relevance of photolysis in toluene was needed. Overall, the findings of this study are considered to have limited applicability to the environment.

Rahm et al. (2005) conducted a study to determine the relative susceptibility of a variety of compounds, including decaBDE, to hydrolysis via nucleophilic aromatic substitution. When decaBDE was reacted with sodium methoxide dissolved in methanol, the estimated half-life for the hydrolysis reaction was 0.028 h, indicating a rapid hydrolysis reaction.

For lower brominated PBDEs, the rate of reaction decreased by roughly a factor of 10 for each bromine removed, relative to decaBDE. The authors concluded that decaBDE would be susceptible to hydrolysis by nucleophilic compounds in the environment. However, given that sodium methoxide is not normally present at significant concentrations in the environment and that reaction catalyzed by mineral surfaces, enzymes, etc., would be the main reactant for hydrolysis, there is a high level of uncertainty in extrapolating these results to the natural environment.

Geller et al. (2006) report the findings of a photolysis experiment on decaBDE. DecaBDE (BDE 209 congener; 98% purity) was dissolved in 3 mL of tetrahydrofuran (THF) at saturation (10 g/L, also containing additional particulates of solid-phase decaBDE), and irradiated with four lamps for a period of up to 48 h. Following irradiation, samples were analyzed by HPLC and GC–MS using electron impact ionization with identification of degradation products based on retention times. The photolysis products included hepta- to nonaBDEs as well as tri- to hexabromodibenzofurans. Comparison with the chromatogram for 2,3,7,8-substituted dibenzofurans indicated that these were not major degradation products. These findings have similar shortcomings to other studies using organic solvents and are not considered environmentally relevant.

Kuivikko et al. (2007) investigated the photodegradation of decaBDE dissolved in isooctane and combined the findings with a model to predict the photodegradation half-life in two marine systems—the Baltic Sea and the Atlantic Ocean. DecaBDE (BDE 209; purity > 98.3%) was dissolved in isooctane (250 ng/mL), the solution was placed in quartz GC-autosampler vials and the vials were exposed to natural light in Helsinki, Finland, in a shallow pool for 60 minutes on October 5. The concentration of parent decaBDE was analyzed four to five times during the irradiation; each time there were three replicates, a dark control and an isooctane blank. The quantity of decaBDE was analyzed using GC–MS. The concentration of decaBDE decreased according to first-order kinetics, with a half-life of approximately 0.03 d.

Kuivikko et al. (2007) reported a quantum yield for decaBDE of 0.28 ±0.04. The quantum yield was used in model simulations to predict the photodegradation half-life in the Baltic Sea (both surface and 10 m mixing layer) and the Atlantic Ocean (40 m mixing layer only). The model simulations assumed environmentally relevant concentrations (3–4 pg/L and 30–40 pg/L) in the water phase and typical summer solar radiation in the Baltic Sea, and also accounted for light attenuation by dissolved and particulate matter. Kuivikko et al. (2007) predicted mixing zone half-lives of 1.8 d (Baltic Sea) and 0.4 d (Atlantic Ocean), which were the same for both decaBDE concentrations.

While the model appears to simulate natural conditions, it is important to consider that decaBDE in the water column would be primarily sorbed to suspended particulates rather than dissolved. Thus, although the degradation of the dissolved phase is predicted to be relatively rapid, this degradation would represent only a very small amount of the total decaBDE present in the water column. Therefore, there is uncertainty as to whether the

predicted half-lives would be a representative portrayal of the persistence and degradation of decaBDE in the water column.

Ahn et al. (2006a) investigated the photochemical debromination of decaBDE sorbed to major components of soil/sediment/mineral aerosols including clay minerals and noncrystalline metal oxides which are known to have electron transferring capacities. The decaBDE used in the study had a purity of 98%. Test matrices included montmorillonite, kaolinite, organic-carbon-rich natural sediment (16.4% OC content), aluminum hydroxide, iron oxide and manganese dioxide. Each of the decaBDE-amended test matrices (250 mg) was combined with 500 μL water and the mixtures were then irradiated with artificial light or natural sunlight. Irradiation with artificial light used four 24-W lamps with peak output at 350 nm for a period of 14 d. Natural sunlight exposure was conducted between July and November 2004, in West Lafayette, Indiana, for a period of up to 101 d (further exposure in November/December did not result in additional degradation of decaBDE). Samples of decaBDE were quantified using HPLC, while the debrominated products were quantified using GC – electron capture detector (GC–ECD).

The dark and light controls showed no signs of degradation although the study reported minor peaks of nona- and octaBDEs in the dark control which were likely impurities in the decaBDE formulation. For the artificial light exposures, half-lives for montmorillonite, kaolinite, sediment and aluminum hydroxide were 36, 44, 150 and 178 d, respectively. In natural light, half-lives for decaBDE using montmorillonite, kaolinite and sediment were 261, 408 and 990 d, respectively, with negligible degradation on aluminum hydroxide (note that these half-lives represent days of continuous exposure to sunlight, as opposed to actual days including light and dark exposure). Degradation of decaBDE was negligible for iron oxide and manganese dioxide under either lighting scheme. It is important to note that all of these half-lives are longer than the light exposure times, suggesting the potential for uncertainty in these estimates. The half-lives for the natural light exposures suggest that decaBDE photodegradation occurs slowly in the natural setting.

The fastest degradation was observed on montmorillonite and kaolinite and these matrices were the focus for identification of debromination products. Identified products for kaolinite and montmorillonite exposed to sunlight included nonaBDEs (i.e., BDE208, -207, -206) and octaBDEs (BDE197 and -196), as well as trace amounts of tri- to heptaBDEs over longer sunlight exposure times. Higher fractions of tri-to heptaBDEs were also observed for the artificial light exposures. Several unidentified octaBDE products were also observed in the chromatograms. The formation of the identified products was consistent with stepwise debromination, initially forming nona-, then octa- and then heptaBDEs.

Ahn et al. (2006b) investigated metal oxide mediated debromination of decaBDE using birnessite (a naturally occurring manganese oxide mineral) in THF:water and water:catechol reactor systems. The purity of the decaBDE used for the experiments was 98%. The amended birnessite was prepared by combining 0.1 mL of a stock solution

containing 1 mg/mL decaBDE dissolved in THF with 50 mg of birnessite in 15-mL test tubes and then air-drying for one day to remove the THF. The first set of experiments examined the degradation of decaBDE sorbed to birnessite in THF:water systems. For these experiments, the decaBDE-amended birnessite was mixed with 5 mL of THF:water solutions at ratios ranging from 0:10 to 10:0. Follow-up experiments examined (i) the reactivity of dissolved decaBDE in THF:water at a ratio of 7:3 by varying the amount of treated birnessite from 0 to 50 mg/mL, and (ii) the role of THF as the hydrogen donor for debromination of decaBDE, determined by measuring the production of succinic acid in the system. All treatments were conducted in triplicate and shaken in the dark for a period of 24 h with subsampling for analysis at various times. A separate set of experiments investigated the degradation of decaBDE sorbed to birnessite in water systems, in the presence of the naturally occurring hydrogen donor, catechol. Catechol was combined (0.003, 0.045 and 46.135 mmol) with 5 mL of water and mixed with 50 mg of decaBDE-amended birnessite. All experimental treatments were shaken in the dark for 23 d with subsampling at various times to track the degradation of decaBDE. DecaBDE (i.e., BDE209) was extracted in THF and analyzed using HPLC while the potential products were quantified using GC–ECD and identified by matching peak retention times with known retention times for lower brominated PBDEs.

The experiments using combined THF:water reactor systems produced rapid degradation of decaBDE with > 75% transformation of decaBDE over the 24-hour period for some THF:water ratios. However, the follow-up experiments determined that THF was reacting with the birnessite to form succinnic acid, and in the process acted as a hydrogen donor for the debromination of decaBDE sorbed to the birnessite. Thus, the relatively rapid observed degradation rates appeared to depend on the presence of THF. Because THF is not normally present in the environment, the rate of decaBDE degradation is not considered realistic with respect to conditions present in the natural environment. The rapid reaction rates in the THF:water experiments did allow for identification of debrominated products. The lower brominated products produced after 24 h included tetra- to nonaBDEs. The reaction appeared to proceed in a stepwise manner with decaBDE being initially degraded rapidly to nonaBDEs, followed by further stepwise debromination to lower brominated PBDEs.

The second set of experiments using water:catechol reaction systems were used to examine potential for decaBDE degradation in the presence of the naturally occurring substance catechol. No significant degradation was observed over the 23-d period in the experiments with 0.003–0.045 mmol catechol. However, slow degradation was observed with 46 mmol catechol, with the mass of decaBDE in the reaction vessels decreasing from approximately 0.1 μmol to 0.085 μmol during the 23-d experiment. Although the products were not quantified for the water:catechol systems, it is possible that they would follow a similar pattern to the THF:water systems. Although the rate of reaction was slow under the simulated natural conditions, it is possible that decaBDE may eventually debrominate over time in the presence of naturally occurring minerals and hydrogen donors, such as catechol.

Stapleton and Dodder (2006) reported the findings of a study of photolytic debromination of decaBDE in house dust. The dust used for the study was a National Institute of Standards and Technology standard reference material (SRM) prepared from vacuum cleaner bag contents from homes, motels and hotels in the U.S. The dust is known to contain PBDE (including decaBDE), and certified PBDE concentrations were available for 15 congeners. Photolysis studies were conducted with both the SRM dust in its existing form and with SRM dust without PBDEs (these were removed by Soxhlet extraction) but then spiked with a known quantity of decaBDE to yield a concentration of 2180 μg/kg dw. Analysis of the "cleaned" dust prior to spiking confirmed that decaBDE was not detected (< 0.2 μg/kg). Samples (0.5 g) of each dust material were placed in UV cuvettes and exposed to natural outdoor sunlight in Gaithersburg, Maryland, between 9 a.m. and 5 p.m. on days when no precipitation was forecast, for a total of 200 h. Three replicates were included for each experiment along with three control samples for each dust material, which were covered in aluminum foil.

The concentrations of decaBDE were found to decrease in both dust materials, with first-order degradation rates of 2.3×10^{-3}/h in spiked dust and 1.7×10^{-3}/h in natural dust, corresponding to half lives of 301 and 408 h in sunlight, respectively. The authors speculated that the longer half-life in natural dust might be due to matrix factors which attenuated the amount of light reaching the decaBDE molecules, or that sorbing materials present in the natural dust were removed by the extraction process, making the spiked decaBDE more available for photodegradation. An additional explanation was that solid decaBDE with a limited surface area for irradiation might be present in the natural house dust. The spiked dust samples were also analyzed for lower brominated degradation products. At the end of the exposure, approximately 38% of the initial decaBDE concentration had been lost or degraded. Part of the loss (i.e., equivalent to approximately 13%) was due to debromination to predominantly the three nonaBDE congeners, but also due to lesser amounts of octa- and heptaBDE congeners. The remaining 25% of the original decaBDE concentration could not be accounted for and was lost to unknown pathways and/or products. The Stapleton and Dodder (2006) study suggested that the presence of BDE201 and -202 may provide a marker for debromination of BDE209. In the OctaBDE commercial mixture, BDE201 makes up a very small component (i.e., from below detection to 0.8%), and BDE203 is not detected.

This study provides reasonably strong evidence that photodegradation of decaBDE on dust can occur under natural conditions in the environment and that lower brominated PBDEs can be formed. The authors noted that while the actual degree of sunlight exposure to household dust might be limited by windows and shading, dust in cars would be subjected to much higher levels of sunlight, making debromination of decaBDE on dust in cars potentially significant.

The Government of the United Kingdom (United Kingdom 2007a) describes the findings of an additional study by Stapleton (2006b) on the debromination of decaBDE in house dust. The methods were similar to those of Stapleton and Dodder (2006) except that the exposure was carried out during the hours of 9 a.m. to 4 p.m. for up to a total of 90 h. Sunlight exposures were conducted between July and August 2004 at a mean temperature

of 27.4°C. Over the 90-hour exposure period, decaBDE concentration in the house dust decreased from 2180 μg/kg dw to 1570 μg/kg dw, indicating that approximately 28% of decaBDE degraded. The start and end concentrations of decaBDE in the dark control were not statistically different. The half-life for decaBDE was estimated at 216 h (continuous sunlight exposure, or 27 d assuming 8 h sunlight per day). Lower brominated PBDEs were detected as degradation products and these included three nonaBDEs, six octaBDEs and one heptaBDE. The mass balance of decaBDE and lower brominated PBDE congeners indicated that approximately 17% of the original decaBDE was unaccounted for, suggesting the formation of alternative (unidentified) products or volatilization of lower brominated PBDEs.

Gerecke (2006) determined the reaction quantum yield of decaBDE on kaolinite and measured light penetration in this mineral in order to calculate the photodegradation rate of decaBDE. The decaBDE used in the study had a purity of 98%. Light penetration into kaolinite was estimated using the Kubelka-Munk theory, which relies on the measurement of light absorption (k) and scattering (s). Thin layers of kaolinite were prepared on quartz glass with 10 different thicknesses prepared for estimating k and s. The kaolinite was spiked with decaBDE in an isooctane/toluene mixture (95/5, v/v). Although the spiking method is not provided in detail, it is presumed that after spiking the solvent was evaporated off, leaving a solid layer of decaBDE-spiked kaolinite on quartz glass, since the light exposures were conducted for either dry kaolinite or wetted kaolinite. The spiked kaolinite layers were irradiated with sunlight at noon on clear summer days in Dubendorf, Switzerland. The experiments were conducted in a water bath to maintain constant temperature and the study included dark controls as well as precautions to avoid light exposure outside of the specified times. The concentrations of decaBDE and potential products were analyzed using GC/MS.

Sunlight had only very limited penetration in the kaolinite below 50 μm and the authors concluded that only decaBDE sorbed to particles at the very surface of soils would have the potential to undergo photolysis. In the experiments with decaBDE-spiked kaolinite, half-lives of 76 and 73 minutes were determined for dry and wet conditions. However, the observed degradation was non-exponential, likely due to the large difference in degradation rate between the upper and lower sides of the kaolinite layer. Under dry conditions, degradation products were identified as lower brominated PBDEs, whereas for wet conditions, the products were not identified, suggesting that the products were not lower brominated PBDEs. These findings demonstrate that while photodegradation of decaBDE sorbed to mineral solids is possible in the environment, the rate and amount degraded is highly dependent on the penetration of light into soil and mineral layers.

Nose et al. (2007) studied the degradation pathways of standard decaBDE (purity not noted, but purchased from Wako Pure Chemical Industries, Ltd.) during hydrothermal treatment. Their evaluation was carried out in a micro autoclave made of stainless steel filled with 40 mL of distilled water. The decaBDE material was initially dissolved in toluene, and then spiked into the autoclave chamber, which was sealed. Temperature was controlled, with the first heating of 25 min to 300ºC and a pressure of 8 MPa. The experiment was repeated at different time intervals (0, 10, 30, 60, 120, 240 and 360 min),

defined as the processing time. The reaction time was defined as the sum of the heating time and the processing time. The chamber was then cooled down to 100ºC using a fan, and then soaked in ice water for 20 min.

The study found some decomposition (~45%) after approximately 12 min at 200°C, and almost complete breakdown (i.e. > 99%) after 10 min at 300°C. Debromination to nonaBDE was determined; however, debromination of meta and para positions to BDE208 and -207 occurred at a faster pace than debromination of the ortho position to BDE206. The experiment was also conducted with other lower brominated PBDEs with similar findings. Thus, the authors concluded that the reactivities of bromine on the para and meta positions were relatively high, while the reactivity of the ortho bromine was extremely low in hydrothermal treatment. The study also confirmed the formation of PBDD/DFs during the study. While having limited applicability to the natural environment, the study appears to confirm the findings of other studies respecting preferential meta- and para-position debromination (e.g., Stapleton et al. 2006; Gerecke et al 2005; Huwe and Smith 2007a, 2007b). As noted in the Canadian screening assessment on PBDEs (Canada 2006; Environment Canada 2006), under certain combustion/pyrolysis and photolysis conditions, all PBDEs (including DecaBDE) can form brominated dibenzofurans and dibenzo-*p*-dioxins. These transformation products are brominated analogues of the Toxic Substances Management Policy Track 1 polychlorinated dibenzofurans and dibenzo-*p*-dioxins. Complete destruction of DecaBDE and any possible breakdown products appears to occur with exposure to temperatures of 800°C and above for 2 seconds (European Communities 2002).

Li et al. (2007) examined the debromination of decaBDE (DE-83R, > 97% purity obtained from Great Lakes Chemical Company) by resin-bound zerovalent iron nanoparticles. The study involved the use of about 50 test tubes prepared with decaBDE in acetone, mixed with an equal volume of distilled water to form an 8-mL solution. Then 2 g of resin containing zerovalent iron was added. The test tubes were capped and shaken for 1 h to 10 d in a water bath (25 ±0.5°C). Analyses were performed using GC with an ECD detector, and HRGC/HRMS. The results demonstrated rapid debromination of BDE209 after about 8 h, which the authors determined to follow first-order transformation kinetics resulting in a rate constant of 0.28 ±0.04/h and half-life of 2.5 h. Disappearance of BDE209 occurred, with the subsequent formation of nona- to triBDEs in a sequential manner. All three nonaBDEs appeared in significant amounts within 1 h, steadily increased for 8 h, then decreased below detection after 24 h. The congener pattern dominance shifted to heptaBDEs after 2 d, and then hexaBDEs were most abundant. After 10 d, pentaBDEs were present in significant amounts.

The authors noted that the identification of reaction products was challenging, mainly due to the lack of appropriate PBDE standards at the time of their experiments, with many peaks not matching any of those for 43 available chemical standards. Issues of co-elution were identified. However, all three nonaBDEs were identified with confidence and two of five octaBDE peaks were identified (i.e., BDE197 and -196). The hepta- to tetraBDEs that were confidently identified included BDE183, -153, -154, -99 and -47 (all contain both para-position bromines). Overall, the presence of unidentified peaks makes it

difficult to conclusively confirm the positional preference of debromination pathways. Li et al. (2007) also conducted an identical study using PCB209, which dechlorinated at a much slower rate than the debromination of BDE209. After 10 d, only 21% of PCB209 was lost, with only the formation of nona- and octaPCBs identified. Overall, the use of zerovalent iron nanoparticles means that this study has uncertain relevance to the environment.

Kajiwara et al. (2008) examined DecaBDE photolysis in plastics under natural sunlight. High-impact polystyrene (HIPS) was added to toluene containing 100 μg/mL of DecaBDE and shaken overnight to achieve complete dissolution; toluene subsequently evaporated in the dark. Crushed television casing and solidified HIPS samples compounded with the technical mixtures were further pulverized in a liquid nitrogen chamber. Resultant materials were screened with a vibrating stainless-steel sieve apparatus. The fine sieved powder (106–300 μm) was used in the irradiation experiment (samples of HIPS+DecaBDE and TV casing). Aliquots of the powdered plastic were transferred to quartz tubes, sealed with Teflon plugs. A subset of HIPS+DecaBDE samples hydrated with 0.5 mL of hexane-washed water was also prepared to examine the effect of water on PBDE photolysis. Tubes were kept inside the laboratory in the dark at room temperature until the sunlight irradiation experiment was performed.

The HIPS+DecaBDE samples showed a gradual disappearance of BDE209 upon exposure to sunlight. No degradation was found in the dark controls throughout the experiment. After one week of exposure to sunlight, the concentration of BDE209 declined to approximately 50% of the initial level, indicating prompt photodegradation in the plastic matrix. Hydrated HIPS+DecaBDE samples showed more rapid degradation of BDE209 than non-hydrated samples. The authors suggested that this accelerated degradation might be explained by the fact that water played a role as a hydrogen donor, promoting debromination.

Kajiwara et al. (2008) state that previous studies have focused on the photodegradation of PBDE dissolved in solvents or absorbed to particles, and that their study is the first to show the photolysis of BDE209 compounded into a polymer matrix. Assuming a first-order reaction, they report a calculated half-life for BDE209 transformation in HIPS of 51 d. The authors highlight that this half-life for plastic is longer than those measured in sand, sediments and soils but equivalent to the half-lives measured in house dust.

During the study, the HIPS+DecaBDE samples showed that concentrations of hexa- to nonaBDE congeners increased several-fold after one week of exposure, and the authors interpreted that debromination of BDE209 resulted to some extent in the formation of lower brominated PBDE congeners. After this first week, the concentrations of hexa- to nonaBDE congeners remained constant or decreased slightly, despite the continuous decrease in BDE209. Congeners BDE47, -99 and -100 were not detected and stepwise debromination of PBDEs was not clearly observed in this study. At the end of the exposure period, the total PBDE concentration was less than 20% of the initial level, and the proportion of BDE209 to total PBDEs had decreased from 90% to 44%. Kajiwara et al. (2008) reported the photolytic formation of tri- to octaBDFs in their HIPS+DecaBDE

samples. The concentration of total PBDFs showed an increase (greater than 40 times) on day 7 of exposure, while BDE209 concentration decreased.

With respect to the TV casing samples, the study showed no disappearance of BDE209 and formation of lower brominated PBDE congeners throughout the 224 d of sunlight irradiation, in contrast to HIPS+DecaBDE samples. Two possible explanations for the differences in degradation profiles were suggested in the study: 1) the difference in initial concentrations of BDE209 (HIPS+DecaBDE and the TV casing samples contained 0.1 and 10% BDE209, respectively) influenced degradation rates; and/or 2) the effects of the other plastic additives in TV casings, such as pigments, UV absorbers, and stabilizers, potentially affected light penetration depth in each plastic sample.

Raff and Hites (2007) examined the importance of photolysis in the removal of PBDEs from the atmosphere by comparing the photolysis frequencies of PBDE congeners to the first-order removal rate constants for oxidant reactions and dry and wet deposition. The study compared atmospheric removal processes affecting PBDE (i.e., focused on BDE47, -99 and -209) fate using a box model simulating the atmosphere above Lake Superior. The model results indicated that photolysis results in the highest loss (90% of the total losses) of BDE-47 and -99 from the atmosphere above Lake Superior. However, for BDE209, more than 90% of the removal is the result of precipitation events. The authors suggested that this explains why the PBDE congener profile found in sediments from Siskiwit Lake (located on a remote island in Lake Superior) and the Great Lakes appear to be enriched in BDE209 compared to other congeners. These results are consistent with congener profiles obtained from air-particle samples collected in the Arctic that showed significant depletion of BDE209 relative to tetra- and pentabrominated PBDE congeners. The main conclusion of the study is that deposition processes control the loss of BDE209 from the atmosphere and are responsible for the enhancement of BDE209 found in sediment samples from the Great Lakes.

Biodegradation

Gerecke et al. (2006) report the findings of follow-up experiments to their 2005 study. The anaerobic biodegradation of technical DecaBDE (98% purity) in digested sewage sludge was investigated using the same test system as Gerecke et al. (2005) but with only single primers, either 2,6-dibromophenol or 4-bromobenzoic acid. The use of each primer resulted in debromination of decaBDE via loss of para-position bromine to BDE208 but the reaction was slow, with a half-life exceeding 700 d. In the absence of a primer, the half-life was longer, up to 1400 d. The authors also conducted field monitoring of decaBDE in a sewage treatment plant to determine whether biodegradation occurred in a full-scale anaerobic digester. Grab samples of influent, reactor and outlet sewage sludge were collected from a WWTP at Dubendorf, Switzerland. The concentration of decaBDE in sludge was observed to decrease between the influent and outlet streams, suggesting that transformation occurred in the full-scale digester. However, the authors cautioned that the relatively short residence time in the reactor (28 d) and low level of replication (i.e., only one set of grab samples) meant that these results should be considered preliminary and not necessarily unequivocal.

The biodegradation of decaBDE (> 98% purity) by cultures of anaerobic bacteria was investigated by He et al. (2006). The cultures of anaerobic bacteria included *Dehalococcoides ethenogenes* 195, *Sulfurospirillum multivorans* and *Dehalococcoides* sp. strain BAV1 (each of which have been shown to dechlorinate organochlorine compounds), as well as an enriched autotrophic culture containing *D. ethenogenes* 195, and an enrichment containing a number of *Dehalococcoides* spp. All cultures were grown on ~500 mM trichlorethane (TCE) with the exception of *Dehalococcoides* sp. strain BAV1, which was grown on vinyl chloride.

The experiments were conducted in 160-mL serum bottles containing a culture medium and carbon source appropriate for each organism. For *D. ethenogenes* (an autotrophic culture), no carbon source was used. Prior to inoculation the test vessels were sealed to ensure anaerobic conditions and then autoclaved for 25 minutes at 121°C. To initiate each experiment, 5 mL of a decaBDE stock solution in trichloroethane (TCE, ~1 μM decaBDE) was added to the test vessel for a final concentration of 1 mM TCE and 0.1 μM decaBDE. The bottles were inoculated with one of the active cultures described above at 5% or 10% v/v. The test samples were incubated in the dark at 30°C for a period of up to 12 months, with weekly sampling of 1 mL of culture for the analysis of decaBDE and degradation products. Each experiment was conducted with two replicates per treatment and repeated at least once to confirm the results. Abiotic control samples were also included in the experimental design. PBDE congeners were detected using GC–ECD.

The experiment with *S. multivorans* exhibited rapid dechlorination of TCE, but no degradation of decaBDE within the first weeks of incubation. After an additional two months of incubation without replacement of TCE, decaBDE was observed to degrade to non-detectable levels, while octa- and heptaBDEs became detectable in both replicates. No degradation was evident in the abiotic controls, indicating that the disappearance of decaBDE was due to anaerobic degradation. These findings suggest that under appropriate conditions, certain bacteria may debrominate decaBDE. No degradation of decaBDE was seen in the experiments using any of the other cultures.

Parsons et al. (2004) investigated the potential for reductive debromination of decaBDE in anaerobic sediment suspensions. The experiments were conducted using sediment collected at Hansweert in the Western Scheldt, which is known to contain high concentrations of decaBDE. A total of 20 g of the sediment were suspended in 20 mL of anaerobic medium and the suspensions were spiked with decaBDE (14 μg/g sediment) and incubated anaerobically at room temperature in the dark (details of the anaerobic test system were not provided). Sterilized controls were also conducted (details of the sterilization procedure was not provided). At varying intervals over an approximately 205-day test period (read from graph), experimental and control samples were extracted in hexane/acetone, subjected to a clean-up process and analyzed using GC–low resolution mass spectrometry (LRMS).

Concentrations of decaBDE in the spiked samples decreased significantly over the first two months of incubation and the decrease coincided with the appearance of new

chromatogram peaks which were tentatively identified from their retention times and mass spectra as nonaBDEs. The concentrations of nonaBDEs were generally too low to be quantifiable and the authors speculated that nonaBDEs were further debrominating to lower brominated PBDE congeners. There is significant uncertainty in these findings with respect to anaerobic sediment debromination since a similar decrease in decaBDE was also found in the sterile controls. The authors suggested that this was due to incomplete control sterilization as confirmed by the production of methane upon addition of lactic, pyruvic and acetic acids to the control preparations. However, it is also possible that photodegradation of decaBDE occurred during sample handling, extraction and analysis. Therefore, although Parsons et al. (2004) found a decrease in decaBDE in anaerobic sediment suspensions, this decrease cannot be attributed unequivocally to anaerobic degradation.

Parsons et al. (2007) report the findings of an additional investigation of reductive debromination of decaBDE in anaerobic sediment microcosms. The experiments were conducted using sediment collected at Hansweert in the Western Scheldt, which is known to contain high concentrations of decaBDE. A total of 10 g of the sediment were suspended in 50 mL of anaerobic medium (containing acetate, lactate and pyruvate). The suspensions were then spiked with decaBDE or individual nonaBDE congeners and incubated at room temperature in the dark. At varying intervals over an approximate 260-day test period (read from graph), experimental and control samples were extracted in hexane/acetone, subjected to a clean-up process and analyzed using GC/MS in selected ion monitoring mode.

There was no measurable decrease in decaBDE in the active and control microcosms, although nonaBDEs were detected in the decaBDE-spiked samples at much higher concentrations than known background levels in the study sediments

Knoth et al. (2007) conducted a monitoring study of PBDEs, including decaBDE, in sewage sludge from 11 municipal WWTPs in Germany. A total of 39 sludge samples from different stages of the treatment process—primary sludge, secondary excess sludge and (dewatered) digested sludge—were collected from March 2002 to June 2003. The samples were subjected to a process of sterilization, freeze-drying, and spiking with stable isotope standards, followed by Soxhlet extraction in toluene, four-column clean-up of the toluene extraction, and reduction to 100 μL. The extracted and cleaned-up sample was analyzed by GC–select ion monitoring (electron ionization) (SIM(EI+))LRMS to quantify decaBDE and lower brominated PBDE congeners.

The congener profiles in the various treatment plants and treatment stages were dominated by decaBDE, which ranged from 97.1 to 2217 ng/g dw. Debromination of decaBDE to lower brominated PBDEs during treatment was either not occurring or too slow to be detected during the total retention time of sludge (11–13 d) in German WWTPs.

La Guardia et al. (2007) examined potential for *in vivo* and environmental debromination of decaBDE in a WWTP and its receiving environment. Concentrations of decaBDE and

lower brominated PBDEs were monitored in sludge, sediments, and fish in the receiving environment of a WWTP located in Roxboro, North Carolina (further study details in Section 2.2). The PBDE congener profile was tracked from WWTP to receiving environment sediments and to biota in order to evaluate whether significant debromination was occurring. For the sludge samples, 17 PBDE congeners were identified in 2002 while 18 PBDE congeners were identified in 2005. The major congener in sludge was BDE209, accounting for 60% (58 800 µg/kg dw) of the total PBDE burden in 2002 and 87% (37 400 µg/kg dw) of the total PBDE burden in 2005. The sludge congener profiles were similar to the technical formulations (PentaBDE and DecaBDE), suggesting minimal debromination in WWTP sludge. In receiving environment sediments the major PBDE congeners included BDE209, -206, -99 and -47, with BDE209 making up > 89% of the total BDE burden in each of the sediment samples. The decaBDE concentration in sediments was highest between 1 and 6 km downstream of the outfall, with maximum levels ranging from 3240 mg/kg OC to 2450 mg/kg OC. The lower brominated PBDE congeners were attributed to the PentaBDE technical formulation, suggesting minimal debromination of decaBDE in surficial sediments.

Tokarz et al. (2008) conducted concurrent PBDE (i.e., BDE209, -99 and -47) experiments using anaerobic sediment microcosms and a cosolvent-enhanced biomimetic system. The sediment microcosms contained natural sediments with no detectable PBDEs collected from Celery Bog Park, West Lafayette, Indiana. The PBDEs were dissolved in a toluene solution and added to sediments, and then the solvent was evaporated off. This mixture was then blended with wet sediments to form a concentration of approximately 5.0 µg/g and 3 µg/g for BDE99 and -47, and BDE29, respectively. The microcosms were fed methanol and dextrose to ensure the formation of anaerobic conditions, and to provide electron donors. Autoclaved control systems were implemented. The biomimetic experiment involved the use of Teflon-capped glass vials with 0.03 mM of BDE209, -99 or -47 mixed with 5.0 mM titanium citrate and 0.2 mM vitamin B12 in 0.33 M TRIZMA buffer solution containing tetrahydrofuran. Controls were used without titanium citrate. Debromination products were identified and quantified using GC–ECD and GC–MS.

Tokarz et al. (2008) notes that cobalamins, such as coenzyme vitamin $B_{12,}$ have the ability to mediate reductive dehogenation of halorganics. Cobalamins from decaying cells have been iscolated from environmental samples. In addition, they indicate that vitamin B_{12} has ubiquitous presence in the environment in anaerobic micro-organisms.

The biomimetic system demonstrated reductive debromination at decreasing rates with decreasing bromination (e.g., half-life of 18 seconds for BDE209 and almost 60 d for BDE47). In natural sediment microcosms, the half-life for BDE209 was estimated to range from 6 to 50 years, with an average of 14 years, based on observations over 3.5 years. After 8 months, BDE47 decreased approximately 30% without a consistent concurrent increase in daughter debromination products. While complete debromination to diphenyl ether was not ruled out, it appeared unlikely since no intermediate products were identified. The researchers speculated that there could have been formation of hydroxylated and methoxylated derivatives of tetraBDE. The researchers synthesized

their data from both systems and proposed major debromination pathways for sediment and biomimetic systems as follows: BDE209 > nonaBDEs (BDE206, -207 -208) > octaBDEs (BDE196, -197) > heptaBDEs (BDE191, -184, 2 unknown heptaBDEs) > hexaBDEs (BDE138, -128, -154, -153) > pentaBDEs (BDE119, -99) > tetraBDEs (BDE66, -47, -49) > triBDEs (BDE28, -17). Specifically, at the end of the 3.5-year study, their analysis of BDE209 degradation in sediments identified BDE208, -197, -196, -191, -128, -184, -138, and -128, as well as 3 unidentified octaBDEs and 2 unidentified heptaBDEs.

The findings of Tokarz et al. (2008) showing prolonged persistence of decaBDE in sediment microcosms are supported by a field study of Eljarrat et al. (2008) and Sellström et al. (2005). Eljarrat et al. (2008) examined the fate of PBDEs in sewage sludge from five municipal WWTPs after agricultural application in Spain in 2005. PBDE concentration in sewage sludge ranged from 197 to 1185 ng/g dw, with BDE209 being the predominant congener, ranging in concentrations from 80.6 to 1083 ng/g dw. Concentrations of BDE209 in soils ranged from 14.6 to 1082 ng/g dw at seven agricultural sites. High concentrations (i.e., 71.7 ng/g dw) were even found at one site that had not received sludge application for four years, illustrating the persistence of BDE209 in soils. Sellström et al. (2005) collected PBDE samples from sites that received past sewage sludge amendments. They sampled soil from three research stations (with reference plots and sewage-sludge-amended plots) and two farms (reference and amended/flooded soils) in Sweden. They determined that decaBDE concentrations in soil ranged from 0.015 to 22 000 ng/g dw. Highest levels were noted at the farm site that had not received amendments for 20 years.

Kohler et al. (2008) examined sediment concentrations and temporal trends of deca-, nona- and octaBDEs utilizing a sediment core from Greifensee, a small lake located near Zürich, Switzerland. In their study, they note that PBDEs first appeared in sediment layers corresponding to the mid-1970s. The authors identified all three nonaBDEs and at least seven octaBDEs (BDE202, -201, -197/204, -198/203, -196/200, -205 and -194) in the lake's surface sediments using reference materials. While deca- and nonaBDE concentrations were found to increase rapidly with time, the increase of the octaBDE concentrations was slower. Congener patterns of the octa- and nonaBDE congeners in the sediments did not change with time, and thus transformation of PBDEs was not said to be observed in the sediments over the 30-year span. However, the authors noted different relative proportions of nona- and octaBDEs in the sediments in comparison to the commercial PBDE mixtures. Notably, they detected BDE202 which is apparently not reported in any of the PBDE mixture and they speculated that its presence could have been due to transformation of decaBDE. Also, they noted that congener patterns of octaBDE were dissimilar to those of the commercial PBDE mixtures, but similar to patterns in house dust and profiles of photodegradation products of decaBDE. Consequently, they suggested biotic and/or abiotic transformation processes are involved between the release of the technical products into the environment and their final residues in sediments that are responsible for the changes of congener patterns that they observed in the sediments of the Swiss lake.

Zhou et al. (2007) evaluated the ability of white rot fungi to degrade BDE209 in a liquid culture medium, and the effects of Tween 80 and ß-cyclodextrin on the degradation of BDE209 by white rot fungi. White rot fungi have been shown to rapidly oxidize and mineralize many aromatic compounds, including PCBs. In order to improve BDE bioavailability, "solubilizing" agents such as the surfactant Tween 80 or cyclodextrin were added to their test systems. The authors note that the application of this technology is most suited for bioremediation applications. Overall, this study has unknown relevance to environmental conditions.

Zhou et al. (2007) utilized 1 mL of decaBDE (98% pure) in dichloromethane added to 250-mL flasks. The solvent was then allowed to evaporate, resulting in a total BDE209 mass of 16 µg coating the bottom of the flask systems. A 100-mL aliquot of aqueous medium (distilled water, malt extract, glucose, peptone and yeast extract mixture) was added to each flask. White rot fungi were inoculated to the liquid culture and the test system was shaken for 10 d in the dark. Identical test systems were also implemented utilizing additions of Tween 80 and cyclodextrin concentrations ranging from 0 to 900 mg/L. Analyses were conducted using HPLC with a UV detector. The test systems with only white rot fungi added showed a decrease of 42.2% over 10 d in the amount of BDE209 in the test system. The sterile controls showed no significant degradation over time. Tween 80 was found to enhance BDE209 degradation at an appropriate concentration (maximum degradation 96.5% over 10 d). Cyclodextrin was also shown to enhance BDE209 degradation (maximum degradation of 78.4% over 10 d). Transformation products were not identified in this study.

Orihel et al. (2009) presented an ongoing study that may provide some key information on the environmental transformation and aquatic bioaccumulation of decaBDE. Their field mesocosm study compares sediment and periphyton BDE209 concentrations from low (dose=0.039 g of DecaBDE), medium (dose=0.28 g of DecaBDE) and high (dose=2.3 g of DecaBDE) treatments against a control mesocosm. Based on preliminary results, the authors suggest that DecaBDE breakdown had occurred since lower brominated PBDE products were observed in surface sediments as soon as 1 month after DecaBDE addition to mesocosm water. In their study, penta- to hepta-BDEs were observed in the medium- and high-mesocosm treatments, but not in the low-treatment or control mesocosms. DecaBDE was only observed in periphyton in the medium- and high-treatment mesocosms. NonaBDE was observed in periphyton in the high mesocosm after 1 month, but not after 4 months. Hexa-, hepta- and octaBDEs were not detected in periphyton after 1 month, but were after 4 months in the medium- and high-treatment mesocosms. In the coming years, the results will be finalized.

3.2.2 Conceptual Model of Transformation in the Environment

The likelihood, rates and potential products of decaBDE debromination will depend upon the medium in/on which it is present, and the rate of various degradation processes (e.g., photodegradation, abiotic degradation, biodegradation). To illustrate the partitioning properties of decaBDE under various possible release scenarios, Level III fugacity modelling using the CHEMCAN model (Webster et al. 2004) was conducted and

parameterized for the "Ontario Mixed Wooded Plain" region. The primary model parameter inputs included

- log K_{ow} = 8.7 (Wania and Dugani 2003; see Appendix C).
- log K_{oa} = 12 (maximum value allowed in model, indicating that decaBDE is essentially non-volatile; estimated values for decaBDE exceed 15).
- degradation rates = negligible in air, water, soil and sediment.[7]

Based on its chemical properties, decaBDE is expected to be primarily sorbed to organic fractions. Table 3-1 summarizes the predicted fractions in each medium for releases to air, water or soil. From Table 3-1 it is apparent that

1. A large fraction (> 96%) of decaBDE in the environment is expected to be associated with either soils or sediments (depending on whether the release is to soil or the aquatic environment) and, within these bulk compartments, decaBDE is associated almost entirely with the solid phase.
2. Although < 3.4% of decaBDE in the environment is expected to be associated with bulk air or bulk water phases, it is expected that of the mass of decaBDE present in bulk air or bulk water > 95% will be sorbed to either aerosol or suspended sediment.
3. Regardless of the release scenario, less than 0.1% of decaBDE in the environment is predicted to be found dissolved in water or in the vapour phase.

Thus, studies that examine the debromination of decaBDE present in soils and sediments or sorbed to particulate and/or biota phases in air or water likely give the best indication of the potential significance of a transformation process in the environment. For studies that examine the debromination of decaBDE in solution or in vapour phase, the significance of transformation is less certain and is likely low since the dissolved/vapour fraction of decaBDE in the environment is predicted to be extremely low. According to Stapleton (2006a),

> *In the environment, it is expected that BDE 209 will be found primarily bound to solids in the water column, and bound to particles in the atmosphere. Therefore, degradation of BDE 209 dissolved in water (or organic solvents) is not expected to be of environmental relevance.*

A further consideration for evaluating the available information on debromination in the environment is how well the experimental conditions represent those that would be expected under natural settings. For example, in experiments that use organic solvents to disperse or dissolve decaBDE, extrapolation to normal environmental conditions is difficult since artificial organic solvents used in laboratory studies are not commonly found in the environment and the solubility of decaBDE in water is much lower than that of organic solvents. Furthermore, organic solvents are often much stronger hydrogen donors than water or other naturally occurring substances, resulting in faster rates and potentially non-representative patterns of decaBDE transformation. In the environment, many different donor molecules would be present, and thus substitution of bromine by

[7] While some degree of transformation is likely to occur in the environment, the intent of the model simulation was to examine the partitioning properties of decaBDE.

hydrogen is unlikely to be the sole route of transformation. This creates uncertainty as to how well degradation studies that use organic solvents would represent expected degradation of decaBDE in the environment. Other experimental conditions also require scrutiny to ensure relevance to those that may be seen in the environment. For instance, experiments using natural sunlight or simulated natural sunlight are likely to better represent natural conditions than those using artificial light. It is also not likely that materials such as zerovalent iron nanoparticles, specialized microbial cultures and fungal cultures will be found in great abundance in the environment.

In general, laboratory studies examining decaBDE transformation were frequently conducted under conditions that were favourable for decaBDE debromination and are not necessarily representative of typical conditions in the environment. Thus, while some transformation of decaBDE in the environment is plausible, data showing accumulations of decaBDE in the environment (e.g., see Canada 2006; Environment Canada 2006b) also imply recalcitrance.

Table 3-1: Predictions of the CHEMCAN Model for decaBDE in the "Ontario Mixed Wooded Plain" region

	1 kg/year to water[1]	1 kg/year to soil[1]	1 kg/year to air[1]
Mass in bulk air (kg)	1.27E-07	2.17E-07	2.23E-03
Fraction of total mass in bulk air	1.46E-06%	7.64E-08%	3.70E-03%
In bulk air, fraction as vapour	0.21%	0.21%	0.21%
In bulk air, fraction sorbed to aerosol	99.79%	99.79%	99.80%
Mass in bulk water (kg)	0.31	0.31	9.49E-02
Fraction of total mass in bulk water	3.38%	0.11%	0.16%
In bulk water, fraction dissolved in water	0.12%	0.01%	0.12%
In bulk water, fraction sorbed to suspended particles	95.25%	95.35%	95.25%
In bulk water, fraction accumulated in fish	4.63%	4.63%	4.64%
Mass in bulk soil (kg)	3.32E-03	274.36	57.59
Fraction of total mass in bulk soil	0.00%	96.80%	95.35%
In bulk soil, fraction in gas phase in air spaces	0.00%	0.00%	0.00%
In bulk soil, fraction dissolved in soil water	0.00%	0.00%	0.00%
In bulk soil, fraction sorbed to soil solids	100.00%	100.00%	100.00%
Mass in bulk sediment (kg)	8.78E+00	8.78	2.71
Fraction of total mass in bulk sediment	96.60%	3.10%	4.49%
In bulk sediments, fraction dissolved in porewater	0.00%	0.00%	0.00%
In bulk sediments, fraction sorbed to sediment solids	100.00%	100.00%	100.00%

Notes:
[1] Releases to other media are assumed to equal zero.

Figure 3-1 integrates the available information regarding possible environmental debromination pathways for decaBDE in a conceptual model while Table 3-2 provides a list of the products which are potentially formed from each debromination process. A tabulated summary of the environmental debromination information is provided in Appendix E.

Studies focusing on decaBDE sorbed to particulates or solid phases conducted in the absence of organic solvents, with natural sunlight, may provide a realistic indication of decaBDE transformation in the environment. Figure 3-2a summarizes the photodegradation pathways for decaBDE sorbed to particulate or solid phases. DecaBDE (i.e., BDE209) sorbed to dust or other dry minerals and particulates appears susceptible to relatively rapid transformation, with half-lives ranging from 76 min (decaBDE sorbed to a thin film of kaolinite; Gerecke 2006) to 408 h (decaBDE sorbed to house dust; Stapleton and Dodder 2006). Transformation appears to follow stepwise reductive debromination to form hexa- to nonaBDEs. A few studies also indicated alternative reaction pathways to either tetra- and pentaBDFs or unidentified products.

DecaBDE (i.e., BDE209) sorbed to particulates in aqueous systems appears to photodegrade at a generally slower rate, with half-lives ranging from 73 minutes (decaBDE sorbed to a thin film of kaolinite in presence of water; Gerecke et al. 2006) to 990 d (decaBDE sorbed to sediment organic carbon and exposed to sunlight; Ahn et al. 2006a). In addition, some studies have also demonstrated significant degradation (up to 71%) over 60–72 h. The identified products included hexa- to nonaBDEs and some lower brominated PBDEs. The potential for photolytic transformation of the sorbed decaBDE appears to be modulated by the ability of sunlight to penetrate water and the sorbing matrix. Gerecke et al. (2006) determined that light penetrated only 50 μm into kaolinite, suggesting that the photodegradation of decaBDE would be mainly limited to that amount of the chemical present on exposed surfaces. In surface waters, sunlight reaching suspended and bottom sediments would also experience some level of attenuation. Thus, it can be concluded that while photodegradation on solid phases can occur at significant rates, only a very small fraction of the total decaBDE in the environment (i.e., adsorbed to particulates or on solid surfaces) which has contact with sunlight would be susceptible to photodegradation. Also, depending on matrix characteristics (e.g., shielding capacity) and level of exposure to sunlight, photodegradation will be limited.

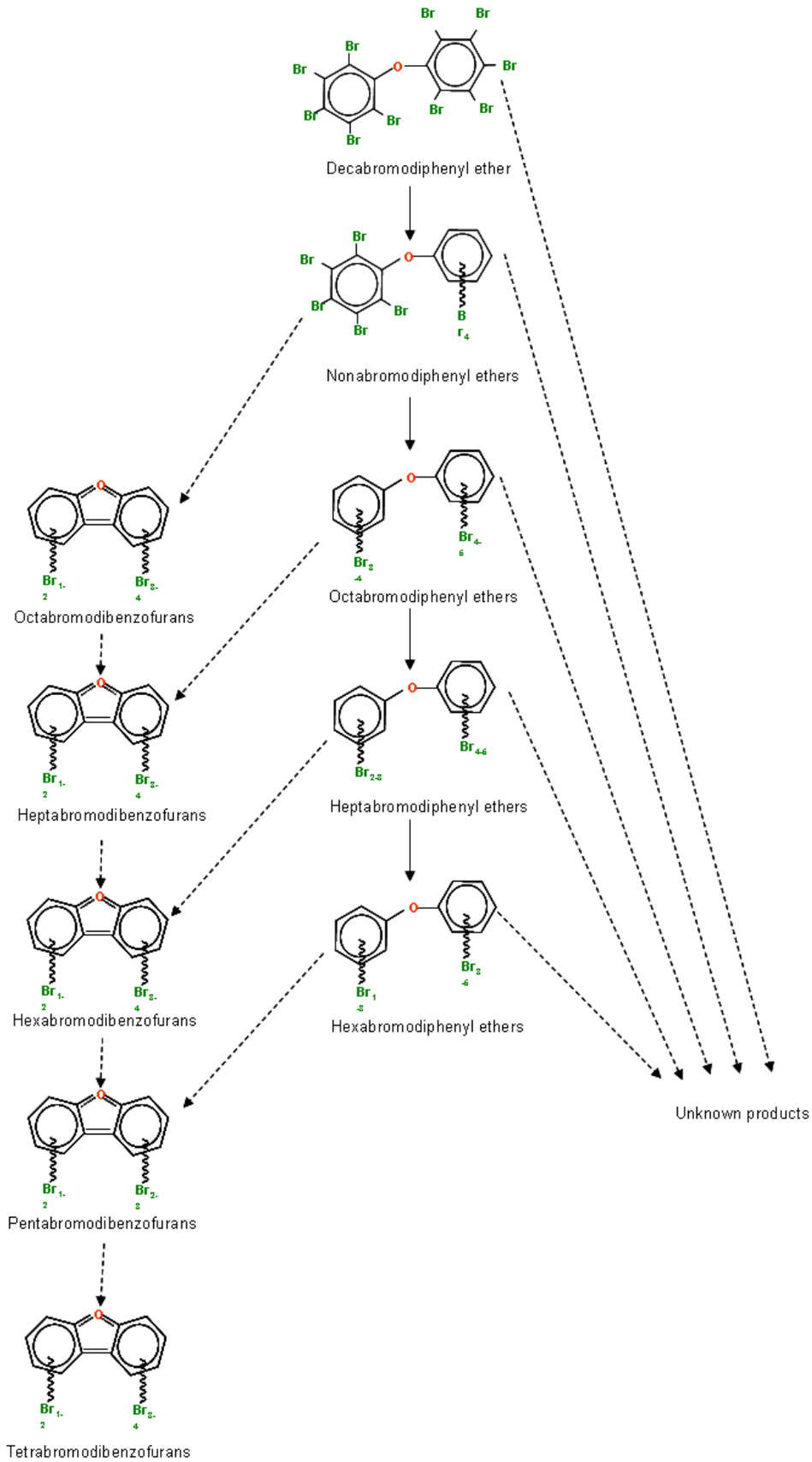
In addition to photodegradation, decaBDE sorbed to solids may also be subject to biodegradation processes in aquatic sediments, soils or WWTPs. Figure 3-2b summarizes biodegradation pathways for decaBDE. The results of biodegradation studies are somewhat mixed. Early studies (MITI 1992; CMABFRIP 2001) focused on decaBDE mineralization and these indicated very little, if any, degradation; however, these studies did not specifically determine the production of potential transformation products. Tokarz et al. (2008) showed that decaBDE in natural sediment microcosms had a half-life that ranged from approximately 6 to 50 years, with an average of 14 years. In laboratory studies using activated sludge, Gerecke et al. (2005, 2006) determined half-lives ranging from 693 to 1400 d depending on the presence/absence of primer, and identified debromination products as octa- and nona-BDEs. In a separate study, He et al. (2006)

observed complete transformation of decaBDE to hepta- and octaBDEs over 2 months with one anaerobic culture (*Sulfurospirillum multivorans*) but negligible degradation with other anaerobic cultures. The monitoring studies of WWTP sludge (Knoth et al. 2007; La Guardia et al. 2007) provide little evidence of decaBDE debromination in WWTPs, possibly because the residence time in WWTPs is too short for significant debromination to be observed. In addition, sediment microcosm studies by Parsons et al. (2004, 2007) fail to provide evidence of significant debromination, although Parsons et al. (2007) did identify small amounts of nonaBDEs. Thus, while the experimental conditions of the activated sludge studies are environmentally relevant, it is possible that the rates of degradation are too slow, or the cultures used are too specific, for the observed debromination to be significant in the environment. Overall, it appears that photodegradation may be more significant than biodegradation for decaBDE sorbed to solids.

Figure 3-2: Conceptual model of possible environmental transformation pathways for decaBDE

A) Photodegradation on Particulates (Dust, Soil, Aerosol and Sediment) in Dry and Aqueous Systems

- Photodegradation of decaBDE sorbed to dust, aerosol, suspended sediment and other solids occurs faster than biodegradation but slower than photodegradation in solution, likely due to limited light penetration into solid surfaces.
- >95% of decaBDE in bulk air and bulk water will be sorbed to either aerosol or suspended sediment.
- However, <3.4% of decaBDE in the environment is expected to be associated with bulk air or bulk water, reducing the significance of photodegradation on solid phases within these compartments.
- DecaBDE sorbed to dust and other surfaces indoors, outdoors and in automobiles could have significant light exposure and potentially significant photodegradation.
- DecaBDE at soil surfaces could also be subject to light exposure and photodegradation.
- Photodegradation of decaBDE sorbed to solid phases may be the most important degradation pathway in the environment.
- Other pathways leading to unidentified products could also occur, including further debromination/transformation of PBDEs or PBDFs and ring cleavage.



B) Biodegradation

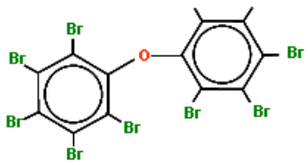
•Biodegradation occurs slowly in activated sludge experiments and may be limited in sediments and soils with less ideal biodegradation conditions.

•However, a large fraction (>96%) of decaBDE in the environment is expected to be associated with either soils or sediments, depending on the compartment of release.

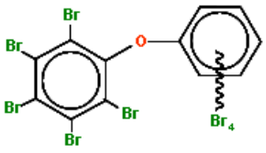
•In soil and sediments, biodegradation will be the primary transformation pathway since light penetration would be limited, reducing photodegradation.

•Given the low rates of biodegradation, the significance of these pathways in the environment as a whole is uncertain.

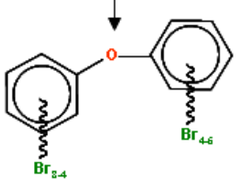
•Although not shown on the figure, it is also possible that other, unidentified products may be formed including substances formed via pathway of ring cleavage.



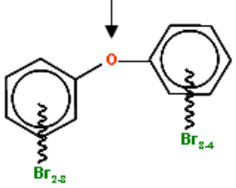
Decabromodiphenyl ether



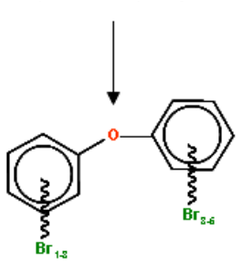
Nonabromodiphenyl ethers



Octabromodiphenyl ethers



Heptabromodiphenyl ethers



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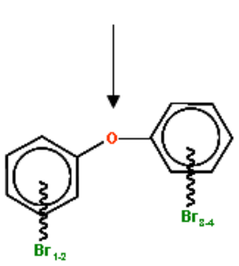
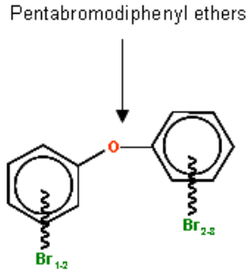


Pentabromodiphenyl ethers

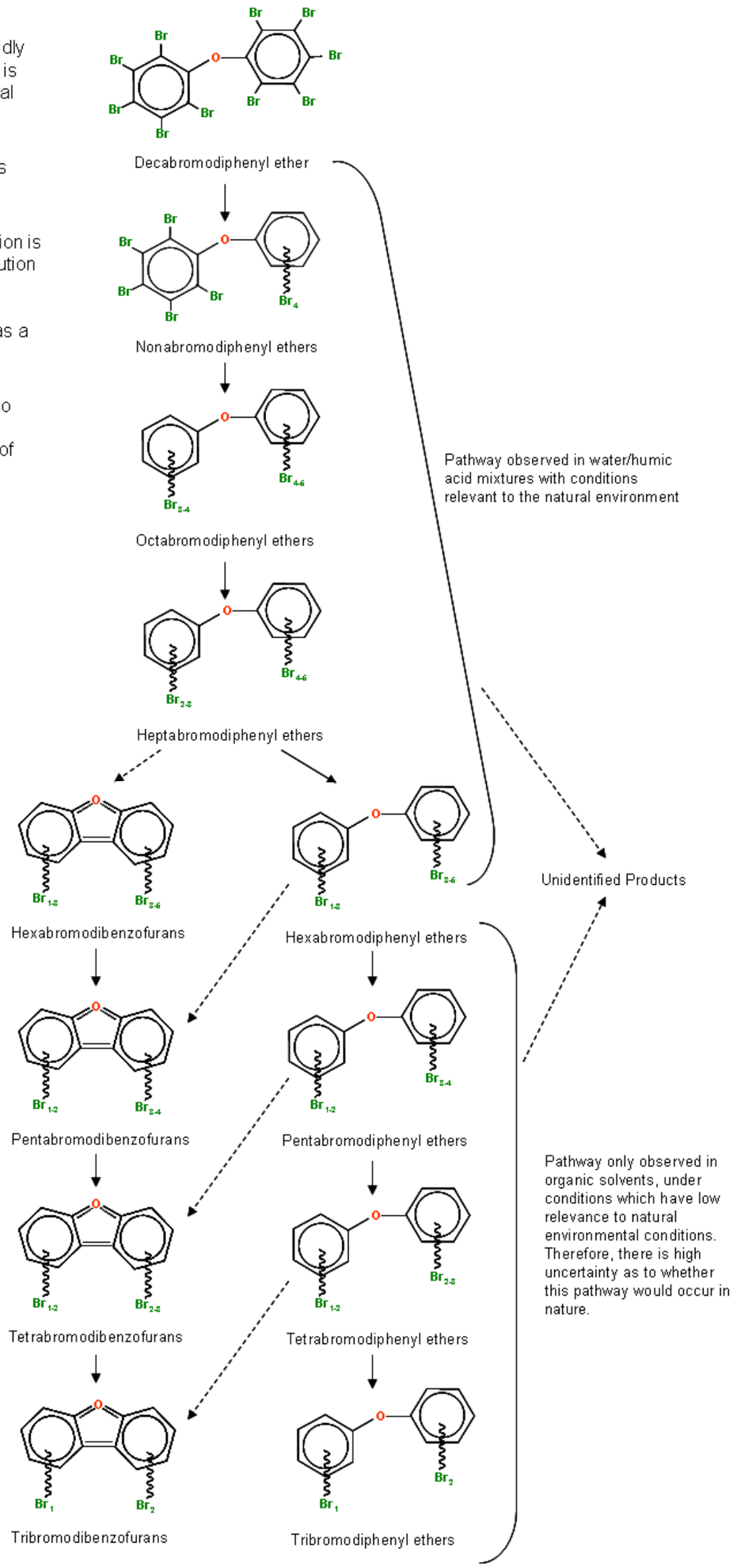


Tetrabromodiphenyl ethers

Lower BDEs

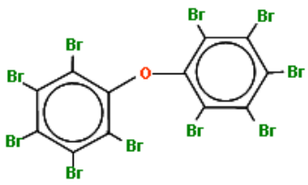
(C) Photodegradation in Solution (Natural and Organic Solvents)

- Photodegradation occurs rapidly for decaBDE in solution which is irradiated by sunlight or artificial light.
- However, less than 0.1% of decaBDE in the environment is predicted to be dissolved in solution.
- Therefore, although the reaction is rapid, photodegradation in solution is not expected to result in significant debromination of decaBDE in the environment as a whole.
- Other pathways leading to unidentified products could also occur, including further debromination/transformation of PBDEs or PBDFs and ring cleavage.

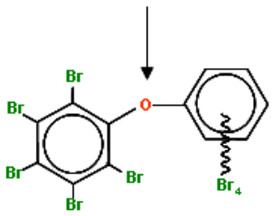


D) Abiotic Degradation (Other than Photodegradation)

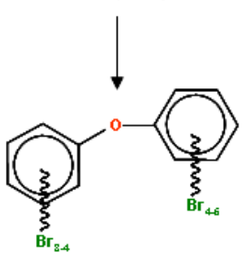
- Abiotic degradation may occur for decaBDE in the presence of reducing agents or sorbed to minerals in the presence of natural hydrogen donors such as catechol (although the observed degradation in the presence of catechol is slow). Most laboratory studies demonstrating abiotic degradation (other than photolysis) appear to have limited relevance to the environment.
- Hydrolysis has also been demonstrated but it is unknown whether this would occur under normal environmental conditions.
- Because there are relatively few studies of abiotic degradation, the significance of abiotic processes is highly uncertain.
- It is possible that abiotic degradation could be a significant, albeit slow, process in soils and sediments where light does not penetrate and biodegradation occurs slowly.
- Other pathways leading to unidentified products could also occur, including ring cleavage.



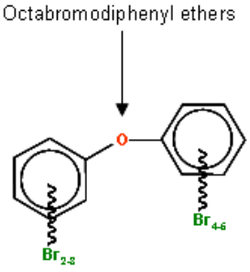
Decabromodiphenyl ether



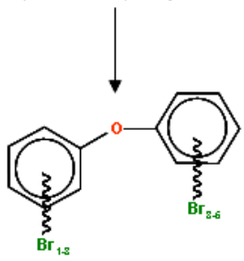
Nonabromodiphenyl ethers



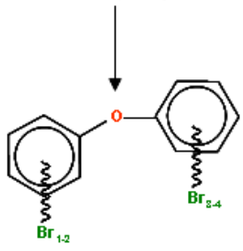
Octabromodiphenyl ethers



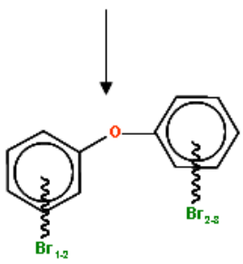
Heptabromodiphenyl ethers



Hexabromodiphenyl ethers



Pentabromodiphenyl ethers



Tetrabromodiphenyl ethers

Lower BDEs

Table 3-2: Summary of decaBDE transformation products observed in laboratory studies with some relevance to environmental settings

		DecaBDE Transformation Process				
Congener Group Formed	**Targeted for Virtual Elimination under CEPA 1999**	**Photodegradation Sorbed to Solids in Dry and Aqueous Systems** (references in brackets)	**Photodegradation Dissolved in Natural Solvents** (references in brackets)	**Photodegradation Dissolved in Artificial Organic Solvents**** (references in brackets)	**Biodegradation** (references in brackets)	**Abiotic Degradation** (references in brackets)
nonaBDEs		X (2,3***,4,9,10,11, 21)	X (8)	X (1,5,6,7,8,19)	X (14,15,16, 21† & ††)	X (12,13**, 20**)
octaBDEs		X (2,3***,4,9,10,11, 21)	X (8)	X (1,5,6,7,8,19)	X (14,15, 21† & ††)	X (12,13**, 20**)
heptaBDEs		X (2,3***,9,10,11, 21)	X (8)	X (1,5,6,7,8,19)	X (15,21† & ††)	X (12,13**)
hexaBDEs	X	X (2,3***,9,21)	X (8)	X (1,5*,6,7*)	X (21† & ††)	X (12,13**, 20**)
pentaBDEs	X	X (9*)		X (1,6)	X (21††)	X (12,13**, 20**)
tetraBDEs	X	X (9*)		X (1,6)	X (21††)	X (12,13**, 20**)
triBDEs		X (9*)		X (1,6)	X (21††)	X (20**)
pentaBDFs		X (21)				
heptaBDFs		X (21)				
hexaBDFs		X (21)		X (18)		
triBDFs		(2,21)		X (8,18)		
tetraBDFs		X (2,21)		X (8,18)		
triBDFs		(21)		X (8,18)		
diBDFs				X (18*)		
monoBDFs				X (18*)		
Unidentified products		X (10,11,21,17)		X (5)		

Notes:

* Indicates that only trace or small amounts were formed.

** Due to the use of artificial solvents, these results are considered to have low relevance to the environment.

***Identification of debromination products was largely inconclusive, although there was some evidence of the formation of hexa- to nonaBDEs.

†In natural sediments.

††In cosolvent enhanced biomemetic system.

BDEs – brominated diphenyl ethers

BDFs – brominated dibenzofurans

The results of Gerecke (2006) have not been included because the decaBDE degradation products were described as lower brominated PBDEs, with no indication of which PBDEs were formed.

The results of Parsons et al. (2004) have not been included because degradation to nona- and lower brominated PBDEs was observed in both the control and treatment groups.

References

1. Watanabe and Tatsukawa 1987
2. Söderstrom et al. 2004
3. Jafvert and Hua 2001
4. Hua et al. 2003
5. Palm et al. 2003
6. Bezares-Cruz et al. 2004
7. Eriksson et al. 2004
8. Geller et al. 2006
9. Ahn et al. 2006a
10. Stapleton and Dodder 2006
11. Stapleton 2006b
12. Keum and Li 2005
13. Ahn et al. 2006b
14. Gerecke et al. 2005
15. He et al. 2006
16. Gerecke et al. 2006
17. Gerecke 2006
18. Hagberg et al. 2006
19. Barcellos da Rosa et al. 2003
20. Tokarz et al. 2008
21. Kajiwara et al. 2008

Although photodegradation experiments with decaBDE dissolved in water potentially represent environmentally relevant conditions, the significance of any observed debromination is uncertain since only a very low fraction of decaBDE would be present in the dissolved phase. Figure 3-2c provides a conceptual model of photodebromination pathways for decaBDE dissolved in water/solvent systems. Two of the associated studies are notable because the results are potentially relevant to the natural environment. Eriksson et al. (2004) observed relatively rapid transformation (half-life = 6.4 h) of decaBDE in water/humic acid mixtures with hexa- to nonaBDEs formed. Because artificial light was used, there is some uncertainty whether the rate would be as fast in natural sunlight. Kuivikko et al. (2006, 2007) observed a photodegradation half-life of 0.3 d for decaBDE in isooctane but conducted modelling to estimate half-lives ranging from 0.2 to 1.8 d in Baltic Sea and Atlantic Ocean surface waters.

Laboratory-based studies on the transformation of decaBDE provide support for a conclusion that transformation to lower BDEs and BDFs should be occurring in the environment. However, there is a dichotomy between the findings of these studies and the results of monitoring studies in terms of realistic support for significant transformation of decaBDE in the environment. If these laboratory-based studies provide accurate depictions of transformation in the environment, one would expect to find similarities in the patterns observed in the environment and those described as transformation products of decaBDE in laboratory studies. One would expect to see widespread occurrence of the decaBDE transformation products in various environmental media, such as sediments. The Canadian screening assessment on PBDEs (Canada 2006) showed how monitoring studies are dominated by congener profiles that are consistent with the PentaBDE and OctaBDE commercial mixtures. The United Kingdom (2007a) notes that the available monitoring data provide limited circumstantial evidence for a correlation between concentrations of decaBDE and lower brominated PBDEs in some environmental matrices (e.g., citing the findings of Voorspels et al. (2006a, 2007b). The United Kingdom (2007a) also notes that, in the study by Gerecke et al. (2005), biodegradation appeared to preferentially remove the para-position bromines from the BDE molecule, but that these PBDEs are not routinely observed in the environment.

Besides photodegradation and biodegradation, decaBDE is susceptible to abiotic debromination in the absence of light exposure as shown under laboratory conditions. Figure 3-2d summarizes the transformation pathways for these other abiotic degradation processes. Keum and Li (2005) observed up to 90% transformation of decaBDE dissolved in deionized water over 40 d in the presence of reducing agents such as zerovalent iron, iron sulphide and sodium sulphide. Li et al. (2007) showed complete disappearance after 8 h in a water-acetone system containing nanoscale zerovalent iron, while Rahm et al. (2005) observed a half-life of 0.028 h for decaBDE dissolved in methanol and reacted with sodium methoxide. Similar to photodegradation studies using organic solvents, there is uncertainty as to how well these studies represent natural conditions and whether such processes may take place under natural conditions.

3.2.3 Implications for Bioaccumulation Assessment

The existing laboratory evidence for the transformation and debromination of decaBDE through environmental processes such as photodegradation, abiotic degradation and biodegradation indicates potential transformation pathways which lead to the formation of

1. lower brominated PBDEs;
2. brominated dibenzofurans; and
3. unidentified degradation products.

However, the laboratory studies also indicate that both the lower brominated PBDEs and PBDFs would, themselves, be susceptible to further degradation and the actual significance of the formation of these products is unknown. Palm et al. (2003) and Eriksson et al. (2004) have shown that the rate of transformation of PBDEs decreases as the number of bromine atoms/molecule decreases. With decreasing bromination, diphenyl ethers have a lessened overlap with absorption spectra of wavelengths >290 nm, which is the range of the solar spectrum at ground level, and thus it is expected that they would be less susceptible to decay than decaBDE. Since lower brominated PBDEs demonstrate slower rates of phototransformation, it could be speculated that in a system with a single input of decaBDE, a steady-state build-up of lower brominated PBDEs could occur. However, some level of continued photodegradation down to the biphenyl ether could also likely occur. The magnitude of the build-up would be difficult to predict since the formation and subsequent degradation of PBDEs depends on their relative rates of formation and degradation in the environment, both of which are not known. In addition, the magnitude of accumulation would depend on decaBDE loading rates to the environment, which are also difficult to predict (United Kingdom 2004).

In order to evaluate whether these decaBDE transformation products have the potential to bioaccumulate or biomagnify in food webs, the BAF and BMF models from Section 3.1 will be used (see Appendix D for model input values). This analysis assumes that the transformations of decaBDE observed in the laboratory studies discussed in this report are also occurring in the natural environment; however, it is acknowledged that this is a subject of uncertainty. As noted earlier, in the absence of metabolism, the BAFs of hexa- to nonaBDEs are predicted to exceed 5000. When metabolic transformation is considered (a more realistic scenario), BAF estimates for hexaBDEs to nonaBDEs still exceed 5000, but are orders of magnitude lower.

BAF estimates for brominated dibenzofuran products were also undertaken. For these transformation products, there was no information that would allow for estimation of k_M, and a value equal to that estimated for decaBDE was chosen for illustrative purposes. It is likely that PBDFs would undergo further metabolism; however, the actual rates of metabolism are unknown. Although the chosen value and resulting BAFs represent reasonable hypothetical predictions, it is important to acknowledge that these predictions are uncertain. Further detail of log K_{ow} and k_M estimates are provided in Appendix D. In the absence of metabolism, the BAFs for penta- and hexaBDFs are predicted to exceed 5000 for the middle trophic level fish, while BAF predictions for tri- and tetraBDFs are

below 5000. With consideration given to metabolic transformation (a more realistic scenario), the status of the BDFs with respect to the BAF criterion of 5000 remains unchanged, but values are somewhat lower overall.

In the absence of metabolism, the BMFs of hexa- to nonaBDEs are predicted to be very high, ranging from ~130 to ~150. However, when metabolic transformation (a more realistic scenario) is considered, the predicted BMFs for hexa- to nonaBDEs ranged from 5 to 6. The metabolism-corrected BMFs exceeded 1 for all metabolites, primarily due to a relatively high dietary assimilation efficiency calculated for these substances. For the BDF products, a similar BMF of 120 to 150 for penta- and hexaBDFs was predicted, while BMFs predictions for tri- and tetraBDFs were 8 and 45, respectively. When corrected for metabolism, the BMF values ranged from 4 to 6 (a similar k_M value was used for all BDFs).

The modelling analysis of potential environmental transformation products of decaBDE predicted BAFs that predominantly exceed 5000 and BMFs exceeding 1 for practically all of the metabolites, because of relatively high dietary assimilation efficiency and optimal $\log K_{ow}$ and $\log K_{oa}$ values for bioaccumulation via passive diffusion. This suggests concerns that the transformation products formed in the environment as a result of the photodegradation, biodegradation, and possibly abiotic degradation, of decaBDE, are potentially bioaccumulative and biomagnifying in food webs, and may result in increased exposure and risk to upper trophic level organisms.

4 Summary and Conclusions

The existing evidence for the bioaccumulation of decaBDE does not support a conclusion of "bioaccumulative" as set out in the *Persistence and Bioaccumulation Regulations*. While most available data show that decaBDE has limited potential to bioaccumulate or biomagnify in the environment, some evidence suggests a higher BAF than previously considered for decaBDE, and some new data suggest possible biomagnification. Some studies also show that levels of decaBDE are steadily rising in some biota, and in some cases, measured concentrations have reached levels that can be subjectively described as high. The modelling undertaken to support this evaluation, however, shows uncertainty associated with metabolism in fish as model-predicted aquatic BAFs range from below the 5000 criterion to well above 5000. Predicted terrestrial carnivore BMF values also range from below 1 to greater than 1 depending on the rate of metabolism assumed. Although less relevant than BAF or BMF, experimental BCF measures are below the 5000 criterion. The substance is shown to be increasing in concentrations in some wildlife species, and some data suggest that decaBDE has reached concentrations in some organisms interpreted to be high. Potential factors limiting the bioaccumulation potential of decaBDE include low assimilation efficiency and/or metabolic transformation.

There is still some uncertainty respecting rates and pathways for metabolic transformation organisms. Debromination has been indicated in both mammalian and fish studies, but the amounts of debrominated PBDE product formed is typically very low

(e.g., representing less than 1%, up to a few percent of the total dose of decaBDE depending on the study). Formation of nona-, octa- and heptaBDEs has been noted for mammals. In addition to these congener groups, hexa- and pentaBDEs have been noted as bioformed in fish. However, the interpretation of results from metabolic transformation studies is at times complicated by incomplete characterization of impurities in the dosed material and/or dosage of many PBDEs concurrently. Some rodent studies have made inferences, based on mass balance evaluations, that rates of transformation may be higher, with one study suggesting that approximately 45% of the total dose of decaBDE was unaccounted for and may have been metabolized to other compounds (like hydroxylated and hydroxymethoxylated PBDEs) and/or bound as inextricable residues.

With respect to chemical transformation in the environment, this review supports the findings of the Ecological Screening Assessment of PBDEs (Environment Canada 2006a, 2006b), which identified photodegradation and biodegradation as likely mechanisms for transformation in the environment. This review also identifies various new studies that quantify rates of degradation and propose chemical transformation pathways. Together, the new and existing studies provide evidence which makes it plausible to conclude that decaBDE is transforming in the environment.

This evaluation found that decaBDE that is sorbed to dry minerals and particulates appears to undergo relatively rapid phototransformation in the presence of sunlight. In addition, decaBDE sorbed to solids may be subject to biodegradation; however, it appears that this process is occurring at a much slower rate than photodegradation. While photodegradation on solid phases can occur at significant rates, only a very small fraction of the total decaBDE reservoir in the environment (e.g., that fraction adsorbed to particulates or on solid surfaces) that has contact with sunlight would be susceptible to photodegradation. Based on fugacity modelling, < 3.4% of decaBDE in the environment is expected to be associated with bulk air or bulk water phases with potential exposure to sunlight. DecaBDE sorbed to solid surfaces (both anthropogenic and natural) could also be exposed to sunlight. Within these phases, light attenuation and matrix shielding would affect overall exposure to sunlight and potential for photodegradation. While most decaBDE in the environment would partition to sediment and soils (fugacity modelling predicts > 96%), biodegradation has been shown to be a very slow process, with half-lives on the scale of a few years to several decades. Some studies have also not shown any obvious transformation in sediments or soil even after several decades. Hence, it is apparent that evidence of transformation in the environment could be very subtle and shielded by existing PBDE congener patterns dominated by the commercial PBDE products. As well, the infrequent historic analysis of higher brominated PBDEs like octa- and nonaBDEs could make it difficult to detect or confirm transformation.

Based on the available laboratory studies with conditions relevant to the environment, it is reasonable to expect that decaBDE may be transformed in the environment, leading to the formation of lower brominated PBDEs, PBDFs and other unknown products. A number of studies have now shown that decaBDE may transform by photodegradation or biodegradation to nona -, octa-, hepta- and hexaBDEs. One study also indicates trace

formations of penta- and tetraBDEs by photodegradation, while another study also indicates biodegradation to penta- and tetraBDEs under enhanced biodegradation conditions. In addition, photodegradation of decaBDE has also been shown to form tri- to octaBDFs as well as unidentified products.

Modelling of BAFs and BMFs played an important supplemental role in this review and was used to suggest whether decaBDE and its transformation products may be bioaccumulative or biomagnify in food chains.

The metabolism-corrected model predicted aquatic BAFs for decaBDE ranged from below the 5000 criterion of the *Persistence and Bioaccumulation Regulations* to well above 5000. The predictions demonstrate the uncertainty associated with the metabolism potential of decaBDE in fish and log K_{ow} determinations for this substance. Given the exceptionally low water solubility limit of decaBDE, it is not expected that this substance will be appreciably taken up from the water phase by aquatic organisms. Although less relevant than BAF or BMF, experimental BCF measures are below the 5000 criterion for decaBDE. With consideration given to decaBDE metabolism, terrestrial BMF predictions (based on a wolf food chain) indicate lack of or a low level of biomagnification of decaBDE.

In the absence of metabolism, the BAFs of all potential metabolites and transformation products of decaBDE were predicted to exceed 5000. When assumptions were made respecting metabolic transformation (a more realistic scenario), almost all proposed transformation products still yielded BAFs exceeding 5000. In the absence of metabolism, the BMFs of potential transformation products are also predicted to be very high; however, when assumptions were made respecting metabolic transformation, the predicted BMFs are significantly lower, but are still all greater than one. This analysis suggests that many possible decaBDE metabolites/transformation products could be highly bioaccumulative and some metabolites may have the capacity to biomagnify in food chains.

Overall, this review confirms that, based on the reviewed materials published up to August 25, 2009, decaBDE is not shown to meet bioaccumulation criteria as defined under the *Persistence and Bioaccumulation Regulations* under CEPA 1999. However, some studies show that levels of decaBDE are steadily rising in some biota, and in some cases, measured concentrations are considered high. In addition, some equivocal evidence suggests potential biomagnification in food chains. Although uncertainties remain, it is reasonable to conclude that decaBDE may also contribute to the formation of bioaccumulative and/or potentially bioaccumulative transformation products, such as lower brominated BDEs, in organisms and the environment.

4.1 Consideration Regarding Related Products

While this review has focused on decaBDE, its analyses and conclusions are relevant to alternative flame retardants with similar chemical structures and use patterns. For instance, decabromodiphenyl ethane (or decaBD ethane)—(1,2-

bis(pentabromodiphenyl)ethane; 1,1”-(ethane-, 1,2-diyl) bis[pentabromobenzene])—is a replacement for the decaBDE commercial product, having the same or similar applications. Both are additive flame retardants used in HIPS and in textiles used in the manufacture of television cabinets, cable insulation and adhesives (Kierkegaard 2007). In Japan, Watanabe and Sakai (2003) have shown that there has been a clear shift in consumption away from the decaBDE commercial product to decaBD ethane.

The only structural difference between decaBD ethane and decaBDE is the carbon bond between the aromatic rings of decaBD ethane (for decaBDE, the aromatic rings are linked with an oxygen atom) (see Appendix F). Based on structural similarities, the two substances likely have similar physical-chemical properties, characteristics of persistence, transformation, and accumulation in organisms (Kierkegaard 2007).

DecaBD ethane has been identified in sewage sludge, both in Canada (Konstantinov et al. 2006) and in Spain (Eljarrat et al. 2005), has been measured in walleye and burbot in Lake Winnipeg (Law et al. 2006), and detected in herring gull eggs from the Great Lakes area of Canada (Letcher et al. 2007). The United Kingdom Environment Agency recently published a detailed risk assessment for this substance (United Kingdom 2007b). While direct risks resulting from toxic effects of this substance were considered low, concerns were identified over this substance’s potential to accumulate in wildlife and transformation to other chemical products. The Agency also identified a need for further work on decaBD ethane to confirm the findings of their assessment, particularly to provide more reliable measures of this substance’s potential to bioaccumulate and degrade in the environment.

Based on concerns expressed for decaBDE in this State of Science report, the similarity in properties between decaBDE and decaBD ethane, the presence of the decaBD ethane in Canadian wildlife, and the potential for decaBD ethane to be used as a large-scale replacement for decaBDE, there is a need to further understand the potential risks from decaBD ethane in the environment and its capacity to accumulate in wildlife and transform to bioaccumulative products. Understanding the risk from alternatives will help to ensure that substitutions of flame retardants are made on an informed basis.

5 Key Uncertainties and Data Gaps

The data for bioaccumulation, biomagnification and trophic magnification of decaBDE are limited, especially for top predators in natural settings. Additional ratio-based assessments of decaBDE in top predators in natural settings, particularly in terrestrial systems, would provide better understanding of the bioaccumulation and biomagnification of decaBDE.

Overall, the relative contribution of environmental transformation of decaBDE to the total loadings of lower brominated PBDEs (i.e., having nine or fewer bromine atoms) in the environment is not known. Studies which trace and/or “fingerprint” the source of lower brominated PBDEs in the environment would help clarify the significance of *in*

vivo and environmental debromination of decaBDE, compared to that resulting from the use of the Penta- and OctaBDE formulations.

It may be beneficial to have BAF or BMF determinations for decaBDE on the total burden of parent decaBDE and the metabolites that result from accumulation and transformation of the parent chemical (e.g., lower BDEs and PBDFs). In most of the studies conducted to date, this has been difficult to accomplish because (i) the lesser debrominated products were also present in the exposure medium (i.e., food or water) and their presence in tissue could thus have resulted from both accumulation and bioformation, or (ii) full identification and quantification of most metabolites has not been conducted.

Model-estimated BCFs, BAFs and BMFs presented in this report for decaBDE and known potential transformation products vary. These relied heavily on $\log K_{ow}$ $\log K_{oa}$ E_D and k_M values, which would benefit from refinement in order to improve the precision of the model predictions.

BAFs, BMFs and other indicators of bioaccumulation or biomagnification have not been measured for some of the known metabolites and transformation products of decaBDE, especially their hydroxylated and methoxyhydroxylated forms. Further study of the behaviour of these products would be useful.

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Appendix A – Biota Concentration Data

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				Detection Limit
							Lipid Weight	Wet Weight	Units	Statistic	
BIRDS OF PREY											
Chen et al. 2007	China	2004–2006	Common kestrel *Falco tinnunculus*	Muscle	6	21%	2150		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Common kestrel *Falco tinnunculus*	Liver	6	21%	2870		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Common kestrel *Falco tinnunculus*	Kidney	6	21%	483		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Sparrowhawk *Accipiter nisus*	Muscle	11	21%	192		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Sparrowhawk *Accipiter nisus*	Liver	11	21%	249		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Sparrowhawk *Accipiter nisus*	Kidney	11	21%	83		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Japanese sparrowhawk *Accipiter gularis*	Muscle	6	21%	75		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Japanese sparrowhawk *Accipiter gularis*	Liver	6	21%	78		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Japanese sparrowhawk *Accipiter gularis*	Kidney	6	21%	33		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Little owl *Athene noctua*	Muscle	6	21%	150		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Little owl *Athene noctua*	Liver	6	21%	96		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Little owl *Athene noctua*	Kidney	6	21%	40		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Scops owl *Otus sunia*	Muscle	6	21%	537		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Scops owl *Otus sunia*	Liver	6	21%	174		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Scops owl	Kidney	6	21%	59		ng/g	Mean	

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				
							Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
			Otus sunia								
Chen et al. 2007	China	2004–2006	Long-eared owl *Asio otus*	Muscle	6	21%	9.5		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Long-eared owl *Asio otus*	Liver	6	21%	107		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Long-eared owl *Asio otus*	Kidney	6	21%	23		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Upland buzzard *Buteo hemilasius*	Muscle	3	21%	21		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Upland buzzard *Buteo hemilasius*	Liver	3	21%	58		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Upland buzzard *Buteo hemilasius*	Kidney	3	21%	29		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Common buzzard *Buteo buteo*	Muscle	3	21%	26		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Common buzzard *Buteo buteo*	Liver	3	21%	71		ng/g	Mean	
Chen et al. 2007	China	2004–2006	Common buzzard *Buteo buteo*	Kidney	3	21%	93		ng/g	Mean	
Chen et al. 2008	United States, NE	1996–2006	Peregrine falcon *Falco peregrinus*	Eggs	114	0		1.4–420 (26)	ng/g	Range (median in brackets)	
de Boer et al. 2004	United Kingdom	Not given	Peregrine falcon *Falco peregrinus*	Liver	6	2 of 6	< 5.7–181	< 0.17–6.7	µg/kg	Range	0.08–9.8
de Boer et al. 2004	United Kingdom	Not given	Peregrine falcon *Falco peregrinus*	Muscle	5	None	53–344	1.8–9.5	µg/kg	Range	
de Boer et al. 2004	United Kingdom	Not given	Peregrine falcon *Falco peregrinus*	Egg	6	4 of 6	< 1.8–108	< 0.08–7.5	µg/kg	Range	Lipid:
de Boer et al. 2004	Sweden	Not given	Peregrine falcon *Falco peregrinus*	Egg	20	9 of 20	< 4–412	< 0.3–21	µg/kg	Range	1.5–200
de Boer et al. 2004	Sweden	Not given	Sparrowhawk *Accipiter nisus*	Liver	4	All	< 82 – < 200	< 3.2 – < 9.8	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Sparrowhawk *Accipiter nisus*	Muscle	5	None	13–275	0.26–2.2	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Sparrowhawk *Accipiter nisus*	Egg	5	2 of 5	< 2.1–38	< 0.16–1.5	µg/kg	Range	

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
de Boer et al. 2004	Sweden	Not given	Kestrel *Falco tinnunculus*	Liver	5	3 of 5	< 5.8–120	< 0.26–5.5	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Kestrel *Falco tinnunculus*	Muscle	5	4 of 5	< 4.2–10	< 0.11–0.29	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Barn owl *Tyto alba*	Liver	5	2 of 5	< 2.6–37	< 0.13–2.5	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Barn owl *Tyto alba*	Muscle	5	4 of 5	< 6.3–14	< 0.5–1.2	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Barn owl *Tyto alba*	Egg	4	1 of 4	< 20–30	< 2–1.7	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Montague's harrier *Circus pygargus*	Egg	4	1 of 4	< 2.1–28	< 0.12–1.3	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Marsh harrier *Circus aeruginosus*	Egg	2	All	< 2 – < 2.4	< 0.09	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Sea eagle	Egg	1	None	6.2	0.48	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Osprey *Pandion haliaetus*	Egg	3	All	< 4 – < 27	< 0.2 – < 1.5	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Golden eagle *Aquila chrysaetos*	Egg	5	All	< 4.1	< 0.2	µg/kg	Range	
de Boer et al. 2004	Sweden	Not given	Merlin *Falco columbarius*	Egg	2	1 of 2	< 43–4.3	< 3.8–0.3	µg/kg	Range	
Herzke et al. 2005	Northern Norway	1993–2000	Peregrine falcon *Falco peregrinus*	Egg	9		Detected		ng/g	Median	
Herzke et al. 2005	Northern Norway	1992–2002	Golden eagle *Aquila chrysaetos*	Egg	15		Detected		ng/g	Median	
Herzke et al. 2005	Northern Norway	1995–2000	Merlin *Falco columbarius*	Egg	9		Detected		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	Barn owl *Tyto alba*	Liver	7	Some	59		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	Barn owl *Tyto alba*	Muscle	7	Most	68		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	long-eared owl *Asio otus*	Liver	6	Most	66		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	Long-eared owl *Asio otus*	Muscle	6	All	< 2.25		ng/g	Detection limit	
Jaspers et al. 2006	Belgium	2003–2004	Buzzard	Liver	16	All	< 2.25		ng/g	Detection	

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
			Buteo buteo							limit	
Jaspers et al. 2006	Belgium	2003–2004	Buzzard *Buteo buteo*	Muscle	16	All	< 2.25		ng/g	Detection limit	
Jaspers et al. 2006	Belgium	2003–2004	Sparrowhawk *Accipiter nisus*	Liver	5	Some	52		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	Sparrowhawk *Accipiter nisus*	Muscle	5	All	< 2.25		ng/g	Detection limit	
Jaspers et al. 2006	Belgium	2003–2004	Kestrel *Falco tinnunculus*	Liver	5	Most	85		ng/g	Median	
Jaspers et al. 2006	Belgium	2003–2004	Kestrel *Falco tinnunculus*	Muscle	5	All	< 2.25		ng/g	Detection limit	
Kunisue et al. 2008	Japan (Coastal)	1994	Steller's sea-eagle *Haliaeetus pelagicus*	Muscle	1	1	<0.5		ng/g	Detection limit	
Kunisue et al. 2008	Japan (Inland)	1994–1995	Golden eagle *Aquila chrysaetos*	Muscle	2	1	<0.5–10		ng/g	Range	
Kunisue et al. 2008	Japan (Inland)	1997	Goshawk *Accipiter gentilis*	Muscle	1	0	120		ng/g		
Lindberg et al. 2004	Captive Sweden	1988–1999	Peregrine falcon *Falco peregrinus*	Eggs	4	2	< 7–9 (8.1)		ng/g	Range (geometric mean in brackets)	
Lindberg et al. 2004	Southern Sweden	1992–1999	Peregrine falcon *Falco peregrinus*	Eggs	8	1	< 20–430 (86)		ng/g	Range (geometric mean in brackets)	
Lindberg et al. 2004	Northern Sweden		Peregrine falcon *Falco peregrinus*	Eggs	8	0	28–190 (80)		ng/g	Range (geometric mean in brackets)	
Lindberg et al. 2004	Northern Sweden	1991–1999	Peregrine falcon *Falco peregrinus*	Eggs	18		110		ng/g	Mean	
Potter et al. 2009	United States, Maryland and Virginia	1993–2002	Peregrine falcon *Falco peregrinus*	Eggs	23	0		0.186–48.2 (6.35)	ng/g	Range (median in brackets)	
Voorspoels et al. 2006b	Belgium	2001–2003	Buzzard *Buteo buteo*	Brain	29	All			ng/g		
Voorspoels et al.	Belgium	2001–2003	Buzzard	Fat	16	All			ng/g		

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				Detection Limit
							Lipid Weight	Wet Weight	Units	Statistic	
2006b			*Buteo buteo*								
Voorspoels et al. 2006b	Belgium	2001–2003	Buzzard *Buteo buteo*	Liver	3	Most	19–190 (79)		ng/g	Range (mean)	
Voorspoels et al. 2006b	Belgium	2001–2003	Buzzard *Buteo buteo*	Muscle	29	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Buzzard *Buteo buteo*	Serum	20	Some	2–58 (15)		ng/g	Range (mean)	
Voorspoels et al. 2006b	Belgium	2001–2003	Owls	Brain	8	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Owls	Fat	1	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Owls	Liver	8	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Owls	Muscle	8	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Owls	Serum	3	Most	9		ng/g	Mean	
Voorspoels et al. 2006b	Belgium	2001–2003	Sparrowhawk *Accipiter nisus*	Brain	7	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Sparrowhawk *Accipiter nisus*	Fat	2	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Sparrowhawk *Accipiter nisus*	Liver	7	Most	15–19		ng/g	Range	
Voorspoels et al. 2006b	Belgium	2001–2003	Sparrowhawk *Accipiter nisus*	Muscle	7	All			ng/g		
Voorspoels et al. 2006b	Belgium	2001–2003	Sparrowhawk *Accipiter nisus*	Serum	2	None	16–36		ng/g	Range	
Vorkamp et al. 2005	Greenland	1986–2003	Peregrine falcon *Falco peregrinus*	Eggs	37	0	3.8–250 (11)		ng/g	Range (median)	
de Boer et al. 2004	Sweden	Not given	Red kite *Milvus milvus*	Eggs	4	3 of 4	< 2.1–29.1	< 0.09–2.3	µg/kg		
FRESHWATER FISH AND INVERTEBRATES											
Hale et al. 2002	United States; mid-Atlantic region	2000	Bluegill *Lepomis macrochirus*	Muscle					µg/kg		

Study Authors	Location	Date	Species	Tissue	Concentration Data						
					Sampling N	Not Detected	Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
Hale et al. 2002	United States; mid-Atlantic region	2000	Bluegill *Lepomis macrochirus*	Remainder of body					µg/kg		
Eljarrat et al. 2007	Spain	Nov. 2004, Nov. 2005	Carp *Cyprinus carpio*	Muscle	2	None	63.7–95.3		ng/g	Range	2–19 pg/g ww
			Barbel *Barbus graellsii*	Muscle	13	1 of 13	nd–707		ng/g	Range	2–19 pg/g ww
de Boer et al. 2003	The Netherlands	1999	Freshwater mussels *Dreissena polymorpha*					< 4 – < 34	µg/kg		
Dodder et al. 2002	Northeastern United States; Lake Superior	1994	Smelt *Osmeru mordax*	Whole body				< 1.5	µg/kg		
Dodder et al. 2002	Lake Ontario	1994	Smelt *Osmeru mordax*	Whole body				< 1.6	µg/kg		
Dodder et al. 2002	Lake of the Ozarks, MO	1999	White crappie; bluegill *Pomoxis annularis; Lepomis macrochirus*	Whole body				< 1.4	µg/kg		
Dodder et al. 2002	Hadley Lake, IN.	1999	White crappie, bluegill *Pomoxis annularis; Lepomis macrochirus*	Whole body				< 1.4	µg/kg		
Dodder et al. 2002	Hadley Lake, IN.	1999	White crappie, bluegill *Pomoxis annularis; Lepomis macrochirus*	Muscle				< 1.4, < 1.3	µg/kg		
Guo et al. 2009	Pearl River Delta, S. China		Bighead carp (farmed) *Aristichthys nobilis*	Skin	8			6.78 ± 6.47	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Bighead carp (farmed) *Aristichthys nobilis*	Gills	8			1.18 ± 1.28	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Bighead carp (farmed) *Aristichthys nobilis*	GIT	8			2.09 ± 3.11	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Bighead carp (farmed) *Aristichthys nobilis*	Liver	8			0.85 ± 2.07	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Bighead carp (farmed) *Aristichthys nobilis*	Muscle	8			0.48 ± 0.56	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Mandarin fish (farmed) *Siniperca chuatsi*	Skin	8			7.68 ± 5.46	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Mandarin fish (farmed) *Siniperca chuatsi*	Gills	8			1.34 ± 1.86	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta,		Mandarin fish (farmed)	GIT	8			3.55 ± 4.60	ng/g dw	Mean (± SD)	10 ng/g dw

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
	S. China		*Siniperca chuatsi*								
Guo et al. 2009	Pearl River Delta, S. China		Mandarin fish (farmed) *Siniperca chuatsi*	Liver	8			1.57 ± 3.05	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Mandarin fish (farmed) *Siniperca chuatsi*	Muscle	8			0.22 ± 0.41	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Northern snakehead (farmed) *Nemipterus virgatus*	Skin	8			1.61 ± 2.09	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Northern snakehead (farmed) *Nemipterus virgatus*	Gills	8			2.22 ± 2.75	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Northern snakehead (farmed) *Nemipterus virgatus*	GIT	8			0.99 ± 2.80	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Northern snakehead (farmed) *Nemipterus virgatus*	Liver	8			0.22 ± 0.59	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Northern snakehead (farmed) *Nemipterus virgatus*	Muscle	8			ND	ng/g dw	Mean (± SD)	10 ng/g dw
Ismail et al. 2009	Lake Ontario	1979–2004	Lake trout *Salvelinus namaycush*	Whole body	29 (4 or 5 per year)	None	2.3 (±0.5)–12 (±5.3)	0.27 (± 0.30)–1.3 (± 0.8)	ng/g	Mean (± SE)	0.23 ng/g ww
Sellström et al. 1998	Sweden	1995	Pike *Esox lucius*	Muscle					µg/kg		
Sellström et al. 1998	Sweden	1995	Pike *Esox lucius*	Muscle			not detected – trace		µg/kg		
Sellström et al. 1998	Sweden	1995	Pike *Esox lucius*	Muscle			not detected – trace		µg/kg		
Wang et al. 2007	China	Sept.–Dec. 2006	Lichen	Whole	1	None		1572	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	March brown *Limnodrilus hoffmeisteri*	Whole	1	None		11.37	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Coccid	Whole	1	None		114	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Zooplankton *Monia rectirostris, Monia micrura, Monia macrocopa*	Whole	1	None		151.9	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Common carp *Cyprinus carpio*	Whole	4	75%		5	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Java tilapia *Tilapia nilotica*	Whole	3	100%		< 1	ng/g dw		1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Leather catfish *Silurus meridionalis*	Whole	4	Some		19.32	ng/g dw	Mean	1 ng/g dw

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				
							Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
Wang et al. 2008	China	Sept.–Dec. 2006	Crusian carp *Carassius auratus*	Whole	5	Some		1.79	ng/g dw	Mean	1 ng/g dw
Wang et al. 2008	China	Sept.–Dec. 2006	Chinese softshell turtle *Chinemys reevesii*	Whole	3	Some		2.63	ng/g dw	Mean	1 ng/g dw
Xiang et al. 2007	China	Apr.–Aug. 2004	Large yellow croaker *Pseudosiaena crocea*	Muscle	13	Some	117.4		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Silvery pomfret *Platycephalus argenteus*	Muscle	10	Some	24.4		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Flathead fish *Platycephalus indicus*	Muscle	17	Some	0		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Robust tongue fish *Cynoglossus robustus*	Muscle	8	Some	0		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Bombay duck *Harpodon nehereus*	Muscle	9	Some	0		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Jinga shrimp *Metapenaeus affinis*	Soft tissue	10	Some	0		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Greasy-back shrimp *Metapenaeus crocea*	Soft tissue	10	Some	30.0		ng/g	Median	
Xiang et al. 2007	China	Apr.–Aug. 2004	Mantis shrimp *Oratosquilla oratoria*	Soft tissue	9	Some	42.47		ng/g	Median	
MAMMAL – HERBIVORE											
Lichota et al. 2004	Vancouver Island, Canada	2001	Marmot *Marmota vancouverensis*	Fat			0.5		µg/kg		
Voorspoels et al. 2007	Flanders, Belgium	2001	Wood mice *Apodemus sylvaticus*	Liver and muscle	12	All	< 17		ng/g		
Voorspoels et al. 2007	Flanders, Belgium	2001	Bank voles *Clethrionomys glareolus*	Liver and muscle	12	All	< 17		ng/g		
MAMMAL – PREDATOR											
Mariussen et al. 2004	Norway	1995	Moose *Alces alces*	Liver	Not reported		0.8		ng/g	Mean	
Christensen et al. 2005	Coastal British Columbia	2003	Grizzly bear *Ursus arctos horribilis*	Fat or muscle	6	Not reported	~ 0.1–2.77		µg/kg	Range (from graph)	
Christensen et al.	Interior British	2003	Grizzly bear	Fat or	6	Not reported	~0.5–41.71		µg/kg	Range (from	

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
2005	Columbia		*Ursus arctos horribilis*	muscle						graph)	
Sørmo et al. 2006	Svalbard	2003	Polar bear *Ursus maritimus*	Blubber	4	0	0.03–0.16 (.09)		ng/g	Range (mean)	
Sørmo et al. 2006	Svalbard	2003	Polar bear *Ursus maritimus*	Blubber	4	0		0.075–0.04 (0.0225)	ng/g	Range (mean)	
Mariussen et al. 2004	Norway	1993–1994	Lynx *Lynx lynx*	Liver	7		0.1		ng/g	Mean	
Muir et al. 2006	Svalbard		Polar bear *Ursus maritimus*	Adipose	15		< 1		ng/g	Mean	
Muir et al. 2006	Svalbard		Polar bear *Ursus maritimus*	Adipose	44		< 1		ng/g	Mean	
Skaare 2004	Svalbard	2001–2003	Polar bear *Ursus maritimus*	Adipose			1		ng/g	Mean	
Verrault et al. 2005	Svalbard, Norway	2002	Polar bear (female) *Ursus maritimus*	Plasma	15	93%		< 0.06 –0.1	ng/g	Range	
Voorspoels et al. 2006a	Belgium	2003–2004	Red fox *Vulpes vulpes*	Liver	30	60%	< 9.1–760		ng/g	Range	
Voorspoels et al. 2006a	Belgium	2003–2004	Red fox *Vulpes vulpes*	Muscle	33	79%	< 3.9–290		ng/g	Range	
Voorspoels et al. 2006a	Belgium	2003–2004	Red fox *Vulpes vulpes*	Adipose	27	85%	< 3.7–200		ng/g	Range	
MARINE FISH											
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Conger eel				< 0.2–0.53	< 0.020–0.029	µg/kg		
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Flounder *Paralichthys olivaceus*				1.9–3.2	0.015–0.022	µg/kg		
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Gray mullet *Mugil cephalus*				< 0.2–0.25	< 0.010–0.013	µg/kg		
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Horse mackerel				< 0.2–1.4	< 0.030–0.047	µg/kg		
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Red sea bream *Pagrus major*				< 0.2–0.74	< 0.010–0.020	µg/kg		
Akutsu et al. 2001	Inland Sea of Seto, Japan	1998	Sea bass *Lateolabrax japonicus*				0.40–0.81	0.0087–0.017	µg/kg		

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				
							Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
Burreau et al. 2004	Lumparn Estuary	Not Given	Roach *Rutilus rutilus*	Muscle	8	5	48		ng/g	median	
Burreau et al. 2004	Lumparn Estuary	Not Given	Perch *Perca fluviatilis*	Muscle	33	21	1.3		ng/g	Median	
Burreau et al. 2004	Lumparn Estuary	Not Given	Pike *Esox lucius*	Muscle	25	21	1.7		ng/g	Median	
Burreau et al. 2006	Baltic Sea	1998	Sprat *Sprattus sprattus*	Muscle	6	3	0.082		ng/g	Median	
Burreau et al. 2006	Baltic Sea	1998	Herring *Clupea harengus*	Muscle	5	1	0.24		ng/g	Median	
Burreau et al. 2006	Baltic Sea	1998	Salmon *Salmo salar*	Muscle	10	7	0.41		ng/g	Median	
Burreau et al. 2006	North Atlantic Ocean	1999	Small herring *Clupea harengus*	Muscle	6	0	0.31		ng/g	Median	
Burreau et al. 2006	North Atlantic Ocean	1999	Large herring *Clupea harengus*	Muscle	6	3	0.039		ng/g	Median	
Burreau et al. 2006	North Atlantic Ocean	1999	Atlantic salmon *Salmo salar*	Muscle	10	0			ng/g	Median	
de Boer et al. 1998	Dutch Coast	1995	Mackerel *Scomber scombrus*	Muscle				< 2	µg/kg		
de Boer et al. 2001	North Sea	1999	Herring *Clupea harengus*	Liver			< 4.6 – < 11	< 0.300 – < 0.520	µg/kg		
de Boer et al. 2001	North Sea	1999	Herring *Clupea harengus*	Filet			< 1.4 – < 2.6	< 0.160 – < 0.250	µg/kg		
de Boer et al. 2001	North Sea	1999	Herring *Clupea harengus*	Milt and eggs			< 1.9 – < 5.0	< 0.130 – < 0.200	µg/kg		
de Boer et al. 2001	North Sea	1999	Cod	Liver			< 0.22 – < 53	< 0.110– 0.380	µg/kg		
de Boer et al. 2001	North Sea	1999	Cod	Fillet			< 8.1 – < 40	< 0.049 – < 0.170	µg/kg		
de Boer et al. 2001	North Sea	1999	Whiting *Merlangius merlangius*	Liver			< 0.4 – < 1.2	< 0.280 – < 0.610	µg/kg		
de Boer et al. 2001	North Sea	1999	Whiting *Merlangius merlangius*	Fillet			< 6.5 – < 13	< 0.042 – < 0.078	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Sprat *Sprattus sprattus*	Whole body			< 10	< 0.5	µg/kg		

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				
							Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
de Boer et al. 2001	Tees Estuary, UK	1999	Whiting *Merlangius merlangius*	Whole body			< 33	< 0.5	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Whiting *Merlangius merlangius*	Muscle			< 33 – < 100	< 0.5	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Whiting *Merlangius merlangius*	Liver			< 1.56	–	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Flounder *Paralichthys olivaceus*	Liver			< 2.7	< 0.5	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Greater sandeel *Hyperoplus lanceolatus*				< 18 – < 22	< 0.2	µg/kg		
de Boer et al. 2003	The Netherlands	1999	Flounder *Paralichthys olivaceus*					< 0.2–< 6	µg/kg		
de Boer et al. 2003	The Netherlands	1999	Bream					< 0.03–< 21	µg/kg		
Fjeld et al. 2004	Svolvaer, Lofoten, Norway	2002	Atlantic cod *Gadus morhua*	Liver	21		0.99		ng/g	Mean	
Fjeld et al. 2004	Varangerfjorden, Norway	1998, 2002	Atlantic cod *Gadus morhua*	Liver	21		0.98		ng/g	Mean	
Guo et al. 2009	Pearl River Delta, S. China		Crimson snapper (farmed) *Lutjanus erythropterus*	Skin	8			1.47 ± 2.72	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Crimson snapper (farmed) *Lutjanus erythropterus*	Gills	8			0.30 ± 0.55	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Crimson snapper (farmed) *Lutjanus erythropterus*	GIT	8			1.06 ± 2.99	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Crimson snapper (farmed) *Lutjanus erythropterus*	Liver	8			0.59 ± 1.25	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Crimson snapper (farmed) *Lutjanus erythropterus*	Muscle	8			0.048 ± 0.14	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Golden thread *Nemipterus virgatus*	Skin	8			15.54 ± 24.81	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Golden thread *Nemipterus virgatus*	Gills	8			2.69 ± 5.47	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Golden thread *Nemipterus virgatus*	GIT	8			ND	ng/g dw	Mean (± SD)	10 ng/g dw
Guo et al. 2009	Pearl River Delta, S. China		Golden thread *Nemipterus virgatus*	Liver	8			0.31 ± 0.83	ng/g dw	Mean (± SD)	10 ng/g dw

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
Guo et al. 2009	Pearl River Delta, S. China		Golden thread *Nemipterus virgatus*	Muscle	8			0.089 ± 0.25	ng/g dw	Mean (± SD)	10 ng/g dw
Sørmo et al. 2006	Svalbard	2003	Polar cod *Boreogadus saida*	Whole body	7	0	0.05–0.42 (0.2)		ng/g	Range (mean)	
Sørmo et al. 2006	Svalbard	2003	Polar cod *Boreogadus saida*	Whole body	7	0		0.006–0.05 (0.024)	ng/g	Range (mean)	
Johnson-Restropo 2005	Coastal Florida	2004	Silver perch *Bairdiella chrysoura*	Muscle	6	All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Coastal Florida	2004	Striped mullet *Mugil cephalus*	Muscle	6	All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Coastal Florida	2004	Spotted seatrout *Cynoscion nebulosus*	Muscle	7	All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Coastal Florida	2004	Red drum *Sciaenops ocellatus*	Muscle	11	All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Coastal Florida	2004	Hardhead catfish *Arius felis*	Muscle	8		4.5		ng/g	Mean	
Johnson-Restropo 2005	Coastal Florida	2004	Atlantic stingray *Dasyatis Sabina*	Muscle	7		0.1		ng/g	Mean	
Johnson-Restropo 2005	Coastal Florida	2004	Spiny dogfish *Squalus acanthias*	Muscle	5		16.9		ng/g	Mean	
Johnson-Restropo 2005	Coastal Florida	2004	Atlantic sharpnose shark *Rhizoprionodon terraenovae*	Muscle	5		514.3		ng/g	Mean	
Johnson-Restropo 2005	Coastal Florida	1993–1994	Bull shark *Carcharhinus leucas*	Muscle	6		39		ng/g	Mean	
Johnson-Restropo 2005	Coastal Florida	2002–2004	Bull shark *Carcharhinus leucas*	Muscle	7		778		ng/g	Mean	
Watanabe et al. 1987	Japan	1981–1985	Mullet *Mugilidae*		4				µg/kg		
Watanabe et al. 1987	Japan	1981–1985	Goby		2				µg/kg		
Watanabe et al. 1987	Japan	1981–1985	Sardine		2				µg/kg		
Watanabe et al. 1987	Japan	1981–1985	Sea bass		1				µg/kg		
Watanabe et al. 1987	Japan	1981–1985	Horse mackerel		1				µg/kg		

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				
							Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
Watanabe et al. 1987	Japan	1981–1985	Mackerel *Scomber scombrus*		1				µg/kg		
Watanabe et al. 1987	Japan	1981–1985	Hairtail		1				µg/kg		
MARINE INVERTEBRATES											
Burreau et al. 2006	Baltic Sea	1999	Zooplankton *Calanoid sp.*	Muscle	3	None	2.1		ng/g	Median	
de Boer et al. 2001	North Sea	1999	Starfish Asteroidea	Pyloric caeca			< 2.0–8.9	< 0.150–0.310	µg/kg		
de Boer et al. 2001	North Sea	1999	Hermit crab	Abdomen			< 1.1–7.0	< 0.1600–0.470	µg/kg		
de Boer et al. 2001	North Sea	1999	Male whelk *Haustellum brandaris*	Whole body			< 1.9 – < 14	< 0.052 – < 0.190	µg/kg		
de Boer et al. 2001	North Sea	1999	Shrimp	Whole body			< 2.4 – < 5.6	< 0.073 – < 0.081	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Shrimp	Whole body			< 100	< 0.5	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Polychaete worm	Whole body			< 50	< 0.5	µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Starfish	Whole body and tentacle			< 9	< 0.5	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Mysid shrimp				< 20 93	< 0.2–0.98	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Copepod				< 37	< 0.3	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Gudgeon				< 6.0 – < 17	< 0.1 – < 0.4	µg/kg		
de Boer et al. 2003	The Netherlands	1999	Marine mussels					< 4–< 5	µg/kg		
Fjeld et al. 2004	Svolvaer, Lofoten, Norway	2002	Blue mussels *Mytilus edullis*	Muscle	20		33		ng/g	Mean	
Fjeld et al. 2004	Varangerfjorden, Norway	2002	Blue mussels *Mytilus edullis*	Muscle	50		3.6		ng/g	Mean	
Sørmo et al. 2006	Svalbard	2003	Ice amphipod *Gammarus wilkitzkii*	Whole body	1 (composite of 5)	0	7.22		ng/g	Mean	
Sørmo et al. 2006	Svalbard	2003	Ice amphipod	Whole body	1 (composite	0		0.28	ng/g	Mean	

Study Authors	Location	Date	Species	Tissue	Concentration Data						
					Sampling N	Not Detected	Lipid Weight	Wet Weight	Units	Statistic	Detection Limit
			Gammarus wilkitzkii		of 5)						
Watanabe et al. 1987	Japan	1981–1985	Mussels *Lampsilis radiata*		5	4 of 5		1.4	µg/kg		0.5
MARINE MAMMALS											
de Boer et al. 1998	Dutch Coast	1995	Minke whale *Balaenoptera acutorostrata*	Blubber				< 1	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	Sperm whale *Physeter catodon*	Blubber				< 3 – < 6	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	Sperm whale *Physeter catodon*	Liver				< 3	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	White-beaked dolphin *Lagenorhynchus albirostris*	Blubber				< 10	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	White-beaked dolphin *Lagenorhynchus albirostris*	Liver				< 1	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	Harbour seal	Blubber				< 10 – < 15	µg/kg		
de Boer et al. 1998	Dutch Coast	1995	Harbour seal	Liver				< 1 – < 2	µg/kg		
de Boer et al. 2001	North Sea	1999	Harbour porpoise	Liver			1.2 – < 7.4	–	µg/kg		
de Boer et al. 2001	North Sea	1999	Harbour porpoise	Blubber			< 2.7 – 26	–	µg/kg µg/kg		
de Boer et al. 2001	North Sea	1999	Harbour seal	Liver	9		< 6.5–160		µg/kg		
de Boer et al. 2001	North Sea	1999	Harbour seal	Blubber			< 1.8–16		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Blubber			< 3.8		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Kidney			< 8.2		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Heart			< 4.9		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Lungs			35		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Brain			< 0.8		µg/kg		
de Boer et al. 2001	North Sea	1999	White-beaked dolphin	Liver			140–318		µg/kg		
de Boer et al. 2001	North Sea	1999	Bottlenose dolphin	Blubber			< 2.5		µg/kg		
de Boer et al. 2001	North Sea	1999	Bottlenose dolphin	Liver			< 6.4		µg/kg		
de Boer et al. 2001	North Sea	1999	Bottlenose dolphin	Kidney			< 3.0		µg/kg		

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				Detection Limit
							Lipid Weight	Wet Weight	Units	Statistic	
de Boer et al. 2001	North Sea	1999	Bottlenose dolphin	Muscle			< 7.7		µg/kg		
de Boer et al. 2001	Tees Estuary, UK	1999	Porpoise	Blubber			< 8.7 – < 9	< 7.5	µg/kg		
Sørmo et al. 2006	Svalbard	2003	Ringed seal *Phoca hispida*	Blubber	6	5	0.02		ng/g	Only detected concentration	
Sørmo et al. 2006	Svalbard	2003	Ringed seal *Phoca hispida*	Blubber	6	5		0.006	ng/g	Only detected concentration	
Johnson-Restropo 2005	Western coast of Florida	1991–1996	Bottlenose dolphin *Tursiops truncatus*	Blubber			0		ng/g	Mean	
Johnson-Restropo 2005	Western coast of Florida	2000–2001	Bottlenose dolphin *Tursiops truncatus*	Blubber		All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Eastern coast of Florida	2001–2004	Bottlenose dolphin *Tursiops truncatus*	Blubber		All	< 0.022		ng/g	MDL	
Johnson-Restropo 2005	Coastal Florida	??	Striped dolphin *Stenella coeruleoalba*	Blubber		All	< 0.022		ng/g	MDL	
Shaw et al. 2007 (BFR)	NW Atlantic coast (Maine to Long Island)	1991 – 2005	Harbour seal *Phoca vitulina*	Blubber	4	2 of 4	nd – 7.4		ng/g	Range	
MARINE/AQUATIC BIRDS											
de Boer et al. 2001	Tees Estuary, UK	1999	Cormorant *Corvus marinus*	Liver			< 17 – < 25	< 0.5	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Common tern *Sterna hirundo*	Eggs			< 5.6–70	< 0.46–7.1	µg/kg		
de Boer et al. 2001	Western Scheldt, The Netherlands	1999	Common tern *Sterna hirundo*	Eggs			4.4–27	0.45–2.9	µg/kg		
de Boer et al. 2004	Sweden	Not given	Great crested grebe *Podiceps cristatus*	Liver	4	3 of 4	< 1.5–9.1	< 0.11–0.52	µg/kg		
de Boer et al. 2004	Sweden	Not given	Great crested grebe *Podiceps cristatus*	Muscle	3	2 of 3	< 8.1–31	< 0.4–1.2	µg/kg		
de Boer et al. 2004	Sweden	Not given	Heron	Liver	4	All	< 2.3 – < 5.7	< 0.08 – < 0.25	µg/kg		
de Boer et al. 2004	Sweden	Not given	Heron	Muscle	5	4 of 5	< 6.3–563	< 0.32–4.5	µg/kg		

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
de Boer et al. 2004	Sweden	Not given	Gannett *Morus bassanus*	Eggs	12	All	< 4 – < 57	< 0.2 – < 2.2	µg/kg		
Gauthier et al. 2008	Canada	1982, 1987, 1992, 1995–2006, April – May	Herring gull *Larus argentatus*	Eggs	10–13 eggs/site/ year pooled homogenates	Not reported		4.5–20	ng/g	Range	
Jaspers et al. 2006	Belgium	2003–2004	Aquatic birds (heron/grebe)	Multiple	12	All	<2.25		ng/g		
Kunisue et al. 2008	Open sea (Asia)	1998–1999	Black-footed albatross *Diomedea nigripes*	Muscle	5	All	<0.5		ng/g	Detection limit	
Kunisue et al. 2008	Open sea (Asia)	1998	Laysan albatross *Diomedea immutabilis*	Muscle	5	All	<0.5		ng/g	Detection limit	
Kunisue et al. 2008	Open sea (Asia)	1999	Northern fulmar *Fulmarus glacialis*	Muscle	5	0	<0.5_4.8 (1.9)		ng/g	Range (mean in brackets)	
Kunisue et al. 2008	Japan (Coastal)	1999–2001	Black-tailed gull *Larus crassirostris*	Muscle	5	All	<0.5		ng/g	Detection limit	
Lam et al. 2007	China – Hong Kong	March – May 2004	Little egret *Egretta garzetta*	Eggs	5	Not reported	2.6 ±1.6 (< 0.5–3.8)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al. 2007	China – Hong Kong	March–May 2004	Black-crowned night heron *Nycticorax nycticorax*	Eggs	5	Not reported	14 ±25 (0.62–59)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al. 2007	China – Xiamen	March–May 2004	Little egret *Egretta garzetta*	Eggs	5	Not reported	0.89 ±0.64 (0.42–2.0)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al. 2007	China – Xiamen	March–May 2004	Black-crowned night heron *Nycticorax nycticorax*	Eggs	5	Not reported	1.8 ±2.2 (< 0.5–5.2)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al. 2007	China – Xiamen	March–May 2004	Chinese pond heron *Ardeola bacchus*	Eggs	5	Not reported	99 ± 130 (3.1–290)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al. 2007	China – Xiamen	March–May 2004	Cattle egret *Bubulcus ibis*	Eggs	5	Not reported	28 ± 36 (< 0.5–75)		ng/g	Mean ±standard	0.5

Study Authors	Location	Date	Species	Tissue	Sampling N	Not Detected	Concentration Data				Detection Limit
							Lipid Weight	Wet Weight	Units	Statistic	
										deviation (range)	
Lam et al. 2007	Quanzhou	March–May 2004	Little egret *Egretta garzetta*	Eggs	5	Not reported	1.6 ± 0.55 (0.88–2.3)		ng/g	Mean ±standard deviation (range)	0.5
Lam et al 2007	Quanzhou	March–May 2004	Black-crowned night heron *Nycticorax nycticorax*	Eggs	5	5	< 0.5		ng/g	Mean ±standard deviation (range)	0.5
Verrault et al. 2005	Svalbard, Norway	2004	Glaucous gull (male) *Larus hyperboreus*	Plasma	12	50%		< 0.05–0.21	ng/g	Range	
Verrault et al. 2005	Svalbard, Norway	2004	Glaucous gull (female) *Larus hyperboreus*	Plasma	15	64%		< 0.05–0.33	ng/g	Range	
Verreault et al. 2004	Bjoroya, Norway	2002–2004	Glaucous gull *Larus hyperboreus*	Plasma	89		410		ng/g	Mean	
Verreault et al. 2004	Bjoroya, Norway	2002–2004	Glaucous gull *Larus hyperboreus*	Eggs	4 pools		23–53		ng/g	Range	
Verreault et al. 2004	Svalbard		Glaucous gull *Larus hyperboreus*	Liver	20		nd–170		ng/g	Range	
OTHER BIRDS											
Kunisue et al. 2008	Japan (Inland)	2001	Common cormorant *Phalacrocorax carbo*	Muscle	4	0	< 0.5–3.5 (1.4)		ng/g	Range (mean in brackets)	
Kunisue et al. 2008	Japan (Inland)	1998	Jungle crow *Corvus macrorhynchos*	Muscle	5	0	36–1800 (440)		ng/g	Range (mean in brackets)	
Mariussen et al. 2004	Norway	1990–1993	Grouse	Liver			0.5		ng/g	Mean	
Voorspoels et al. 2006b	Belgium	2001, 2003	Passerines	Liver and muscle	25	All	< 17		ng/g		
VEGETATION											
Schlabach et al.	Skoganvarre,	2002	Moss	n/a	1			0.025	ng/g	Mean	

							Concentration Data				
Study Authors	**Location**	**Date**	**Species**	**Tissue**	**Sampling N**	**Not Detected**	**Lipid Weight**	**Wet Weight**	**Units**	**Statistic**	**Detection Limit**
2002	Norway										
Schlabach et al. 2002	Valvik, Norway	2002	Moss	n/a	1			0.12	ng/g	Mean	

Appendix B – Bioaccumulation, Biomagnification and Trophic Magnification Data

Species, Food Web or Predator:Prey	Ecosystem	Study Type	Tissue	Location	Endpoint	Result	Statistic	Reference	Notes
Pelagic food web (zooplankton, whitefish, white sucker, emerald shiner, goldeye, burbot, walleye) (*Calanoid sp., Coregonus clupeaformis, Catostomus commersoni, Notropis atherinoides, Hiodon alosoides, Lota lota, Stizostedion vitreum*)	Aquatic	Field	Whole body	Lake Winnipeg	TMF (lipid normalized)	3.6	Regression slope	Law et al. 2006	Statistically significant slope ($P < .001$)
Fish:fish	Aquatic	Field	Whole body	Lake Winnipeg	BMFs (lipid normalized)	0.1–6.8	Range	Law et al. 2006	Generally superseded by TMF from the same system but still useful supporting info
Fish:invertebrates	Aquatic	Field	Whole body	Lake Winnipeg	BMFs (lipid normalized)	0.2–34	Range	Law et al. 2006	Generally superseded by TMF from the same system but still useful supporting info
Pelagic food web (zooplankton, sprat, herring, salmon) (*Sprattus sprattus, Clupea harengus, Salmo salar*)	Aquatic	Field	Whole body	Baltic Sea	TMF (lipid normalized)	~0.6 (not significantly different from 0)	Regression slope	Burreau et al. 2006	Lowest trophic level (zooplankton) had much higher concentrations than fish; the feeding relationships were not described
Pelagic food web (roach, perch, pike) (*Rutilus rutilus, Perca fluviatilis, Esox lucius*)	Aquatic	Field	Whole body	Baltic Sea	TMF (lipid normalized)	~0.80 (not significantly different from 0)	Regression slope	Burreau et al. 2004	Lowest trophic level (zooplankton) had much higher concentrations than fish; the feeding relationships were not described
Fish:fish	Aquatic	Field	Whole body	Baltic Sea	BMFs (lipid normalized)	0.02–1.3	Range	Burreau et al. 2004	
Fish:zooplankton	Aquatic	Field	Whole body	Baltic Sea	BMFs (lipid normalized)	0.04–0.11	Range	Burreau et al. 2006	Unclear if fish feed on sampled zooplankton
Fish:fish	Aquatic	Field	Whole body	Baltic Sea	BMFs (lipid normalized)	1.71–5	Range	Burreau et al. 2006	
Juvenile Lake trout *Salvelinus namaycush*	Aquatic	Lab	Whole body	na	BMF (lipid normalized)	0.3	Mean	Tomy et al. 2004	It is unlikely that steady state was reached during the experiment and the BMF was estimated using the kinetic method

Species, Food Web or Predator:Prey	Ecosystem	Study Type	Tissue	Location	Endpoint	Result	Statistic	Reference	Notes
Carp *Cyprinus carpio*	Aquatic	Lab	Whole body	n/a	BCF (wet weight)	< 3 000		MITI 1992	Tissue MDL divided by water solubility, high uncertainty
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Carcass	n/a	BMF (lipid normalized)	0.022	Range	Stapleton et al. 2006	decaBDE only
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Carcass	n/a	BMF (lipid normalized)	0.089	Mean	Stapleton et al. 2006	decaBDE + BDE metabolites
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Serum	n/a	BMF (lipid normalized)	0.35	Mean	Stapleton et al. 2006	decaBDE only
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Serum	n/a	BMF (lipid normalized)	0.52	Mean	Stapleton et al. 2006	decaBDE + BDE metabolites
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Liver	n/a	BMF (lipid normalized)	1.28	Mean	Stapleton et al. 2006	decaBDE only
Rainbow trout *Salvelinus namaycush*	Aquatic	Lab	Liver	n/a	BMF (lipid normalized)	1.73	Mean	Stapleton et al. 2006	decaBDE + BDE metabolites
Polar cod:ice ampipod *Boreogadus saida: Gammarus wilkitzkii*	Marine	Field	Whole body	Svalbard, Norway	BMF (lipid normalized)	0.03	Mean	Sørmo et al. 2005	
Grey seals *Halichoerus grypus*	Marine mammal	Captive	Blood	North Sea	BMF (lipid normalized)	8.3–20.8	Range	Thomas et al. 2005	
Grey seals *Halichoerus grypus*	Marine mammal	Captive	Blubber	North Sea	BMF (lipid normalized)	0.025–0.063	Range	Thomas et al. 2005	Based on day 30 concentration
Grey seals *Halichoerus grypus*	Marine mammal	Captive	Blubber	North Sea	BMF (lipid normalized)	0.044–0.11	Range	Thomas et al. 2005	Based on day 83 concentration (unlikely to represent steady-state conditions)
Grizzly bear *Ursus arctos horribilis*	Terrestrial	Field	Fat and muscle	Coastal and interior British Columbia	Bioaccumulation slope	negative (not significantly different from 0)	Regression slope	Christensen et al. 2005	A positive bioaccumulation slope would provide evidence of biomagnification or trophic magnification
Earthworm *Phylum annelida*	Terrestrial	Lab	Whole body		Soil BSAF(lipid and OC normalized)	0.044–0.73 (0.3)	Range (mean)	Sellstrom et al. 2005	
Rat	Terrestrial	Lab	Whole body	n/a	BMF (wet weight)	0.05	Mean	Huwe and Smith 2007a, b	
Cow	Terrestrial	Lab	Adipose	n/a	BMF (lipid	0.037	Mean	Kierkegaard et	Based on mean silage

Species, Food Web or Predator:Prey	Ecosystem	Study Type	Tissue	Location	Endpoint	Result	Statistic	Reference	Notes
			tissue		normalized)			al. 2007	concentration
Cow	Terrestrial	Lab	Adipose tissue	n/a	BMF (lipid normalized)	0.31	Mean	Kierkegaard et al. 2007	Based on silage concentration at end of experiment
Cow	Terrestrial	Lab	Whole body	n/a	BMF (lipid normalized)	0.026	Mean	Kierkegaard et al. 2007	Based on mean silage concentration
Cow	Terrestrial	Lab	Whole body	n/a	BMF (lipid normalized)	0.21	Mean	Kierkegaard et al. 2007	Based on silage concentration at end of experiment
Sunfish *Lepomis gibbosus*	Aquatic	Field	Whole body	Marlowe Ck., Roxboro, NC	BSAF (lipid and OC normalized)	0.0018	Mean	La Guardia et al. 2007	
Crayfish *Campbarus punctic ambarus sp. C*	Aquatic	Field	Whole body	Marlowe Ck., Roxboro, NC	BSAF (lipid and OC normalized)	0.013	Mean	La Guardia et al. 2007	
Various (see comments)	Aquatic	Field	Whole body	Pearl River Delta, China	BSAF (lipid and OC normalized)	0 – 0.4	Range	Xiang et al. 2007	Large yellow croaker (omnivore), silvery pomfret (omnivore), flathead fish (benthivore), robust tonguefish (benthivore), Bombay duck (piscivore), jinga shrimp (benthic invertebrate), greasy-back shrimp (benthic invertebrate), mantis shrimp (benthic invertebrate)
Fish (see comments)	Aquatic	Field	Whole body	River Vero, Spain	BSAF (lipid and OC normalized)	0.0011 to 0.0013	Range	Eljarrat et al. 2007	Barbell and carp
Aquatic oligochaete	Aquatic	Lab	Whole body	n/a	Accumulation	Minimal		Ciparis and Hale 2005	
Horse mussel *Modiolus modiolus*	Aquatic	Field	Whole body	Vancouver Island, Canada	Sediment BSAF (lipid and organic carbon normalized)	0–3.53	Range	DeBruyn et al. 2009	
Marine mammal: fish	Marine	Field	Blubber and whole fish	North Atlantic, coast of Maine, USA	BMF (lipid normalized)	0.67–1.3	Range (ratio of predator tissue concentration to prey tissue concentration)	Shaw et al. 2009	Harbour seal (blubber):American plaice (whole body); Harbour seal (blubber):Atlantic mackerel (whole body); Harbour seal (blubber):White hake (whole body)
Marine food web: beluga	Marine	Field	Blubber and	Eastern	TMF (lipid	0.3 (statistically	Regression	Tomy et al. 2008	Tissues sampled: blubber—

Species, Food Web or Predator:Prey	Ecosystem	Study Type	Tissue	Location	Endpoint	Result	Statistic	Reference	Notes
whale, narwhal, walrus, arctic cod, shrimp, clams, deepwater redfish, mixed zooplankton *Delphinapterus leucas*, *Monodon monoceros, Odobenus rosmarus, Boreogadus saida*, *Pandalus borealis*, *Hymenodara glacialis*, *Mya truncata, and Serripes groenlandica*, *Sebastes mentella*)			whole organism	Canadian Arctic	normalized)	significant negative slope)	slope		beluga whale, narwhal and walrus; whole organism—arctic cod, shrimp, clams, deepwater redfish, and mixed zooplankton
Marine mammal: fish	Marine	Field	Blubber and whole organism	Eastern Canadian Arctic	BMF (lipid normalized)	<1	Range (trophic level adjusted ratio of predator tissue concentration to prey tissue concentration)	Tomy et al. 2008	Beluga (blubber):cod (whole body); Beluga (blubber):redfish (whole body)
Fish: invertebrate	Marine	Field	Blubber and whole organism	Eastern Canadian Arctic	BMF (lipid normalized)	<1	Trophic level adjusted ratio of predator tissue concentration to prey tissue concentration	Tomy et al. 2008	Cod (whole body):zooplankton (whole body)
Marine food web: beluga whale, ringed seal, arctic cod, Pacific herring, Arctic cisco, pelagic amphipod, and arctic copepod (*Delphinapterus leucas, Phoca hispida, Boreogadus saida, Clupea pallasi, Coregonus autumnalis, Thermisto libelluala, Calanus hyperboreus*)	Marine	Field	Blubber, liver, and whole organism	Western Canadian Arctic	TMF (lipid normalized)	Not statistically significant	Regression slope	Tomy et al. 2009	Tissues sampled: blubber—Beluga whale, ringed seal; liver—arctic cod, Pacific herring, Arctic cisco; whole organism—pelagic amphipod, and arctic copepod
Marine mammal: fish	Marine	Field	Blubber and liver	Western Canadian Arctic	BMF (lipid normalized)	0.3–0.9	Range (trophic level adjusted ratio of predator tissue concentration to	Tomy et al. 2009	Beluga whale (blubber):cod (liver); ringed seal (blubber):herring (liver); Beluga whale (blubber):Arctic cisco (liver)

Species, Food Web or Predator:Prey	Ecosystem	Study Type	Tissue	Location	Endpoint	Result	Statistic	Reference	Notes
							prey tissue concentration)		
Fish: invertebrate	Marine	Field	Liver and whole body	Western Canadian Arctic	BMF (lipid normalized)	4.8–12.7	Range (trophic level adjusted ratio of predator tissue concentration to prey tissue concentration)	Tomy et al. 2009	cod (liver):calanus (whole body); cod (liver):thermisto (whole body)
Freshwater food web: water snake, northern snakehead, mud carp, common carp, crucian carp, prawn, and Chinese mysterysnail (Enhydris chinensis, Channa argus, Cirrhinus molitorella, Cyprins carpio, Carassius auratus, Macrobrachium nipponense, Cipangopaludina chinensi)	Aquatic	Field	Whole body	Reservoir near Qinyuan City, South China	TMF (lipid normalized)	0.26 (negative slope, p=0.53)	Regression slope	Wu et al. 2009	Authors interpret result as statistically significant

Appendix C – Selection of Log K_{ow} for QSAR Modelling of decaBDE

The log K_{ow} for commercial DecaBDE of 6.265 determined at 25°C by CMABFRIP (1997a) is frequently cited in the literature. This value was determined using the generator column method with a composite material received from Great Lakes Chemical Corp., Albemarle Corp. and Bromine Compounds Ltd. and consisting of 97.4% decaBDE, 2.5% nonaBDE and 0.04% octaBDE. The analytical method used was GC-ECD and, using this method, the mean concentration measured in the aqueous material was 0.04 µg/L, while the mean concentration in octanol stock solution was 0.0738 g/L. This resulted in a K_{ow} value of 1.845×10^6, or log K_{ow} of 6.265. It is of interest that the log K_{ow} determined for decaBDE by CMABFRIP was in fact lower than those which they determined for the commercial OctaBDE (6.29; CMABFRIP 1997b) and PentaBDE technical products (6.59; MacGregor and Nixon 1997) using the same procedure. This is not expected, due to the increased hydrophobicity and increased K_{ow}, which would be expected with increased bromination. This also does not reflect available information respecting commercial DecaBDE's solubility in water. CMABFRIP (1997c) determined that the limit of water solubility was < 0.1 µg/L at 25°C, while for commercial OctaBDE (CMABFRIP 1997d) it was determined to be 0.5 µg/L, and 13.3 µg/L for commercial PentaBDE (Stenzel and Markly 1997). The available QSAR estimates for decaBDE indicate that it is predicted to be in the range of 2.841×10^{-8} to 2.6078×10^{-4} µg/L at 25°C. Thus, one can expect that difficulties measuring substances with such low solubility in water could have affected the determination of log K_{ow} by CMABFRIP (1997b). The European Communities (2002) also indicate that the presence of trace amounts of octanol in the water phase of this study may have had an apparent effect on the water concentration and the log K_{ow} value. For these reasons, the log K_{ow} for DecaBDE of 6.265 is considered too low and not appropriate for BAF and BMF modelling purposes.

Watanabe and Tatsukawa (1990) reported a significantly higher log K_{ow} of 9.97. Their approach, which applied to other PBDEs, showed a pattern of increased K_{ow} with increasing bromination using a reverse-phase HPLC method.

Overall, the range of reported and measured values of logK_{ow} for decaBDE indicate a significant degree of uncertainty and measurement error. Log K_{ow} measurements and estimates in this report range from 6.3 to 12.9. Other available estimates include 7.68, 10.37, 10.14, 9.44, 12.11 and 11.48 (from: www.vcclab.org/lab/alogps/start.html). The comprehensive Syracuse Research Corporation database used for the KOWWIN program contains 11 135 values for log K_{ow}. The maximum measured value in this training set is 11.3 and the upper 99.5 percentile is 7.8 (personal communication from JA Arnot to Environment Canada 15 February 2008; unreferenced). Therefore, most of the model estimates for decaBDE are greater than presently measured values for all other organic chemicals. Analytical limitations and high levels of measurement error are the principle reasons for lack of log K_{ow} values generally above 8.0 (e.g., Wania and Dugani 2003). Wania and Dugani (2003) reported a log K_{ow} value of 8.7 for decaBDE as the most reliable input for long-range transport modelling based on the establishment of a QSPR (quantitative structure-property relationship) for higher brominated PBDEs and using the

three solubility concept. This value was judged to represent a more realistic $logK_{ow}$ value for modelling purposes and was used for bioaccumulation modelling in this report. The $logK_{ow}$ value of 8.7 was further used to adjust the predicted $logK_{ow}$s of the expected metabolites of decaBDE using the Experimental Value Adjustment (EVA) method outlined in the KOWWIN program. The EVA method takes into account structural differences between the metabolite and parent compound (decaBDE) and adjusts the preferred value of 8.7 accordingly.

Appendix D – Model Input Parameters for BAF–QSAR and Terrestrial Biomagnification Model Predictions for DecaBDE Transformation Products

(a) BAF–QSAR input parameters for lower brominated PBDEs, hydroxylated BDEs and hydroxymethoxy-BDEs

Metabolite	Log K_{ow}	Median k_M (1/d) (184 g fish at 15°C
Hydroxymethoxy-octaBDEs	6.5[1]	0.02[4]
Hydroxymethoxy-hexaBDEs	4.7[1]	0.02[4]
Hydroxymethoxy-pentaBDEs	3.9[1]	0.02[4]
Hydroxymethoxy-heptaBDEs	5.6[1]	0.02[4]
Hydroxy-hexaBDEs	4.7[1]	0.02[4]
Hydroxy octaBDEs	6.4[1]	0.02[4]
Hydroxy-nonaBDEs	7.3[1]	0.02[4]
nonaBDEs	7.8[2]	0.02[5]
octaBDEs	6.9[3]	0.03[6]
heptaBDEs	6.0[2]	0.01[7]
hexaBDEs	5.1[2]	0.001[8]
pentaBDEs	4.3[1]	0.001[9]

Notes:
[1] Estimated using KOWWIN using the EVA method with a reference logK_{ow} of 8.7 for decaBDE.
[2] Estimated using the KOAWIN estimation model and with input of corrected logK_{ow} from KOWWIN EVA method.
[3] Maximum measured value (Watanabe and Tatsukawa 1990).
[4] k_M based on Tomy et al. (2004) and normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and 15°C. These metabolites are expected to undergo further metabolism; however, the actual rates of metabolism are unknown.
[5] Information regarding the half-life and k_M of nonaBDEs is lacking in the literature and a half-life of 26 d (equal to that of decaBDE) was therefore assumed. Normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and 15°C.
[6] Based on a half-life of 19 d (Tomy et al. 2004) and normalized to weight of middle trophic level fish in Arnot and Gobas model and 15°C.
[7] Based on a half-life of 38 d (Stapleton et al. 2004, Tomy et al. 2004—estimates ranged from 19 to 346 d) and normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and 15°C.
[8] Based on a half-life of 58 d (Stapleton et al. 2004, Tomy et al. 2004—estimates ranged from 23 to 231 d) and normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and 15°C.
[9] Based on half-life of 50.7 d (Stapleton et al. 2004, Tomy et al. 2004—estimates ranged from 43 to 173 d) and normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and 15°C.

(b) Terrestrial biomagnification model input parameters for lower brominated PBDEs, hydroxylated BDEs and hydroxymethoxylated BDEs

Name and Structure	Log K_{ow}	Log K_{oa}	E_D[9]	k_M (1/d) (wolf BW normalized 80 kg
Hydroxymethoxy octaBDE	6.5[1]	17.2[2]	99%	0.004[5]
Hydroxymethoxy pentaBDE	3.9[1]	13.4[2]	99%	0.004[5]
Hydroxymethoxy hexaBDE	4.7[1]	14.6[2]	99%	0.004[5]
Hydroxymethoxy heptaBDE	5.6[1]	15.9[2]	99%	0.004[5]
Hydroxy-hexaBDE	4.7[1]	13.4[2]	99%	0.004[5]
hydroxy-octaBDE,	6.4[1]	15.9[2]	98%	0.004[5]
hydroxy-nonaBDE	7.3[1]	17.2[2]	96%	0.004[5]
NonaBDEs	7.8[3]	13.7[2]	90%	0.007[6]
OctaBDEs	6.9[4]	12.4[2]	98%	0.003[7]
HeptaBDEs	6.0[3]	11.1[2]	99%	0.004[8]
HexaBDEs	5.1[3]	9.8[2]	99%	0.004[8]
PentaBDEs	4.3[3]	8.6[2]	99%	0.004[8]

[1] Estimated using KOWWIN using the EVA method with a reference logK_{ow} of 8.7 for decaBDE.
[2] Estimated using KOAWIN and with input of corrected logK_{ow} from KOWWIN EVA method.
[3] Lowest QSAR estimates from the Supporting Working Document (Environment Canada 2006b).
[4] Maximum measured value (Watanabe and Tatsukawa 1990).
[5] Average based on longest observed half-lives for decaBDE, nonaBDEs and octaBDEs by Huwe and Smith (2007) and normalized to BW of wolf in Gobas Model (80 kg).
[6] Based on longest half-life of 22.6 d for nonaBDEs observed by Huwe and Smith (2007) and normalized to BW of wolf in Gobas Model (80 kg).
[7] Based on longest half-life of 64.8 d for octaBDEs observed by Huwe and Smith (2007) and normalized to BW of wolf in Gobas Model (80 kg).
[8] Based on half-life of 43.7 d (average of half-lives for nona- and octaBDEs) and normalized to BW of wolf in Gobas Model (80 kg).
[9] Estimated according to Kelly et al. (2004) based on E_D for humans.

(c) BAF–QSAR and terrestrial biomagnification model input parameters for brominated dibenzofurans

Product	Smiles	Log K_{ow}[1]	Log K_{oa}[2]	Aquatic k_M[3] (1/d)	Mammal k_M[4] (1/d)	E_D
triBDBFs	C12=CC(Br)=C(Br)C=C1OC3=C2C=C(Br)C=C3	3.0	7.1	0.02	0.004	99%
tetraBDBFs	C12=CC(Br)=C(Br)C=C1OC3=C2C=C(Br)C(Br)=C3	3.9	8.2	0.02	0.004	99%
pentaBDBFs	C12=CC(Br)=C(Br)C=C1OC3=C2C=C(Br)C(Br)=C3Br	4.8	9.5	0.02	0.004	99%
hexaBDBFs	C12=C(Br)C(Br)=C(Br)C=C1OC3=C2C=C(Br)C(Br)=C3Br	5.6	10.7	0.02	0.004	99%

[1] Estimated using KOWWIN using the EVA method with a reference logK_{ow} of 8.7 for decaBDE.
[2] Estimated using KOAWIN and with input of corrected logK_{ow} from KOWWIN EVA method.

[3] k_M for these metabolites is illustrative only and is consistent with a half-life of 26 d and normalized to weight of middle trophic level fish in Arnot and Gobas (2004) model and15^{o}C. These metabolites are expected to undergo further metabolism; however, the actual rates of metabolism are unknown.

[4] Average based on longest observed half-lives for decaBDE, nonaBDEs and octaBDEs by Huwe and Smith (2007a, 2007b) and normalized to body weight of wolf in Gobas model (80 kg); k_M for these metabolites is illustrative only and is consistent with a half-life of 26 d. These metabolites are expected to undergo further metabolism; however, the actual rates of metabolism are unknown.

Appendix E: Summary of Environmental Degradation and Debromination Studies for decaBDE

(a) Photodegradation

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Watanabe and Tatsukawa (1987)	Photodegradation	97% decaBDE and 3% nonaBDE	Artificial UV Light	Dissolved in organic solvents	NR	Tri- to octaBDEs; possibly brominated furans
Söderström et al. (2004)	Photodegradation	Traces of octa- and nonaBDEs	Natural sunlight and artificial UV light	Sorbed to thin layer of silica gel, sand, soil or sediment	NR	Reductive debromination with products including primarily hexa- to nonaBDEs; tetra- and pentaBDFs
Jafvert and Hua (2001)	Photodegradation	98%	Artificial and natural sunlight	Sorbed to hydrated surfaces of glass and silica sand particles, humic acid-coated silica particles, and glass surfaces in contact with aqueous solutions	12–71% degradation over 60–72 h	Identification of debromination products was largely inconclusive, although there was some evidence of the formation of hexa- to nonaBDEs.
Hua et al. (2003)	Photodegradation	NR	Artificial sunlight or natural sunlight	Precipitated onto quartz glass, silica particles and humic acid-coated silica particles; hydrated	44–71% degradation over 60–72 h; humic acid slowed the rate of decaBDE decay	Small amounts of nona- and octaBDE
Palm et al. (2003)	Photodegradation	NR	Artificial xenon lamps	Dispersed in organic solvents	Half-life ~ 30 minutes	Three isomers of nonaBDE formed, then six isomers of octaBDE, then debromination to several isomers of heptaBDE, and finally to trace amounts of hexaBDE. Approximately 75% of decaBDE degradation followed a pathway of debromination while the products of the remaining 25% could not be determined.

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Palm et al. (2003)	Photodegradation/oxidation	NR	Simulated sunlight or hydroxyl radicals	Sorbed to aerosol (silicon dioxide)	$< 6 \times 10^{13}$ cm^3 $molecule^{-1}s^{-1}$	Products not determined
Bezares-Cruz et al. (2004)	Photodegradation	NR	Natural sunlight	decaBDE dissolved in hexane	99% reduction in the decaBDE concentration in as little as 30 minutes	Products ranging from tri- to nonaBDEs were observed.
Eriksson et al. (2004).	Photodegradation	98% decaBDE	Artificial UV light	Organic solvents / water mixtures	4×10^{-4}/s in the methanol/water mixture (half-life ~ 0.5 hr), 6.5×10^{-4}/s in methanol (half-life ~ 0.3 hr), 8.3×10^{-4}/s in pure tetrahydrofuran (half-life ~ 0.23 hr)	3 nonaBDEs, at least 7 octaBDEs, 8 heptaBDEs and small amounts of hexaBDEs
Eriksson et al. (2004).	Photodegradation	98% decaBDE	Artificial UV light	Water / humic acid mixture	3×10^{-5}/s in water with humic acid (half-life ~ 6.4 hr	3 nonaBDEs, at least 7 octaBDEs, 8 heptaBDEs and small amounts of hexaBDEs
Geller et al. (2006)	Photodegradation	98%	Artificial light	Dissolved in tetrahydrofuran	NR	Photolysis products included hepta- to nonaBDEs as well as tri- to hexabrominated dibenzofurans
Kuivikko et al. (2006)	Photodegradation	>98.3%	Natural sunlight	Dissolved in iso-octane; modelling to determine rates in ocean	Half-life of 0.03 days in iso-octane Predicted half-lives in the Baltic Sea of 0.02 days (surface) and 1.2 days (mixing layer) and in the Atlantic Ocean of 0.09 days (mixing layer)	NR
Kuivikko et al. (2006, 2007)	Photodegradation	> 98.3%	Natural sunlight	Dissolved in iso-octane; modelling to determine rates in ocean	Half-life of 0.03 days in iso-octane; mixing zone half-lives of 1.8 days (Baltic Sea) and 0.4 days (Atlantic Ocean)	Mixing zone half-lives of 1.8 days (Baltic Sea) and 0.4 days (Atlantic Ocean), which were the same for both decaBDE concentrations

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Ahn et al. (2006a)	Photodegradation	98%	Artificial light and natural sunlight	Sorbed to montmorillonite, kaolinite, organic-carbon-rich natural sediment (16.4% OC content), aluminum hydroxide, iron oxide and manganese dioxide	For artificial light, half-lives ranged from 36 to 178 days For natural light, half-lives for montmorillonite, kaolinite and sediment were 261, 408 and 990 days, respectively (negligible degradation on other matrices)	Identified products for kaolinite and montmorillonite exposed to sunlight included nonaBDEs (BDE208, -207 -206), octaBDES (BDE197, -196) as well as small amounts of tri- to heptaBDEs
Stapleton and Dodder (2006)	Photodegradation	NR	Natural sunlight	Either native decaBDE in house dust or decaBDE spiked house dust	$2.3x10^{-3}$/hr in spiked dust and $1.7x10^{-3}$/hr in natural dust, corresponding to half-lives of 301 and 408 h in sunlight, respectively	Spiked dust: lower brominated PBDEs including hepta-, octa- and nonaBDE congeners; 54% of the degradation products were not identified
Gerecke (2006)	Photodegradation	98%	Natural sunlight	DecaBDE (BDE209) sorbed to kaolinite. Irradiated dry or in water	Half-lives of 76 and 73 minutes were determined for dry and wet conditions, respectively; dependent on light penetration	Lower brominated PBDEs (under dry conditions); unidentified products (under wet conditions)
Stapleton (2006b)	Photodegradation	NR	Natural sunlight	Either native decaBDE in house dust or decaBDE spiked house dust	Half-life for decaBDE was estimated at 216 h	Three nonaBDEs, six octaBDEs and one heptaBDE. Mass balance analysis of decaBDE determined that approximately 17% of the original decaBDE was unaccounted for, suggesting the formation of alternative (unidentified) products or volatilization of lower brominated PBDEs

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Hagberg et al. (2006)	Photodegradation	NR	Artificial light	DecaBDE (BDE209) dissolved in toluene	NR	Mono- to hexa-substituted PBDFs; the majority of the products were tetra- to hexaBDFs; lower brominated PBDEs not monitored
Barcellos da Rosa et al. (2003)	Photodegradation	98%	Artificial light	DecaBDE (BDE209) dissolved in toluene	$3x10^{-4}$/s	Hepta- to nonaBDEs
Kajiwara et al. (2008)	Photodegradation	NR	Natural sunlight	HIPS (high-impact polystyrene) added to toluene and DecaBDE (BDE209) (100ug/L); toluene evaporated off in dark. A subset of samples hydrated with water to examine effect on photolysis. Crushed TV casing also tested.	HIPS + DecaBDE samples: 50% BDE209 reduction after 1 week. Half life of BDE209 in HIPS estimated at 51 days. Hydrated samples showed faster degradation than nonhydrated. No degradation in dark controls. No degradation of BDE209 in crushed TV casing samples during 224 days of sunlight irradiation.	HIPS + DecaBDE samples: hexa to nona BDE congeners increased several-fold after 1 week exposure, then levels remained constant or decreased slightly. At end of exposure, proportion of BDE209 of total PBDEs had decreased from 90% to 44%. Study confirmed photolytic formation of tri- to octaBDFs. Total PBDFs increased (>40 times) by day 7 of exposure, with decreasing BDE209 (HIPS + DecaBDE samples). No debromination products measured from crushed TV casing samples.

NR – not determined or not reported.

(b) Other Abiotic Degradation

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Keum and Li (2005)	Reductive debromination	NR	DecaBDE (i.e., BDE209) reducing agents – zerovalent iron, iron sulphide, and sodium sulphide.	Dissolved in ethyl acetate	Up to 90% transformation of decaBDE after 40 d	Stepwise debromination with mono- to hexaBDE congeners present after 40 days (started with higher brominated PBDEs)
Li et al. (2007)	Reductive debromination	Commercial DE-83R DecaBDE (> 97%, Great Lakes Chemical)	Nanoscale zerovalent iron	Dissolved in acetone, distilled water added. 25 ± 0.5°C	Half-life was 2.5 h	Stepwise debromination to form tri- to nonaBDEs
Rahm et al. (2005)	Hydrolysis (nucleophilic aromatic substitution)	NR	Reaction with sodium methoxide	Dissolved in methanol	Half-life for the hydrolysis reaction was 0.028 h	NR
Ahn et al. (2006b)	Metal oxide-mediated debromination	98%	Birnessite	Sorbed to birnessite in THF:water and water:catechol systems	THF:water – >75% transformation of decaBDE in 24 h Catechol:water – degradation only observed for highest concentration of catechol (15% degradation over 23 days)	In THF:water – nonaBDEs (BDE207, -208), octaBDEs (BDE196, -197), heptaBDEs (BDE183, -190 and eight unknowns), hexaBDEs (BDE138, -153, -154 and five unknowns), pentaBDEs (BDE99, -100, -118 and one unknown), and tetraBDEs (BDE49, -47, -66). Not determined for water:catechol

NR – not determined or not reported

(c) Biodegradation

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
MITI 1992	Biodegradation	NR	Activated sludge	Aerobic conditions	No degradation after 2 weeks	NR
CMABFRIP (2001)	Biodegradation	97.4% decaBDE, 2.5% nonaBDE and 0.04% octaBDE	Sediment/water system	Anaerobic conditions	< 1% mineralization observed over 32 weeks	NR
He et al. (2006)	Biodegradation	98%	Anaerobic bacteria	DecaBDE (i.e., BDE209) dissolved in TCE and inoculated with anaerobic culture/medium	Degradation only observed with one culture (with *S. multivorans*) in which 0.1 μM decaBDE degraded to non-detectable levels over 2-month experiment	Octa- and heptaBDEs were detectable at the end of the experiment
Knoth et al. (2007)	WWTP monitoring (Biodegradation)	NR	WWTP sludge (primary, secondary and digested)	Field WWTP	NR	An increase in the proportion of lower brominated PBDE congeners was not observed, indicating no transformation of decaBDE during the total WWTP retention time
Gerecke et al. (2005)	Biodegradation	97.9% decaBDE, 2.1% nonaBDEs	Sewage sludge + primers (1 or more of 4-bromobenzoic acid, 2,6-dibromobiphenyl, tetrabromobisphenol A, hexabromocyclododecane and decabromobiphenyl)	Anaerobic conditions	DecaBDE (i.e., BDE209) decreased by 30% within 238 d Rate constant of 1 x 10-3 d-1	2 nonaBDEs and 6 octaBDEs. This was indicative of reductive debromination; loss of bromine from the para and meta positions

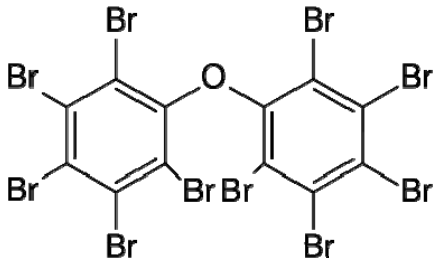
Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Gerecke et al. (2006)	Biodegradation	97.9% decaBDE, 2.1% nonaBDEs	Sewage sludge + single primers (either 2,6-dibromophenol or 4-bromobenzoic acid)	Anaerobic conditions	Half-lives of 700 days (with primer) and 1400 days (without primer) were observed. Monitoring at an operating WWTP found that the concentration of DecaBDE in sludge decreased between the influent and outlet streams.	In the experiments using a primer, decaBDE transformed slowly to BDE208.
La Guardia et al. (2007)	WWTP monitoring (biodegradation)	n/a	WWTP Sludge	NR	Minimal evidence of debromination in WWTP sludge or sediments	NR
Parsons et al. (2004)	Reductive debromination in sediments	NR	Anaerobic sediments	Anaerobic sediment suspensions in anaerobic medium Sediments collected from Western Scheldt	Significant decrease in decaBDE over 2 months; however, results are highly uncertain since similar decrease was observed in abiotic control	NonaBDEs and possibly lower brominated PBDEs
Parsons et al. (2007)	Reductive debromination in sediments	NR	Anaerobic sediments	Anaerobic sediment suspensions in anaerobic medium Sediments collected from Western Scheldt	No statistically significant decrease in decaBDE over 260 days	NonaBDEs detected in decaBDE-spiked samples, although degradation of decaBDE was not significant

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Tokarz III et al. (2008)	Reductive debromination in sediments	NR	Anaerobic sediments and cosolvent-enhanced biomimetic system	Natural sediments with no detectable PBDEs collected from Celery Bog Park, West Lafayette, Indiana. PBDEs dissolved in a toluene solution added to sediments, then evaporated off. This mixture was then blended with wet sediments. Biomimetic experiment involved the use of Teflon-capped glass vials with 0.03 mM of BDE209, -99 or -47 mixed with 5.0 mM titanium citrate and 0.2 mM vitamin B12 in 0.33 M TRIZMA buffer solution containing tetrahydrofuran.	The biomimetic system demonstrated reductive debromination at decreasing rates with decreasing bromination (e.g., half-life of 18 seconds for BDE209 and almost 60 d for BDE47) In natural sediment microcosms, the half-life for BDE209 was estimated to range from 6 to 50 years, with an average of 14 years, based on observations over 3.5 years	Proposed pathway for both systems combined: BDE209 > nonaBDEs (BDE206, -207 -208) > octaBDEs (BDE196, -197) > heptaBDEs (BDE191, -184, two unknown heptaBDEs) > hexaBDEs (BDE138, -128, -154, -153) > pentaBDEs (BDE119, -99) > tetraBDEs (BDE66, -47, -49) > triBDEs (BDE28, -17) Specifically, at the end of 3.5 years, their analysis of BDE209 degradation in sediments identified BDE208, -197, 196, -191, -128, -184, -184, 138, and -128, as well as three unidentified octaBDEs and two unidentified heptaBDEs

Study Authors	Type of Study	Substance Purity	Degradation Mediator	Experimental Conditions	Degradation Rate	Degradation Products
Kohler et al. (2008)	Debromination in sediments	NR	Natural lake sediments	Field – lake sediment cores	Cores show DecaBDEs appear in layers corresponding to 1970s, >30 years.	Despite high persistence of Deca in sediments, study suggests environmental debromination occurs, as shown by the detection of a shift in congener patterns of octa- and nonaBDE in sediments, compared to the respective congener patterns in technical PBDE products (e.g. presence of BDE202). Suggest biotic/abiotic transformation between the release of technical products and final residues in sediment.

NR – not determined or not reported.

Appendix F: Structural Diagrams for DecaBDE and DecaBDethane

Substance Name and CAS Number	Structure
Decabromodiphenyl Ether CAS: 1163–19–5	
Decabromodiphenyl Ethane CAS: 84852–53–9